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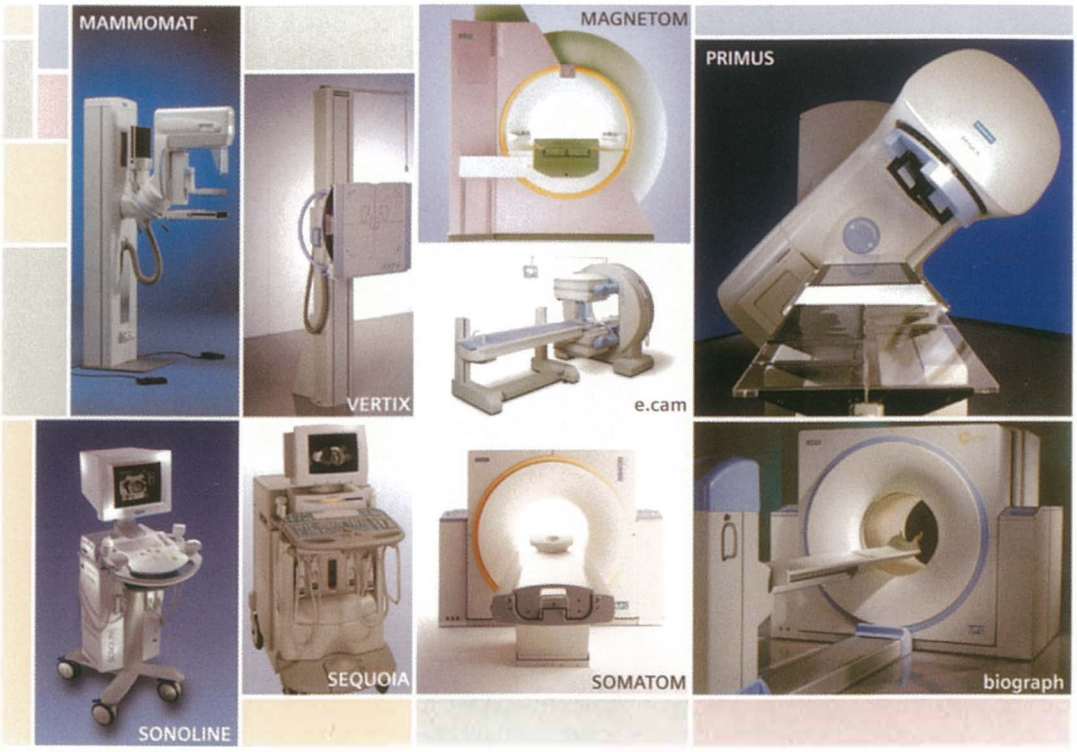

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The 80th Anniversary of Clinical Institute of Radiology Ljubljana

Guest Editor
Prof. Vladimir Jevtič, PhD, MD

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CONTENTS

Radiology and Oncology is covered in *Biomedicina Slovenica, Chemical Abstracts, EMBASE / Excerpta Medica, and Sci Base*

Editorial

History of the Clinical Institute of Radiology in Ljubljana on its 80 the anniversary (1923 -2003)

This issue of Radiology & Oncology is dedicated to 80th anniversary of the Clinical Radiology Institute in Ljubljana, the leading radiological institution in Slovenia since its establishment in 1923. Dr. Vladimir Jevtič presents a short history of the Institute. He especially points out that the main problems throughout the whole history were similar and included the lack of professional and economical independence, as well as the shortage of radiologists and radiographers. Despite serious obstacles 80 years of the Clinical Radiology Institute is a history of successful development of the republic's central radiological institution, a success based on determination, unity and professional integrity of all its members and management.

Drs. D. Vidmar and A. Perovič state that sonographically guided hydrostatic reduction of intussception, which is the most common cause of bowel obstruction in early childhood represents the method of choice because of its high efficiency, lack of complications and radiation exposure.

Drs. V. Jevtič and N. Majcen evaluate the role of magnetic resonance imaging (MRI) in demonstrating evolution of hemispherical spondylosclerosis, a painful syndrome of unclear etiology. They were able to demonstrate some typical MRI features which could be of clinical relevance.

Drs. M. Glušič and E. Brenčič rewiev the current concepts of imaging of acute pancreatitis and its relationship to pancreatic functional tests and treatment planning. The importance of radiological investigations has been increasing in the last decade as the result of introduction of more sophisticated imaging techniques.

Dr. P. Berden rewievs the clinical indications, the technique of performance as well as limitations of magnetic resonance angiography of the portal venous system.

Drs. M. Šurlan and P. Popovič rewiev the current knowledge concearning radiological evaluation and interventional procedures in patients with hemodyalisis vascular access stressing the importance of endovascular prophylactic dilatation of stenosis, the placement of stents and of recanalisation of trombosed fistulas and grafts. In their second contribution they discuss a complex topic of interventional radiological management of the complications following renal transplantation. Variety of complications may occur after renal transplantation the most frequent being renal and perirenal fluid collections and abnormalities of the vasculature and collecting system. Different interventional procedures can be used for treatment such as percutaneous drainage of fluid collections, percutaneous transluminal renal angioolasty without or with stents in renal artery stenosis and some other procedures in relatively uncommon urologic complications.

The rest of the contributions include presentations of several rare but interesting cases. Drs. M. Ogulin and B. Jamar present a case of annular pancreas causing extrahepatic biliary obstruction, Drs. P. Gregorič and A. Perovič stress the clinical importance of portal venous gas detec-

ted by abdominal sonography, Dr. V. Jevtič contribute with demonstration of radiographic, computed tomographic and magnetic resonance imaging appearances of primary V-cutting zone of resorption of lumbar vertebral body in a case of Paget's disease and with two cases of calcified cervical intervertebral disc in a child and a thoracic disc calcification in an adult with posterior herniation. Finally Dr. J. Markota concludes the issue with an interesting case of unusual radiographic changes of a patient with gout.

The editor thanks to the authors for their contribution to this issue of Radiology & Oncology dedicated to an extraordinary jubilee, 80th anniversary of the Clinical Institute of Radiology in Ljubljana.

Guest Editor
Professor Vladimir Jevtič

Radiol Oncol 2004; 38(4): 269-73.

Sonographycally guided hydrostatic reduction of childhood intussusception

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Background. *Intussusception is the most common cause of bowel obstruction in children under two years of age. The proximal part of the bowel and its mesentery (the intussusceptum) enter within that part immediately beneath it (the intussuscipiens). Being pulled by peristalsis the mesenterial vessels get compressed which result in ishaemia of the bowel wall. Most intussusceptions are ileocolic. The diagnosis can be confirmed by a contrast enema or ultrasound. Sonography demonstrates a so-called »target-within-a-target« pattern (in cross-section) with thickened edematous bowel wall with or without vascularisation and prestenotic dilatation with increased peristalsis. Therapeutic reduction can be attempted by a contrast enema (following diagnostic procedure) or by air, both under fluoroscopic monitoring, or by normal saline under sonographic guidance.*

Patients and methods. *We detected sonographically intussusception in three girls of 15, 16 and 18 months having typical clinical signs. We continued with hydrostatic reduction under the sonographic guidance. The reduction was attempted with a saline enema on body-temperature, introduced by the equipment for contrast enema. The bottle of normal saline was hung up 1 m over the examination desk. We needed few liters of saline to replace lost liquids due to the incomplete occlusion of rectum. Meanwhile we monitored the moving of the intussusceptum back into the proximal direction. Criteria for a succesful reduction were the disappearance of the intussusceptum and the passage of fluid through the ileocecal valve.*

Results. *Success was proven in all three girls. No complications occured and the pain relieved immediately after the procedure. There were no signs of intussusception on sonography after 2 and 12 hours. We saw a slightly edematous wall of ileocecal valve and terminal ileum. Due to their exellent clinical conditions they were discharged from hospital after a second sonography.*

Conclusions. *Sonographically guided hydrostatic reduction of intussusception in children is a method of choice because of its high efficiency and lack of radiation exposure. No complications have been reported to date.*

Key words: intussusception-therapy-ultrasonography; child

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Introduction

Intussusception is the most common cause of bowel obstruction in early childhood, with the peak incidence between 6 months and 2

years of age.[1] It is typically presented with an acute onset of colicky abdominal pain. However, the clinical symptoms can be confusing, with recurring non-specific abdominal symptoms, especially when the bowel obstruction is not complete. Intussusception occurs when one segment of the bowel (the intussusceptum) becomes telescoped into the immediately distal segment of bowel (the intussuscipiens). Once trapped, the invaginated segment is propelled by peristalsis farther into the distal segment, pulling its mesentery along it. Intussusception can develop in any free-moving part of the bowel. However, the vast majority of childhood cases are ileocolic;[2] that is, the ileum becomes telescoped through the ileocecal valve into the cecum. The mesenteric vessels of the intussusceptum become compressed, which leads to ishemia and eventually bowel necrosis, particularly at the apex of the intussusceptum.

The diagnosis of intussusception is made by a contrast enema or ultrasound. At the time of diagnosis, a radiologist should attempt the reduction of intussusception. A therapeutic reduction can be attempted by a contrast enema (following diagnostic procedure) or by air insufflation, both performed under the fluoroscopic monitoring. Recently, a new technique - hydrostatic reduction with ultrasound guidance - has been recognized to be equally successful.

Patients and methods

Three female patients, 15, 16, and 18 months of age, with a severe colicky abdominal pain and clinically suspected intussusception were evaluated by sonography. The ileocolic intussusception was detected in all three cases. The examinations were performed with Toshiba PowerVision 6000 SSA-370A ultrasound scanner. A diagnostic procedure was continued with hydrostatic reduction under the sonographic guidance. The reduction was attempted with a normal saline enema, warmed to body temperature, introduced by the equipment for contrast enema. To achieve the appropriate hydrostatic pressure within the bowel lumen, the bottle of normal saline was hung up 1 m above the examination desk. To form a tight seal and to reduce leakage of fluid, the catheter was placed in the rectum through the rubber ring, which was taped to gluteal region. However, the occlusion was still incomplete and we had to use several litters of saline to replace lost fluid. During the fluid instillation, we monitored how the intussusceptum proceeded in the proximal direction. The criteria for a successful reduction were the disappearance of the intussusceptum, followed by the reflux of fluid from the cecum through the ileocecal valve into the terminal ileum. The mean time of the reduction procedure was 15 minutes.

Results

All three attempts at sonographically guided hydrostatic reduction of intussusception were successful. No complications occurred. The pain relieved immediately after the procedure and the patients recovered quickly. The follow-up sonography scan was performed 2 and 12 hours after the procedure. In all three cases follow-up scans showed a slightly oedematous wall of ileocecal valve and terminal ileum and a small quantity of free intraperitoneal fluid. Due to the excellent clinical condition of the patients, they were discharged from hospital after the second follow-up sonography.

Disscusion

Intussusception is the most common cause of bowel obstruction in early childhood, with the peak incidence between 6 months and 2 years of age.[1] It is typically presented with an

acute onset of colicky abdominal pain; however, when the bowel obstruction is not complete, the clinical symptoms can be confusing with the recurring non-specific abdominal symptoms. Intussusception occurs when one segment of the bowel (the intussusceptum) becomes telescoped into the immediately distal segment of the bowel (the intussuscipiens). Once trapped, the invaginated segment is propelled by peristalsis farther into the distal segment, pulling its mesentery along it. The mesenteric vessels of the intussusceptum get compressed, the ishemia and eventually bowel necrosis ensue. The apex of the intussusceptum is the part most prone to the development of pathologic changes. Most cases of intussusception are idiopathic, with no identifiable lesion acting as the lead point. Rarely, a mechanical lead point, such as intestinal polyp, Meckel diverticulum, duplication cyst or lymphoma, can be found. Even more rare is the intussusception in correlation with cystic fibrosis, Henoch-Schonlein purpura, hemophilia or intussusception after operative abdominal procedures.[2] Intussusception can begin in any free-moving part of the bowel; however, the ileocolic intussusception is the most common, with the ileum being telescoped through ileocelcal valve into the cecum. Less frequently, small - intestinal (segment of small intestine enters into the immediately distal segment of the small intestine), colo-colic (analogous pathology in the colon) or ileocecal (ileocecal valve is the lead point) types can develop.

The diagnosis of intussusception is made by a contrast enema or ultrasound. Many pathologic conditions of the bowel wall produce the characteristic sonographic appearance of a »target« sign on transverse images. In intussusception this appearance is even more characteristic with a »target-within-a-target« sign on transverse images and »hayfork« sign on longitudinal scans (Figures 1, 2).

The inner target represents the intussusceptum and the outer target is produced by

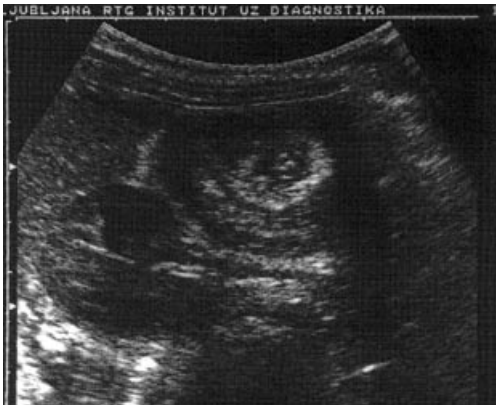

Figure 1. Sonography (transverse scan): »target-within-a-target« sign.

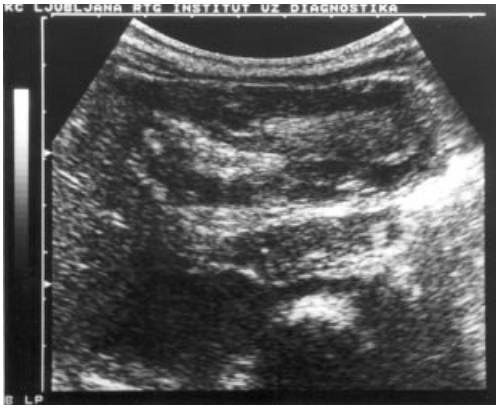

Figure 2. Sonography (longitudinal scan): »hayfork« sign.

the intussuscipiens. The wall of the intussusceptum is oedematous and therefore hypoechoic on sonography scans. With Doppler imaging the presence of compromised/absent blood flow in the bowel wall can be demonstrated, which is important in differentiation from the acute inflammation. The bowel loops proximal to the »target-within-a-target« sign are dilated with increased peristalsis. In all three patients that we examined, the ileocolic intussusception was detected and the described characteristic signs were clearly visible. Ultrasonography is highly accurate in the diagnosis of intussusception with a specificity of 100% and a sensitivity of 88 - 93%.[3,4]

After the diagnosis is made, the primary aim should be a non-operative reduction of the intussusception. Up until recently, the non-operative management included a contrast enema (barium) or air insufflation, both performed under the fluoroscopic monitoring. As ultrasound became the imaging modality more and more used also for bowel pathology, the conditions for sonographically guided reduction of the intussusception were formed. Free intraperitoneal fluid and signs of mechanical ileus are not contraindications for this procedure; the procedure is successful also in children with fluid in the peritoneal cavity.[1,5] As the saline fills the colon, the apex of the intussusceptum is observed under the continuous ultrasound guidance as it proceeds in the proximal direction. The criteria for a successful reduction are the disappearance of »target-within-a-target« sign, visualization of ileocecal valve and reflux of fluid from the cecum into the terminal ileum (Figure 3).[5,6]

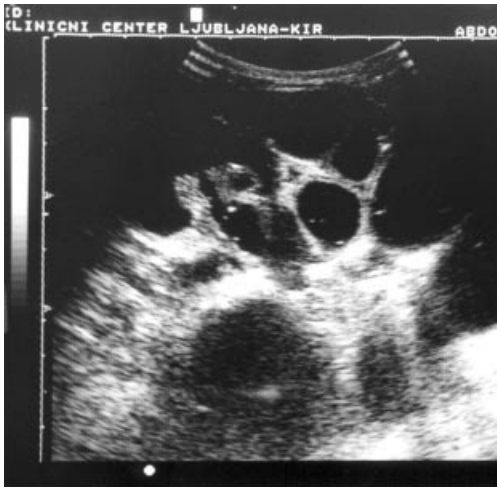

Figure 3. Sonography: ileocecal valve and fluid within the ileum.

To achieve the appropriate hydrostatic pressure within the bowel lumen (at least 60 mmHg, not more than 120 mmHg), the canister used to run normal saline into the rectum should be placed 1 m above the examination desk.[1] To avoid hypothermia, the fluid should be warmed to body temperature. Even though the catheter was placed in the rectum through the rubber ring, which was taped to gluteal region, the leakage of saline around the catheter was substantial in all three patients. We needed several litters of saline to replace lost fluid. All three attempts at a sonographically guided hydrostatic reduction of intussusception were successful, which was confirmed by two follow - up sonographic scans performed 2 and 12 hours after the procedure. According to published data, the sonographically guided hydrostatic reduction of childhood intussusception is curative in 76 - 93% of cases.[6-10] Riebel *et al.* reported that, in comparison with other conservative methods of reduction, the success rate of sonographically guided hydrostatic reduction of intussusception was not substantially worse (versus that of pneumatic reduction) or was even higher (versus that of barium enema reduction).[8]

At both follow-up sonographic scans all three patients had slightly edematous wall of ileocecal valve and terminal ileum, which is in accordance with other studies.[1] No complications occurred. The pain relieved immediately after the procedure and the patients recovered quickly. The review of the available literature also did not reveal any complications of this procedure.[1,6-10] The mean time of the reduction procedure was 15 minutes, which is similar to previously published reports.[6]

Conclusion

From our initial experience and review of the literature we can conclude that the sonographically guided hydrostatic reduction of childhood intussusception is a simple, safe and very successful technique that does not expose the child to ionizing radiation and can in many cases replace operative management.

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Demonstration of evolution of hemispherical spondysclerosis by contrast enhanced Gd-DTPA magnetic resonance imaging

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Background. *The purpose of the study was to estimate the value of Gd-DTPA magnetic resonance imaging (MRI) in demonstrating the evolution of hemispherical spondylosclerosis (HSS).*
Patients and methods. *In eighteen patients with chronic low back pain and typical radiographic findings of HSS seen on plain films, Gd-DTPA MRI of the lumbar spine was performed. MRI morphological and signal intensity appearances of HSS were analysed and compared with radiographic changes.*
Results. *On the basis of MRI features, three distinct groups of cases were identifiable. Within the first group the region of dome-shaped osteosclerosis demonstrated low signal intensity on T1-weighted precontrast spin-echo images, high signal intensity on T2-weighted images and diffuse contrast enhancement on T1-weighted postcontrast images, findings compatible with bone marrow oedema and hyperaemia. The second group showed high signal intensity vertebral body corners surrounded by low signal intensity area, which indicated the combination of fat accumulation and the sclerotic bone. In the third intermediate group anterior disco-vertebral junctions revealed a mixture of MRI appearances characteristic of the first and the second group.*
Conclusions. *Gd-DTPA MRI is capable of demonstrating a spectrum of features which reflect the evolution of HSS. These typical appearances showed by MRI could be of eventual clinical relevance in following the progression of HSS.*

Key words: spinal diseases; magnetic resonance imaging; gadolinium DTPA; osteosclerosis

Introduction

The term HSS was used by Dihlmann[1] to describe a peculiar form of localised erosive spondylopathy with well defined radiographic findings. The most typical features include dome-shaped osteosclerosis which usually affects the anteroinferior portion of the vertebral body with the base at the endplate. Commonly, similar appearances are seen in the neighbouring vertebra. Narrowing of the corresponding disc space indicating disc degeneration is present in the majority of cases. There is a predilection for L4, L5 and L3 vertebral bodies and the toracolumbar junction.

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HSS mainly affects middle-aged women and is clinically characterized by chronic low back pain. Since its first description in literature as nonneoplastic scleroses,[2] different terms like traumatic lesion of the disco-vertebral junction,[3] discogenic vertebral scleroses,[4] idiopathic segmental sclerosis[5] have been used to indicate the possible aetiology of the disease entity. However, the exact aetiology and pathogenesis of HSS still remains uncertain. Experience with MRI of this condition, especially with the use of paramagnetic contrast agents is limited.[6,7]

The purpose of this study was to investigate the morphological and signal intensity MRI appearances of HSS, to compare these features with the conventional radiography and to estimate the value of Gd-DTPA MRI in demonstrating the evolution of HSS.

Patients and methods

18 patients (12 women, 6 men, mean age 45 years) with radiographic findings typical of HSS seen on AP and lateral plain films of the lumbar spine were selected for the study. In 5 patients, lateral conventional tomograms were also available. In order to be included in the study, the radiographs were required to demonstrate dome-shaped osteosclerosis with the base at the intervertebral disc affecting the anterior part of the vertebral body. Other radiographic findings which were also noted included bone erosions of the endplate at the base of sclerosis, new bone production along the anterior vertebral body contour, the presence of an anterior ostephyte and the disc space narrowing.[1] Additional inclusion criteria were chronic low back pain and normal erythrocyte sedimentation rate. In six patients, the exacerbation of low back pain was registered during the last several weeks. MRI of the lumbar spine was performed on a 1.5 T superconducting scanner (Magnetom, Siemens) one to three weeks after plain films. A spin-echo sequence with T1W images (TR 500 ms, TE 15 ms, 2 averages) and T2W images (TR 1900 ms, TE 80 ms, 2 averages) was used, followed by the postcontrast examination immediately after Gd-DTPA (0.1 mmol / kg body weight) was injected intravenously as a bolus through a cannula. The field of view was 28 cm with a data acquisition matrix of 256 x 256. The region of the lumbar spine was presented in a sagittal plane with 5 mm thick consecutive slices. The axial images of the intervertebral discs and the neighbouring parts of the vertebral bodies were obtained at the levels of HSS seen on plain films.

Conventional radiographs and MR images were analysed in a qualitative fashion for morphological and signal intensity differences between normal and pathological disco-vertebral junctions. On the basis of MRI features, an attempt has been made to reconstruct the eventual evolution of HSS.

Results

Three distinct MRI patterns of disco-vertebral junction abnormalities were found at the levels of HSS seen on the plain film radiography. Within the first group of three patients the region of dome-shaped osteosclerosis demonstrated low signal intensity on T1-weighted precontrast images, high signal intensity on T2-weighted images and a diffuse intense contrast enhancement on T1-weighted postcontrast images. There was also focal Gd-DTPA accumulation within the endplate erosions at the base of sclerosis. In one of the patients, these typical MRI appearances were seen at the anterosuperior part of the vertebral body L5 while the neighbouring anteroinferior part of the vertebral body of L4 showed MRI findings characteristic of the second group (Figures 1a, 1b, 1c).

The second group comprised five patients with MRI features of high signal intensity an-

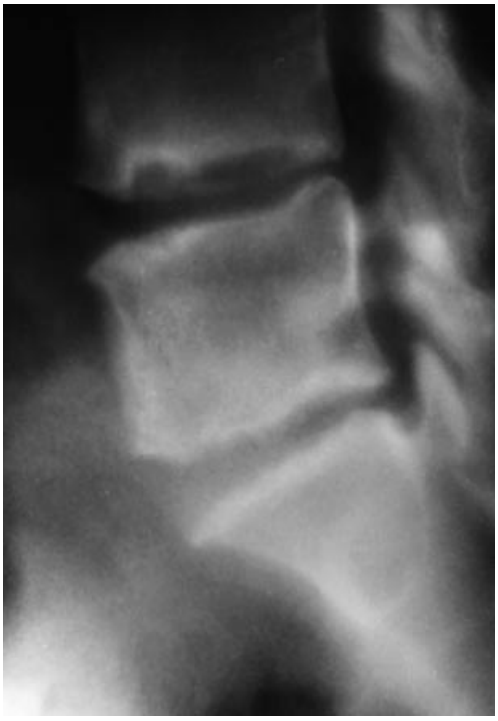

Figure 1a. Initial HSS of the vertebral bodies L4 and L5. Lateral conventional tomography shows discrete osteosclerosis of the vertebral body L4 and typical erosions affecting the anterior thirds of the endplates at the levels of L4 and L5 (big arrow heads).

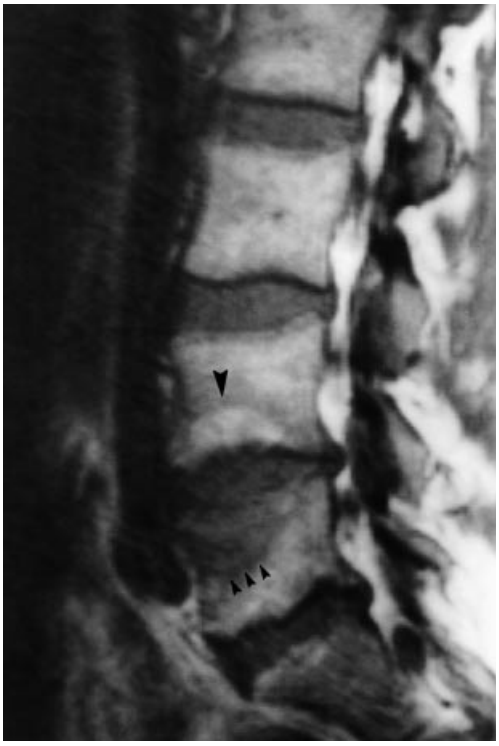

Figure 1b. T1 - weighted sagittal (TR 500 ms, TE 15 ms) precontrast image reveals high signal intensity anteroinferior part of the vertebral body L4, findings consistent with fat accumulation (big arrow head) and low signal intensity anterosuperior part of the vertebral body L5 indicating bone marrow oedema (small arrow heads). Note also that the erosion of the vertebral body L4 is sharply corticated while the erosion at the level of L5 shows indistinct contour.

terior portions of the vertebral bodies on T1-weighted precontrast and T2- weighted images surrounded by a band-like low signal intensity area. No contrast enhancement could be registered within the region HSS of (Figures 2a, 2b, 2c). The accumulation of paramagnetic contrast agent within the endplate erosions was seen in four of these patients. In one of the cases, typical MRI changes developed during the six months following a disc surgery.

In the remainder of the twelve patients, disco-vertebral junctions demonstrated heterogenous MRI appearances which represented a mixture of signal intensity changes and morphological findings from the first and the second group (Figure 3). A focal contrast enhancement was seen within the erosions of the vertebral body endplates. In two of the patients from this intermediate group, MRI showed the posterior disc herniation and in one the presence of ischemic spondylolysis localised at the same level as HSS.

The erosions of the vertebral body endplates typically localised at the anterior junctions of the peripheral bone rim and the cartilaginous endplate (Figures 3a, 3b, 3c, 3d, 3e) were identified by MRI in all of the patients. In three cases the erosions were not seen on the plain films. With the exception of six patients belonging to the first and the third group, in whom indistinct cortical bone was

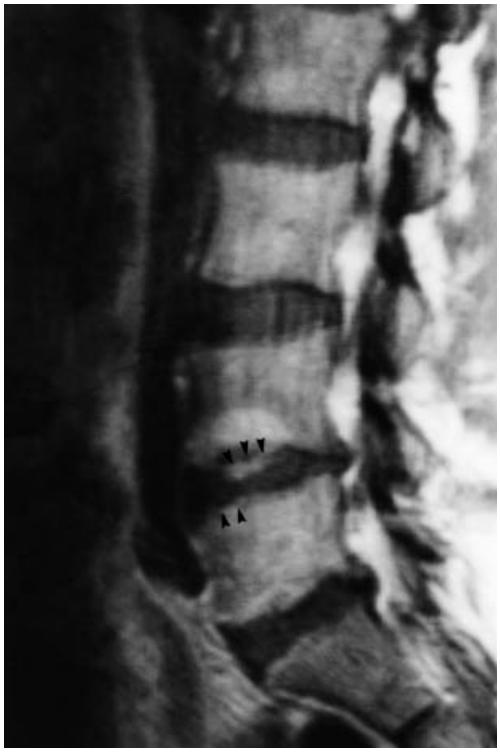

Figure 1c. T1-weighted sagittal postcontrast image demonstrates contrast accumulation within the region of bone marrow oedema of the vertebral body L5 and within the endplate erosions (small arrow heads).

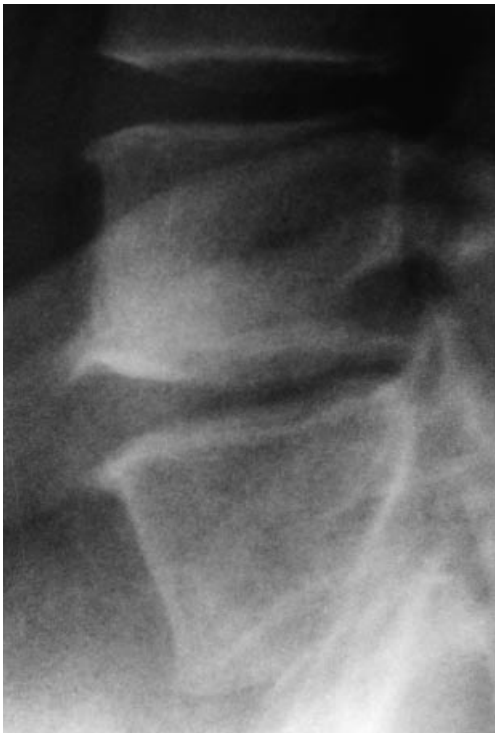

Figure 2a. HSS following disc surgery at the level of L4-L5. Lateral plain film with typical radiographic findings.

revealed (Figure 1); in the rest of the vertebral bodies the erosions were clearly corticated. Additional erosions localised more dorsally were demonstrated in two patients from the second and six from the third group (Figures 4a, 4b, 4c).

Varying amounts of new bone production along the anterior vertebral body contour contiguous with the anterior ostephytes (Figure 4) were revealed on the plain films in patients from all three groups. However, the signal intensity MRI appearances of the periosteal apposition differed between the groups. Within the first and the second group new bone production was of low signal intensity on T1-weighted precontrast images and of high signal intensity on T2-weighted and T1-weighted postcontrast images (Figures 3, 4). Similar findings were revealed in one case from the third group, while the rest demonstrated the linear high signal intensity area along the anterior surface of the vertebral bodies on all MRI sequences (Figure 2).

The anterior ostephytes were more pronounced in patients from the second and the third group. Their signal intensity characteristics were similar to MRI features of the neighbouring vertebral body bone marrow.

At the level of HSS an absence of normal high signal intensity on T2-weighted images affecting the anterior part or the whole disc space indicating disc degeneration was revealed in all of our patients with the exception of focally increased signal intensity within the bone erosions.

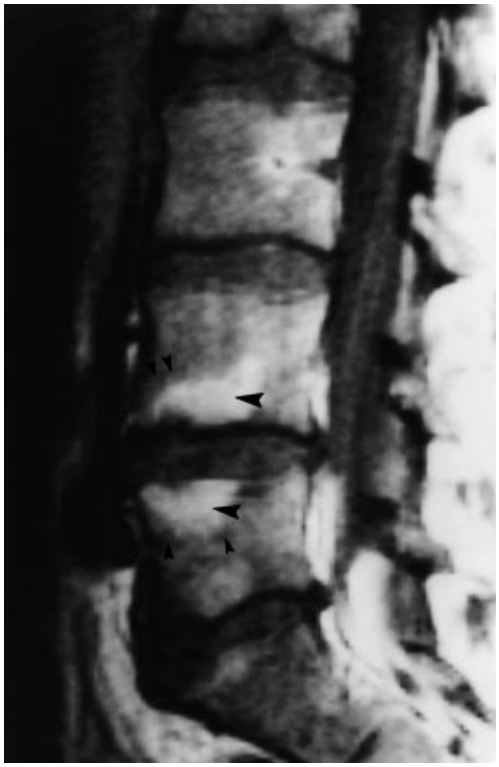

Figure 2b. T1-weighted sagittal (TR 500 ms, TE 15 ms) image reveals high signal intensity fat conversion within the bone marrow and the ostephytes (big arrow heads) surrounded by low signal intensity sclerotic bone (small arrow heads).

Figure 2c. T1-weighted postcontrast sagittal image shows minute contrast enhancement within the endplate erosion (small arrow head). There is no contrast accumulation within the vertebral bodies and the ostephytes.

Discussion

The exact aetiology and evolution of HSS is not completely clear. Therefore, different terms indicating possible aetiology or radiographic features have been applied to this entity.[1-5] In comparison to other radiological techniques, MRI proved to have a unique ability not only to present an excellent anatomical image but also to reflect closely pathophysiological and pathoanatomical changes in different osteoarticular diseases. The experience with MRI of HSS is limited[6,7] especially with the use of paramagnetic contrast agent GD-DTPA and no detailed description of the eventual evolution of MRI changes has been made.

MRI findings of low signal intensity on T1-weighted spin-echo images and of high signal intensity on T2-weighted images within the area of bone sclerosis seen in the patients from the first group (Figure 1) have already been reported.[6,7] In addition we were able to demonstrate a marked contrast enhancement affecting the same region. These MRI features were compatible with the presence of bone marrow oedema and hyperaemia. With the exception of different distribution, which has been focal in HSS and widespread in early degenerative disc disease, there was an obvious resemblance in MRI appearances between described changes in HSS and Modic I lesions[8] in which the existence of fibrovascular tissue below the vertebral body endplates was

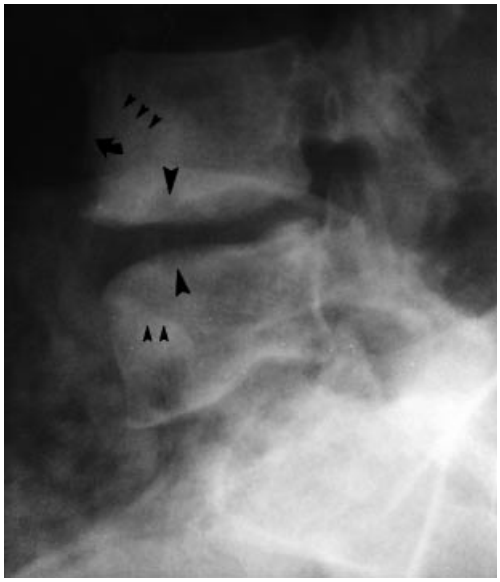

Figure 3a. HSS at the level of L4-L5 disc. Lateral radiograph shows osteosclerosis affecting the anterior parts of the vertebral bodies (small arrow heads), erosions of the endplates (big arrow heads) and periosteal apposition at the anterior contour of the vertebral body L4 (arrow).

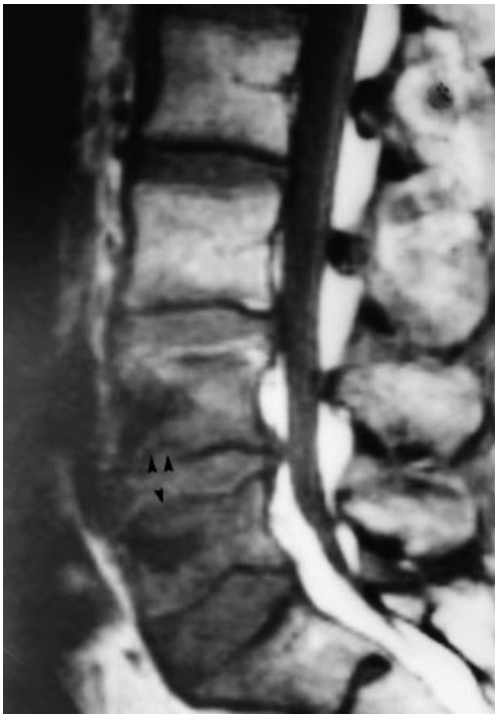

Figure 3b. T1-weighted sagittal (TR 500 ms, TE 15 ms) precontrast image reveals nonhomogenous low signal intensity anterior parts of the vertebral bodies L3, L4 and L5 and the erosions of the endplates (small arrow heads).

proved histologically. Furthermore, we showed similar focal MRI findings at the anterior disco-vertebral junctions in patients with ankylosing spondylitis, having early Romanus lesions.[9] Some histological studies proved that the bone erosions of the vertebral bodies at the attachment of the annulus fibrosus in spondylitis anterior were occupied by the vascularised granulation tissue.[10,11] Within the so-called Type a localised disco-vertebral destructive lesions, which radiographically resemble HSS, Cawley *et al*[12] revealed a peripheral focal infiltration by cartilaginous and fibrous tissue, new bone deposition with thickened trabeculae and oedematous bone marrow, histological evidence of trauma with persistent pressure. These similar histological and MRI appearances seen in various disco-vertebral lesions are evidently nonspecific phenomena and reflect the limited reactive abilities of the osteoarticular system to etiologically different joint diseases.

Within the second group of patients, high signal intensity anterior portions of the vertebral bodies on T1-weighted precontrast and T2-weighted images, surrounded by a band-like low signal intensity area without contrast enhancement on T1-weighted postcontrast images indicated a bone marrow fat conversion combined with a sclerotic bone (Figure 2) nonspecific reaction which has also been described in longstanding disc degeneration[8] or advanced nonactive sacroiliitis.[13]

The most numerous was the third intermediate group of cases in which a combination of signal intensity changes and varying degrees of contrast enhancement represented a mixture of MRI appearances characteristic of the first and the second group. MRI features within the anterior parts of the vertebral bodies were compatible with a combination of

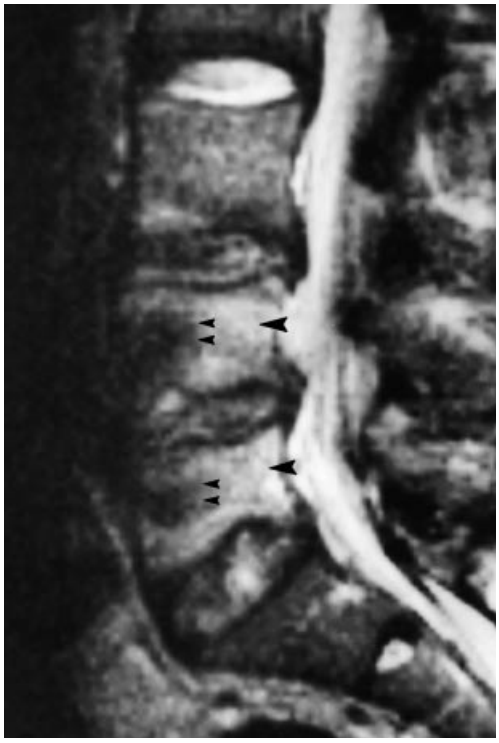

Figure 3c. T2-weighted sagittal (TR 1900 ms, TE 80 ms) image demonstrates widespread high signal intensity bone marrow oedema (big arrow heads) surrounding low signal intensity osteosclerosis (small arrow heads).

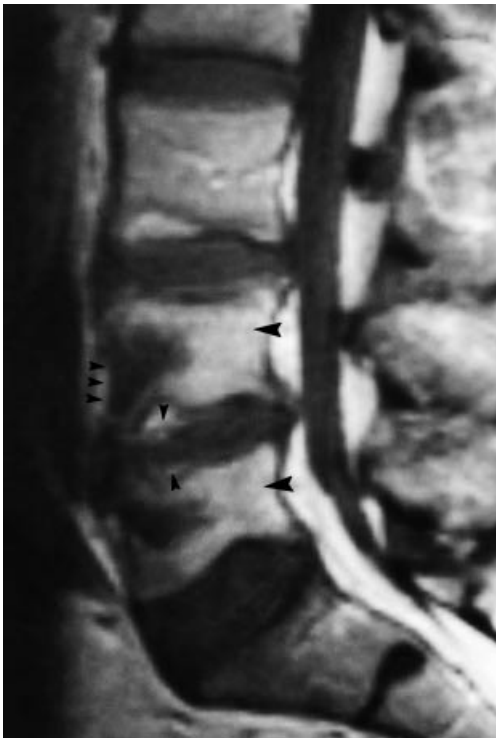

Figure 3d. T1-weighted sagittal postcontrast image shows diffuse contrast enhancement within the vertebral bodies (big arrow heads) and linear enhancement within the endplate erosions and along the anterior surface of the vertebral body L4 (small arrow heads).

bone marrow oedema and hyperaemia, fat accumulation and sclerotic bone (Figure 3). The distribution of these changes within the individual vertebral bodies suggested the possibility that the initial bone marrow oedema at the vertebral body margins has been progressively replaced by fatty and sclerotic bone marrow, findings similar to those seen in progressive disc degeneration.[8]

The reported incidence of the endplate erosions in HSS varies.[1,4] In three of our patients the erosions were not identified on the plain films. However, with the use of MRI as a tomographic method, they were demonstrated in all the cases. This finding supports the opinion of some authors that the anterior defect of the vertebral body endplate represents the initial lesion[1,14] and that the common feature in HSS is the herniation of disc material into the vertebral body[4] which might be provoked by the subclinical endplate trauma.[3] The fact that the erosions were not clearly corticated in several of the patients from the first and the third group while the majority of cases from the third and all from the second group showed corticated erosions, suggests the possibility of advancing process of cortical healing. Gd-DTPA contrast accumulation within the erosion was consistent with the histological findings of the focal replacement of the disc and the disc-bone border by vascularised fibrous tissue in peripheral disco-vertebral destructive lesions reported by Cawley *et al.*[12]

The frequent location of HSS at the lumbosacral and the toracolumbar junctions has

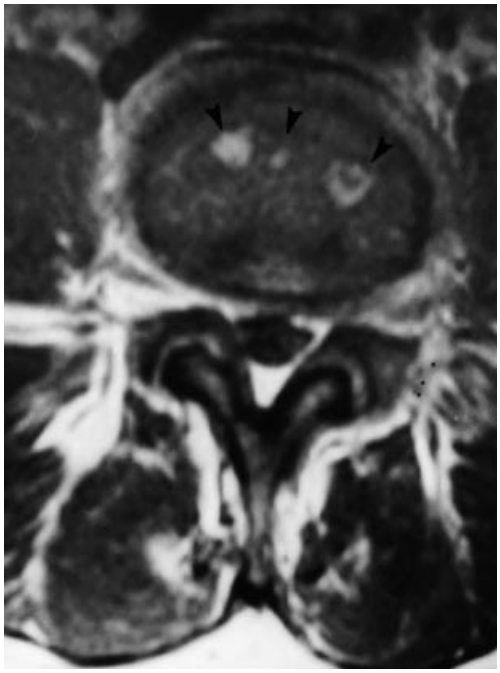

Figure 3e. T1-weighted axial postcontrast image at the level of the lower endplate of L4 demonstrates distribution of bone erosions (big arrow heads) at the border between the epiphyseal ring and the cartilage endplate.

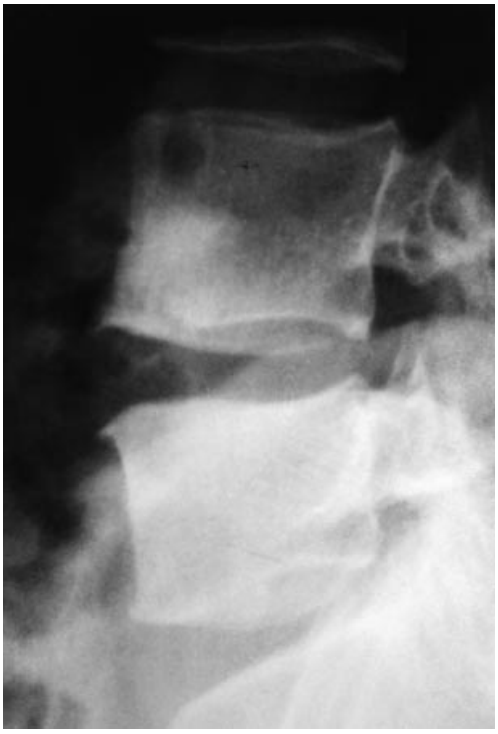

Figure 4a. Disc degeneration at the level of L4-L5 with HSS affecting the anteroinferior part of the vertebral body L4. Lateral radiograph shows dome-shaped osteosclerosis (small arrow heads), an anterior erosion of the endplate (big arrow head) and periosteal apposition along the anterior surface of the vertebral body (arrow).

been attributed to the increased stress at these levels produced by the transition of a mobile to a fixed part of the axial skeleton. It has been postulated that the increase in the segmental mobility due to tears affecting the periphery of the intervertebral disc causes the overstretching of the anterior longitudinal ligament and provoke the osteoblastic production of the marginal osteophytes.[1] Indeed high signal intensity of the new bone production and of the contiguous ostephytes on T2-weighted and T1-weighted postcontrast images (Figures 3, 4) consistent with the presence of oedema and hyperaemia may indicate the instability of the corresponding vertebral dynamic segment. On the other hand, the organized periosteal apposition and marginal ostephytes of high signal intensity on all MRI sequences (Figure 2), due to a fat transformation, presumably represent the end stage of stabilization of abnormally mobile vertebral segments at the level of HSS.

The clinical relevance of different MRI features in HSS is not clear. Modic *et al*[8] suggested the possibility that early disc degenerative changes with the presence of subchondral bone marrow oedema might be symptomatic. Stäbler *et al*[15] showed that the Schmorl's nodes with adjacent bone marrow oedema were seen more frequently in symptomatic than in asymptomatic patients. It would seem therefore that the cases of HSS surrounded by widespread bone marrow oedema could eventually represent symptomatic or »active« lesions. It is of interest to note that all six patients with recent exacer-

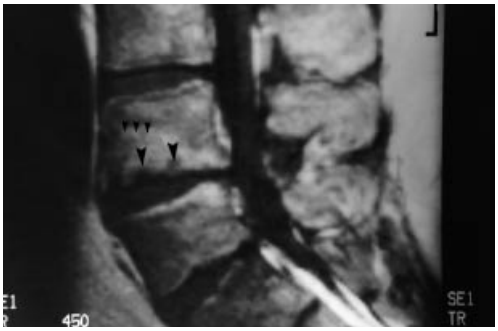

Figure 4b. T1-weighted sagittal (TR 450 ms, TE 15 ms) precontrast image demonstrates high signal intensity fat accumulation within the region of sclerosis seen on the plain film (small arrow heads). Note several erosions of the lower endplate of L4 (big arrow heads).

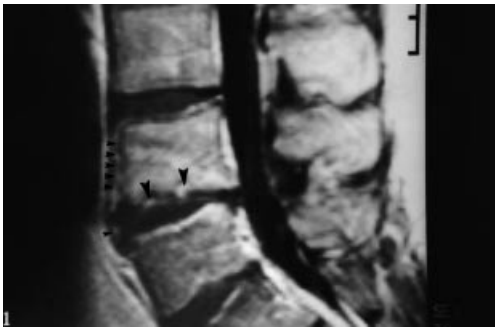

Figure 4c. T1-weighted Gd-DTPA postcontrast sagittal image reveals linear contrast enhancement at the anterior contour of vertebral body L4, the anterior part of the annulus fibrosus L4-L5 (small arrow heads) and within the endplate erosions (big arrow heads). There is no contrast accumulation in the region of osteosclerosis.

bation of low back pain belonged to the first and the second groups in which varying amounts of bone marrow oedema and hyperaemia were demonstrated by MRI. Analogously, it can be assumed that the cases of HSS similar to Modic II lesions[8] seen within the second group of our patients are presumably less symptomatic or »nonactive«.

Although the lack of histology in our study precludes definite conclusions on the basis of the histological results of others[3,4,7,8,10-12] and according to MRI appearances in our patients the eventual evolution of HSS could be postulated. The findings are in favour of the theory that the initial lesion could be anterior erosion at the junction between the epiphyseal ring and the cartilage endplate[1,3,4,14] followed by the instability of the corresponding vertebral dynamic segment. Due to a compromised vertebral body integrity and resulting instability, the bone underlying erosion is exposed to a persistent pressure, which similarly to the initial disc degeneration provokes bone marrow oedema and hyperemia.[8] The process of repair resembling healing of the fracture[12] ensues, characterized by the ingrowth of vascular fibrous tissue into the erosion and the vertebral body and consecutive new bone production within the bone and along its anterior surface. A favourable outcome may eventually result in which the cortication of the endplate erosion, the reinforcement of the vertebral body in the form of bone sclerosis and the anterior disco-vertebral junction shown as the organized periosteal apposition and marginal osteophytes lead ultimately to the spontaneous stabilization of the corresponding vertebral dynamic segment. In cases with an unfavourable outcome the instability persists and may eventually be followed by new bone erosions and the progressive disc degeneration. In our study, MRI proved to be a sensitive indicator of these complex dynamic changes.

It is concluded that GD-DTPA MRI is capable of demonstrating the evolution of HSS. It seems to enable the differentiation of a spectrum, including the initial phase characterized by a compromised integrity of the anterior endplate, followed by the intermediate phase of reactive changes, leading finally to the stabilization of the abnormally mobile vertebral dynamic segment. MRI appearances could be of eventual clinical relevance in following the progression of HSS.

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Radiologic imaging of acute pancreatitis

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Background. *Acute pancreatitis comprises a broad spectrum of inflammatory changes in the gland and has a variety of causes. The most common are alcoholism and biliary tract disease.*
This paper discusses the current concepts of diagnosis and the relationship between morphology as depicted by imaging, pancreatic function tests and treatment planning.
Conclusions. *Our understanding of the morphologic changes produced by the inflammatory process has expanded in the last decade as more sophisticated imaging techniques have been used for evaluation.*

Key words: pancreatitis - radiography; acute disease

Introduction

Pancreatitis is defined and classified according to clinical, morphologic and histological criteria. Acute pancreatitis is defined as an acute inflammatory process of the pancreas, caused by activation of the proteolytic enzymes within the gland.[1]

The most common etiologic factors of acute pancreatitis are cholelithiasis and alcohol abuse, the others are rarer (trauma, surgery, ERCP, viral and bacterial infections, pancreatic tumours, drugs, hyperlipoproteinemia, primary hyperparathyroidism, idiopathic).

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The clinical spectrum of acute pancreatitis varies from mild to severe disease.

Mild acute pancreatitis is the most common form of the disease, and is characterized macroscopically by interstitial - oedematous inflammation of the gland. Microscopic findings include infiltration of the interstitial space with leukocytes, occasional small scattered foci of acinar cell necrosis, and both intra- and peripancreatic fat necrosis. Patients with mild pancreatitis typically present with sudden abdominal pain and tenderness. Fever and leukocytosis may be present.

Patients with severe acute pancreatitis may initially experience symptoms similar to those in mild disease, but often the pain is worse and they often show additional clinical and laboratory evidence of systemic dysfunction and multisystem failure. The length of hospitalization is longer and may be complicated by hypotension, pulmonary insufficiency, renal failure, gastrointestinal bleeding,

metabolic acidosis, hypocalcaemia, disseminated intravascular coagulation, and sepsis.[1,2]

Clinical diagnosis

The clinical diagnosis of acute pancreatitis is straightforward when patients present with appropriate symptoms and when laboratory findings are confirmatory. Diagnostic difficulties arise when patients present with nonspecific symptoms that mimic other acute abdominal conditions or when laboratory tests are non-diagnostic. Serum amylase and lipase levels represent the most commonly used laboratory tests to diagnose pancreatitis. These test values are imperfect disease indicators, however, because they may be normal in up to 10% of patients. Serum amylase increase is not specific for acute pancreatitis, the height of serum amylase and/or lipase does not correlate with the severity of pancreatitis.[2,3]

Objective assessment of the severity of an acute attack of pancreatitis is critical to patient management, because the clinician must be able to differentiate mild acute (interstitial) pancreatitis from severe acute (necrotizing) pancreatitis. Approximately 50% of patients present with mild acute pancreatitis and require only minimal supportive therapy and limited hospitalization. Mortality is rare in this group unless a serious complication occurs or the disease evolves into a more severe form. Patients with severe acute pancreatitis have mortality rates of 10-20%, with death often due to shock associated with early respiratory and renal failure or sepsis.

High-risk patients who may benefit from aggressive clinical and surgical management must therefore be identified.
Disease severity can be evaluated objectively using the Ranson criteria:
- At admission:
  Age over 55 years
  White cells over 16000/ mm^3
  Blood glucose over 11 mmol/l
  LDH over 5.83ukat/l (350UI/l)
  AST over 2.0 ukat/l (250UI/l)
- During initial 48h:
  Hematocrit decrease over 10%
  Blood urea increase over 5 mg/100 ml
  Calcium less than 8 mg/100 ml (2 mmol/l)
  PO2 less than 8 kPa (60 mm Hg)
  Base deficit over 4 mmol/l (mEq/l)
  Fluid deficit over 6000 ml

Three or more Ranson criteria depicted during first 48 hours indicate severe acute pancreatitis. Patient with six or more Ranson criteria is very likely to die.

Radiologic diagnosis

The primary roles of radiologic imaging in patients with suspected pancreatitis are:
- to confirm or exclude the clinical diagnosis of acute pancreatitis,
- to determine, if possible, the cause of the disease,
- to stage disease severity,
- to detect complications,
- to provide imaging guidance for percutaneous therapy.

Contrast-enhanced CT is the imaging modality of choice for evaluating patients with acute pancreatitis.[2-5] It can accurately diagnose and stage the disease as well as provide the necessary information for percutaneous management. Sonography, endoscopic retrograde cholangiopancreatography, and angiography play secondary roles. Ultrasound is primarily used to detect biliary stones and to follow the size of pancreatic fluid collections and pseudocysts. Diagnostic endoscopic retrograde cholangiopancreatography is seldom performed during acute attacks of pancreatitis, because it may exacerbate the disease or introduce infection. Endoscopic retrograde cholangiopancreatog-

raphy with sphincterotomy may be performed to alter the course of disease in patients with suspected biliary pancreatitis. Angiography is reserved for diagnosis and treatment of vascular complications.[4]

The value of MRI in acute pancreatitis remains to be established. Thus far, it has not been widely applied in the care of patients with acute pancreatitis, though it can be used in patients unable to receive iodinated contrast media.[6,8]

On conventional SE and GRE T1-weighted images, the normal pancreas is approximately isointense with the liver. On T2-weighted images the normal pancreas is moderately hyperintense compared with the liver. T1-weighted breath hold images after bolus injection of extracellular contrast agent (gadolinium) show intense parenchymal enhancement which peaks before the arrival of contrast in the superior mesenteric vein, and fades fairly quickly. The liver-specific contrast medium manganese-DPDP is also taken up by the pancreatic exocrine tissue, where it produces an increase in signal intensity on T1-weighted images.

Oedema of the pancreas causes diffuse signal reduction on T1-weighted images and reduced enhancement after intravenous contrast agent. In more severe cases unenhanced gradient echo T1-weighted images may show peripancreatic oedema as areas of low signal extending into the fat surrounding the gland. Dynamic post-contrast acquisition is a sensitive method for demonstrating the presence and extent of pancreatic necrosis, shown as areas of diminished or absent parenchymal enhancement. Exudate and fluid collections within or around the pancreas can be seen on T1, but are also well shown on T2-weighted images, where they appear as areas of high signal intensity. T2-weighted images also give a clear distinction between the fluid and solid components of localized exudates and pseudocysts. This is important in patients who are candidates for percutaneous drainage of pancreatic collections, which often appear as areas of homogeneous low attenuation on computed tomography even when the collection is mostly solid. Because of the high sensitivity of gradient echo images to susceptibility effects produced by fresh bleeding, the presence of a hemorrhagic component in acute pancreatitis is probably detectable more readily and over a longer time course by MR than by CT, although the clinical value of this finding is still uncertain.

MRCP - MR cholangiopancreatography

MRCP provides a non-invasive alternative to diagnostic ERCP, particularly useful in patients in whom ERCP is technically difficult or unsuccessful (e.g. following gastric or pancreatic surgery, or in patients with tight strictures of the main duct).

MRCP relies on extreme T2 weighting which effectively eliminates signal from all tissues except stationary free water protons, so that the images display only those structures containing localized fluid collections. With careful selection of the imaging volume, a demonstration of the pancreatic duct and the main bile ducts can be obtained.

MRCP has the advantage of being completely non-invasive, however it does not offer the opportunity to carry out therapeutic procedures, it is also contraindicated in patients with metallic clips or stents, with exception of titanium, relatively lower resolution also limits the demonstration of branch ducts, except when they are evidently dilated.

CT should be performed at the peak of pancreatic arterial perfusion using thin-section (5 mm or less) scanning.

CT finding of mild acute pancreatitis is a slight to moderate increase in the size of the pancreas. Although pancreatic enlargement is typically diffuse with associated contour irregularity, it can be segmental, most commonly involving the head. As disease severity increases, evidence of peripancreatic inflam-

matory stranding is seen, manifesting as increased density to the surrounding fatty tissue, thickening of the peripancreatic fascial planes, and heterogeneity of the pancreatic parenchyma. Rupture of the secondary pancreatic ductules leads to both intra- and extravasation of pancreatic secretions. In such cases, CT reveals both intrapancreatic and peripancreatic fluid collections. They are composed of blood, pancreatic enzymes, fluid, and debris. Acute collections have fluid density, lack an inflammatory capsule or wall, and are confined by the anatomic space in which they form. They are most commonly seen adjacent to the pancreas in the anterior pararenal space and the lesser sac. Extensive collections may spread throughout the peritoneal cavity, and may dissect as far as the neck, pericardium, pleura, mediastinum, and groin. Solid organ involvement within the hilar regions of the spleen, kidneys, and liver can also occur.

Pancreatic necrosis is defined as focal or diffuse areas of unenhancing, nonviable pancreatic parenchyma. Regions of necrosis show no or diminished enhancement. In a large series of patients who underwent both CT and surgery, Berger et al. showed CT to have an overall accuracy of 87% in diagnosing necrosis. CT specificity was 100% when necrosis involved more than 30% of the gland. A 21% false-negative rate was seen in patients with minor (less than one third) necrosis, which fell to 11% in those patients with major (greater than 50%) necrosis.[4]

Local complications of acute pancreatitis

Acute fluid collections may be seen in 40 - 50% of patients with acute pancreatitis. Approximately 50% are small and resolve spontaneously. Collections which persist may evolve into pancreatic pseudocysts, a process requiring at least 4 weeks.

Pseudocysts are encapsulated collections which appear marginated on CT because of a nonepithelialized inflammatory fibrous wall or capsule. Approximately half of all pseudocysts resolve spontaneously, especially uninfected pseudocysts less than 5 cm in diameter. Those which do not may either stabilize or decrease slightly in size, remaining clinically asymptomatic, or they may enlarge and cause complications. Major pseudocyst complications shown by CT include arterial pseudoaneurysm formation, with or without cyst hemorrhage, superinfection (suggested by air within the cyst), venous occlusion (suggested by perigastric and mesenteric varices), biliary and gastrointestinal tract obstruction, and solid organ invasion.

Local septic complications

Sterile necrosis, defined as pancreatic necrosis without infection, must be differentiated from both infected necrosis and pancreatic abscess. The mortality of infected necrosis (39-67%) is significantly greater than that associated with sterile necrosis (13-14%) These entities may be distinguished if evidence of bacterial infection is obtained by CT-guided fine-needle aspiration.[9] Accurate differentiation of infected pancreatic necrosis from pancreatic abscess is also essential. Infected necrosis carries a much higher morbidity and mortality than does pancreatic abscess.[9]

Pancreatic abscess is a localized collection of pus located near the pancreas. In contrast to infected necrosis, which can develop at any time during the course of acute pancreatitis, pancreatic abscesses typically present 4 weeks or more after the onset of symptoms. A pancreatic abscess may be diagnosed by CT if a fluid collection containing liquid pus is identified adjacent to a normally enhancing pancreas.

Pancreatic ascites presents as free intraperitoneal fluid containing high amylase levels. It is usually seen in more severe forms of pancreatitis, only rarely in association with a normal-appearing pancreas.

Pancreatic haemorrhage may be seen in cases of traumatic pancreatitis, as well as in cases of severe acute pancreatitis where significant hemorrhagic had necrotizing components coexist.

Besides local complications severe forms of acute pancreatitis are often accompanied by systemic complications - hypotension, pulmonary insufficiency, renal failure, gastrointestinal bleeding, metabolic acidosis, hypocalcaemia, disseminated intravascular coagulation, sepsis.

Staging of acute pancreatitis[2-5,9]

CT scan provides important information in accordance with the Balthazar-Ranson criteria of severity, where inflammatory process and gland necrosis are separately graded.

Inflammatory process (a):		
Grade A	Normal pancreas	0
Grade B	Focal or diffuse enlargement of pancreas	1
Grade C	Pancreatic and peripancreatic inflammatory changes	2
Grade D	Single fluid collection	3
Grade E	Two or more fluid collections and/or the presence of gas in/or adjacent to pancreas	4

Gland necrosis (b):	
No necrosis	0
Less than 30%	2
30-50%	4
Greater than 50%	6

CT severity index (CTSI): a+b.

Patients with a CTSI of 0-3 show a 3% mortality rate and an 8% morbidity rate, whereas in patients with a CTSI of 7-10, mortality and morbidity rates are 17 and 92% (Figure 1).

CT retains a disadvantage of exposure to x rays, which is of importance in younger patients and in patients in whom CT examination is repeated.

Value of CT examination is limited in patients, in whom intravenous contrast agent is contraindicated, due to severe renal insufficiency or allergy.

In patients with suspected severe acute pancreatitis, early CT study is suggested by many authors for confirmation of the diagnosis and detection of severity of disease. It should be done within 24-48 hours from the beginning of the disease. CT should be repeated in 7-8 days, or earlier in case of clinical deterioration.

Conclusions

Pancreatic necrosis, depicted by CT scan, and peripancreatic inflammatory collections are the two prognostic indicators of severity of acute pancreatitis. The most serious complications develop in patients with peripancreatic collections and some of these patients die. Such complications occur mostly in individuals with associated early or late pancreatic necrosis. In most individuals fluid collections tend to resolve spontaneously if the pancreas maintains its integrity.

The limitations of the early CT examination are related to potential development of complications in patients with a normal pancreas, as well as to the development of late pancreatic necrosis in these individuals. For these reasons, patients with peripancreatic

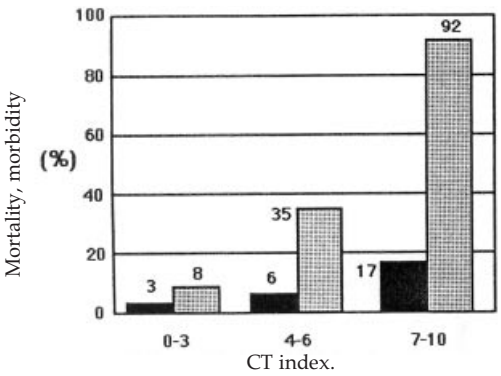

Figure 1. Mortality, morbidity according to CT index.

fluid collections, pancreatic necrosis, or both should undergo follow-up CT examination in 7-8 days or earlier if their clinical condition deteriorates.

To improve the prognostic accuracy of the initial CT examination, a CT severity index, based on combined assessments of peripancreatic inflammatory collections and degree of pancreatic necrosis, was developed.

Patients with a high CT severity index (7 to 10 points) showed a 92% morbidity and a 17% mortality, whereas those with a low severity index (0 to 3 points) had 3% morbidity and no mortality.

Detection of pancreatic necrosis, either at the first examination or at follow-up study, or a high CT severity index at the initial CT examination are ominous predictive indicators of severe pancreatitis, which enable the radiologist to identify a group of patients in whom most life-threatening complications will occur.

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review

Magnetic resonance angiography of the portal venous system

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Background. *Imaging of the portal venous system is necessary in different clinical conditions. Three-dimensional (3D) contrast-enhanced magnetic resonance angiography (MRA) is useful in obtaining high quality portal vein images. A fast gradient-echo MR imaging sequence with minimum repetition time and echo time is used. Up to 40 ml of paramagnetic contrast is injected into peripheral vein as a bolus. The Arrival of contrast medium in the aorta is preferably detected with an automated system, when breath-hold sequence is started, and repeated two times, to depict arterial and venous phase. Maximum-intensity-projection (MIP) imaging is the usual postprocessing method.*

Conclusions. *In patients with portal hypertension, MRA can present collateral pathway and patency of the portal vein or portosystemic shunt. In portal vein thrombosis MRA provides information about the location and length of portal vein obstruction and helps in therapeutic strategy decision. MRA is a proper technique in Budd-Chiari syndrome, where it is important to determine the location and length of hepatic outflow obstruction. MRA is a very good modality before liver transplantation to depict vascular anatomy and portal vein patency, and after liver transplantation to image possible liver complications. Its limitations include inappropriate positioning of the 3D acquisition slab, respiratory motion artefacts, and metal implants (e.g. pacemaker).*

Key words: portal vein; magnetic resonance angiography; vascular patency

Introduction

The portal venous system must be evaluated before planning the treatment of patients with portal hypertension, portal vein thrombosis, hepatic veins occlusion or a tumour of the liver, pancreas, or bile duct.

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Colour Doppler ultrasonography (US), catheter angiography, computed tomographic angiography (CTA) and magnetic resonance angiography (MRA) can be used for portal venous system imaging. Colour Doppler US is non-invasive, relatively inexpensive and can provide information on portal blood flow, but it is operator and patient dependent (acoustic window, uncooperative patient).[1] Catheter angiography (indirect portography, percutaneous transhepatic portography, and direct splenoportography) are invasive and limited by flow dynamics.[2] CTA can demonstrate the portal venous system in a short time, but this

technique uses ionizing radiation and requires a large amount of iodinated contrast material.[3]

Three-dimensional (3D) contrast-enhanced magnetic resonance angiography (MRA) was initially used manly in arteriography of the aorta and renal arteries.[4,5] Respiratory motion had previously been a major source of artefact in abdominal imaging. With faster sequences it has become evident that this technique has greater capability in abdominal and liver imaging. MRA has the ability to cover large regions of interest within a single breath hold. Superb images of the portal, mesenteric and systemic veins can be obtained routinely. Source images can be obtained to help evaluate parenchymal lesions. The combination of parenchymal and vascular information allows accurate staging of liver neoplasm. In addition, vascular complications of hepatic transplantation can be clearly demonstrated with MRA.[6] Its advantages over digital subtraction angiography (DSA) include its large field of view, its short imaging time, and its non-invasive nature and low risk of complications, which permit repeated studies.

In this article, we briefly discuss some technical aspects of 3D contrast enhanced MRA, reformatting methods and limitations. We present the advantages of MRA in different clinical conditions - portal hypertension, portal vein thrombosis, Budd-Chiari syndrome and liver transplantation.

Technique

A fast 3D spoiled gradient-echo MR imaging sequence with minimum repetition time and echo time is used, with low flip angles ranging from 20° to 30°. Tissue contrast is low; vascular contrast is achieved with high intravascular concentrations of gadolinium, which is injected intravenously as a bolus. By the time extra cellular contrast agent reaches the portal vein, it is rather diluted. That is why a dose of 0.2 to 0.3 mmol/kg body weight and flow rates from 2.5 to 4 ml/sec are recommended.[7]

The best images are obtained when the acquisition of the central portion of k-space corresponds with the maximum concentration of contrast material in the vessels of interest.[6] The range of contrast travel times is broad and depends on patient age, cardiovascular status and hydration state; it is advisable to perform timing with a test bolus,[8] or preferably, with an automated triggering, in which the vessel of interest is continuously scanned until the signal intensity reaches a specified level - at that time the 3D sequence is started.[9] Centric phase encoding is used in conjunction with these techniques; central k space, which is responsible for image contrast, is acquired at the beginning of the scan, when vascular contrast is maximal.[10]

These timing strategies are generally used to optimize the arterial-phase imaging. Acquiring two additional 3D volumes after the optimized arterial-phase imaging almost always yields good visualization of the portal and systemic veins with one or both of the later sequences.[6]

Our typical portal MRA examination consists of axial fast spin-echo MR imaging through the region of interest. This is useful for the evaluation of liver and spleen and helps determine landmarks for the 3D volume. A contrast bolus - 40 ml of Magnevist with flow-rate 2.5 ml/s is then injected. Smartprep (GE Medical Systems) is used to automatically detect arrival of contrast medium in the aorta. The arterial-phase breath-hold sequence in coronal plane is acquired 5 seconds after the arrival of contrast medium in the abdominal aorta. The first-phase sequence is followed immediately by two additional acquisitions with 10 seconds intersequence delay. One sequence takes 14 to 20 seconds, depending on volume of interest. Before the examination, patients were instructed in breath-holding in the same posi-

tion. A final post-MR angiography axial fat-saturated spoiled gradient-echo sequence is performed to help visualize parenchymal lesions.

Data processing

Data processing is an important aspect of 3D contrast-enhanced MRA. A variety of reformatting techniques are now available to the radiologist, and each technique has its own strengths and weaknesses, which can lead to pitfalls and artefacts in inexperienced hands. It is important to be well versed in different reformatting techniques.

Maximum-intensity-projection (MIP) imaging is the most common method. It is well suited to contrast-enhanced MRA, particularly arterial-phase imaging, in which background signal is low and arterial contrast is high. MIP images obtained from the entire data set are almost always contaminated by artefacts, which can limit the visibility of vessels. Image quality can be improved by obtaining sub volume MIP images or by manually editing the entire data set.[6] Subtraction of a precontrast data set from the arterial- phase data to eliminate background noise is problematic in the abdomen, where any discrepancy in breath holding between acquisitions can result in misregistration artefact. Arterial-phase images of the abdomen are almost always diagnostic without subtraction (Figure 1a) as long as good bolus timing has been achieved. Subtraction is much more useful in the venous phase.[11] Even in imperfect subtraction conditions due to different breath-hold positions between each acquisition, excellent suppression of arterial signal and enhancement of adjacent organs can be obtained after post-processing by using a partial volume MIP technique (Figure 1b).[12] We routinely use subvolume MIP in three orthogonal and oblique projections if necessary (depending on patient anatomy). We improve portal venous images by subtracting the arterial phase from the portal venous phase image using available software.

However, source images should be examined too, because nonocclusive thrombus can easily be missed on MIP images and subtraction is an artificial technique. Volume rendering, surface rendering, and virtual endoscopy may be useful in certain applications.[13]

Clinical applications

Contrast-enhanced 3D MRA can demonstrate the intrahepatic and extrahepatic portal venous system as well as hepatic veins. Clinical

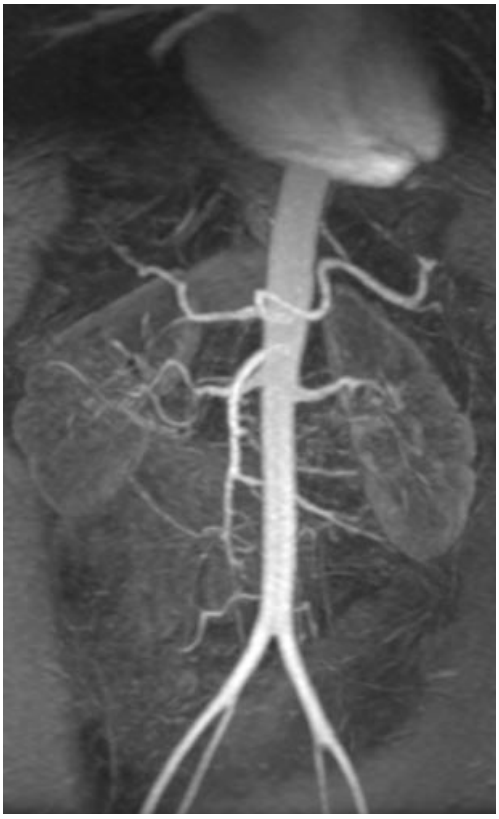

Figure 1a. 3D contrast-enhanced MRA in patient with suspected portal occlusion, arterial phase, subvolume MIP. Superb visualization of the abdominal aorta and visceral arteries.

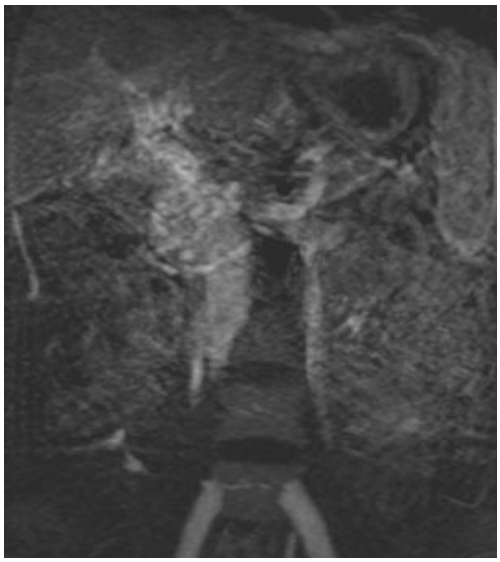

Figure 1b. The same patient, portal phase, arterial phase is subtracted. A network of small collateral vessels despite normal portal vein is seen in hepatic hilus - cavernous transformation of the portal vein.

applications of this method include portal hypertension (portosystemic shunt, portal vein obstruction, and hepatic vein obstruction), hepatic encephalopathy, hepatocellular carcinoma and pancreatobiliary tumours and liver transplantation (pre-and posttransplantation evaluation).[14] In patients with portal hypertension, MRA can be used to evaluate portosystemic shunt, hepatopetal collateral pathways, and obstruction of the portal or hepatic veins. In planning treatment for hepatic encephalopathy, it is important to identify the causative portosystemic shunt. In patients with hepatocellular carcinoma or pancreatobiliary tumours, one must determine the presence or absence of portal vein invasion when planning treatment.

Portal hypertension

Different pathologic conditions like liver cirrhosis, chronic hepatitis, and Budd-Chiari syndrome can lead to portal hypertension and hepatic encephalopathy. It is important to evaluate portosystemic collateral pathways because such patients are at high risk of hepatic coma and massive haemorrhage from oesophagogastric varix. Portosystemic shunts can be formed anywhere in the abdomen and include oesophagogastric varix, paraumbilical vein, mesenteric-gonadal, mesenteric-retroperitoneal, intrahepatic portosystemic, and splenorenal shunts. Therefore, it is necessary to examine the whole abdomen.[14]

Hepatic encephalopathy is caused by a massive portosystemic shunt, which can be easily detected with MRA, as well as oesophagogastric varices, especially when MRA is performed with arterial-phase subtraction (Figures 2a, 2b).

Artificial portosystemic shuts (surgical or percutaneous transhepatic-TIPS) are used to reduce flow and pressure in varix and diminish the risk of variceal bleeding. 3D contrast MRA can be a useful guide for shunt planning and it can present shunt patency. MRA assesses patency of any kind of shunt, as long as metallic clips do not obscure portal venous anatomy. TIPS shunts are more difficult to assess due to metallic stents. If the stainless steel stent is used to bridge the portal and

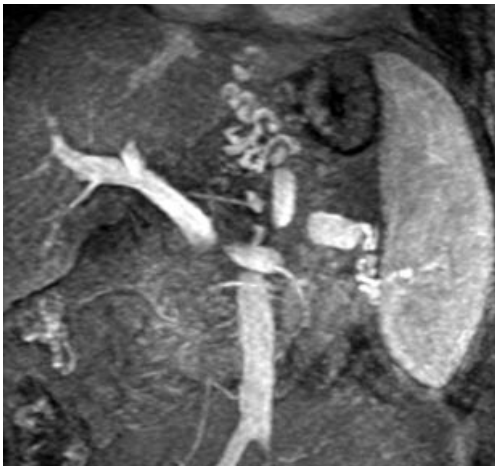

Figure 2a. 3D contrast-enhanced MRA in patient with portal hypertension, subvolume MIP. Portal vein is clearly shown, as well as oesophagogastric varix. Enlarged spleen and distal abdominal aorta is visible.

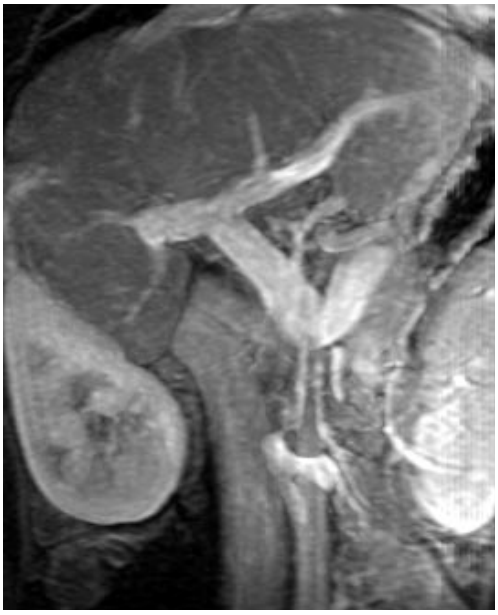

Figure 2b. The same patient with portal hypertension, oblique view. Major intrahepatic branches of portal vein are shown to better extent.

systemic venous systems, the lumen cannot be evaluated by MRA.[7] In conjunction with phase contrast techniques, shunt flow volume can be determined non-invasively.[15]

Portal vein thrombosis

Different pathologic conditions can cause portal vein thrombosis, like pancreatitis, portal hypertension, trauma, malignancy and coagulopathy.[16] The acutely thrombosed portal vein is expanded with thrombus and contains no flow. Over time portal venous collateral pathways develop in both hepatopetal and hepatofugal directions. Hepatopetal collateral pathways include the cavernous transformation of the portal vein that develops in main portal vein obstruction, the dilated pancreaticoduodenal venous arcades that develop in superior mesenteric vein obstruction and the dilated gastroepiploic, short gastric, and coronary veins that develop in splenic vein obstruction. Cavernous transformation of the portal vein represents a network of small collateral vessels (Figure 1b). It is identified by characteristic enhancement pattern in the hepatic hilum during portal venous phase of MRA.[17]

It is important to assess portal venous patency in these diseases. MRA provides information about the location and length of portal vein obstruction and also about portal collateral pathways.[18] In potential candidates for liver transplantation, it is necessary to evaluate portal venous patency.[19]

Budd Chiari

Budd-Chiari syndrome is a rare disorder characterized by hepatic outflow occlusion and is difficult to diagnose by any method.[7] It is caused by various conditions including congenital or idiopathic obstruction, hepatic vein thrombosis, hepatic veno-occlusive disease after liver transplantation, and hepatic tumours. The major symptoms are ascites, hepatomegaly, and abdominal pain. It is classified into three types according to the location of the occlusion. Type 1 is defined as occlusion of the inferior vena cava, type 2 as occlusion of major hepatic veins and type 3 as obstruction of the small centrilobular venules. Imaging methods should help in decision, whether it can be treated with anticoagulants, surgery, or interventional procedures. In planning treatment, it is important to determine the location and length of hepatic outflow obstruction.[14] MRA is an appropriate technique to diagnose hepatic vein occlusion. Budd-Chiari can be confirmed by observing absence of hepatic veins and more heterogeneous enhancement of peripheral liver.[7]

Liver transplantation

Patent portal vein is required for liver transplantation. When ultrasound fails to adequately visualize the portal vein, MRA offers a safe and accurate imaging of portal venous

anatomy and anatomy of others important veins - inferior vena cava, superior and inferior mesenteric vein, splenic vein and varices.[7]

Following liver transplantation, MRA is a very good modality to image possible liver complications. In suspicion of allograft ischemia, portal vein is the first to evaluate, since blood supply to the liver is primarily via this vessel. The most common site of stenosis is at the anastomosis. Hepatic artery is much smaller in calibre and eventual stenosis of this vessel is difficult to assess.[20] Thrombosed artery results in ischemia of the common bile duct which can lead to biliary strictures and leaks. Inferior vena cava (IVC) anastomoses may also become flow limiting - IVC is generally well depicted on portal phase of MRA.

Before MRA, T1- and T2-weighted images should be performed to look for possible other postoperative complications - abscesses, fluid collections, hepatic masses. With fast breath-hold sequences even biliary obstruction may be evaluated.

Limitations

General contraindications to MR imaging (pacemakers, aneurysm clips, or claustrophobia) also apply to contrast-enhanced 3D MRA, which has several other limitations.

This technique is unable to demonstrate the flow direction of the portal venous system, unlike phase-contrast or time-of-flight MRA.[15] Metallic clips, stents, and embolization coils can cause considerable artefact and obscure important structures. Artefacts from respiratory motion and peristaltic bowel movement degrade image quality, especially in debilitated patients who are unable to hold their breath. When subtraction techniques are used, respiratory misregistration also degrades image quality.[14] Even when the study is optimal, the resolution of gadolinium-enhanced MR angiography is relatively low compared with that of conventional angiography, and visualization of small peripheral arteries is very limited.[6] There is a risk of allergic reactions to contrast media, although the incidence is low. Gadolinium-enhanced MR angiography, although less expensive than conventional angiography, is still an expensive examination.

Conclusion

A precise assessment of the portal vein and collateral pathways is potentially helpful for treatment and planning purposes. Three-dimensional gadolinium-enhanced MRA is helpful in evaluating the anatomy of the portal venous system and its pathologic conditions, such as portosystemic shunt, portal vein thrombosis, portal vein invasion by hepatic and pancreatobiliary tumours, and hepatic vein obstruction.[21] It is frequently useful in both pre- and postoperative imaging of artificial portosystemic shunts and liver transplant recipients.

A variety of methods to increase the speed of 3D acquisition are under investigation.[22,23] Time-resolved MR angiographic techniques allow repeated acquisition of a volume of interest during the passage of the contrast material bolus. Novel imaging and reformatting techniques may allow faster acquisition or improved resolution. Echoplanar imaging may become a viable alternative to the standard gradient-echo acquisition.[6]

Several intravascular contrast agents are currently undergoing clinical trials. These agents have much longer vascular half-lives and may allow high-resolution imaging of the arterial and venous system with appropriate respiratory gating.[24,25]

MRA combines speed, excellent contrast, and relative simplicity and has been applied to virtually all regions of the body from the brain to the extremities.

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review

Interventional radiology in haemodialysis fistulae and grafts

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Background. *The aim of the paper is to review the role of interventional radiology in the management of haemodialysis vascular access. The evaluation of patients with haemodialysis vascular access is complex. It includes the radiology/ultrasound evaluation of the peripheral veins of the upper extremities with venous mapping and the evaluation of the central vein prior to the access placement and radiological detection and treatment of the stenosis and thrombosis in misfunctional dialysis fistulas. Preoperative screening enables the identification of a suitable vessel to create a haemodynamically-sound dialysis fistula. Clinical and radiological detection of the haemodynamically significant stenosis or occlusion demands fistulography and endovascular treatment. Endovascular prophylactic dilatation of stenosis greater than 50% with associated clinical abnormalities such as flow-rate reduction is warranted to prolong access patency. The technical success rates are over 90% for dilatation. One-year primary patency rate in forearm fistula is 51%, versus graft 40%. Stents are placed only in selected cases; routinely in central vein after dilatation, in ruptured vein and elastic recoil.*

Conclusions. *Thrombosed fistula and grafts can be declotted by purely mechanical methods or in combination with a lytic drug. The success rate of the technique is 89-90%. Primary patency rate is 8% to 26% per year and secondary 75% per year.*

Key words: kidney failure, chronic; hemodialysis; catheters, indwelling; radiology, interventional

Introduction

Renal failure is treated by dialysis or renal transplantation. About 63% of patients are treated with haemodialysis, 9% with peritoneal dialysis and 28% with renal transplantation.[1] Haemodialysis can be performed through central venous catheters (internal jugular, subclavian and femoral vein) or through a permanent arteriovenous access. Long-term patency of central catheters is low, because they are prone to infection and thrombosis.[2] Chronic haemodialysis is performed by the autogenous arteriovenous fistula and the synthetic bridge graft. Native fistulas are the most durable type of vascular access and least prone to complications.[3] A recent American article reported 54% primary patency for grafts at one year, compared with 75-91% for native fistulas.[4] Peritoneal dialysis

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is performed after placement of an abdominal catheter and uses the peritoneum as the exchange membrane. Renal transplantation has become the treatment of choice for end-stage renal disease (ESRD) because of long-term survival and improved life quality in cases when transplantation is performed instead of haemodialysis. Because of the limited long-term patency of haemodialysis grafts or fistulas, repeated surgical or percutaneous radiological repair is necessary to preserve the long-term patency of the dialysis access, and preservation or improvement of the renal function. Percutaneous radiological treatment provides the least invasive procedure, intended to prolong life of the dialysis grafts or fistulas.

Radiological management and endovascular treatment haemodialysis fistulas and grafts

The first indications for interventional radiology were stenoses, which were difficult to treat surgically because of their location (central veins) or because of their propensity to early recurrence. The first radiological reports in 1980s were disappointing due to inadequate armamentarium and the inexperience of radiologists.[5] In the last decade, a decisive turning point came with the availability of high-pressure balloons, hydrophilic guidewires, stents, declotting techniques such as the pulse-spray and thromboaspiration, which helped radiologist to improve success rates.[3,6] The advantages of the endovascular approach-minimal invasiveness, better imaging and better venous preservation - rapidly became very clear. The publication of the American DOQI guidelines in 1997 gave the first official recognition to the value of interventional radiology for the treatment of both stenoses and thrombosis.[7]

Radiological patient evaluation prior to access placement

The role of preoperative screening is twofold: first, to identify usable vessel to create autogenous fistulas or a synthetic bridge graft, and second to detect occult central venous stenosis. Radiological screening is performed by ultrasound, venography, and in future it will also be done by MRI.[8-10] This helps surgeons to create native or good synthetic grafts fistulas. Native fistulas, which are never used and early graft failures, are associated with the common problem of inadequate vessel (artery or vein) selection. Patients with ESRD often have multiple venepunctures and numerous intravenous accesses inserted and thus increased likelihood of venous stenosis or occlusion. Robbin found that 77% [40 of 52] of patients had a history of previous central catheter placement or major surgery in which a central catheter may have been placed.[10] Thirteen patients [25%] had a history of catheter placement on the same side as the planned haemodialysis access.

A patient who has had prior central venous catheterisation (jugular or subclavian) should be screened with venography and ultrasound to exclude occult central venous stenosis. Surratt *et al*. found a 40% prevalence of subclavian vein stenosis in patients who had previously had dialysis catheterisation in this venographic study.[11] Occult central venous stenosis is nearly always asymptomatic, and becomes unmasked only when high venous flow occurs due to the ipsilateral fistula or graft. Patients may develop marked arm swelling, which can impair wound healing as well as impede the use of the vascular access. Therefore, if central venous stenosis is detected, serious consideration should be given to placing access in the opposite extremity.

With ultrasound, vessels can be assessed for size, stenosis, and occlusion.[9-13] Although ultrasound can be used to evaluate the venographic anatomy of the arm, venography can provide the access surgeon with a »road map«

of the entire arm. It also excludes central venous stenosis.[8,9] Venography is performed by puncturing the dorsal vein of the hand. Opacification of the forearm veins is obtained after placement of a tourniquet in the upper arm in order to create congestion. The venous status (presence, absence, mean diameter, stenosis, and occlusion) of the cephalic and basilic vein in the forearm, at the elbow and in the upper arm can be evaluated as well as the patency of the central veins. Care should be taken to image the axillary vein with the arm in abducted position to avoid pseudostenosis. In cases of contraindications to iodine injection (for example allergy or previously non-dialysed patients), carbon dioxide gas or gadolinium can be used as contrast agents.[13]

Radiological stenosis detection and indication for treatment

The underlying stenosis is predominantly present at the venous anastomosis of the graft, in the revealed area of the forearm native fistulas and in the venous outflow far from the anastomosis of upper arm fistulas.[14] The underlying stenosis is unmasked in more then 85% of cases thrombosis the grafts and in almost all cases for native fistulas.[14,15] Thrombosis occurs roughly 10 times more frequently on prosthetic grafts then in native fistulas.[15]

Among the numerous methods described for stenosis screening, radiologists can use ultrasound and MRI examinations.[7,16] Ultrasound provides both anatomic and physiologic information that can be useful in screening and problem-solving.[17] The typical ultrasound findings in access stenosis include areas of locally increased velocity (greater than 400 cm/sec) and/or turbulence and the detection of hypoechogenic material consisting of intimal hyperplasia forming an anatomically visible stenosis. Duplex ultrasound is able to diagnose efferent vein stenoses with an accuracy of 96%, synthetic graft stenoses with an accuracy of 86%, and anastomotic stenoses in native fistulas with an accuracy of 81%.[16,18] Doppler measurement of velocity is then multiplied by the cross-sectional area of the graft to give volume flows in millilitres per minute. Normal graft usually has volume flows well in excess of 1300 ml/minute, and it has been shown that volume flow less than 300 ml/minute or 450 ml/min is correlated with impending graft failure.[19] Ultrasound evaluation can be performed on abnormalities on puncture sites including pseudoaneurysms, aneurysm dilatations of cephalic vein (which usually are not clinically significant) and aneurismal dilatation in PTFE grafts, which can be very significant. Patients with non-invasively established haemodynamic significant stenosis or occlusion require fistulography and endovascular treatment (Figures 1, 2, 3).[4,20,21]

Dialysis fistulography is the least invasive method. Fistulography can be performed with the dialysis needles in place. The only, if any, risks involved in the procedure are the risk of contrast allergy and further reduction in residual renal function,[21] The latter consideration can be obviated with carbon dioxide or gadoliniums fistulography in selected cases.[13] A well-performed fistulogram is the foundation of all percutaneous interventions in haemodialysis access (Figures 1a, 2a, 4a). The technique of fistulography has been well-described.[5] A complete fistulogram must show the entire ingraft or fistulas from the arterial side all the way through to the venous circulation, with evaluation of the venous outflow, and the central veins (Figure 1a).

DOQI guidelines 10 and 17 recommend treating stenoses greater than 50% only when there are concomitant clinical abnormalities and flow-rate reduction or pressure changes.[7] Dilatation of asymptomatic stenosis detected by systematic colour flow duplex ultrasonography was beneficial only for »virgin« grafts (those that had never been previously dilated

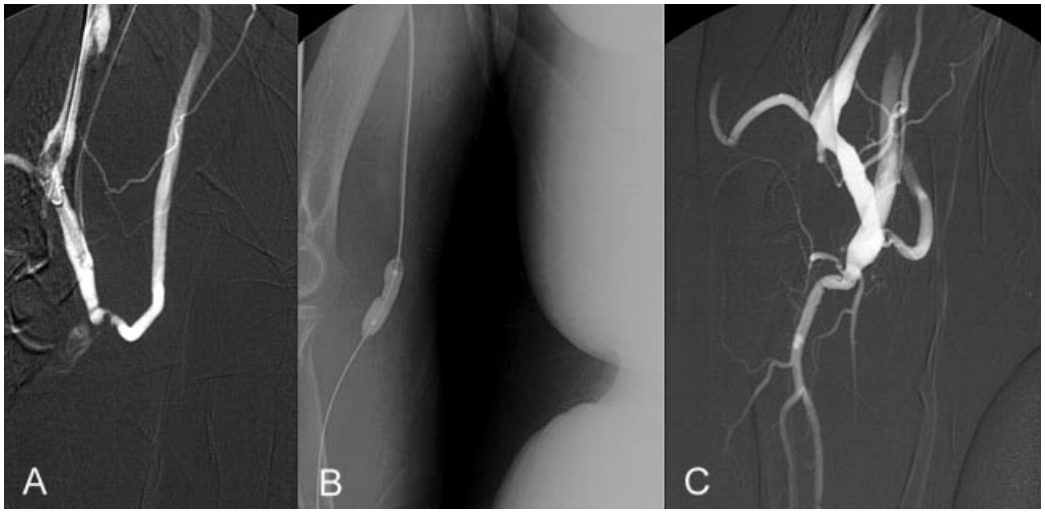

Figure 1. Fistulography. a. Stenosis in the anastomosis fistulae between brahial artery and cephalic vein, due to neointimal hyperplasia. b. PTA with 8 mm balloon; c. the result is a good anastomosis patency

or revised). The treatment of haemodynamically significant stenosis reduces the rate of thrombosis and prolongs the average use-life of the access.[8, 22-26]

Nevertheless, clinical examination should remain the key detection method. Clinical abnormalities include direct palpation of the stenosis under the skin and localised loss or reinforcement of the thrill. Stenosis of the arterial inflow or stenoses located in the anastomotic area can be responsible for a fistula, which is too flat, or for vacuum phenomena during dialysis. Stenosis in the cannulation areas can make routine needling difficult. Venous stenosis located far from the anastomosis causes congested fistula with loss of thrill, increased compression times after dialysis with formation of aneurysms and the development of collaterals. Finally, limb oedema indicates central venous obstruction (Figures2a, 4a). However, once haemodynamically significant stenosis is detected, the optimal timing of treatment for the prevention of thrombosis remains to be determined for both fistula and graft.

Dialysis-access thrombosis is usually detected clinically. A graft that no longer has a palpable pulse or thrill is clearly thrombosed and does not need any further evaluation for the diagnosis of access failure. Native fistulas, on the other hand, characteristically develop only thrombosis of segments of their venous outflow, and ultrasound and/or fistulography can be extremely helpful in delineating the length of thrombosis, as well as the character of venous reconstruction above the thrombosis site. Fistulography is usually done in anticipation of the percutaneous treatment of thrombosis and is extremely useful in planning revascularisation.

Endovascular treatment, haemodialysis access, native fistulas and grafts

Treatment of venous stenosis is important clinically because it preserves the access sites for future use.

Percutaneous angioplasty

Percutaneous transluminal angioplasty, which is an outpatient procedure, successfully treats over 80% of stenosis in both native and synthetic fistulas and in both venous and arterial outflow tracts.[4,6,14,20,21] Angioplasty

can be performed on both anastomotic and more proximal lesions including central stenoses (Figures 1a, 1c, 2b, 2c). Prospective angioplasty of all venous stenoses that narrow the lumen by more than 50% improves fistula function and prolongs access survival. Angioplasty therapy is safe, effective, and easily performed, and has become a standard therapy for the management of venous stenosis affecting dialysis-access fistulas and graft.[6,20,21]

Direct fluoroscopy observation is necessary to perform angioplasty, with digital capability being very advantageous. Heparinisation is unnecessary. During dilatation, flow is occluded for only short intermittent periods, and clotting of the access therefore rarely occurs. Because the procedure is potentially very painful, the patient requires adequate sedation.[21] Patients must be monitored very closely during this process. Since the requirements for adequate sedation varies considerably, the dosage must be carefully titrated.

Before angioplasty fistulography was performed to evaluate the anatomy and pathology of the fistulas or graft and its draining veins up through the superior vena cava (Figures 1a, 2a, 4a). The needle is inserted anterograde into the shunt-leading vein or graft. A hydrophilic-coated, steerable, 0.035-inch Terumo guidewire is passed up through the stenoses to the level of central veins. In cases where the vein is tortuous or occluded in a short segment, guidewire manipulation is also required with diagnostic catheter support, which can be a considerable challenge. A balloon catheter is passed over the guidewire and advanced to the most central lesion. It is important for the balloon to be slightly larger than the affected vein and that a high-pressure balloon is used.[4,21] The basic technique of angioplasty as applied to the access graft and associated venous range is dependent upon the use of a large diameter, high-pressure balloon catheter. Unless the veins are unusually small, a 6-8 mm balloon is used in both graft and peripheral veins and a 12 mm balloon is used centrally. A pressure of 10 atmospheres is routinely utilized. If this is not effective in breaking the lesion, pressures of 15 and 20 atmospheres are sequentially applied. The balloon is allowed to remain expanded for one to two minutes, with each in-

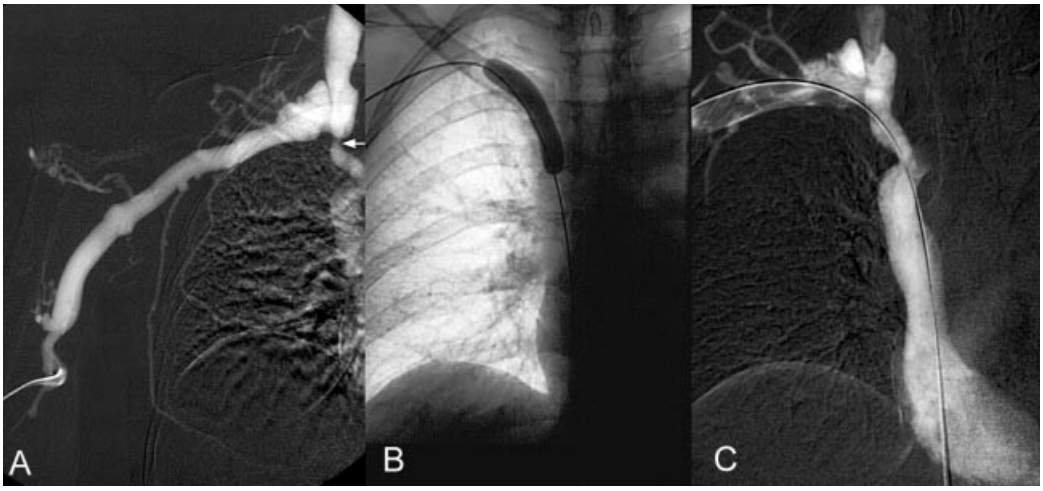

Figure 2. A patient with malfunctional forearm dialysis fistulas and oedema of the arm. Fistulography. a. High grade stenosis of the right brachiocephalic vein (arrow); b. PTA with the high pressure balloon; c. the result is a good patency.

flation. Multiple dilatations are used for resistant lesions. Post-procedural fistulography is performed to assess and document the results of the therapy (Figures 1c, 2c, 4d). The ability to measure intra-access blood flow immediately after the procedure may also prove beneficial.[18] Haemostasis is obtained following the removal of the vascular sheath by manual compression. Using a bandage is relatively inexpensive and very effective in most cases.

Patency rates with angioplasty vary according to the follow-up period and the types of access and vessel treated. Unfortunately stenoses do recur relatively rapidly. The largest series of angioplasty grafts have high success rates ranging from 94 to 98% in unassisted (or primary) patency rates, ranging from 17 to 40% at one year.[4-6,21] Over the period of a year, secondary patency rates of 82% and 85% were obtained in the forearm and in the upper arm respectively, but with more frequent reintervention in the upper arm (11 vs. 18 months). The initial success rate generally ranges from 80-94%.[14,21] The unassisted patency rate for angioplasty generally ranges from 31-45% per year. The only two large series for forearm native fistulas reported a success rate ranging from 91-95% and one-year primary patency rates from 44-51%.[4,5,21] This article also presents the positive influence of the age of the vascular access on the outcome dilatation of fistulas. Manninen reports a significantly poorer outcome when stenoses were located near the anastomosis and when the feeding artery was »small«.[22] When the outcomes of each of the lesions were compared to that of the anastomosis group, long venous, midgraft and subclavian groups had statistically inferior unassisted patency rates.[4,21] The poorest results e to be found in the literature are those associated with simple dilatation of the central vein (23 to 29% primary patency rate at 6 months).[26] Angioplasty treatment of venous stenosis based on prospective monitoring for increasing stenosis is effective. It results in a decreased incidence of thrombosis and extended usability of the dialysis vascular accesses.[4-6,21]

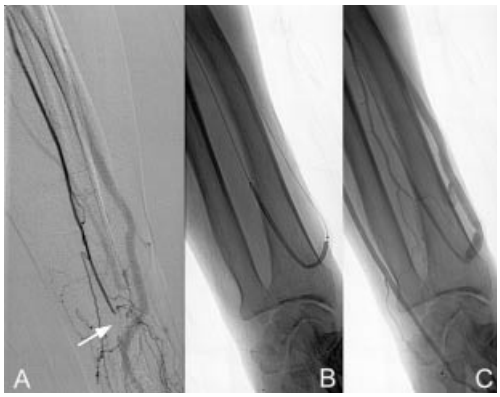

Figure 3. Reduced blood flow through the left forearm fistulas. a. Arteriography shows the occlusion of the radial artery above the fistulas (arrow), which functions through the collaterals of the palmar arch. b. Transarterial recanalisation and dilation of the occlusion. C: Higher blood flow through the fistulas.

Complications associated with venous angioplasty include severe allergy to iodine, infection, thrombosis and vein ruptures. Loss of vascular access following vessel rupture or thrombosis has become extremely rare, since stents and catheter thrombectomy are available. After a seemingly effective dilatation, some lesions recoil, which may occur immediately or over a period of days after the procedure.[4,20,21] Deaths related to procedures are extremely rare.[4]

Use of stents for venous stenosis associated with dialysis vascular access

The role of the endoluminal stent in the management of venous stenosis of dialysis access is unclear. Although stents can help manage some access difficulties, they are not permanent solutions. There is a consensus concerning the value of stent placement for the rupture of an outflow vein after balloon angioplasty, when prolonged balloon inflation fails[23] and for treatment of stenosis recoil. Elastic lesion may first completely dilate with the angioplasty balloon; significant residual

stenosis occasionally remains, suggesting an elastic lesion. The use of stent is suggested in cases of the rapid recurrence of stenotic lesion following angioplasty (Figure 4). Are stents significant for delaying restenosis? Same authors report intervals between interventions that double in length after stent placement, others disagree.[4,24-26] The presence of residual stenosis or developed thrombosis after stent placement might indicate insufficient pre-dilatation, and placement of stent during incomplete declotting procedures. In general, a 50% post-angioplasty stenosis may be used as a threshold for further intervention with stent. However, all authors of recent literature agree that only self-expandable stents should be placed in dialysis access and that indication must be selected. The diameter of the stent must be at least 1 or 2 mm larger than the diameter of the largest balloon used to dilate the stenosis. Its length should be as short as possible, to cover the lesion only. The stent must not be placed in stenotic lesions that cannot be dilated or a stenotic lesion at a venous anastomosis. At the venous anastomosis, a forearm fistula or graft stent can overlap the basilic vein and prevent the future creation of transposed brachiobasilic fistulas, or protruded stents into the subclavian vein from the final arch of the cephalic vein in which it could induce stenosis, which would preclude the future use of the basilic and axillary veins for direct fistula drainage of an upper arm graft. A stent placed in the subclavian vein must not overlap the ostium of a patent internal jugular vein. This vein is essential for placing the central catheter or for bypassing a subclavian stenosis. In order to avoid inducting stenosis on the contra-lateral trunk, a stent placed in the right or left brachiocephalic vein must not protrude into the superior vena cava. The stent must be placed in the brachiocephalic trunk only, without overlapping either the subclavian or internal jugular ostium. This means, however, that future access

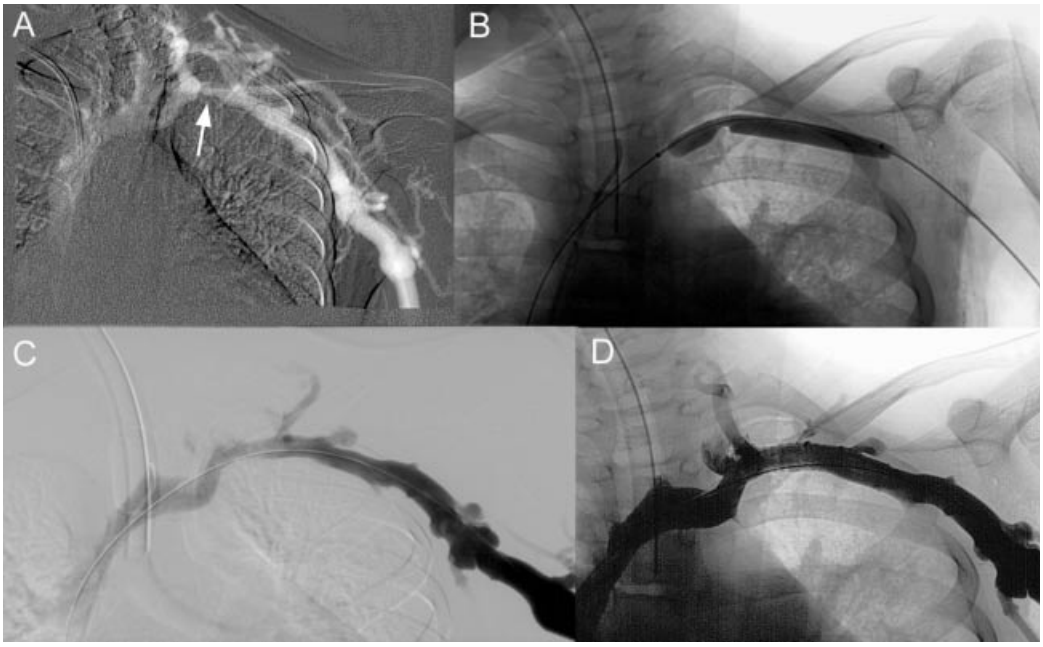

Figure 4. A patient with malfunctional dialysis fistulas and oedema of the arm. Fistulography. a. High grade stenosis of the subclavian vein with collateral flow (arrow). b. PTA with high pressure balloon of 12 mm diameter. c. Relatively good result after the PTA; d. better result after the placement of the stent.

sites must be anticipated before placing the stents. Authors recommend primary stent placement due to poor results after simple central vein dilatation (Figures 4b, 4c, 4d).[24,25]

Primary patency rate following the venous use of stents is relative poor, being approximately 20% per year. However, with aggressive re-intervention, the cumulative patency rate is roughly around 70% per year. Modalities that have been used for stent maintenance include thrombolysis, stent angioplasty, new stent, and the use of the Simpson or Redha atherectomy device. As previously mentioned, the principal difficulty underlying the inability to maintain patency is the development of neointimal hyperplasia.

The six-month and one-year stent primary patency rate in central veins was only 42% and 20%. Haage (84% and 54% respectively) and Mickely (90% and 70% respectively) obtained better results.[24,25]

The most typical complication is inaccurate placement or migration of stents.[24,25] Only two casualties have been reported linked to stent placement, by infection and presumed right atrium perforation.[4]

Endovascular treatment of dialysis fistula thrombosis

The most common complication of permanent vascular access are thromboses which, when not mended, account for 80 to 85% of access loss. The major predisposing factor is stenosis on vein side anastomosis of the vascular access, responsible for 85% of thrombosis.[2,4,6,14,20,21] Other causes of fistula thrombosis include arterial stenosis, post-dialysis fistula compression, hypotension, increased haematocrit levels, hypovolemia, or hypercoagulable states.[4,6,27] The therapeutic options of fistula thrombosis include surgical thrombectomy, thrombolysis with thrombolytic agents, thromboaspiration and mechanical dissolution. If these modalities are successful, a fistulogram can then be performed and detected stenoses treated with angioplasty or surgical revision.[4-6,14,20]

Surgical thrombectomy is a quick, outpatient procedure, has a very low complication rate, and is initially successful in 90% of cases.[4,21] However, failure to correct the underlying outflow stenosis leads to rapid rethrombosis.

The endovascular declotting techniques published to date can be divided into pharmaco-mechanical and purely mechanical methods. The earliest attempts to treat fistula thrombosis with thrombolytic agents, such as urokinase and streptokinase, originally yielded disappointing results.[27] However, recent dosing adjustments and technical advances have improved the success rate and reduced the incidence of bleeding in patients for whom there is no contraindication to thrombolytic therapy.[28] For example, the use of the pulse-spray technique, which combines thrombolytic therapy with hydromechanical clot disruption, rapidly established access patency in over 90% of cases with minimal complications. The pulse-spray method consists of placing two multi-sideholed catheters with tip-occluding wires in a criss-cross fashion in the thrombosed graft. The forceful and rapid injection of 0.2-0.3 ml aliquots of a 10 ml mixture combining 250,000 units of urokinase and 5000 units of heparin are than applied every 30 sec. to each catheter with a tuberculin syringe. The successful administration of tissue plasminogen activator has also been reported.

Pharmaco-mechanical methods include thrombolytics at low or high dose, infused locally through regular needles or specific catheters for some minutes or hours before detachment or the crushing of residual thrombus with a balloon, more rarely with an aspiration catheter. These were previously described as the Bookstein »pulse-spray« method, the Cynamon »lyse and wait« method, the Goodwin and Craggbrush infusion technique.[28-30] The commonly occurring

venous stenosis should be corrected.

Purely mechanical methods include clot extraction or disruption methods with the Trerotola and Sharaffuddin balloon-based methods, the Beathard spray-spray with saline method, the manual catheter-directed thromboaspiration, the Schmitz-rode rotating pigtail and all kinds of declotting devices. The major concern regarding these techniques is the possibility of clinically significant pulmonary emboli developing. To reduce the possibility of large pulmonary emboli, a device has been developed which consists of a high speed rotating camp tip which pulverizes the clot into tiny particles such as: Arrow-Trerotola PTD, Hydrolyser, Amplatz Thrombectomy device and many others that are likely to appear.

The technical success rates for pharmacomechanical and mechanical methods are 89-95%. Long-term primary patency rates are relatively poor in all reports, ranging from 8 to 26% per year.[4,21] However, secondary patency rates of 75% have been reported.[4] Treatment results of native fistula are better.[4,21,31]

The rates of significant complications range from 0-9%.[4,21] Complications include thrombus migration and vessel rupture, regarded as a significant complication, as well as infection, ischemic hand, remote or local bleeding, requiring transfusion or surgery, pseudoaneurysms and fluid overload.[4,5,31-33]

The mortality rate is low. Literature reports data on just six casualties linked to the declotting procedure. The causes of death were pulmonary or septic embolism and hemiplegia due to paradoxical embolism in patients with a patent foramen ovale and a right-to-left shunt.[4]

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review

Interventional radiological management of complications in renal transplantation

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Background. *The most frequent radiologically evaluated and treated complications in renal transplantation are perirenal and renal fluid collection and abnormalities of the vasculature and collecting system. Renal and perirenal fluid collection is usually treated successfully with percutaneous drainage. Doppler US, MRA and digital subtraction angiography (DSA) are most important in the evaluation of vascular complications of renal transplantation and management of the endovascular therapy.*

Conclusions. *Stenosis, the most common vascular complication, occurs in 1% to 12% of transplanted renal arteries and represents a potentially curable cause of hypertension following transplantation and/or renal dysfunction. Treatment with percutaneous transluminal renal angioplasty (PTRA) or PTRA with stent has been technically successful in 82 to 92% of the cases, and graft salvage rate has ranged from 80-100%. Complications such as arterial and vein thrombosis are uncommon. Intrarenal A/V fistulas and pseudoaneurysms are occasionally seen after biopsy, the treatment requires superselective embolisation. Urologic complications are relatively uncommon; they consist predominantly of the urinary leaks and urethral obstruction. Interventional treatment consists of percutaneous nephrostomy, balloon dilation, insertion of the double J stents, metallic stent placement and external drainage of the extrarenal collections. The aim of the paper is to review the role of interventional radiology in the management of complications in renal transplantation.*

Key words: kidney transplantation; renal artery obstruction; radiology interventional; angioplasty, balloon

Introduction

Renal failure is treated by dialysis or renal transplantation. Renal transplantation has become the treatment of choice for end-stage renal disease (ESRD) because of long-term survival and improved life quality in cases when transplantation is performed instead of hemodialysis.[1-3] The most frequent renal transplantation complications are the following: renal and perirenal fluid collections, decreased renal function, and abnormalities of the vascular, tubular system, and renal parenchyma. Postoperative fluid collections are common, after transplantation, and they include haematomas, seromas, urinomas,

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lymphoceles and abscesses. Vascular complications of transplantation include occlusion or stenosis of the arterial or venous supply, arteriovenous fistulas, and pseudoaneurysms. Urological complications consist predominantly of urinary leaks and urethral obstruction. Percutaneous and endovascular management of these complications has become an important component in the management of transplant patients and has led to further improvement in graft salvage rates.

Perirenal fluid collections

Perinephric fluid collections are the most common complications in renal transplantation, occurring in approximately 50% of transplant patients.[4] The majority of these are asymptomatic, but approximately 15 to 20% may cause symptoms secondary to local mass effect that can produce pain, hydronephrosis, lower extremity oedema and compromise transplant function.[4,5] These fluid collections can also become secondarily infected. The majority of these collections can be detected by ultrasound (US). US-guided aspiration and drainage is essential for the correct diagnosis and management of symptomatic post transplant fluid collections. However, computer tomography (CT) often delineates fluid collections and their anatomic relationship to adjacent structures better than US, particularly in obese patients. In addition, puncture and drainage can be performed with CT guidance in cases when US fails to demonstrate access to the collection.

Lymphoceles

Lymphocele formation is a late complication of renal transplantation caused by lymphatic obstruction or leak. It can occur in up to 15% of renal transplantations.[6,7] Simple needle aspiration of the fluid using sterile technique will make the diagnosis. At biochemical analysis, a lymphocele has the same levels of protein, urea nitrogen, creatinine, electrolytes, and occasionally, lipids as serum, so differentiation from urinoma, haematoma, seroma, or abscess is possible.[8-10] Most lymphoceles are discovered incidentally, are small or sterile and asymptomatic, and do not require therapy. If symptomatic, they are usually secondary to local mass effect, with compression of the urethra resulting in hydronephrosis, compression of the iliac vein with resultant lower extremity oedema, or deep venous thrombosis, or a combination of both. Only rarely cases do secondary compression of renal artery lead to postoperative hypertension. Symptomatic lymphoceles can be treated with either percutaneous or surgical techniques. Despite the relatively high success rates of surgical treatment (90% success rate), open surgery carries more risks, greater morbidity and longer hospitalisation.[10-13] The operation of choice is laparoscopic peritoneal marsupialisation (fenestration) of the cyst into the peritoneal cavity. The major advantage of the laparoscopic approach is the absence of postoperative *ileus*, with the opportunity to continue the enteral immunosuppressive regimen and a lower recurrence rate (4%).[5,13] Percutaneous therapy varies from simple aspiration to placement of a drain (Figures 1a, 1b), with or without sclerotherapy. Percutaneous needle aspiration is the usual technique for diagnosis, but simple percutaneous aspiration of the lymphocele results in 80%-90% recurrence rate and an infection of 25%-50%.[5,14-16] Indwelling catheter drainage alone has been reported in most series as reaching cure rates of 50%-80%.[8,12,17,18] The introduction of a sclerosing agent in conjunction with catheter drainage has improved success rates to 79%-94%, with recurrence rate up to 31%.[9,10,12,19-22] A number of agents, including povidone iodine, bleomycin, alcohol, doxycycline and talc have been used for sclerosis, but none of these agents universally. The major advantage of percutaneous

drainage and sclerotherapy is the very low incidence of major complications. The most frequently reported complication is secondary infection of the lymphocele, with reported rates of 7%-17%.[14,23-25] The major disadvantages of percutaneous drainage and sclerotherapy are the need for multiple treatments and the requirement of the catheter to be left in place for a significant period of time.

Urinomas

Urinomas due to extravasations of urine from the renal pelvis, urethra, or ureteroneocystostomy usually occur in the first 1-3 weeks following transplantation and may result from of disruption of the ureterovesical anastomosis, incomplete bladder closure, ischemia of the collecting system, post biopsy injury, or severe obstruction. The diagnosis is usually made by US or CT. Characterisation of the fluid can be achieved by obtaining a sample via US-guided aspiration and determining the creatinine concentration of the sample. After confirmation of the diagnosis, percutaneous catheter placement is indicated. Anterograde pyelography is the best test for confirming the source of leak. It is discussed in the section on urinary complications.

Haematomas

Postoperative perirenal haematomas occur frequently but are usually small and asymptomatic and should be considered a normal sequel to surgery.[6,26] In US, the acute haematoma appears as a fluid collection with echogenic debris. Later the clot lysis-decreased echogenicity appears. The CT appears as fluid collection with hyper attenuating areas prior to intravenous contrast material administration, a finding which is consistent with fresh blood. Size, location, and growth determine the significance of these collections. An increase in size may indicate the need for interventional management or surgery. Large haematomas can cause symptoms secondary to mass effect but usually only present a problem when they become secondarily infected. If the patient is asymptomatic and there are no signs of infection, these collections can be treated conservatively if the haematoma does not increase in size. Aggressive interventional percutaneous drainage can successfully resolve an infected haematoma but larger calibre drains (12 or

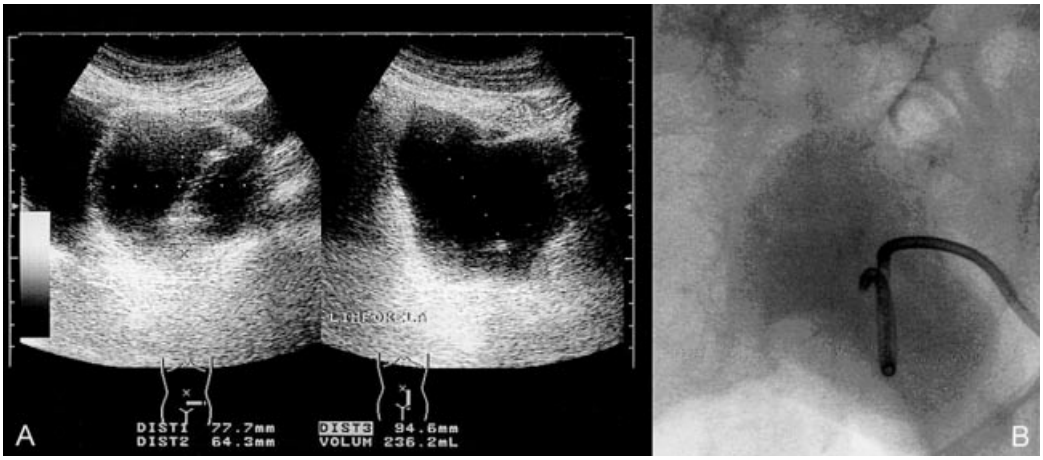

Figure 1. Lymphocele. a. US image demonstrates a large hypoechoic fluid collection with single septation in the left side of the pelvic cavity. **b.** Percutaneous catheter drainage (X-rays)

14 Fr) are usually required. Immediate postoperative haematoma can be secondary to graft rupture or injury to the vascular pedicle. Emergency surgery is mandatory in these cases.

Perirenal abscess

Perirenal abscess is an uncommon early complication of renal transplant. When a patient has fever and US or CT demonstrates perirenal collection with air, the diagnosis of perirenal abscess is clear. The treatment of abscess is based on antibiotics together with a percutaneous or surgical drainage procedure depending on the size of the abscess and the clinical course. Bouali *et al* and Lang report success rates of 67% and 96% in a series of 31 and 33 cases of abscesses treated percutaneously together with low rate in complications.[27,28] Percutaneous drainage under US or CT guidance avoids the risk inherent in surgery and anaesthesia, saves considerable time and meets greater patient acceptance. We recommend insertion of a percutaneous catheter into abscess to obtain a specimen for culture, and to drain the pus-filled cavity in order to hasten recovery and shorten the duration of anti-microbial therapy.

Vascular complications

Vascular complications are found in less than 10% of renal transplant recipients, but they are an important cause of graft dysfunction.[6,29-35] The most frequent vascular complications of renal transplantation include stenosis, thrombosis, arteriovenous fistulas, aneurysms, and pseudoaneurysms in the graft artery and in the recipient iliac arterial system. In contrast to other causes of transplant dysfunction, vascular complications are associated with high morbidity and mortality. The diagnostic screening methods are ultrasound with duplex and colour Doppler modes and three-dimensional (3D) gadolinium (Gd)-enhanced magnetic resonance angiography (MRA).[36,37] Digital subtraction angiography (DSA) remains the standard procedure for final diagnosis and checking endovascular treatment of vascular complications. Endovascular management is the initial treatment of choice for vascular complications in renal transplantation.

Renal artery stenosis

Stenosis, the most common vascular complication of renal transplantation, occurs in 1%-12% of transplanted renal arteries and represents a potentially curable cause of hypertension following transplantation and/or renal dysfunction.[30-34,39-42] The cause of transplant renal artery stenosis (TRAS) is multifactorial and includes surgical technique (clamp injury to the vascular endothelium, perfusion pump cannulation injury of the donor vessel, faulty suture technique), angulations due to disproportionate length between graft and iliac artery, kinking of the renal artery, type of allograft, immunological factors, and cytomegalovirus arteritis.[40,41] In long-standing transplants, progression of underlying arteriosclerosis in the recipient iliac artery can also be a source of arterial stenosis. The incidence of renal artery stenosis is more common in cadaver transplant (most renal allografts originate in cadavers), and it can occur as early as two days or as late as several years after the procedure.[38,43] In cadaver transplant, allograft revascularisation is usually performed as an end-to-side anastomosis with the external iliac artery. When a living, related donor is used, an end-to-side anastomosis with the external iliac artery (AIE) may still be used, but an end-to-end anastomosis with the internal iliac artery (AII) is often preferred.[44] The clinical presentation often mimics that of rejection, but a diagnosis of renal allograft arterial stenosis may be strongly suggested on the basis of sonography and

biopsy. A renal allograft biopsy is generally performed prior to angiography in order to rule out chronic rejection of other forms of renal parenchyma disease. Doppler US has become the preferred screening modality for stenosis of the transplanted renal artery.[45] Doppler ultrasound has been reported to have a sensitivity of 85% to 100% and specificity of 86% to 100% in the diagnosis of TRAS.[5,44-48] Data suggest that this technique is highly accurate, but is highly dependent upon the experience of the ultrasonographer. In obese patients, colour Doppler evaluation can be difficult and MRA or CT angiography can be helpful as an adjunctive non-invasive diagnostic test for TRAS. The gold standard for diagnosis of transplant renal artery stenosis remains DSA, being diagnostic in 93% of cases.[49] It should be performed in patients with clinical and Doppler US findings of TRAS or in patients with clinical findings of stenosis despite normal US results. However, it is invasive and in patients with marginal renal function it can induce acute tubular necrosis secondary to contrast toxicity. A useful alternative to nephrotoxic iodinated contrast agents is provided by CO^2 or Gadolinium DSA.

The options available to correct stenosis of the renal artery include conservative treatment, transluminal angioplasty (PTRA) with or without stenting and surgery. If the stenosis is relatively minor and the blood pressure controllable with medication, it is reasonable to continue with conservative treatment. The conservative option becomes particularly attractive if the lesion is unsuitable for PTRA or when PTRA is used but it fails. Surgery should only be undertaken for graft arterial stenosis if the other alternatives of PTRA or conservative treatment are not appropriate. PTRA has become accepted as the initial procedure of choice in the treatment of renal allograft arterial stenosis, both in allograft renal and native iliac artery stenoses (Figures 2a, 2b, 2c). The best approach to end-to-end anastomoses (AII) is from the contralateral-femoral artery, whereas end-to-side anastomoses (AIE) are usually more accessible from an ipsilateral approach. Non-selective pelvic arteriography was performed to exclude inflow lesions by means of a femoral approach as previously described. A narrowing of greater than 50% of the luminal diameter was considered haemodynamically significant. All patients who underwent interventional radiological treatments received intra-arterial boluses of heparin (5000 IU) during the procedure. The technical success of PTRA was defined as a residual stenosis of less than 30%

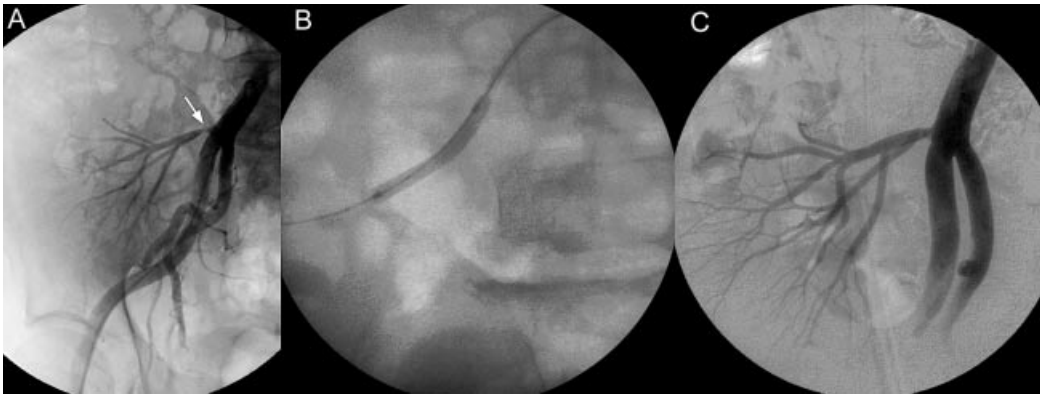

Figure 2. Transplant renal artery stenosis. **a.** Cadaver renal transplantation, with a single renal artery and end-to-side anastomosis to the right external iliac artery. Arteriogram shows a stenosis of the transplant renal artery (arrow). **b.** Balloon dilatation. **c.** Repeat arteriogram after PTRA with 5-mm diameter angioplasty balloon.

after angioplasty and no flow-limiting intimal flap. Clinical success was defined as more than 15% reduction in serum creatinine level, more than 15% reduction in mean diastolic blood pressure with the number of anti-hypertensive medications equal to that before PTRA, or more than 10% reduction in mean diastolic blood pressure with a reduction in the number of anti-hypertensive medications.[40] Technical success has ranged from 82% to 93%, and graft salvage rates have ranged from 80%-100%.[30-34,41-50] Although up to 20% of cases will develop recurrent stenosis, but are usually amenable to repeat PTRA,[30-34,39,43,50] periprocedural morbidity is generally low, and significant complications have been reported in 0% to 12% of cases.[44,51] Complications are usually related to puncture site complications and rarely to distal extremity embolisation, extensive arterial dissection, renal artery thrombosis and renal artery rupture.[30-34,39,43,50] The success of stent placement used in combination with PTRA for a wide variety of vascular lesions suggests that deployment of metallic stents may be useful in those with recurrent stenosis of the transplant renal artery, failure to satisfactorily eliminate the stenosis after repeat balloon dilatation, intimal dissection with luminal compromise, and stenosis involving the renal arterial ostium (Figures 3a, 3b, 3c, 3d). Stent deployment in six patients with recurrent stenosis was evaluated in retrospective study.[52] At almost three years post procedure, all arteries were patent without significant stenosis, and no additional interventions were required. The role of stent placement for the treatment of TRAS and its effect on long-term patency has yet to be investigated.

Renal artery thrombosis

Renal artery thrombosis is an uncommon complication of transplantation and usually occurs in the early postoperative period, almost invariably leading to graft loss. The most common causes are faulty surgical anastomoses, persistent hypotension, dehydration, and procoagulant conditions such as lupus anticoagulant, severe acute rejection, and progression of a stenosis to thrombosis. Spontaneous late thrombosis of the renal artery is a rare event, and renal artery stenosis is an obvious risk factor. Thrombosis of long-term grafts after intervention such as angiography or attempted angioplasty is more common than spontaneous renal artery thrombosis. Renal artery thrombosis after PTRA may be minimized by keeping the patient well anti-coagulated during the procedure and by the intra-arterial administration of nitro-glycerine in order to prevent vascular spasm. The diagnosis is relatively simple when duplex and colour Doppler techniques fail to demonstrate intra renal venous and arterial flow, with angiography providing confirmation prior to intervention.[53] In selected cases, the early diagnosis of vascular thrombosis may en-

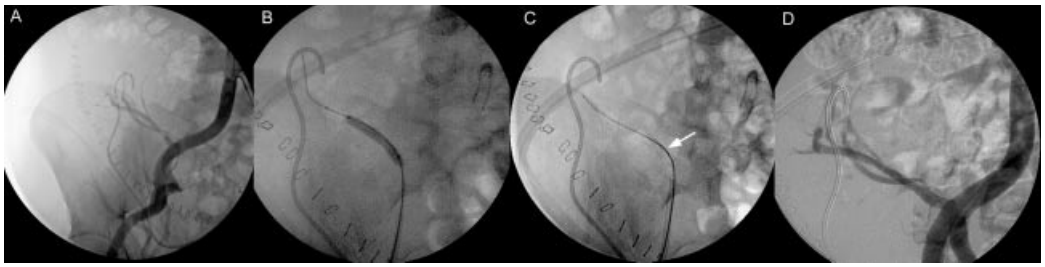

Figure 3. Transplant renal artery stenosis. **a.** Cadaver renal transplantation, with end-to-side anastomosis to the right external iliac artery. Arteriogram shows a stenosis of the transplant renal artery. **b.** Balloon dilatation. **c.** Metallic stent after incomplete PTRA (arrow). **d.** Repeat arteriogram after procedure shows a widely patent transplant renal artery with no residual stenosis.

able graft salvage thrombolytic treatment or clot aspiration. The use of thrombolytic therapy in treating renal artery thrombosis has been reported as successful.[44,54,55]

Intrarenal arteriovenous fistulas and pseudoaneurysms

Intrarenal arteriovenous fistulas and pseudoaneurysms are occasionally seen after biopsies. They occur in 1%-18% of renal biopsy.[5,56,57] Intrarenal arteriovenous fistulas may appear when an artery and vein are lacerated; pseudoaneurysms result when only the artery is lacerated. Arteriovenous fistulas within the kidney are usually asymptomatic, and normally resolve spontaneously. When lesions are sizable, marked arteriovenous shunting may result in renal ischemia, hypertension or hematuria.[6,57] They are easily identified at colour and Doppler US. Helical CT is a good alternative when US cannot define the nature of the lesion. However, a negative US and CT evaluations does not exclude the diagnosis, and, if clinical suspicion remains, these patients should be evaluated with angiography. When symptomatic or large, intrarenal arteriovenous fistulas and pseudoaneurysms may be effectively treated with selective arterial catheterisation and embolisation. Transcatheter embolisation is a safe and effective alternative to surgery; however, it may result in a segmental infarction and impaired renal function if the feeding vessel is embolised. Treatment requires superselective embolisation in order to preserve the maximum amount of renal parenchyma (Figures 4a, 4b). A potential complication is occlusion of the main renal artery due to migration of the embolising agent. Coils are typically chosen as the embolic agent for arteriovenous fistulas because of the greater control in deployment when compared to Gelfoam embolisation.[3] Perini *et al*[58] performed embolotherapy in 21 patients with renal allografts and vascular complications. Technical success was achieved in 20 of 21 patients (95%) without serious complications, and no long-term graft dysfunction was noted in 58% of those treated.

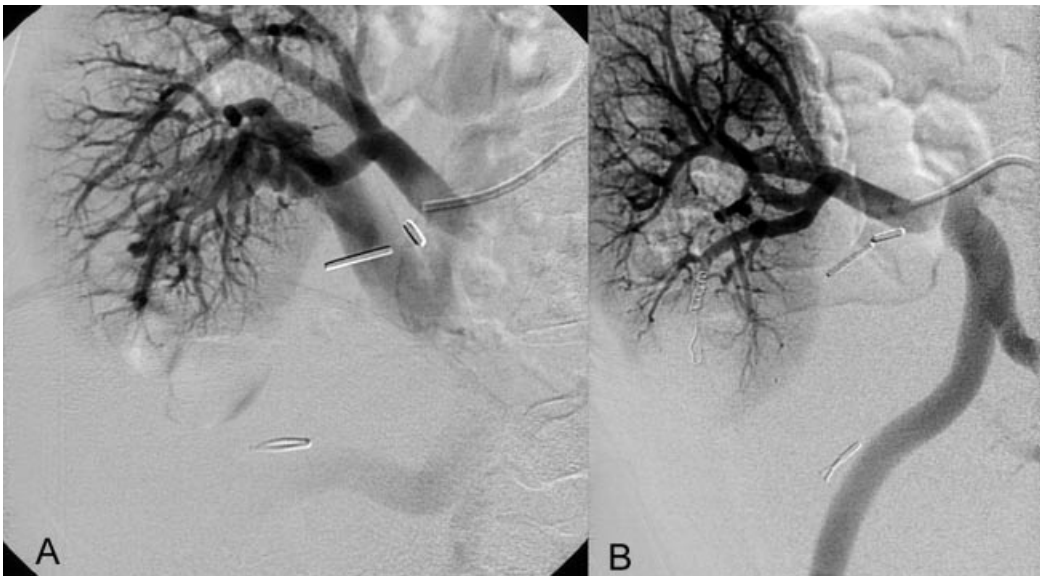

Figure 4. Intrarenal arteriovenous fistula following biopsy. **a.** Angiogram obtained with selective injection of the main renal artery shows early filing of the transplant renal vein. **b.** On an angiogram obtained after subselective embolization with coils, early draining veins are not visualized.

Renal vein thrombosis

Renal vein thrombosis is an uncommon complication that usually occurs in the first postoperative week. When occurring in the early post transplant period it is usually associated with surgical complications and often results in the loss of the graft. At a later stage, when graft function has stabilised, its development may be associated with underlying disorders such as glomerulonephritis, immunosuppressive therapy, acute rejection, or extension of lower extremity venous thromboses.[9,58,59] The clinical features, with the effect on urine output, vary from primary non-functioning, similar to that seen with arterial thrombosis, to sudden loss of urine output and rising creatinine in a graft which is different than in one functioning perfectly. The clinical signs are striking, often with severe pain resulting from rapid local graft swelling. The usual outcome of renal vein thrombosis is infarction, and transplant nephrectomy is usually performed to prevent infection.[53] Whenever such a diagnosis is made, surgery is always urgent, because, apart from the necessity to relieve the pain, delay is associated with an increasing risk of graft rupture, which may result in catastrophic graft haemorrhage. The sonographic diagnosis dependents mainly on the Doppler portion of the examination. On grey-scale US images, the allograft may appear swollen and hypoechoic. At Doppler US examination, venous flow is absent, and the arterial waveform shows reversed, plateauing diastolic flow.[6,60,61] The diagnosis is confirmed by venography with selective venous catheterization and therapeutic thrombolysis. There are several case reports in the literature on the attempts to treat transplant renal vein thrombosis with arterial, venous, or a combination of arterial and venous thrombolysis and percutaneous mechanical thrombectomy, but results vary.[55,59,62] It is uncommon to achieve complete thrombolysis, but partial lysis of the vein may result in a marked clinical improvement.

Urological complications

Urological complications after renal transplantation are relatively uncommon. They predominantly consist of urinary leaks and urethral obstruction. The quoted incidence of urological complications ranges from 5% to 14%.[6,63-66] Urologic complications can be divided into early and late categories. Early complications are defined as those that occur within three months of the transplant, and late complications are those that occur after this period of time.[64] Leaks tend to occur early, although obstruction may occur at any post transplant stage. These complications must be diagnosed early, because delays in diagnosis can result in the loss of transplanted kidney as well as increased patient morbidity and mortality. The most commonly performed interventional procedure in the renal transplant patient is percutaneous antegrade nephrostomy for urinary obstruction or leak.[44,67]

Urethral obstructions

Urinary tract obstruction can occur early or late after renal transplantation and are observed in less than 5% of patients.[6,53,65,66,68] Patients with obstruction are typically asymptomatic, and the diagnosis is made during evaluation because of a rising serum creatinine level and is confirmed by ultrasound examination of the transplanted kidney. The common causes of obstruction are urethral stricture or kinking (accounting for more than 50% of obstruction), urethral blood clot, and urethral compression from lymphocele.[5,44] Less common etiologies of obstruction include oedema or narrowing at the ureteroneocystostomy, pelvic fibroses, fungal debris, or compression from an extrinsic mass such as adjacent haematoma or lymphoadenopathy. More than 90% of urethric stenoses occur within the distal third of the urethra and may appear days or years after

transplantation.[68,69] Once obstruction is suggested by US, antegrade pyelography can be obtained in order to confirm the diagnosis, to provide detailed, anatomic definition of the type and level of the obstruction, and to serve as an access route for percutaneous management. Interventional radiology treatment of urinary tract obstruction consists of percutaneous nephrostomy, balloon dilatation, insertion of double J stents, stent placement, or correction of the source of extrinsic compression of the collecting system, such as a lymphocele. If obstruction is secondary to ureteroneocystostomy oedema or urethral blood clot, external diversion through a percutaneous nephrostomy catheter often provides temporary relief of obstruction until the oedema subsides or the clot has passed spontaneously (Figure 5).[5,44,65] The nephrostomy is removed only after an antegrade nephrostogram has confirmed that the urinary tract is unobstructed.

Urethral stenosis in the early postrenal transplant period can be safely and effectively treated by percutaneous dilatation and stenting, with few side effects and long-term success.[70,71] This method is especially efficient in patients who develop urethral stenosis within three months of the transplant. In patients with urethric strictures developing three months after transplantation or later, percutaneous stenting is of limited significance and most patients require surgical correction. Bhagat *et al*[70] and Fontaine *et al*[71] reported a success rate of 69% and 62% of early obstruction (within three months) and 33% and 16% of late obstruction (after three months) in a series of 41 and 44 cases treated percutaneously with urinary diversion by percutaneous nephrostomy, balloon dilatation and urethral stenting. We advise the application of urethral dilatation to short fibrotic strictures, particularly those located at the ureterovesical junction. A high-pressure balloon, selected according to urethra and stricture size, was inserted and advanced to the stricture and inflated for 1-4 min. When the pressure dents on the balloon disappeared, the stricture had been controlled, and an 8-9 Fr double J stent was passed into the urethra (Figure 6). Usually the stent is kept indwelling for 9 to 18 weeks, after which it is removed cystoscopically.[5] However, surgical reconstruction may be required for long or recurrent stricture, and strictures refractory to balloon dilatation.

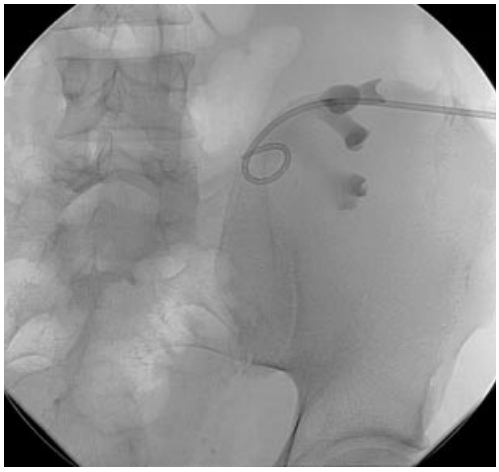

Figure 5. Urinary tract obstruction. Function is maintained by percutaneous nephrostomy.

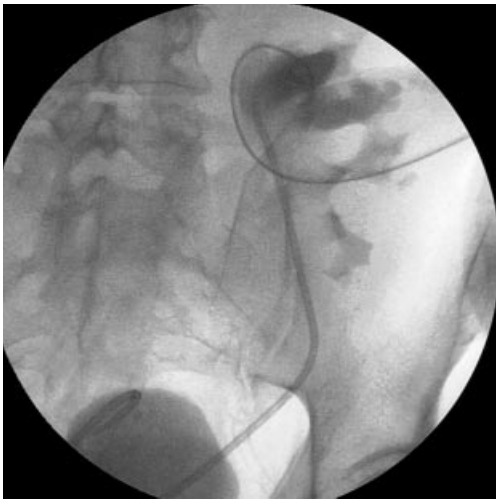

Figure 6. Urinary tract stenosis. An 8-F double J stent is placed across the stenosis after dilatation.

Urine leaks (urinomas)

Urine leaks are relatively rare complications following transplantation, occurring at the frequency of approximately 1% to 5%, and appearing in the early postoperative period as pain, swelling, and discharge from the wound.[65,68,72] Urethral extravasations producing urinoma can be caused by graft rejection, urethral necrosis due to ischemia, or inadequate surgical technique.[63] Most urine leaks occur at the ureteroneocystostomy, possibly due to vascular insufficiency, or along the anterolateral surface of the bladder where the ureteroneocystostomy has been performed.[44,68,70] Leaks may also occur from the proximal urethra, renal pelvis, or calyces secondary to distal urethral obstruction, renal infarction, or percutaneous renal biopsy. Diagnosis is usually made by US evaluation of the transplant, which reveals perinephric fluid collections that is relatively anechoic but may contain septations. Initial management should include percutaneous aspiration and fluid analysis to distinguish it from a lymphocele, which reveals an elevated creatinine level. A percutaneous nephrostogram and drainage can be both diagnostic and therapeutic, and effective in the treatment of urine leaks in renal transplant patients.[65,67,70] Definitive therapy can be carried out surgically or percutaneously with urethral stenting, double-J stents and urinary diversion by percutaneous nephrostomy.[70,71,73] Matalon *et al* have reported a success rate of 87% in a series of 23 cases of urinary leaks treated percutaneously with urinary diversion.[74] Fontaine et al treated 17 patients with transplant urinary leaks with percutaneous nephrostomy and nephroureteral stent and achieved successful closure of the urinary fistula in 10 patients (59%).[71] Benoit *et al* described successful closure of urinary leaks in all 7 patients (100%) with percutaneous nephrostomy and stent insertion.[72] The duration of catheter drainage typically ranges from 6-17 weeks.[5,65,71,74]

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case report

Annular pancreas causing extrahepatic biliary obstruction

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Background. *Annular pancreas is an uncommon congenital abnormality, consisting of a flat band of pancreatic tissue, which encircles duodenum or extrahepatic biliary duct. We present a case of obstructive jaundice, caused by annular pancreas.*

Case report. *A 46 years old female was admitted because of a sudden onset of abdominal pain, vomiting and jaundice. For the last six years she occasionally noticed her skin was light yellow, in the last year she felt distension in the upper abdomen, especially after fatty meals.*

Conclusions. *Two US examinations, the first one six months before the admission, showed dilated hepatic ducts. The reason of dilatation was unclear, even after the endoscopic US examination. At operation an almost complete obstruction of the common hepatic duct was found, caused by a narrow band of pancreatic tissue.*

Key words: pancreas abnormalities; bile duct obstruction, extrahepatic; cholangiopancreatography, endosopic retrograde

Introduction

Annular pancreas is an uncommon congenital abnormality. It consists of a flat band of pancreatic tissue completely encircling the second portion of the duodenum or the extrahepatic bile duct.[1]

It may manifest clinically in the neonate (52%) or remain asymptomatic until adulthood (48%).[1] It can represent an incidental finding during an endoscopic retrograde cholangiopancreatography (ERCP).[2]

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In adults, symptoms of annular pancreas usually present at the age of 20-50 years as a duodenal obstruction, rarely as a biliary tract obstruction.[2]

Jaundice associated with an annular pancreas has been presumed to result from pancreatitis,[2,3] but some reports suggest that an annulus is, by itself, capable of causing a significant biliary obstruction.[4,5]

We present a case of obstructive jaundice, caused by annular pancreas, without pancreatitis.

Case report

A 46 years old female presented to the emergency room suffering from an acute abdominal pain and vomiting. At physical examina-

tion she was jaundiced. Liver function tests were abnormal (AST 7.14, ALT 8.78, bilirubin 57/42, γGT 2.92), but levels of lipase and amylase were normal. The US examination showed thickened gallbladder wall, proximally dilated common bile duct and moderate dilatation of the right and the left intrahepatic ducts. No gallstones or masses were seen (Figure 1).

For the last six years the patient occasionally noticed having light yellow coloured skin. In the last year she felt distension in the upper abdomen after meals, especially in the afternoon, after eating fatty food. She never vomited, had no diarrhoea and was afebrile. Difficulties gradually increased and 6 months ago she underwent the first US examination. A concrement, a few mm in size, was found in the infundibulum of the gallbladder. The common bile duct was dilated up to 1 cm above the ampulla of Vater. The cause of dilatation was not clear.

The patient underwent ERCP.

Duodenum and papilla of Vater were normal. A 2 mm long stricture of the common bile duct with prestenotic dilatation was seen, intrahepatic ducts were also dilated, the cystic duct was not obstructed (Figure 2).

The pancreatic duct opened into the common ampulla and had a normal course. There was no accessory pancreatic duct visible.

The endoscopic US examination confirmed a short, smooth stenosis of the choledochal duct and a dilatation of the hepatic duct, looking benign. Some small concrements were found in the gallbladder. There were no signs of tumour like masses (Figure 3).

A congenital anomaly was suspected and with the patient in a good condition, despite a long history of symptoms, the surgeon de-

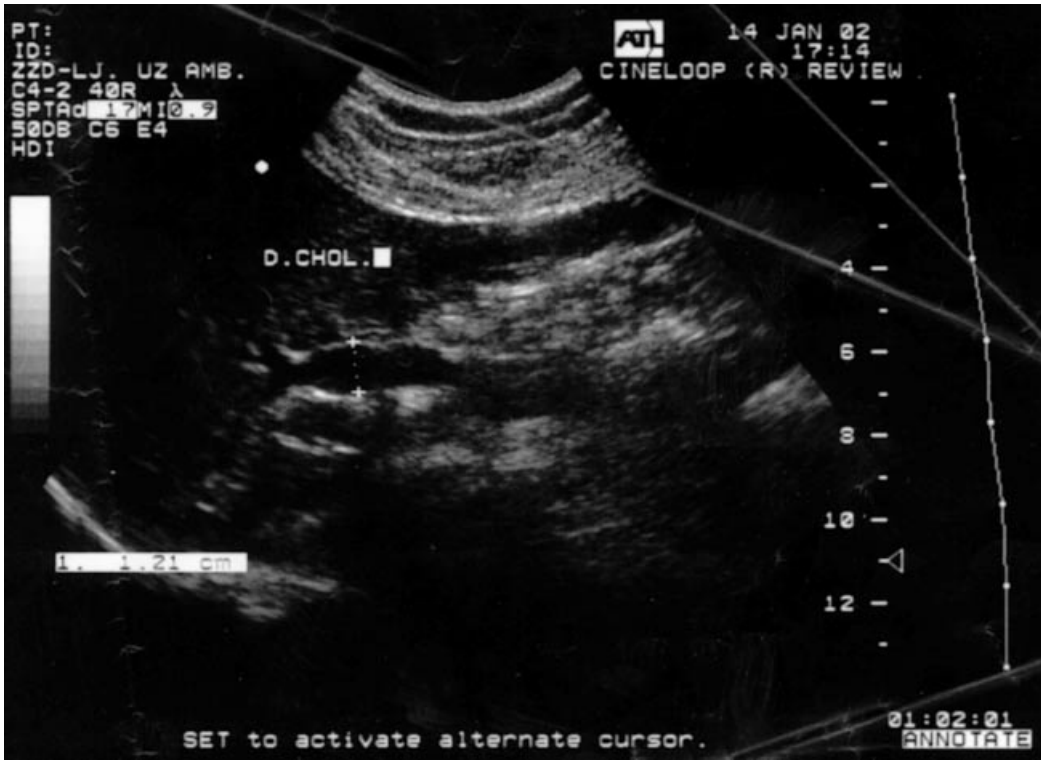

Figure 1. Ultrasound: dilatation of hepatic ducts.

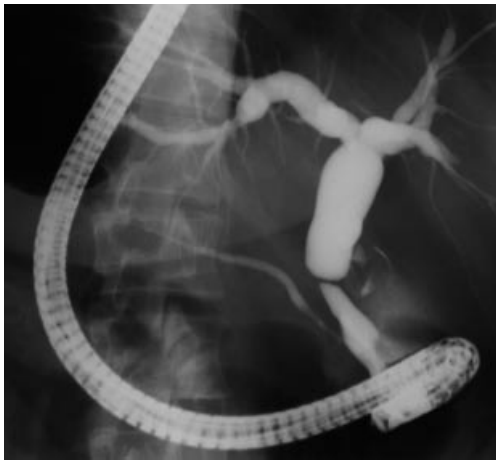

Figure 2. ERCP: stenosis of common hepatic duct proximally.

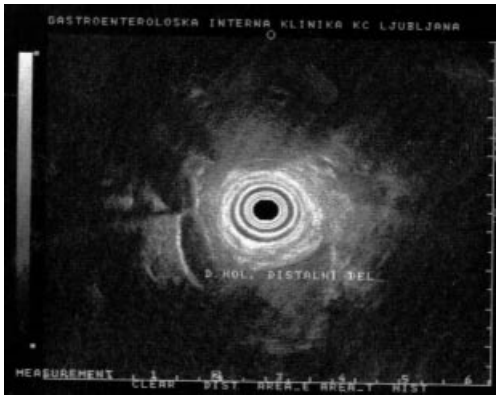

Figure 3. Endoscopic ultrasound: stenosis.

cided to perform abdominal laparotomy without any additional imaging procedures.

At intraoperative cholangiography, an almost complete obstruction of the common bile duct was found (Figure 4). It was caused by a narrow band of pancreatic tissue encircling the common bile duct. A cholecystectomy and a hepaticojejunal anastomosis were done.

Discussion

Annular pancreas is a congenital anomaly of rotation and fusion of primitive intestine and

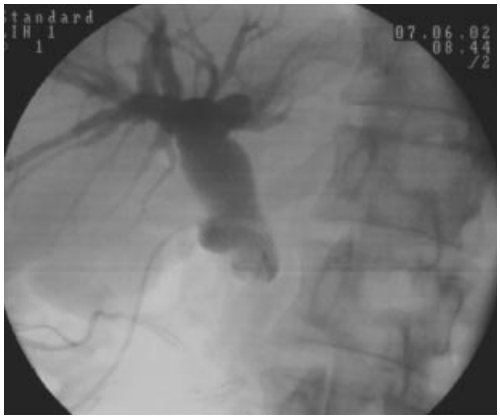

Figure 4. Intraoperative cholangiography: stenosis causing complete obstruction of common hepatic duct.

its associated structures, which may cause duodenal obstruction in the neonatal period or remain silent throughout life.[2] At postmortem, the incidence has been quoted as between 5-15/100.000. ERCP has led to a much more frequent diagnosis of annular pancreas in as many as one in 150 patients undergoing this procedure.[4]

There are three theories concerning the formation of the annular pancreas:
a) hypertrophy of both, the ventral and the dorsal ducts, resulting in a complete ring,
b) adherence of the ventral duct to the duodenum before rotation and
c) hypertrophy or adherence of the left bud of a paired ventral primordium.[1]

There is a clear distinction between the annular pancreas encircling duodenum presenting in children and that in adults, probably due to different severity of stenosis.[6]

In neonates, the symptom of severe duodenal obstruction is vomiting on the first day of life. There is often previous history of polyhydramnion. A number of other anomalies such as intestinal malrotation, duodenal atresias, and cardiac anomalies are often present as well.[1,6]

In older children and adults, the onset is more insidious, with intermittent clinical problems. Nausea, vomiting, and epigastric

pain are the main complaints due to the duodenal obstruction.[2,4] Pancreatic stenosis of the duodenum is usually not sufficient to cause symptoms until there the provocating factors - peptic ulcer disease or pancreatitis - supervene.

The incidence of associated gastric and duodenal ulcers ranges from 26 to 48%, and pancreatitis develops in 15 to 30% of patients.[1,2]

It has been suggested that annular pancreas may be the cause of extrahepatic biliary obstruction because of pancreatitis in the parenchyma, encircling the duodenum and bile duct, although jaundice is rare.[3]

In our case, no laboratory or clinical signs of pancreatitis were present. Laboratory data (AST 7.14, ALT 8.78, bilirubin 57/42, γGT 2.92, normal lipase and amylase) were suggestive of biliary obstruction with the abnormal liver function.

The US examination is the first in patients with abdominal symptoms. ERCP is the procedure of choice in patients with biliary obstruction, especially if the findings at US are equivocal. A diagnosis of annular pancreas encircling biliary tract is not always made preoperatively.[2,7] ERCP fails to make the diagnosis of annular pancreas if there is the obstruction of the duct leading to annular pancreas, if the duct of the annular pancreas does not empty into the main pancreatic duct or if there is a high grade of duodenal obstruction.[2]

In our case, no anomaly of the pancreatic duct was shown at ERCP.

In reviewing literature, not many cases of annular pancreas, causing biliary obstruction, without pancreatitis or duodenal obstruction, were found.

Adamo and co-authors reported it as being very interesting because of obstructive jaundice being the first symptom of this anomaly.8 Another article presents three cases of annular pancreas with the obstructive jaundice, but the obstruction was due to carcinoma of ampulla of Vater.[4,9]

In another case annular pancreas was causing a dilatation of biliary and pancreatic ducts, without obstructive symptoms and with normal liver tests. This patient underwent CT because of the other suspected disease and the dilatation was an accidental finding.[10]

Conclusions

The prevalence of annular pancreas in Slovenia is not known. In spite of such congenital anomaly being rare and much rarely being the cause of obstruction of the extrahepatic biliary tract we should keep it in mind as a differential diagnostic possibility. But, as other authors suggest, an annular pancreas should not be considered a satisfactory explanation for the obstructive jaundice until other possibilities have been fully excluded.[4]

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case report

Clinical importance of portal venous gas detected by abdominal sonography: a report of two cases

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Background. *Portal venous gas (PVG) can be a sign of serious disease and a predictor of poor clinical outcome. However, it can also occur as a transient phenomenon with little clinical significance, especially following blunt abdominal trauma and various diagnostic and therapeutic procedures.*

Case reports*. We describe two patients with PVG detected on abdominal sonography, who had very similar sonographic findings but a completely different clinical outcome. The first patient was a 70-year-old man in whom PVG was the consequence of mesenteric infarction; the patient died in hospital shortly after the admission. Our second patient was a 26-year-old man who was injured in a motor vehicle accident. He was clinically stable and needed no aggressive treatment. PVG proved to be a transient phenomenon that cleared spontaneously within a day.*

Conclusions. *Doppler sonography is a sensitive and specific modality for the detection of PVG. In patients with a serious underlying disease, sonography can identify the cause of PVG, so that the appropriate therapy may be undertaken without delay. If the aetiology is unclear, the decision to undertake further expensive and potentially harmful diagnostic procedures should be based on the patient's clinical status.*

Key words: portal vein; embolism, air - ultrasonography; ultrasonography, Doppler

Introduction

Portal venous gas (PVG) in adults is a rare entity. In the past, PVG was regarded as a sign of serious disease (mostly intestinal ischemia), predicting a poor clinical outcome in most cases. In the recent years, however, the routine use of sonography in the evaluation of a variety of conditions, in particular blunt abdominal trauma, has led to the recognition of a number of clinically unimportant causes of PVG.[1]

We describe two patients with PVG detected by abdominal sonography who had a completely different clinical outcome.

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Case reports

Our first patient was a 70-year-old man presenting with severe abdominal pain, who had been found lying on the floor in his home a

few hours before the admission. On initial evaluation by a traumatologist and an abdominal surgeon, he was disorientated and showed signs suggestive of an acute abdomen. The abdominal sonography revealed PVG, but there was no definitive evidence of bowel ischemia, although the patient's clinical status was compatible with this diagnosis. At the operation, performed on the same day, a necrotic distal ileum and proximal colon were resected. The patient died on the following day. The autopsy examination confirmed bowel ischemia caused by occlusion of the superior mesenteric artery.

The second patient was a 26-year-old man who was involved as a driver in a traffic accident and was suspected of having blunt abdominal injuries. After the initial evaluation by a trauma surgeon, he underwent the abdominal sonography, which showed mild contusion of the right kidney and PVG, but there were no definite signs of bowel injury, and no free fluid or gas was detected in the abdomen. CT of the abdomen likewise failed to reveal evidence of bowel injury. The patient was haemodynamically stable and the abdominal pain gradually subsided. The follow-up abdominal sonography on the following day was within the limits of normal. PVG was no longer present, and the patient was discharged from hospital.

Discussion

Gas in the portal vein can be visible on a plain radiograph, but newer imaging techniques, such as computed tomography and sonography - especially Doppler sonography - are more sensitive and specific for its detection. Small amounts of PVG detected by Doppler sonography may not be demonstrated by CT.[2,3]

Sonography is usually the first diagnostic modality used in the assessment of a patient with suspected abdominal disease. In our hospital, sonography is performed routinely in all patients suffering from blunt abdominal trauma.

PVG can be diagnosed both with the grey-scale and the Doppler techniques.

On the grey-scale examination, PVG is seen as small echogenic foci moving rapidly in the direction of blood flow in portal veins. The high acoustic impedance of gas is responsible for this phenomenon.[4] Initially the foci are visible mainly in the main portal vein and its major branches. Later, they may be seen also in the periphery of the liver parenchyma. Besides, gas bubbles, red-blood-cell aggregates can also be visualized moving in the lumen of a vein, but gas is readily distinguished from them by its greater echogenicity and velocity (Figure 1).[1]

In a Doppler sonogram, gas within a vessel has a characteristic appearance. It produces an artefact of bidirectional, vertical, high-amplitude spikes, accompanied by a characteris-

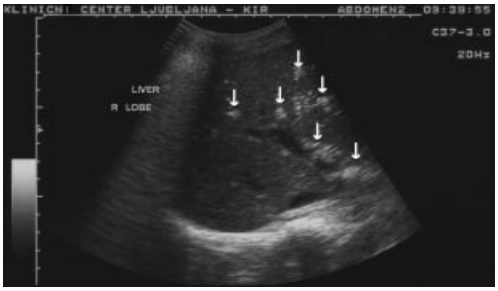

Figure 1. Grey-scale sonogram showing small echogenic gas bubbles (arrows) in the portal veins and liver parenchyma.

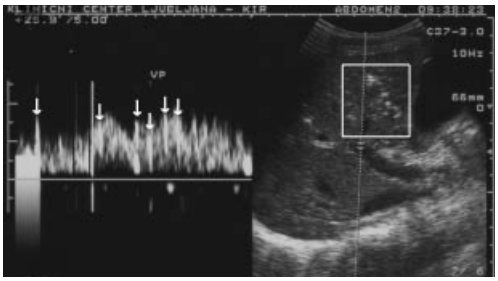

Figure 2. Doppler sonogram of a portal vein showing multiple high-amplitude spikes (arrows), typical of intravascular gas.

tic noise that disturbs the normal Doppler tracing of the portal vein (Figure 2).[5]

Gas may come into the portal vein by the direct infiltration through the damaged intestinal wall (to an intestinal venule or a mesenteric vein), or it may be produced within the portal vein by bacteria that have traversed a damaged intestinal wall.[6]

The causes of PVG can be divided into four groups:

a. *intestinal wall alterations* (mesenteric ischemia, inflammatory bowel disease);
b. *bowel distension*, occurring spontaneously or due to traumatic or iatrogenic causes;
c. *intra-abdominal sepsis* (diverticulitis, abdominal wall gangrene, pylephlebitis, necrotizing enterocolitis, perforation of a gastric ulcer);
d. *other causes* (transplantation, intestinal pneumatosis, corticosteroid therapy, chronic pulmonary disease).[7]

The most common cause of PVG is bowel ischemia. PVG resulting from *bowel ischemia* has been shown to have a poor prognosis, with a mortality rate of 75-90%.[7] A recent study has suggested that the prognosis may be more favourable if PVG is the result of mesenteric ischemia without associated bowel necrosis.[8] The causes of mesenteric ischemia include arterial and venous mesenteric thrombosis, hypoperfusion associated with nonocclusive vascular disease, embolic disease, and disease processes leading to the intestinal obstruction.[7]

Most patients with PVG due to bowel ischemia, as is the case with our first patient, are in a critical condition. The demonstration of PVG on the abdominal sonography should prompt a search for associated abnormalities, such as thickening or ischemia of the bowel wall, occlusion of the superior mesenteric artery, absent peristalsis, bowel distension, mesenteric oedema, ascites, fluid collections or intestinal pneumatosis. If any of these abnormalities occur in association with clinical signs suggestive of an acute abdomen, laparotomy must be undertaken without delay. If none of these abnormalities are found and the patient is clinically stable, further imaging studies, such as contrast-enchanced CT or angiography, should be performed before laparotomy.

Bowel distension can produce a minimal mucosal disruption that allows intraluminal gas to become intravascular. PVG secondary to bowel distension can occur in association with

1. *iatrogenic dilatation of the stomach and bowel* (endoscopic procedures, e.g. colonoscopy, sclerotherapy for gastric varices, ERCP or barium enema);
2. *paralytic ileus, mechanical obstruction, acute gastric dilatation*;
3. *blunt trauma and barotrauma*.[7]

Most patients in the first two groups can be managed medically. The mechanical obstruction of the bowel requires a causal treatment.

PVG has been reported to occur in less than 1% of patients with blunt abdominal trauma.[1] In these patients, PVG does not necessarily imply bowel perforation or necrosis. Benign PVG in the setting of blunt abdominal trauma is thought to result from acute pressure changes that occur at the time of injury and force intraluminal gas into the bowel wall, where it is absorbed into the portal circulation. The finding of additional abnormalities on sonography, such as an aperistaltic, distended bowel with a thickened, ischemic wall, or the presence of free fluid, calls for appropriate therapeutic measures. When no additional abnormalities are present, the further management should be governed by the clinical status. If this is stable and the patient has no major problems, as is the case with our second patient, there is no need to carry out expensive and potentially harmful investigations (contrast-enhanced CT, angiography). Only a plain radiograph of abdomen on the left lateral side should be obtained to rule out a possible perforation of the bowel (free gas),

and a follow-up sonography should be carried out after 24 hours to check if the PVG has cleared.

Several *infectious abdominal processes*, including diverticulitis, appendicitis, cholecystitis and colitis, are associated with PVG. The pathogenesis of PVG occurring in conjunction with the abdominal infection is not fully understood. It can be the result of septicaemia in the mesenteric and portal veins (pylephlebitis) or increased fermentation of carbohydrates within the bowel.7 If there is no associated liver abscess, the medical treatment is sufficient in most cases.

Another possible mechanism for the development of PVG is a portosystemic shunt, allowing gas to enter portal veins from the systemic venous circulation, where it may occur as a consequence of penetrating trauma, recent surgery or various procedures, such as central venous catheter placement.

Conclusions

Sonography is usually the first diagnostic examination in the evaluation of a patient with abdominal troubles. In our hospital, it is performed routinely in all patients suffering from blunt abdominal trauma who do not require an immediate laparotomy. The Doppler mode is particularly sensitive and specific for the detection of gas in the portal vein. The finding of PVG on the abdominal sonography in a patient who appears to be severely ill warrants a careful search for other abnormalities. In experienced hands, sonography can reveal a serious abdominal disease underlying PVG, thus making it possible to initiate an appropriate treatment without delay. If the cause of PVG is not apparent on sonography, the decision to undertake further, more demanding and potentially harmful investigations (contrast-enhanced CT, angiography) must depend on the patient's clinical status.

We must bear in mind that PVG is not always a sign of serious illness but may occur as a transient, clinically insignificant phenomenon, especially following abdominal trauma and various diagnostic and therapeutic procedures.

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case report

Radiographic, computed tomographic and magnetic resonance imaging appearances of primary V-cutting zone of resorption of lumbar vertebral body in Paget's disease

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Background. *Paget's disease of bone typically demonstrates three evolutionary phases with the characteristic radiographic findings. The incipient stage is manifested by an advancing lytic zone of resorption. Unlike the skull and the long bones the primary advancing zone of rarefication has not been clearly demonstrated within the spine and pelvis.*

Case report. *A 62-year-old man with histologically proved polyostotic Paget's disease was admitted to the hospital due to the exacerbation of pain at the toracolumbar junction with deterioration during the night. Due to slight elevation of acid phosphatase and clinical signs indicating prostatic hypertrophy the possibility of metastatic prostatic carcinoma has been raised. A bone scintigraphy with technetium phosphonate showed the expected marked increased uptake affecting numerous bones with the typical radiographic signs of Paget's disease. Besides these findings, there was also moderate focal accumulation within the right dorsal half of the vertebral body of L1. Conventional tomography, computed tomography (CT) and magnetic resonance imaging (MRI) demonstrated the primary cutting zone of resorption affecting the same region.*

Conclusions. *We present a unique case of Paget's disease with primary V-cutting zone within the undeformed vertebral body of L1 and spontaneous progression to the midphase of the disease on a three year follow-up.*

Key words: osteitis deformans; spinal diseases - radiography; tomography, x-ray computed; magnetic resonance imaging

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Introduction

Paget's disease of bone typically demonstrates three evolutionary phases with the characteristic radiographic findings. The initial lytic phase is followed by the midphase with a mixture of lytic and sclerotic changes leading finally to the late sclerotic phase of the disease. The incipient stage is manifested

by an advancing lytic zone of resorption. Unlike the skull and the long bones the primary advancing zone of rarefication has not been clearly demonstrated within the spine and pelvis.[1] It has been stressed that the most of vertebral lesions reported in literature as the primary lytic phase of the disease actually represent the secondary bone resorption following the compression of the vertebral bodies.[2]

In this case report, we describe radiographic, computed tomographic (CT) and magnetic resonance imaging (MRI) findings of primary cutting zone of the resorption within the undeformed vertebral body of L1. A three-year follow-up revealed a progressive transition to the midphase of Paget's disease.

Case report

A 62-year-old man with histologically proved polyostotic Paget's disease was admitted to the hospital due to the exacerbation of pain at the toracolumbar junction with deterioration during the night. The physical examination of the toracolumbar spine demonstrated the limited mobility without neurological impairment. Pertinent laboratory findings included extremely elevated levels of serum alkaline phosphatase and urine hydroxyproline. Due to slight elevation of acid phosphatase and clinical signs indicating prostatic hyperthrophy the possibility of metastatic prostatic carcinoma has been raised. A bone scintigraphy with technetium phosphonate showed the expected marked increased uptake affecting the right frontal area, the vertebras C3, T1, T5, L2, L3, L4, L5, the left scapula, the pelvic bones and the proximal part of the left femur. Besides these sites, there was also moderate focal accumulation within the right dorsal half of the vertebral body of L1. The plain films of the affected areas demonstrated features typical for the midphase of polyostotic Paget's disease with the exception of the L1 vertebra. A careful analysis of the conventional tomography of L1 revealed the discrete triangular osteolytic area affecting the right dorsal part of the vertebral body localized just bellow the lower end plate and the dorsal cortex (Figure 1a). The osteolytic area correlated well with the region of moderately focally increased uptake on the bone scintigraphy. MRI of L1 showed the triangular low signal intensity area on T1-weighted spin-echo images, which was of high signal intensity on T2-weighted images and demonstrated the intense Gd-DTPA contrast enhancement on T1-weighted postcontrast images (Figure 1b). Since the clinical, scintigraphic and radiologic findings were in favour of the eventual metastatic disease secondary to prostatic carcinoma CT guided bone biopsy was pro-

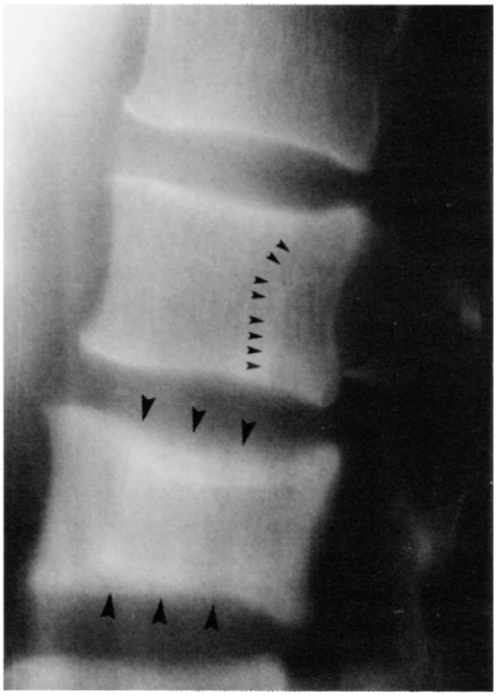

Figure 1a. Lateral conventional tomography of undeformed L1 vertebral body shows discrete triangular primary cutting cone zone of resorption localised bellow the lower endplate and the dorsal cortex (small arrow heads). Note also »picture-frame» appearance of the vertebral body L2 (big arrow heads), typical for the midphase of Paget's disease.

posed. The CT examination of L1 vertebra showed an oval purely lytic lesion below the dorsal cortex without any enlargement of bone contours (Figure 1c). The patient ultimately refused the bone biopsy and was discharged from the hospital. He was lost from our control for one and a half year. During that time the patient was treated intermittently with calcitonin and diphosphonates but laboratory examinations demonstrated only slightly decreased levels of serum alkaline phospahatase and of urine hydroxyproline. The repeated CT examination of L1 vertebra (Figure 2a) showed that the lesion progressed centrifugally for approximately 6-7 mm. The lesion, which at the first examination was purely lytic, on the control examination demonstrated mixed lytic/sclerotic features. The advancing edge of the lesion remained osteolytic and it looked as it was followed by osteosclerosis. In comparison with the initial examination the most interesting new finding was some enlargement of the bone contour seen along the posterior border of the vertebral body of L1 (Figure 2b). The repeated bone scintigraphy was similar to the first examination and revealed no additional focal accumulation, which could be expected in case of eventual metastatic disease. On a three year follow-up radiography, CT and MRI demonstrated the further progression of the lesion with a mixture of lytic/sclerotic findings typical for the midphase of Paget's disease. The slight compression of the lower end plate of L1 was evident reflecting the weakening of the pagetic bone (Figure 3).

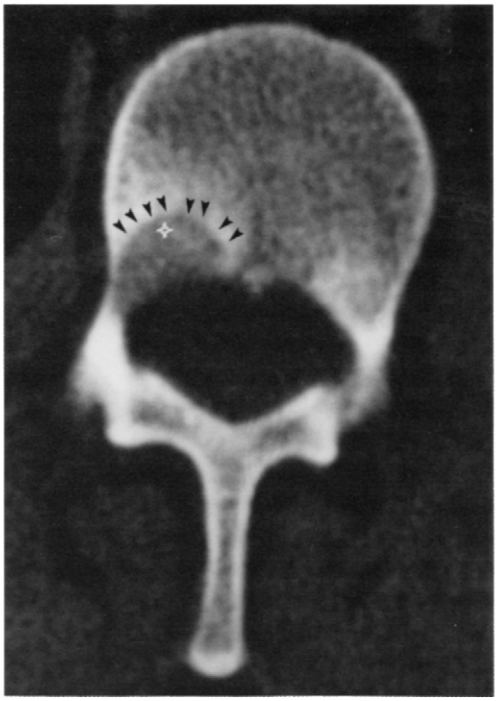

Figure 1c. Initial CT scan of L1 reveals an oval purely osteolytic lesion without evident bone enlargement (small arrow heads).

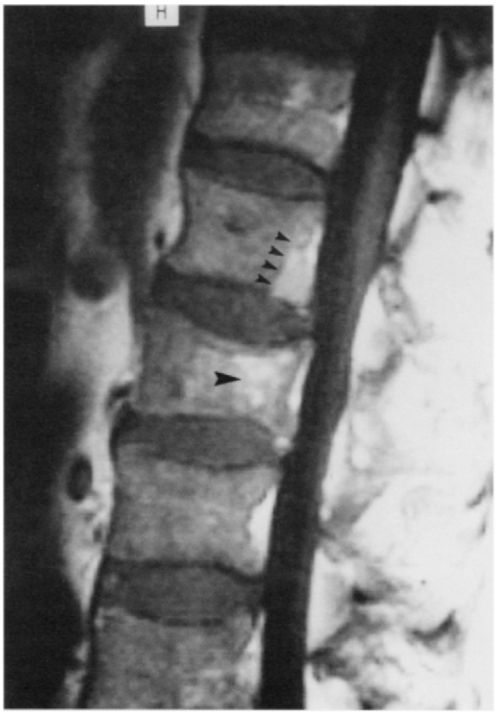

Figure 1b. Sagittal T1-weighted (500/15) spin-echo Gd-DTPA postcontrast image demonstrates marked contrast enhancement within the primary lytic zone of L1 (small arrow heads) and the central part of L2 vertebral body (big arrow head)

Discussion

The primary lytic phase of Paget's disease of bone is characterised by an advancing zone of bone rarefication reflecting the intense osteoclastic activity. The average rate of progression in the untreated patients has been estimated at about 1 cm/year (1mm /month). The focal bone resorption is quickly followed by a marked osteoblastic repair which tends to compensate the loss of the bone.[3] This focal bone balance between resorption and formation may demonstrate considerable variations.[2] Therefore, the sclerotic phase may be quickly changed into the mixed or the lytic appearances of the disease. The advancing zone of rarefication can be easily revealed radiographically within the skull as *osteoporosis circumscripta* or within the long bones as V-shaped cutting cone. However, within the trabecular bones like pelvis and spine, the primary lytic zone has not been clearly demonstrated.[1] The first radiologic evidences in these areas are typically sclerotic features of the disease.[3] According to Maldague and Malghem[2] most of the vertebral lesions, reported in literature as demonstrating the cutting cone of primary lytic phase, actually followed the final sclerotic phase of the disease. The secondary resorption within the sclerotic pagetic bone usually occurs due to some provoking factors, most commonly the compression of the vertebras, and has been never observed at the level of undeformed vertebral bodies. In this respect our case is unique. An osteolytic zone has been clearly demonstrated by the conventional tomography and CT examination within the undeformed vertebral body of L1 (Figures 1a, 1c). Furthermore the primary zone of rarefication affected only a part of the vertebral body of L1. In other re-

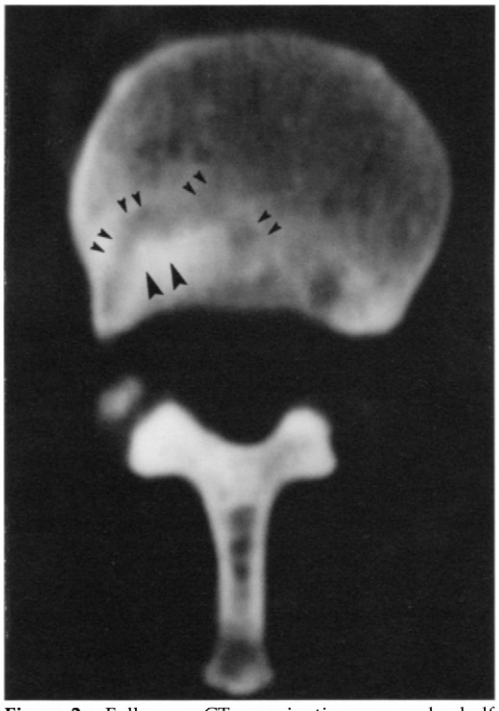

Figure 2a. Follow-up CT examination one and a half year later shows centrifugal enlargement of the lesion. The advancing lytic cutting cone (small arrow heads) is followed by osteosclerosis (big arrow heads).

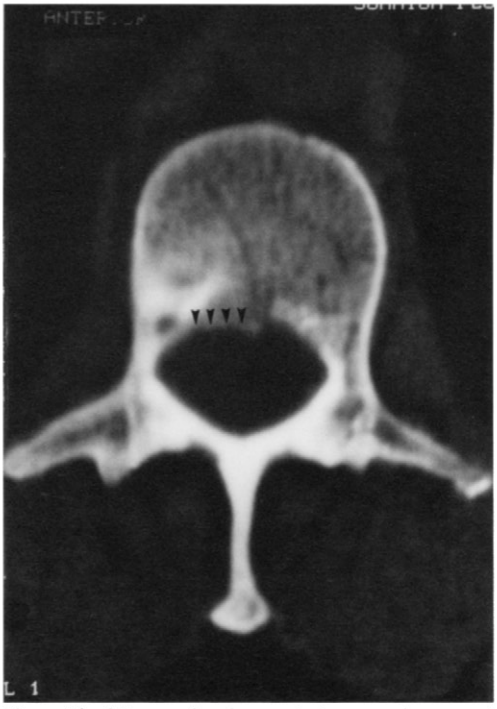

Figure 2b. CT scan also demonstrates an enlargement of bone contour at the dorsal aspect of the vertebral body of L1 (small arrow heads)

ported cases not only the compression of the vertebral bodies was seen, but the rarefication was more diffusely distributed.[2,4,5] There are no reasons to believe that in this particular case the zone of rarefication appeared secondarily within the preexisting pagetic bone. At the initial examination the lesion was purely osteolytic and there was no enlargement of the vertebral body which would be present in a case of previous sclerotic phase of the disease. The scintigraphic finding of only moderately increased uptake of technetium phosphonate within the primary resorption zone seen in our patient has been reported in literature at the initial phase of Paget's disease.[2] MRI appearances of high signal intensity on T2-weighted images and marked contrast enhancement on T1-weighted postcontrast images (Figure 1b) within the primary lytic zone of L1 and the central part of vertebral body of L2 represented oedema and hyperaemia consistent with the replacement of fat and haematopoietic elements of normal bone marrow with hypervascular fibroblastic tissue characteristic of Paget's disease.[1] The radiologic features on a follow-up examination were also in favour of the existence of primary lytic zone within the vertebral body of L1. Two of the four cardinal radiographic appearances of Paget's disease were shown, the enlargement of bone contours and an advancing resorption zone. A typical bone enlargement not seen at the initial examination appeared only after one and a half year reflecting activation of the subperisteal cortex (Figure 2a). At that time there was also the evident progression of primary cutting cone for about 6-7 mm. The primary zone of resorption was quickly followed by secondary osteosclerosis, which may reflect a higher remodelling rate of the trabecular bone.[2] The rapid process of sclerosis could be eventually attributed to antiosteclastic therapy. However, the influence of treatment was probably limited since the levels of serum alkaline phospahatase and urine hydroxyproline were not significantly decreased after one year and a half.

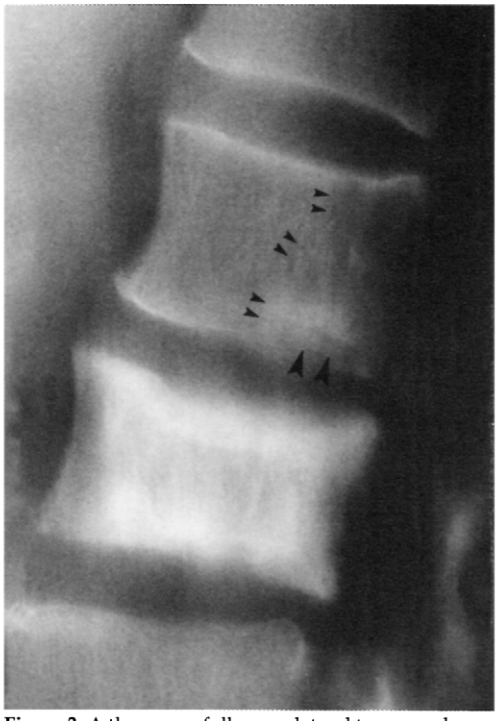

Figure 3. A three year follow-up lateral tomography reveals mixed lytic/sclerotic features typical for the midphase of Paget's disease. The lesion has progressed and affects nearly half of the vertebral body L1 (small arrow heads). Note initial concavity of the lower end plate in the region of weakened pagetic bone (big arrow heads).

Without bone biopsy the definite diagnosis of Paget's disease still remains hypothetic. On the other hand the eventual bone biopsy at the beginning of the lesion of vertebral body of L1 could substantially change the natural evolution of the disease. Anyhow, the diagnosis of metastatic disease after a three year follow-up was not realistic. Relevant clinical, laboratory and scintigraphic findings were all negative. Besides, there were no radiologic sings of local cortical destruction and extension of the osteolytic lesion of L1 into the surrounding soft tissues which would be expected in the metastatic disease. The lesion has progressed within the periosteal envelope

of bone, findings characteristic of a midphase of Paget's disease. Finally, the radiological changes of the vertebral body of L1 on the follow-up examinations were comparable to the neighbouring vertebras with all typical signs of a midphase of Paget's disease.

In summary, we presented a unique case of Paget's disease with primary V-cutting cone within the undeformed vertebral body of L1 and spontaneous progression to the midphase of the disease on a three year follow-up. Pertinent radiographic, CT and MRI findings were described.

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case report

A calcified cervical intervertebral disc in a child and a thoracic disc calcification in an adult with posterior herniation-radiographic, computed tomography and magnetic resonance imaging findings

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Background. *Nucleus pulposus calcification in children is a relatively rare but well known clinical syndrome, usually localized at the level of the cervical spine. The exact aetiology still remains uncertain. Calcifications of the intervertebral discs in adults differ from the childhood variety. They are mainly degenerative in nature and occur at the level of midthoracic and upper lumbar spine. Potentially serious complications, posterior herniation of calcified disc may occur in both entities.*

Case reports*. We report two cases of the calcification of the nucleus pulposus in a seven-year-old boy at the level of C7-T1 and a case of calcified intervertebral disc T11-T12 in a forty-five-year-old woman, with massive posterior herniation. Remission of symptoms was achieved with a conservative therapy alone. Clinical, radiographic, computed tomographic and magnetic resonance imaging (MRI) findings were analyzed in an attempt to investigate similarities and differences between both disease entities.*

Conclusion. *Massive posterior herniation of calcified nucleus pulposus in a child was treated conservatively with a favourable outcome. A disappearance of symptoms followed quick resolution of herniated calcified masses. In adult variety extruded thoracic disc calcification was of a permanent type with no tendency towards spontaneous resolution and remission of symptoms after the conservative therapy. MRI seems to be able to depict disc calcification before a conventional radiography. The widening of affected discs in a paediatric patient was also better demonstrated by MRI. It would seem to support the theory of an increased intradiscal pressure as the precursor of annulus fibrosus ruptures and consecutive calcified disc herniations.*

Key words: intervertebral disc; calcinosis; intervertebral disc displacement; spinal disease - radiography, tomography; x-ray computed; magnetic resonance imaging

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Introduction

Nucleus pulposus calcification in children is a relatively rare but well known clinical syndrome, usually localized at the level of the cervical spine. More than hundred and thirty cases have been reported in the literature

with an increasing number of new reports in the last decade.[1-4] The disease entity has been attributed to trauma,[5,6] inflammation,[7,8] or increased hydrostatic pressure within the disc,[3] but the exact aetiology still remains uncertain. The prognosis of discal calcifications in children is good.[3,9,10] As a rule pain resolves and there is a spontaneous resorption. Although the benign nature of the disease has been emphasized by many authors, the herniation of the calcified nucleus pulposus through the ruptured annulus fibrosus may occasionally occur.[1,2,5,6,11-14] This rare but potentionally serious complication with radiologic signs of the extradural space occupying lesion raises the question of an eventual operative therapy.[12-14] In several cases, which have been reported in literature,[1,9,11] the remission of symptoms occurred with a conservative treatment.

Calcifications of the intervertebral discs in adults differ from the childhood variety and may reflect numerous systemic diseases. Disc calcifications are more frequent and often affect not only the nucleus pulposus but also the annulus fibrosus. In adults calcific deposits are mainly degenerative in nature and occur at the level of midthoracic and upper lumbar spine which are relatively nonmobile. Therefore, a posterior massive displacement of a calcified intervertebral disc into the spinal canal is a rare complication.[1]

Radiographic and computed tomography (CT) findings in herniated disc calcifications are well known. However, magnetic resonance imaging (MRI) appearances have not been often reported in the literature.[15,16] In the last decade, the significance of MRI as a primary radiologic diagnostic modality in different spinal disorders has been increasingly gaining in relevance. Therefore, the radiologists and the clinicians must be aware of the MRI findings of these rare but potentionally serious complications of the intervertebral disc calcifications.

We report two cases, a case of the calcification of the nucleus pulposus in a seven-year-old boy at the level of C7-T1 and a case of the calcified intervertebral disc T11-T12 in a forty-five-year-old woman, with the massive posterior herniation. The remission of symptoms was achieved with a conservative therapy alone. Clinical, radiographic, CT and MRI findings were analyzed in an attempt to investigate similarities and differences between both disease entities.

Case reports

Case I

A seven-year-old-boy presented with lower neck pain, limited movements and stiff neck, a day following gymnastic activity at school. He could not remember having had any significant trauma. There were no neurological findings. The patient was afebrile, his ery-

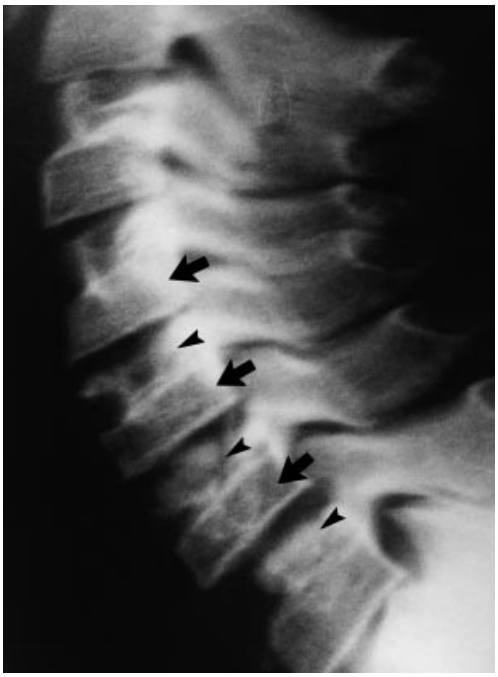

Figure 1a. Initial lateral radiograph showing calcification of the nucleus pulposus at the levels C4-C5, C5-C6, C6-C7 (arrow heads) and mild flattening of the vertebral bodies C4, C5 and C6 (arrows).

throcyte sedimentation rate and a white blood cell count were within normal limits. Initial lateral radiographs demonstrated calcifications of the nucleus pulposus at the levels C4-C5, C5-C6, C6-C7, C7-T1 and the mild flattening of the vertebral bodies C4, C5 and C6 (Figure 1a). MR imaging revealed a signal void within the intervertebral discs C4-C5, C5-C6, C6-C7 and C7-T1, findings consistent with nucleus pulposus calcifications and slight disc expansions most pronounced at the level C7-T1 (Figure 1b). Following the treatment with a cervical collar on an outpatient basis he became asymptomatic after four weeks. The patient was admitted to hospital for the second time one year later with mild left-sided torticallis. He complained of lower neck pain radiating to the shoulders. There was no history of trauma. Neurological and laboratory investigations were normal. A control lateral radiograph of the cervical spine showed disc calcifications at the levels of C4-C5, C5-C6 and C7-T1. However, there was no nucleus pulposus calcification of C6-C7 disc which was most pronounced at the initial examination (Figure 2a). CT scan revealed minimal calcifications within the intervertebral disc C6-C7 and a narrow calcified tract in the midline leading to the rupture of the annulus fibrosus. The calcified nucleus pulposus was nearly completely herniated posteriorly and distributed quite evenly throughout the whole epidural space behind the vertebral body of T1. The narrowing of the spinal canal was slightly more pronounced on the left side (Figure 2b). On MR imaging nucleus pulposus calcifications within the disc and the epidural space were demonstrated as a signal void (Figure 2c). The signal intensity of the vertebral bodies C6 and C7 was decreased on T1-weighted images and increased on T2 weighted spin echo images, findings consistent with bone marrow oedema (Figure 2d). In comparison to radiography and CT examination MR imaging clearly revealed that there was no compression of the spinal cord. The patient was discharged from the hospital after a cervical collar was applied. On a control clinical examination six weeks later he was asymptomatic. Control CT showed only minimal remnants of the calcified herniated nucleus pulposus in the midline, the abundance of epidural masses being resorbed (Figure 3).

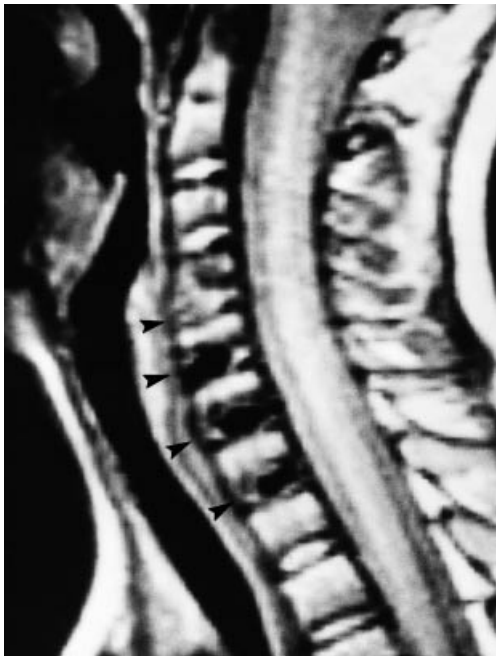

Figure 1b. Mid-sagittal PD-weighted spin echo image (TR 2200 ms, TE 20 ms) reveals signal void within the calcified intervertebral discs C4-C5, C5-C6, C6-C7 and C7-T1 (arrow heads) with slight disc expansion most pronounced at the level C6-C7. Note that the signal void at this level extends far posteriorly into the annulus fibrosus indicating the possibility of its rupture.

Case II

A forty-five-year-old female complained of pain radiating from the lower to the upper thoracic region for many years. The pain was aggravated by thoracic flexion but was not increased by coughing. There was no preceding history of any significant trauma. She experienced hypoesthetic area localized dorsally on the right side at the level of T10-T11. There

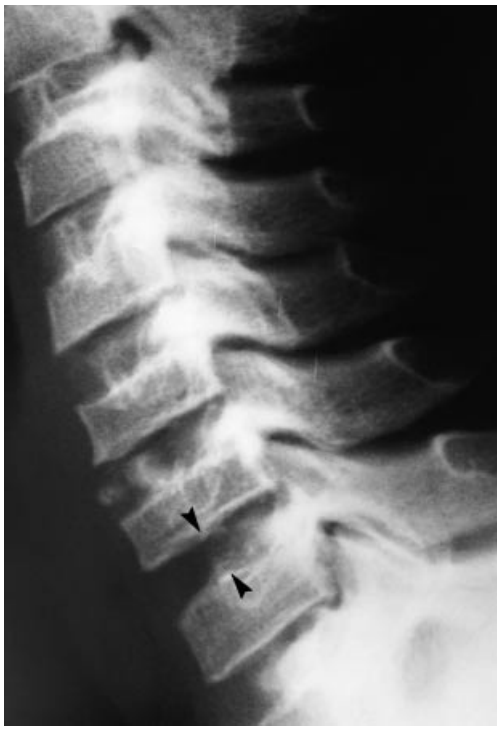

Figure 2a. Follow-up radiograph taken one year later shows disappearance of the nucleus pulposus calcification at the level C6-C7, which was most pronounced at the initial examination (arrow heads) and consecutive narrowing at the disc space.

was no neurological deficit and the laboratory findings were within normal limits. Lateral roentgenogram and conventional tomography of the thoracic spine showed findings typical of haemangioma of the vertebral body T10 and a calcified area in the posterior third of the intervertebral disc T11-T12. The dorsal herniation of the calcified material into the spinal canal behind the vertebral body D12 was also evident (Figure 4a). CT examination revealed the calcification of the nucleus pulposus and a narrow calcified tract leading to the rupture of the annulus fibrosus. Extruded calcified material was globular in appearance and localized in the midline within the epidural space (Figure 4b). The calcified nucleus pulposus and herniated masses were of low signal intensity on all MRI sequences.

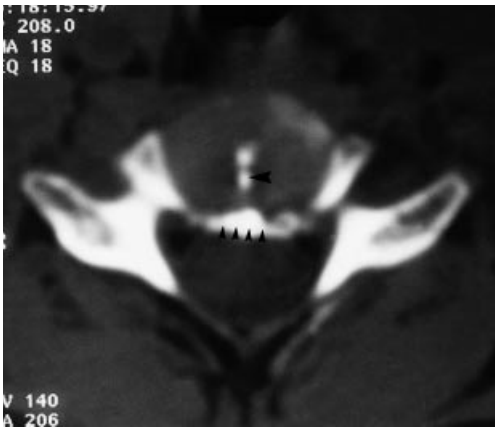

Figure 2b. CT examination of the intervertebral disc C6-C7 demonstrates hyperdense narrow tract in the midsagittal plane (big arrow head) leading to the rupture of the annulus fibrosus. Herniated calcified nucleus pulposus is quite evenly distributed within the epidural space (small arrow heads)

MR imaging showed no compression of the spinal cord. The signal intensity of the neighbouring vertebral bodies T11 and T12 was normal (Figure 4c). The patient was treated with analgesics on an outpatient basis and her pain diminished in intensity but did not disappear completely. On a follow-up radiographic examination two months later the herniated calcified masses remained unchanged.

Discussion

The herniation of the calcified nucleus pulposus in children through the rupture in the annulus fibrosus is an unusual complication. Only forty cases including ours have been published to date.[1,2,5,6,11-14] The calcified nucleus pulposus may be displaced in different directions. However the posterior and posterolateral extrusion into the spinal canal with the neurological signs of the spinal cord or the nerve-root compression may occasionally occur. This is the most serious complication of disc calcification in children.[12-14] Clinical symptoms in our patient did not dif-

fer from similar cases reported in literature and included limited movements, stiff neck, mild torticollis and lower neck pain. There was no history of significant trauma or laboratory signs indicating the inflammation. With the exception of several patients,[12-14] who were treated surgically in majority of cases including ours, the remission of symptoms was achieved with a conservative therapy alone. The disappearance of symptoms in our patient coincided with a quick resolution of the herniated calcified nucleus pulposus which occurred during six weeks. Good prognosis in children may be attributed not only to a quick resolution but also to a pliability of calcified masses which has been proved surgically.[14]

The calcific deposits in adults predominantly consist of hydroxyapatite and may be of impermanent or permanent type.[15] In our case a longstanding clinical history and a radiographic follow-up examination which showed no signs of resolution suggested the permanent type of calcification. The results of the conservative treatment in our adult patient were not favourable. Her pain did not resolve completely, which was in accordance with the radiographic signs of a persistent calcification within the spinal canal. In the absence of the significant neurological im-

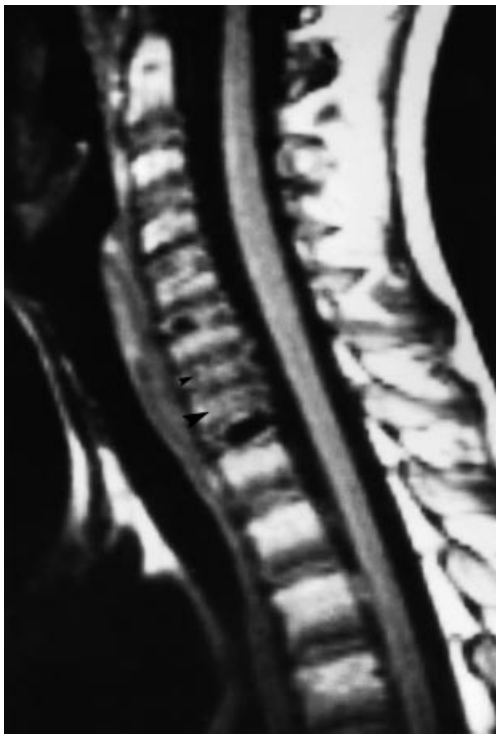

Figure 2c. control sagittal MR T1-weighted spin echo image (TR 500 ms, TE 15 ms) reveals disappearance of sagittal void at the level of herniation (small arrow head). Extruded calcified low signal intensity nucleus pulposus cannot be identified since the CSF space is of similar low signal intensity on T1-weighted image. It is of interest to note the decreased signal intensity of the vertebral body C7 (big arrow head) consistent with bone marrow oedema.

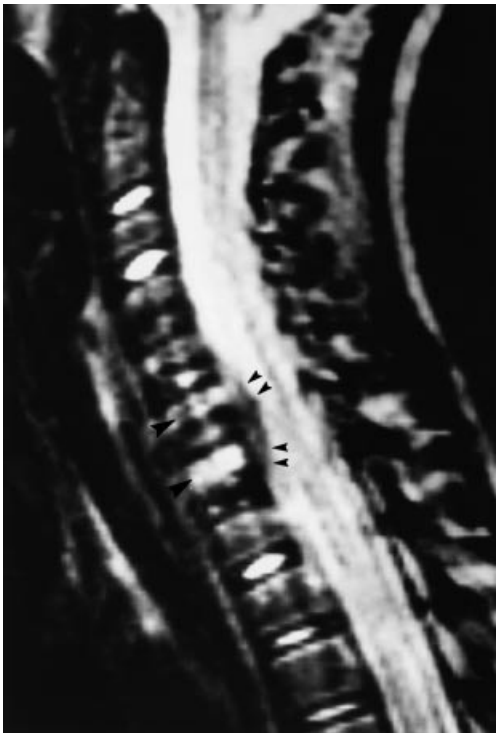

Figure 2d. On T2-weighted spin echo image (TR 2200 ms, TE 80 ms) high signal intensity bone marrow oedema is seen within the vertebral bodies of C6 and C7 (big arrow heads). Low signal intensity extruded calcified nucleus pulposus is clearly demonstrated due to neighbouring high signal intensity CSF on T2-weighted (small arrow heads).

pairment a surgical treatment was not justified.

Radiographic signs in our young patient were fairly typical. The usual findings of cervical multilevel disc calcifications were demonstrated. However, the distribution of calcified masses throughout the individual disc spaces differed. At the levels of C4-C5, C5-C6 and C7-T1 the calcification appeared to be flattened and more widespread. Since the normal nucleus pulposus lies eccentrically on the border between the medial and posterior third of the disc space, the widespread distribution of calcified masses suggests the intradiscal displacement of the calcified nucleus pulposus into the fissures of the annulus fibrosus, findings clearly demonstrated on CT examination. Radiographic appearances were to some extent reminescent of discographic findings in the degenerative disc disease.[17] Fragmentation with a tendency towards the anterior displacement was revealed at the level C5-C6. The flattening of the vertebral bodies C4, C5 and C,6 seen also in our patient, has been frequently reported in literature.[3,9,10]

The mechanical stress is an important factor in modeling of the normal vertebral bodies. It has been proved that, in the absence of normal weight-bearing, the height of the vertebral bodies increase.[18] Conversely, the flattening of the vertebral bodies presumably indicates that the increased stress has been evenly distributed throughout the whole endplate reflecting the morphological appearance of widespread distribution of the calcified nucleus pulposus within the intervertebral disc. In comparison to other disc calcifications the nucleus pulposus calcification at the level of C6-C7, which ultimately herniated, was more abundant and globular in appearance. The initial MR examination (Figure 1b) revealed not only the expected signal void within the calcified intervertebral discs but also a slight disc expansion, most pronounced at the level of C6-C7, consistent with an increased intradiscal pressure, which was not so clearly demonstrated on the plain film radiography. Similar MRI findings have been already reported in discs without visible

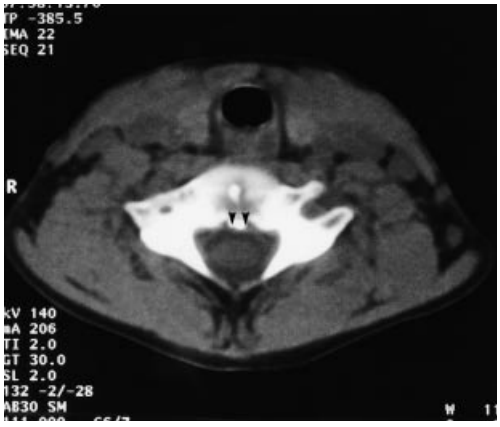

Figure 3. Control CT examination done after six weeks at the level of C6-C7 reveals a nearly complete resolution with only minimal remnants of the calcified herniated nucleus pulposus within the epidural space (arrow heads).

Figure 4a. Conventional tomography of the lower thoracic spine demonstrates calcified nucleus pulposus of the T11-T12 disc space (big arrow heads) and dorsal extrusion of the calcified material behind the vertebral body of T12 (small arrow heads). Findings typical of cavernous haemangioma of the vertebral body T10 are also seen (arrow)

calcifications by Swischuk and Stansberry[16] who supposed that it could represent the initial phase of the disease entity. In our case the rupture of the annulus fibrosus occurred at the level of C6-C7 in which the disc expansion was most pronounced. These findings are in favour of the theory of an elevated intradiscal pressure.[3] On the basis of the MRI appearances it could be assumed that an initial phase of the disease may be characterized by an expansion of the intervertebral disc, presumably due to the osmotic absorption of water caused by an increased content of calcium salts within the nucleus pulposus. An increased intradiscal pressure makes the disc more vulnerable and ultimately leads to ruptures within the annulus fibrosus with a consecutive intradiscal displacement and/or extrusion of the calcified nucleus pulposus and disc decompression. These MR appearances also suggest that MR is probably more sensitive than radiography in depicting a disc widening and possibly that the most expanded discs are at highest risk for eventual herniations. On a follow-up radiography (Figure 2a) the disappearance of disc calcification C6-C7 together with pain distribution suggested the possibility of the dorsal disc herniation, which has been proved on CT examination (Figure 2b).

The most common site of the calcified nucleus pulposus is at C6-C7,[19] which as in our case, has also been a frequent location of herniations.[1,2,11] It could be attributed to the increased stress at this level produced by the transition of a mobile cervical to a fixed thoracic part of the axial skeleton. The narrowing of the disc space following the herniation (Figure 2a) indicated a disc decompression. Due to its well known high sensitivity for the

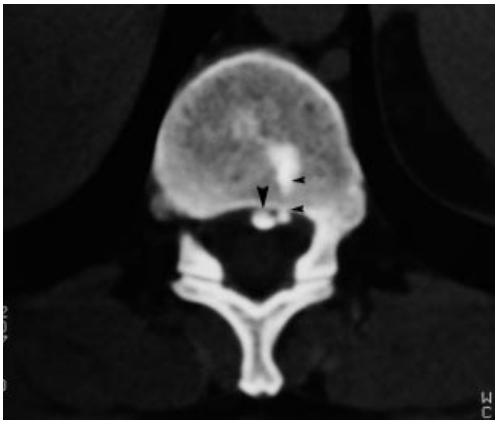

Figure 4b. CT examination at the level of T11-T12 disc space reveals calcified nucleus pulposus and narrow hyperdense tract directed to the left para-saggital rupture of the annulus fibrosus (small arrow heads). Extruded calcified material is globular in appearance and localized in the mid-saggital plane of the epidural space (big arrow head).

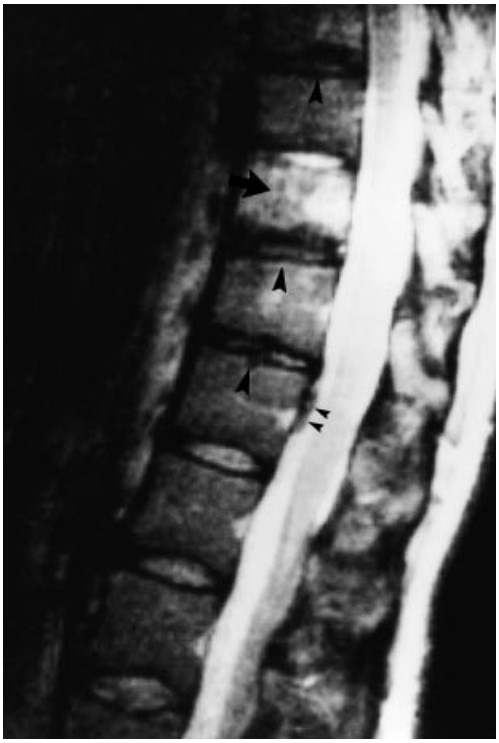

Figure 4c. Sagittal MR T2-weighted spin echo image (TR 1900 ms, TE 80 ms) shows decreased signal intensity of the intervertebral discs T8-T9, T10-T11 and T11-T12 (arrow heads) and normal signal intensity of the neighbouring vertebral bodies. The extruded low signal intensity calcified masses are clearly seen against high signal intensity CSF (small arrow heads). There is no compression of the spinal cord. High signal intensity haemangioma of T10 vertebral body is also demonstrated (arrow).

demonstration of calcified structures CT is the method of choice in suspect calcified disc herniations. In our patient CT clearly showed the posteriorly and caudally extruded calcified nucleus pulposus as a hyperdense space occupying lesion evenly distributed within the epidural space. The distribution reflected operatively proved a soft toothpaste like consistence of the calcified nucleus pulposus.[14] One of the important advantages of MRI is its inherent high contrast resolution which enables the noninvasive presentation of the spinal cord and evaluation of the eventual compression in disc herniations. In our case a good demonstration of low signal intensity herniated calcified masses within the epidural space without a cord compression was possible only on T2-weighted images due to neighbouring high signal intensity CSF space (Figure 2d). The most interesting MRI appearances, which have not been reported previously,[1,16] included the low signal intensity of the vertebral body C7 on T1-weighted images and the high signal intensity of the vertebral bodies C6 and C7 on T2-weighte images, the findings which were not present at the examination done before the disc herniation. These findings, consistent with bone marrow oedema and hyperaemia, were similar to those described at the initial stage of the disc degeneration[20] and may presumably explain the radiographically proved early degenerative changes which occur following disc calcifications in children.[10] Care has to be taken not to misinterpret these MRI appearances as the presence on an infectious discitis.

In our adult patient the radiographic (Figure 4a) and CT findings (Figure 4b) were similar to changes seen in a child with disc calcification. However, there was a substantial difference in morphology of the epidural calcified masses, which were globular in the adult variety, indicating its hard consistence and nonpliability. MRI findings of the low signal intensity disc spaces at the levels of T8-T9 and T10-T11 without radiographic evidence of calcifications and degeneration resembled to those seen in the case report by Swischuk and Stansberry.[16] It seems to indicate the high sensitivity of MRI which is able to detect disc calcifications at an earlier stage than the conventional radiography. MRI findings of the normal signal intensity of bone marrow within vertebral bodies were in accordance with the absence of significant disc degenerative changes (Figure 4c).

In summary, the massive posterior herniation of the cervical pediatric nucleus pulposus was treated conservatively with a favourable outcome. A disappearance of symptoms followed the quick resolution of herniated calcified masses. In adult variety the extruded thoracic disc calcification was of a permanent type with no tendency towards the spontaneous resolution and remission of symptoms after a conservative therapy. MRI seems to be able to depict the disc calcification before the conventional radiography.[16] The widening of affected discs in a child was also better demonstrated by MRI. It would seem to support the theory of an increased intradiscal pressure as the precursor of annulus fibrosus ruptures and consecutive calcified disc herniations.[3] The most interesting MRI findings, which have not been reported, included bone marrow changes consistent with the beginning of the disc degeneration in a child. Since these MRI appearances may resemble to early inflammatory changes, care has to be taken not to misdiagnose an infectious discitis.

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case report

Unusual radiographic changes of a gout patient

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Background. *Gout is a metabolic disorder that results in hyperuricemia and accumulation of uric acid crystals (urats) in tissues, especially joint cartilage. The gouty arthritis presents as acute attacks of arthritis leading eventually to chronic gouty arthritis. In 80% of cases it first occurs in the matatarsophalangeal (MTP) joint of the great toe and is more frequent in male population.*

Case report. *We present a case of unusual radiographic changes accompanying gouty arthritis. A 63 year old female complained about swelling of the first MTP joint on the right, right knee, about stiffness of feet and hands'digits and about backache. First symptoms started to appear 30 years ago. In the time of examination radiographs displayed degenerative changes of the majority of presented joints, bilateral sacroiliitis and osseous ankylosis of both insteps. Microscopic examination showed urate crystals in the samples of the synovial fluid aspirated from the knee. The histological findings of the synovial tissue after the synovectomy were also in favour of gouty arthritis.*

Conclusions. *Radiographs are the most important imaging modality in the diagnostic process of gout. However, radiographic differential diagnosis can be difficult, since the findings overlap with other conditions which cause arthritis and osteoarthritis especially in longstanding gout, elderly patients and females. The diagnosis must be often confirmed with the help of laboratory and histological findings.*

Key words: arthritis, gout - radiography

Introduction

Gout is a metabolic disorder that results in hyperuricemia and leads to monosodium urate crystals being deposited in various sites in the body, especially joint cartilage.[1]

The commonly reported overall prevalence of gout is 6 per 1000 population for men and 1 per 1000 population for women.[2] The prevalence is increasing with age to rates of 24 per 1000 in men and 16 per 1000 in women.[3] A part of this increase may be increased physicians' awareness of atypical gout.[4]

The classic radiographic findings are asymmetrical soft tissue swelling with or without calcifications (gouty tophi); well-defined erosions, often with sclerotic borders or overhanging edges, without marked osteoporosis.[1] Joint space width may appear relatively wide.[5] It typically affects the first metatarsophalangeal (MTP) joint.

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Case report

A 63-year-old female has been admitted to the department of rheumatology in our hospital for several times over the last few years. She was complaining about swelling of the right great toe and right knee at times, about stiffness of feet and hands' digits and about a backache. First symptoms started to appear 30 years ago. Laboratory tests showed hyperuricemia. In the department of radiology we performed radiography of feet, ankles, knees, hands, sacroiliac joints and thoracic spine. The radiograph of feet demonstrated state after bilateral operation of hallux valgus deformity, osteoarthritis of metatarsophalangeal (MTP) joints and fibular subluxation of digits. The bone structure of MTP joints of great toes was demineralised. We saw osteophytosis of ankles and osseous ankylosis of both insteps (Figure 1). The radiograph of knees showed effusion and marked hyperostotic osteophytosis. Degenerative joint disorders were seen on the radiograph of hands. On the sacroiliac joints the radiograph joint space irregularity could be seen: pseudodilatations, especially of the left joint space, indistinct outlines, fine erosions and juxta-articular sclerosis more pronounced on the side of iliac bone (Figure 2); the finding of sacroiliitis was additionally confirmed by the computed tomography. The thoracic spine radiograph demonstrated Forestier's ankylosing hyperostosis.

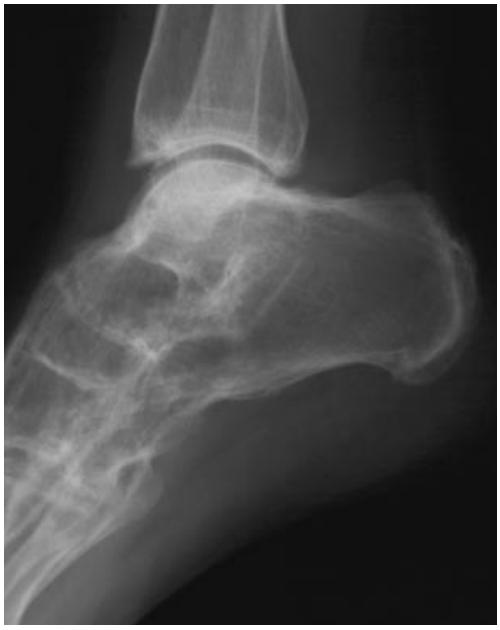

Figure 1. A lateral radiograph of the ankle shows osseous ankylosis of instep in patient with gout.

A diagnostic puncture of the right knee's effusion was obtained. Intra- and extracellular urate crystals were found in the synovial fluid using a polarizing microscope. The histological findings of the synovial tissue after the synovectomy of the right knee pointed to gouty arthritis.

She also underwent the resection of the subcutaneous node on the left elbow. The hystological findings of the node showed large aggregations of uric acid crystals surrounded by abundant macrophages, scattered lymphocytes and foreign body type cells. The finding is typical of gouty tophi.

Despite the unusual radiographic presentation, the diagnosis of gout was established with the help of laboratory and histological findings.

Discussion

Despite centuries of recognition of gout, the proper diagnosis of gout is still problematic.[5] It has been only in the past 35 years that a significant research in the area of gout has been undertaken.[6] The classic presentation of gout is as an acute monoarthritis often in the first MTP joint or in other joints, such as knee. Gout is a common problem in middle-aged males, from the fourth to sixth decade, but has an increasing recognition in elderly ones with unique and often atypical features.[7] Acute attacks of gout are less common in elderly people, in whom it presents as a chronic polyarticular disease in a much different fashion making proper diagnosis less likely.[8-10] In

these cases gout can be misdiagnosed and confused with other arthritides or osteoarthritis.

In younger patients, hyperuricemia and gout are overwhelmingly observed in men. However, it has been recognized with an increasing frequency particularly in the elderly female population. Although historically only about 5% of all patients with gout were women, recent observations suggest that female gouty arthritis occurs far more commonly than previously suspected.[6] After menopause, serum uric acid levels in women approach those in men. As many women as men are newly diagnosed as having gout when they are older than 60 years, and more women than men are diagnosed when they are older than 80 years.[8] Many of these patients also had underlying osteoarthritis. There is also the increased female-male ratio in atypical gout.[4]

Although the location of gout is mostly in the MTP joint of great toe (followed by the other joints of foot, ankles, knees, hands), in case of atypical gout it occurs on uncommon places even for the first time.

Gout is seldom the cause of backache. Sacroiliitis is a rare manifestation of gout. In the literature there are only a few descriptions of gout affecting spine. When lumbosacral spine is involved, the same radiographic changes are described as on the peripheral joints - soft tissue tophi, bone erosions with sclerotic borders and overhanging edges. There has been a case report of epidural tophus, which caused thoracic myelopathy

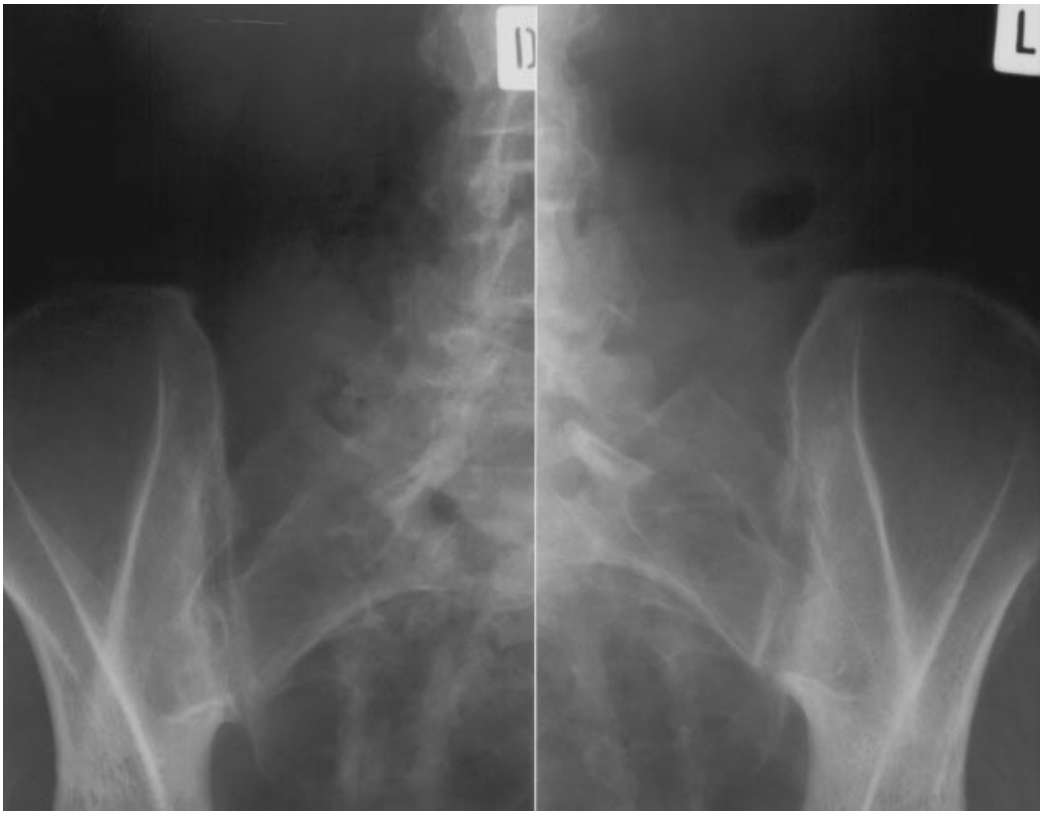

Figure 2. Sacroiliitis shown by radiography; indistinct joint space and sclerosis more pronounced on the side of the iliac bone.

due to the compression.[11] Gout seldom presents with ankylosis. There are some descriptions of ankylosis in advanced cases of chronic gout beginning in youth.[12]

Radiographs are the most important imaging modality when diagnosing gout. However, the radiographic differential diagnosis can be difficult, since the findings overlap with other conditions which cause arthritis and osteoarthritis especially in longstanding gout, elderly patients and females.[13] Therefore, the radiographic diagnosis of gout remains an important challenge and must often be confirmed with the help of laboratory and histological findings.

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Zgodovina Kliničnega inštituta za radiologijo v Ljubljani - 80. obletnica (1923-2003)
Historia Magistra Vitae

Jevtič V

Izhodišča. Prikazana je kratka zgodovina Kliničnega radiološkega inštituta, Univerzitetnega kliničnega centra v Ljubljani od njegove ustanovitve l. 1923. Skozi celotno zgodovino so se na poti razvoja Inštituta pojavljali številni problemi. Najpomembnejši, s katerimi se soočamo tudi danes, vključujejo pomanjkanje strokovne in ekonomske samostojnosti. Posledica tega je nezmožnost spremljanja hitrega tehnološkega razvoja svetovne radiologije ter nezadostno število radiologov in radioloških inženirjev, ki so čezmerno obremenjeni z rutinskim delom, kar povzroča težave v učni in raziskovalni dejavnosti. Kljub resnim oviram vodstvo Inštituta ter entuzijazem vseh radiologov in radioloških inženirjev zagotavljata nenehni strokovni in tehnološki razvoj, ki je osnova današnje visoko kvalitetne diagnostične in intervencijske radiologije. Mnogo izrednih dosežkov ne bi bilo brez nekaj izrednih oseb, ki so položile trdne temelje današnjega Inštituta.
Zaključki. 80 let Kliničnega inštituta za radiologijo v Ljubljani je zgodovina uspešnega razvoja vodilne radiološke institucije v republiki, ki so jo skupaj z vodstvom zagotovili odločnost, enotnost in profesionalnost vseh zaposlenih.

Radiol Oncol 2004; 38(4): 269-73.

Dezinvaginacija s hidrokolonsonografijo pri otroku

Vidmar D, Višnar Perovič A

Izhodišča. Invaginacija je najpogostejši vzrok črevesne obstrukcije pri otrocih, mlajših od dveh let. Proksimalni del črevesa in njegov mezenterij (intususceptum) se uvihata v distalni del (intussuscipiens), nato ju peristaltika vleče naprej, pri čemer se komprimira mezenterialno žilje. Najpogostejša je ileokolična invaginacija. Diagnozo lahko postavimo z irigografijo ali ultrazvokom. Ultrazvočni znaki so t.i. »kokarda v kokardi«, zadebeljena stena z ali brez znakov prekrvavitve ter prestenotična dilatacija z ojačano peristaltiko. Dezinvaginacijo lahko izpeljemo s kontrastnim sredstvom ali z zrakom pod rentgensko kontrolo ali s fiziološko raztopino pod ultrazvočno kontrolo.

Bolniki in metode. Pri treh deklicah v starosti 15,16 in 18 mesecev smo ob tipični klinični sliki ultrazvočno diagnosticirali ileokolično invaginacijo in nadaljevali z dezinvaginacijo pod ultrazvočno kontrolo. S pomočjo sistema za uvajanje barija pri irigografiji smo v široko črevo dovajali fiziološko raztopino, ogreto na telesno temperaturo. Posoda s tekočino je bila 1 m nad preiskovalno mizo. Količina uporabljene tekočine je bila več litrov, ker je zaradi nepopolne okluzije rektuma precej tekočine odteklo. Ultrazvočno smo opazovali potovanje vrha invaginata nazaj v proksimalni smeri. Poseg smo zaključili, ko je izginila tipična slika »kokarde v kokardi« in je prišlo do refluksa tekočine v terminalni ileum.

Rezultati. Poseg je bil uspešen in je potekal brez komplikacij pri vseh treh deklicah. Bolečine so takoj po posegu prenehale. Ob kontrolnih pregledih po 2 in 12 urah ni bilo več znakov invaginacije. Nekoliko edematozna je bila stena valvule Bauchini in terminalnega ileuma. Klinično stanje deklic je bilo normalno, tako da so bile odpuščene v domačo oskrbo neposredno po drugi ultrazvočni kontroli.

Zaključki. Dezinvaginacija s hidrokolonsonografijo je zaradi visoke učinkovitosti in odsotnosti ionizirajočega sevanja metoda izbora. Zapleti doslej niso opisovani.

Radiol Oncol 2004; 38(4): 275-84.

Prikaz napredovanja hemisferične spondiloskleroze s slikovno magnetno resonanco in paramagnetnim kontrastnim sredstvom Gd-DTPA

Jevtič V, Majcen N

Izhodišča. Namen študije je bil ugotoviti vrednost slikovne magnetne resonance (MRI) s paramagnetnim kontrastnim sredstvom Gd-DTPA v prikazu napredovanja hemisferične spondiloskleroze (HSS).

Bolniki in metode. Pri 18 bolnikih s kronično bolečino v križu in tipičnimi radiografskimi znaki HSS smo opravili Gd-DTPA MRI lumbalne hrbtenice. Primerjali smo morfološke spremembe in spremembe intenzitete signala z radiografskimi znaki HSS.

Rezultati. Na podlagi MRI smo primere razdelili v tri skupine. V prvi skupini je bilo radiografsko področje osteoskleroze hipointenzivno na T1-poudarjenih spin-eho slikah, hiperintenzivno na T2-poudarjenih slikah in je kazalo difuzno nabiranje paramagnetnega kontrastnega sredstva. Opisane spremembe so bile združljive s kostnim edemom in hiperemijo. V drugi skupini so bili robovi vretenc na vseh slikovnih sekvencah hiperintenzivni, obdani s hipointenzivno cono in se v njih ni nabiralo kontrastno sredstvo. Opisano je izražalo kombinacijo maščevja in kostne skleroze. Pri preostalih bolnikih so MRI znaki predstavljali kombinacijo sprememb vidnih v prvi in drugi skupini.

Zaključki. Gd-DTPA MRI kaže zapletene histološke spremembe v teku napredovanja HSS. Spremljanje značilnih MRI znakov ima lahko klinični pomen pri sledenju bolezni.

Radiol Oncol 2004; 38(4): 285-90.

Radiološka diagnostika akutnega pankreatitisa

Glušič M, Brenčič E, Popovič P

Izhodišča. Akutni pankreatitis zajema širok spekter vnetnih sprememb parenhima pankreasa, najpogostejša vzroka sta alkoholizem in žolčni kamni, drugi pa so redkejši.
V članku obravnavamo diagnostične metode, povezavo med morfološkimi ugotovitvami, laboratorijskimi testi in načinom zdravljenja.
Zaključki. Moderne diagnostične metode, ki so danes na voljo, so pripomogle k boljšemu poznavanju morfoloških sprememb, ki jih akutni pankreatitis povzroča.

Radiol Oncol 2004; 38(4): 291-7.

Magnetnoresonančna angiografija splenoportalnega sistema

Berden P

Izhodišča. Splenoportalni venski sistem moramo prikazati pri različnih kliničnih stanjih. Tridimenzionalna (3D) magnetnoresonančna angiografija (MRA) s kontrastom splenoportalni sistem dobro prikaže. Uporabljamo hitre sekvence z gradientnim odmevom ter najkrajšim možnim časom ponovitve in odmeva. Skozi periferno veno injiciramo do 40 ml paramagnetnega kontrastnega sredstva. Za zaznavanje kontrasta v aorti je najbolje uporabiti avtomatiziran sistem, ki nato tudi sproži slikanje. Slikanje opravimo v zadržanem dihu in dvakrat ponovimo, da prikažemo arterijsko in vensko fazo. Za obdelavo slik po preiskavi največkrat uporabljamo tehniko projekcije maksimalne intenzitete (MIP).
Zaključki. Pri bolnikih s portalno hipertenzijo MRA dobro prikaže kolaterale ter prehodnost portalne vene ali portosistemskega šanta. MRA pokaže mesto in dolžino zapore portalne vene pri trombozi portalne vene in pomaga pri odločitvi o načinu zdravljenja. Je ustrezna tehnika pri sindromu Budd-Chiari, kjer je pomembno določiti mesto in dolžino zapore jetrnih ven. MRA je zelo dobra metoda pred presaditvijo jeter, saj odlično prikaže anatomijo in prehodnost portalne vene, in tudi po presaditvi za odkrivanje morebitnih zapletov. Omejitev MRA predstavljajo metalni vsadki (npr. srčni vzpodbujevalnik), nezadovoljiv prikaz pa lahko dobimo pri nepravilni nastavitvi področja slikanja in premikanju zaradi dihanja.

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Radiol Oncol 2004; 38(4): 299-308.

Intervencijska radiologija pri hemodializnih fistulah

Šurlan M, Popovič P

Izhodišča. Namen članka je podati pregled vloge intervencijske radiologije pri obravnavanju hemodializnih žilnih pristopov. Obravnava bolnika, preden oblikujemo žilni pristop za hemodializo, vključuje radiološko ali ultrazvočno diagnostiko perifernega žilja zgornje okončine in ugotavljanje prehodnosti centralne vene. Kasneje pa vključujeodkrivanje in zdravljenje zožitev ali tromboz v slabo delujočih dializnih fistulah. Predoperacijski pregled omogoča izbiro ustreznih žil za oblikovanje dobro delujoče dializne fistule. Klinično in radiološko odkrivanje hemodinamsko pomembnih zožitev ali zapor vključuje fistulografijo, ki ji po potrebi sledi endovaskularno zdravljenje. Razširitev zožitev, ki so večje od 50%, je upravičena, ker podaljšuje prehodnost žilnega dostopa. Tehnični uspeh razširitve je 90%. Enoletna primarna prehodnost razširjene fistule na podlahti znaša 51%, pri graftih pa 40%. Žilne opornice postavljamo le v izbranih primerih; rutinsko pa v centralno veno po razširitvi, v primeru raztrganja vene ali prekomerne elastične zožitve po razširitvi.

Zaključki. Prehodnost tromboziranih fistul ali graftov lahko zagotovimo z mehaničnimi postopki ali v kombinaciji s trombolitičnim zdravljenjem. Tehnični uspeh dosežemo v 89% - 90%, primarno enoletno prehodnost pa le v 8% - 26%, vendar sekundarna prehodnost dosega 75%.

Radiol Oncol 2004; 38(4): 309-21.

Radiološka diagnostika in zdravljenje zapletov presajene ledvice

Popovič P, Šurlan K

Izhodišča. Namen članka je podati pregled vloge intervencijske radiologije v diagnostiki in zdravljenju zapletov po presaditvi ledvic. Zapleti nastanejo na žilah, sečnih izvodilih ter v ledvici in njeni okolici v obliki patoloških tekočinskih formacij. Diagnostiko, načrtovanje in vodenje znotrajžilnega zdravljenja žilnih zapletov opravljamo z Dopplerskim ultrazvokom (UZ), magnetno resonanco (MR) in z digitalno subtrakcijsko angiografijo (DSA).
Zaključki. Najpogostejši žilni zaplet presajene ledvice je zožitev ledvične arterije, ki se pojavi pri 1% - 12% bolnikov. Nezdravljena zožitev je lahko razlog za arterijsko hipertenzijo in odpoved ledvice, čeprav je zaplet potencialno ozdravljiv. Zožitev zdravimo s perkutano transluminalno ledvično angioplastiko (PTRA) z uporabo žilne opornice ali brez nje, ki je za hipertenzijo uspešna v 82% - 92% primerov, ohranitev presajene ledvice pa je možna v 80% - 100%. Zapleti, kot sta arterijska in venska tromboza, so redki. Po biopsijah se občasno pojavijo znotraj ledvične arteriovenske fistule in psevdoanevrizme, ki jih zdravimo s superselektivno embolizacijo. Najpogostejši urološki zapleti so zožitve, zapore in fistule sečevodov, ki jih zdravimo s perkutano nefrostomo, balonsko dilatacijo, vstavitvijo dvojne J kateterske opornice ali kovinske opornice. Tekočinske patološke kolekcije, kot so limfokele, abscesi, hematomi zdravimo s perkutano drenažo.

Radiol Oncol 2004; 38(4): 323-7.

Anularni pankreas, vzrok izvenjetrne zapore: prikaz primera

Ogulin M, Jamar B

Izhodišča. Anularni pankreas, redka prirojena anomalija, je trak pankreatičnega tkiva, ki obkroža dvanajstnik ali izvenjetrni žolčni vod. Prikazujemo primer anularnega pankreasa, ki je povzročil izvenjetrno zaporo odtoka žolča.
Prikaz primera. 46-letna ženska je prišla v urgentno ambulanto zaradi nenadnih bolečin v trebuhu, bruhanja in zlatenice. V zadnjih šestih letih je občasno opazila rumenkasto obarvanje kože, v zadnjem letu pa je občutila napetost v zgornjem delu trebuha, predvsem po mastnih obrokih.
Zaključki. Dve ultrazvočni preiskavi, prva pred šestimi meseci, sta pokazali razširitev glavnega izvenjetrnega voda in jetrnih žolčnih izvodil. Vzrok za razširitev je ostal nejasen, tudi po endoskopski ultrazvočni preiskavi. Pri operaciji so našli skoraj popolno zaporo izvenjetrenega žolčnega voda, ki jo je povzročal traček pankreatičnega tkiva.

Radiol Oncol 2004; 38(4): 353-62.

Radiol Oncol 2004; 38(4): 329-32.

Klinični pomen odkritja plina v portalnih venah jeter pri ultrazvočnem pregledu trebuha: prikaz dveh primerov

Gregorič P, Višnar-Perovič A

Izhodišča. Dokaz plina v veni porte je ponavadi znak resnega obolenja v trebuhu in pomeni slabo prognozo.
Z napredkom ultrazvočne diagnostike v zadnjih letih odkrijemo zrak v portalni veni tudi kot prehoden pojav brez kliničnih posledic in sicer pri topi poškodbi trebuha in nekaterih diagnostičnih in terapevtskih postopkih.
Prikaz primerov. Predstavljamo dva bolnika. Obema smo pri ultrazvočni preiskavi trebuha odkrili znake plina v portalnih venah, potek bolezni pa je bil diametralno nasproten. Prvi bolnik je bil star 70 let. Utrpel je infarkt mezenterija, čigar posledica je bil plin v portalnih venah. Umrl je nekaj ur po prihodu v bolnišnico. Drugi bolnik je bil star 26 let. Poškodovan je bil kot voznik v prometni nesreči. Med hospitalizacijo je bil klinično stabilen in ni potreboval invazivnega zdravljenja. Ugotovili smo, da se je pojavil plin v portalni veni prehodno zaradi tope poškodbe trebuha.
Zaključki. Ultrazvočna preiskava trebuha z uporabo Dopplerja je občutljiva in specifična metoda za dokaz plina v portalnih venah. Z ultrazvočno preiskavo lahko tudi odkrijemo obolenje v trebuhu, ki je vzrok za ta pojav in skrajšamo čas do potrebnega zdravljenja. Če z ultrazvočno preiskavo ne odkrijemo jasnega vzroka za plin v portalnih venah, se moramo o nadaljnih, potencialno nevarnih diagnostičnih in terapevtskih postopkih odločati glede na klinično sliko, ker je pojav plina v portalnih venah lahko tudi prehodnega značaja.

Radiol Oncol 2004; 38(4): 333-8.

Radiografski, računalniškotomografski in magnetnoresonančni znaki primarne cone kostne resorpcije v korpusu ledvenega vretenca pri Pagetovi bolezni

Jevtič V

Izhodišča. Potek Pagetove bolezni kosti ima tri razvojne faze z značilnimi radiografskimi znaki. V začetni fazi je prisotna napredujoča cona kostne resorpcije. Primarna napredujoča cona osteolize ni bila nikoli jasno prikazana na hrbtenici in medenici, kot je bila na lobanji in dolgih kosteh.

Prikaz primera. 62-letni bolnik s histološko potrjeno diagnozo poliostotske Pagetove bolezni je bil sprejet v bolnico zaradi bolečin v višini torakolumbalnega prehoda, ki so se ponoči stopnjevale. Zaradi zmerno zvišane kisle fosfataze ter kliničnih znakov hipertrofije prostate je bil postavljen sum na karcinom prostate. Kostna scintigrafija s tehnecijevim fosfonatom je pokazala pričakovano nabiranje radiofarmaka na številnih kostnih elementih s tipičnimi radiografskimi znaki Pagetove bolezni. Razen omenjenega se je pokazalo zmerno žariščno nabiranje radiofarmaka tudi v desni dorzalni polovici korpusa vretenca L1. V istem področju so klasična tomografija, računalniška tomografija (CT) ter magnetnoresonančna tomografija (MRI) prikazale primarno cono kostne resorpcije.

Zaključki. Prikazan je edinstven primer Pagetove bolezni s primarno cono kostne resorpcije v morfološko nespremenjenem korpusu vretenca L1. Po treh letih zasledovanja je prišlo do spontanega napredovanja v srednjo fazo bolezni.

Radiol Oncol 2004; 38(4): 353-62.

Radiol Oncol 2004; 38(4): 339-47.

Kalcifikacija medvretenčne ploščice vratne hrbtenice pri otroku in prsne hrbtenice pri odraslem z dorzalno hernijacijo - radiografski, računalnoškotomografski ter magnetnoresonančni znaki

Jevtič V

Izhodišča. Kalcifikacije nukelusa pulposusa pri otroku so sorazmerno redek, vendar dobro znan klinični sindrom, ki se običajno pojavlja na vratni hrbtenici. Natančen vzrok še vedno ni znan. Kalcifikacije medvretenčne ploščice odraslega so večinoma posledica degeneracije in se pojavljajo v višini spodnje prsne in zgornje ledvene hrbtenice. Pri obeh entitetah lahko nastane resna komplikacija, posteriorna hernijacija kalcificiranega diskusa.

Prikaz primera. Prikazana sta dva primera kalcificirane medvretenčne ploščice, prvi pri 7-letnem otroku v višini C7-T1 ter drugi pri 45-letni ženski v višini T11-T12, oba z razsežno dorzalno hernijacijo. V prvem primeru je dosežena remisija s konservativnom zdravljenjem. Na podlagi analize radiografskih, računalniškotomografskih ter magnetnoresonančnih (MRI) sprememb smo ugotovili podobnosti in razlike med obema entitetama.

Zaključki. Masivna dorzalna hernijacija kalcificiranega nukleusa pulposusa pri otroku je uspešno zdravljena konzervativno. Izginotje kliničnih znakov je sledilo hitri resorpciji kalcificirane duskus hernije. Pri odraslem po konzervativnem zdravljenju ni prišlo do resorpcije kalcificirane diskus hernije in kliničnih znakov izboljšanja. Z MRI je možen zgodnejši prikaz kalcifikacij. Tudi razširitev prizadetih medvretenčnih ploščic pri otroku je boljše prikazana z MRI. Razširitev kalcificiranih medvretenčnih ploščic podpira teorijo zvišanega intradiskalnega pritiska kot vzroka rupture anulusa fibrozusa in posledične hernijacije nukleusa pulposusa.

Radiol Oncol 2004; 38(4): 349-52.

Nenavadne radiografske spremembe pri bolnici s putiko

Markota J

Izhodišča. Putika je metabolna motnja, ki je povezana s hiperurikemijo in nalaganjem kristalov sečne kisline (uratov) v tkiva, zlasti v sklepni hrustanec. Za urični artritis so značilni ponavljajoči se napadi akutnega artritisa, ki se lahko nadaljuje v kronično obliko. V 80% primerov se bolezen začne v metatarzofalangealnem sklepu nožnega palca in je pogostejša pri moških.
Prikaz primera. Prikazali smo primer nenavadnih radiografskih sprememb pri uričnem artritisu. 63-letna bolnica se je pritoževala nad občasnim otekanjem bazalnega sklepa desnega nožnega palca in desnega kolena, okorelostjo stopal in prstov rok ter bolečinami v hrbtu. Prve težave so se pojavile pred tridesetimi leti. Rentgenogrami so pokazali degenerativne spremembe večine slikanih sklepov, obojestranski sakroiliitis in kostno ankilozo obeh nartov. Pri pregledu sinovijske tekočine kolena s polarizacijskim mikroskopom so ugotovili uratne kristale. Tudi histološki izvid sinovije po sinovektomiji je potrdil urični artritis.
Zaključki. Radiografija je najpomembnejša slikovna metoda pri diagnostiki putike. Diferencialna diagnostika pa je lahko težavna, kadar se spremembe prekrivajo z drugimi artritisi ali osteoartrozo, zlasti pri dolgotrajni putiki, starejših bolnikih in ženskah. V teh primerih končno diagnozo potrdimo z laboratorijskimi in histološkimi izvidi.

Notices

*Notices submitted for publication should contain a mailing address, phone and/or fax number and/or e-mail of a **Contact** person or department.*

Thoracic oncology

January 14-16, 2005

The »4th Annual Clinical Cancer Update« will be held in North Lake Tahoe, USA.

Contact UCSF Office of Continuing Medical Education, P.O. Box 45368, San Francisco, CA 94145-0368, USA; or call +1 415 476 4251; or fax +1 415 502 1795; email info@ocme.ucsf.edu; or see http:// www.cme.ucsf.edu

Radiation oncology

March, 2005

The ISRO international teaching course on »Palliative Care in Cancer Treatment« will take place in Dar es Salaam, Tanzania.

See http://www.isro.be

Brachytherapy

May 5-7, 2005

The Annual Brachytherapy Meeting GEC-ESTRO and pre-meeting workshop on breast cancer will take place in Budapest, Hungary.

Contact ESTRO office, Avenue E. Mounier, 83/12, B-1200 Brussels, Belgium; or call +32 775 93 40; or fax +32 2 779 54 94; or e-mail info@estro.be; or see http://www.estro.be

Clinical oncology

May 13- 17, 2005

The ASCO Meeting will be offered in Orlando, USA.

E mail enews@asco.org; or see http://www/asco.org

Lung cancer

July 3-6, 2005

The »11th World Conference on Lung Cancer« will be offered in Barcelona, Spain.

Contact Heather Drew, Imedex, Inc., 70 Technology Drive, Alpharetta, GA 30005 USA; or call +1 770 751 7332, or fax +1 770 751 7334; or e-mail h.drew@imedex.com, or see www.imedex.com/calenders/oncology/htm

Radiotherapy

September 24-29, 2004

The »8th Biennial ESTRO Meeting on Physics and Radiation Technology for Clinical Radiotherapy« and »Pre-Meeting Workshop on Image-Guided Radiotherapy« will take place in Lisboa, Portugal.

Contact ESTRO office, Avenue E. Mounier, 83/12, B-1200 Brussels, Belgium; or call +32 775 93 40; or fax +32 2 779 54 94; or e-mail info@estro.be; or see http://www.estro.be

Radiation oncology

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See http://www.isro.be

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October 17-19, 2005

The IASLC workshop »Biology and Prevention of Lung Cancer« will be offered in Woodstock, Vermont, USA.

Contact Taryn Klocke at Envision Communications; call +1 770 763 5690; or see www.lungcancerprevention.net

Oncology

October 30 - November 3, 2005

The ESTRO 24 / ECCO 13 Conference will take place in Paris, France.

Contact FECS office, Av. E. Mounier, 83/4, B-1200 Brussels, Belgium; or call +32 7759340; or fax +32 2 7795494; or e-mail info@estro.be; or see http://www.fecs.be

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Activity of »Dr. J. Cholewa« Foundation for Cancer Research and Education - A Report for the Last Quarter of 2004

The Dr. J. Cholewa Foundation for Cancer Research and Education has continued to support various activities associated with cancer research and education in Slovenia throughout the last quarter of 2004. Several other initiatives were examined and carefully thought of in the aforementioned time period. The topics discussed included the possibility of the Foundation co-sponsoring the publication of the book dedicated to children survivors of various cancer diseases, the cooperation with the Emergency Ward at the Clinical Centre in Ljubljana and the problems associated with the public calls for Dr. J. Cholewa Foundation for Cancer Research and Education grant applications. Several other problems associated with all these ideas and endeavours were thoroughly discussed during the course of 2004 and also during the last time period mentioned previously. It is worth mentioning that many study and research grants have been already bestowed to researchers from various scientific fields associated with oncology in Slovenia and that many of them were also given grants and means to attend scientific meetings, congresses, conferences and symposia dealing with oncology worldwide. The Foundation is also determined to continue to support the regular publication of ĽRadiology and Oncology« international scientific journal, which is edited, published and printed in Ljubljana, Slovenia, as it has done over the last couple of years.

In one of the meetings of the Supervisory and Executive Boards of the Foundation held previously in the year 2004 the financial situation was thoroughly evaluated and discussed. It has to be acknowledged that various public and privately owned enterprises find it ever more difficult to contribute financially to help running day to day operations of the Foundation and its many scopes of activity. Several new initiatives and suggestions were discussed and evaluated during the recent meetings of the Foundation to address this serious problem. It is important to note that many public companies and private individuals remain committed to support the Dr. J. Cholewa Foundation for Cancer Research and Education and its activities.

Tomaž Benulič, MD
Borut Štabuc, MD, PhD
Andrej Plesničar, MD

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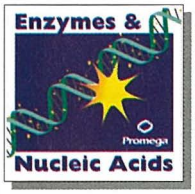

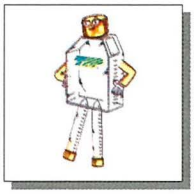

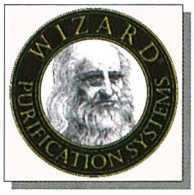

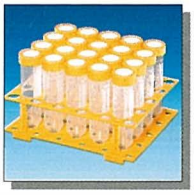

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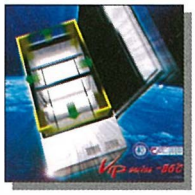

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General instructions • Radiology and Oncology will consider manuscripts prepared according to the Vancouver Agreement (*N Engl J Med* 1991; **324:** 424-8, *BMJ* 1991; **302:** 6772; *JAMA* 1997; **277:** 927-34.). Type the manuscript double spaced on one side with a 4 cm margin at the top and left hand side of the sheet. Write the paper in grammatically and stylistically correct language. Avoid abbreviations unless previously explained. The technical data should conform to the SI system. The manuscript, including the references may not exceed 15 typewritten pages, and the number of figures and tables is limited to 4. If appropriate, organize the text so that it includes: Introduction, Material and methods, Results and Discussion. Exceptionally, the results and discussion can be combined in a single section. Start each section on a new page, and number each page consecutively with Arabic numerals.

Title page should include a concise and informative title, followed by the full name(s) of the author(s); the institutional affiliation of each author; the name and address of the corresponding author (including telephone, fax and e-mail), and an abbreviated title. This should be followed by the *abstract page*, summarising in less than 200 words the reasons

for the study, experimental approach, the major findings (with specific data if possible), and the principal conclusions, and providing 3-6 key words for indexing purposes. Structured abstracts are preferred. If possible, the authors are requested to submit also slovenian version of the title and abstract. The text of the report should then proceed as follows:

Introduction should state the purpose of the article and summarize the rationale for the study or observation, citing only the essential references and stating the aim of the study.

Material and methods should provide enough information to enable experiments to be repeated. New methods should be described in detail. Reports on human and animal subjects should include a statement that ethical approval of the study was obtained.

Results should be presented clearly and concisely without repeating the data in the tables and figures. Emphasis should be on clear and precise presentation of results and their significance in relation to the aim of the investigation.

Discussion should explain the results rather than simply repeating them and interpret their significance and draw conclusions. It should review the results of the study in the light of previously published work.

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Evans R, Alexander P. Mechanisms of extracellular killing of nucleated mammalian cells by macrophages. In: Nelson DS, editor. *Immunobiology of macrophage.* New York: Academic Press; 1976. p. 45-74.

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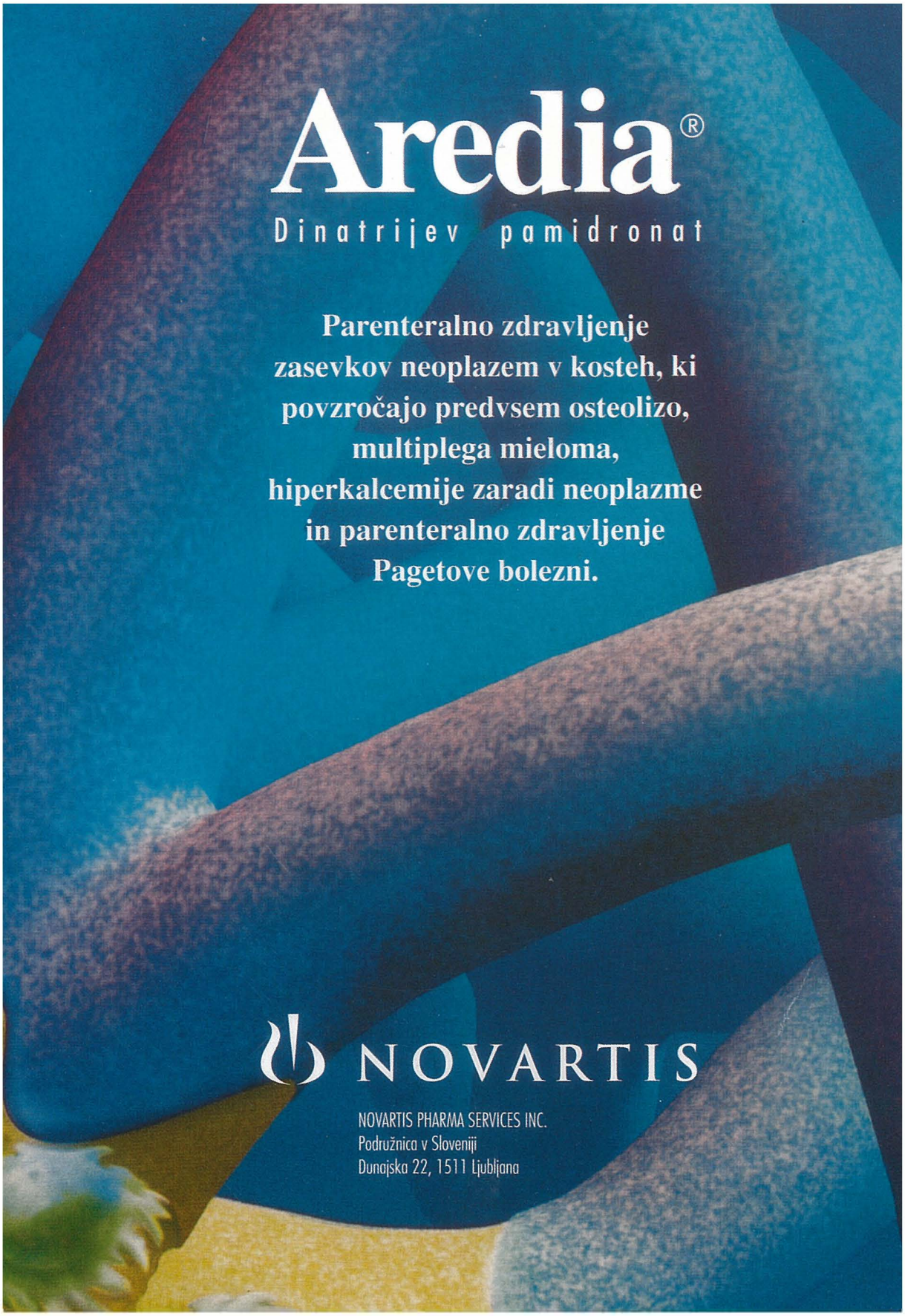
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