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

case report

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

Primož Gregorič, Alenka Višnar-Perovič

Clinical Radiology Institute, University Clinical Centre Ljubljana, Slovenia

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.

Received 3 November 2004
Accepted 23 November 2004

Correspondence to: Primož Gregorič, MD, Clinical Radiology Institute, University Clinical Centre Ljubljana, Zaloška 7, SI-1000 Ljubljana, Slovenia; Phone: +386 1 839 48 63; E-mail: primoz.gregoric@guest.arnes.si

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-

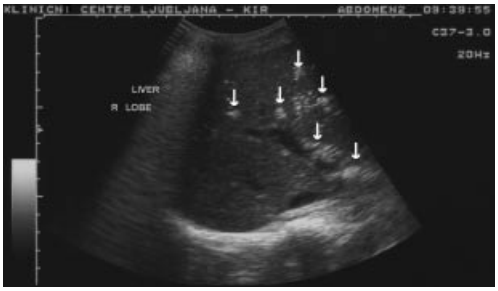

Figure 1. Grey-scale sonogram showing small echogenic gas bubbles (arrows) in the portal veins and liver parenchyma.

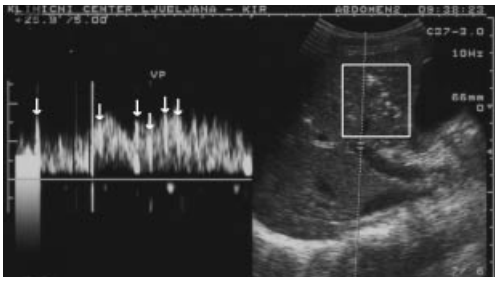

Figure 2. Doppler sonogram of a portal vein showing multiple high-amplitude spikes (arrows), typical of intravascular gas.

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.

References

1. Brown MA, Hauschildt JP, Casola G, Goink BB, Hoyt DB. Intravascular gas as an incidental finding at US after blunt abdominal trauma. *Radiology* 1999; **210:** 405-8.

2. Chevallier P, Peten E, Souci J, Chau Y, Padovani B, Bruneton JN. Detection of portal venous gas on sonography, but not on CT. *Eur Radiology* 2002; **12(5):** 1175-8.

3. Maher MM, Tonra BM, Malone DE, Gibney RG. Portal venous gas: detection by gray-scale and Doppler sonography in the absence of correlative findings on computed tomography. *Abdominal Imaging* 2001; **26:** 390-4.

4. Strohm WD, Romele UE. Die Bedeutung der sonographie bei dem nachweis von gasansammlungen in der pfortader oder in lebervenen. *Medizinische Klinik* 1994; **89:** 538-42.

5. Schultze CG, Blum UK, Haag K. Hepatic portal venous gas: imaging modalities and clinical significance. *Acta Radiologica* 1995; **36:** 377-80.

6. Lafortune M, Trinh BC, Burns PN, Breton G, Burke M, Dery R, et al. Air in the portal vein: sonographic and Doppler manifestations. *Radiology* 1991; **180:** 667-70.

7. Sebastia C, Quiroga S, Espin E, Boye R, Alvarez-Castells A, Armengol M. Portomesenteric vein gas: pathologic mechanisms, CT findings and prognosis. *Radiographics* RSNA 2000; **20:** 1213-24.

8. Ohtsubo K, Okai T, Yamaguchi Y, Watanabe H, Motoo Y, Matsui O, et al. Pneumatosis intestinalis and hepatic portal venous gas caused by mesenteric ischemia in an aged person. *Gastroenterology* 2001; **36:** 338-40.