Multislice computed tomography of pulmonary embolism: spectrum of findings

Šerif Bešlić, Faruk Dalagija, Vesna Đurović

Institute of Radiology, Clinical Center, University of Sarajevo, Bosnia and Herzegovina

Background. *The purpose of this study is to analyse the contribution of multislice computed tomography (MSCT) as a diagnostic method in the diagnosis of pulmonary embolism (PE) and spectrum of findings in our material.*

Methods. *During the period of one and a half year, we found PE in 25 patients (15 males and 10 females). The average age of the patients was 54.4 years (25 - 74). The examination was performed by »Somatom Volume Zoom« Siemens CT machine with four row detectors, with retrospective ECG gating, collimation 4 x 2.5 mm and reconstructed section with 0.8 mm. Contrast medium (130 ml) and 10 ml of saline was applied, administered with a flow rate of 3.5 ml/s and with time delay of 22 seconds.*

Results. *During the examination, we found embolism of the main branches of pulmonary artery in 14 (56%) patients, at the right branch in 10 (40%), at the left one in 4 (16%), and bilateral pulmonary embolism in 11 (44%) patients. Subsegmental pulmonary emboli were noticed in 8 (32%) patients. Pulmonary infarct was found in 12 (48%) patients, and was followed up with ipsilateral pulmonary artery dilatation in 11 (44%) cases, redistribution of the circulation and pulmonary artery branches dilatation in infarct zone in 9 (36%) cases, contrast enhanced consolidation of pulmonary parenchyma in 10 (40%), rag zones of ground glass attenuation in 15 (60%), haemorrhage in 21 (84%), striped and reticular pulmonary drawing in 11 (44%), and mosaic olighemy in 3 (12%)cases. Thrombi were rare, found only in the R/L atrium in 2 (8%)cases, pericardial haemorrhage in 1 (4%), mediastinal lymph nodes in 1(4%) case, sudden cut off of peripheral branch leading to infarct apex in 1 (4%), and haemoptysis in 1 (4%) case. In addition to deep vein thrombosis, heart failure was found as aetiology factor in 7 (28%) and malignancy in 3 (12%) cases.*

Conclusions. *MSCT is an excellent non-invasive method for visualization of thrombus in the pulmonary artery. In our study, we have more often found embolism of the right branch of pulmonary artery, and pleural effusion, infarct contrast enhanced consolidation of pulmonary parenchyma, ground glass attenuation zone, ipsilateral pulmonary artery dilatation, circulation redistribution with pulmonary artery branches dilatation nearby infarct zone. This diversity of findings cannot be noticed by any other method, with the possibility of making alternative diagnosis, which has led MSCT in the foreground when pulmonary embolism diagnostics is at stake.*

Key words: pulmonary embolism – diagnosis; X-ray computed - methods

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Correspondence to: Prof. Šerif Bešlić, MD, PhD, Institute of Radiology, Clinical Center University of Sarajevo, Bolnička 25, Bosnia and Herzegovina; Fax +387 33 444 553; E-mail: sbeslic@bih.net.ba

Introduction

Pulmonary embolism (PE) is an obstruction of any pulmonary artery branch by clot which could be brought from varicose veins in legs, tendency to blood clotting (because of existing malignancy, after prostate gland operation, in gynaecologic diseases, in heart failure and arrhythmias, etc).[1]

It is a frequent disease in risk hospitalized patients. An embolus tears off from the deep leg veins, divides in fragments in the right heart, from where it is rinsed in the lung, where the emboli of different size splash the lung.[2,3]

PE can cause immediate death or the patient is breathless, has chest pain, his face and body become blue, neck veins swell up, his breathing is low and rapid, has palpitations, coughs and coughs up white pus mixed with blood (sputum).[1,4,5]

Thrombosis can cause pain and swelling of the leg, red colouring, warmth and tension could occur, but clinical evaluation is mostly unreliable and can cause serious mistakes, and most of people with deep vein thrombosis (DVT) have no any symptoms.[6]

Sudden dyspnoea without changes on plain chest radiography could be one of the manifestations.[1]

PE prevalence found on autopsy is 15-26%, and it is greater than the one found in hospitalized patients.

Owing to organs hypoxia, especially brain, 1/6 patients lose consciousness (syncope). The embolism attacks can repeat several times, so high blood pressure in pulmonary circulation appears (pulmonary hypertension), with bad prognosis and most patients die after 5 years.[1]

Due to the above, all cases of pulmonary embolism must be taken seriously.

Vein thrombosis is a frequent disease, with an incidence of 1-2 persons/ per 1000/ per year. Approximately 3000 persons get vein thrombosis in Slovenia every year. Non-treated thrombosis of popliteal and/or femoral vein will lead to PE in approximately 50% patients, of whom 10% patients die of pulmonary embolism.[3]

PE is the third most frequent cause of death in US with almost 50 000 deaths every year, with annual frequency of 300 000 to 600 000 (on average about 500 000) accidents. Many of PE events passed undiscovered because clinical indications and PE symptoms are non-specific. A fast and accurate diagnosis can save 100 000 lives every year. An accurate diagnosis is therefore a great challenge.[7,8]

The mortality caused by PE is high, partly because of relapse. The mortality in non-treated patients is 30%, and in the patients with anticoagulation therapy, 10%. Repeated PE appears in 0.4-0.5% patients with acute PE. Access to the treatment of patients with PE has to be fast and interdisciplinary. Clinical suspicion of PE has to be confirmed or denied by diagnostic methods, preferably with non-invasive, precise and easily accessible methods.

It is important to exclude PE, because of the haemorrhage which can occur during anticoagulant therapy. The haemorrhage risk increases by 2% every day during the anticoagulant therapy.[3]

It is known that 20% of DVT of the calf will have propagation proximately, but sometimes thrombosis staying in the calf should not lead to embolism. Should we wait and look at the insurance policy in situation like that, or should we start with diagnostic imaging of the affected extremity?

Differential diagnosis of PE is very wide and includes many conditions, from life-endangered diseases to anxiety.[3]

There is no any warning signs, symptoms or laboratory tests, which suggest PE.[2]

In 2/3 of patients with the suspicion of PE, other diseases were diagnosed.[9]

PE diagnosis is clinical, laboratory (simple test in bed D-Dimmer essay), ECG, plain ra-

diography of chest (RTG), gas analysis in arterial blood, lung scintigraphy with technetium 99, transthoracic or transesophageal US of the heart and veins, angiography of pulmonary artery, which until recently, was the most reliable diagnostic method, and sometimes the therapy method as well.[1,3,8]

The chest angiographies were normal in 10% cases, and most of its abnormalities were non-specific. Non-specific shade, pleural effusion, atelectasis or elevation of hemidiaphragm, and vascular alternation, i.e. focal ipsilateral pulmonary artery enhancement (Fleschner's sign or so called the ankle sign) could be found. The pulmonary artery branch embolism can be seen as the lung infarct, a wedge shaped shadow with its top oriented centrally and base peripherally. Usually, the right interlobar pulmonary artery is affected. Because of the clot presence, focal olighemy may occur, but is very rare and results from the vascular obstruction (Westermarks sign).

Lung infarct can develop immediately or in 2-3 days after embolism, usually peripherally or in the lower lung zones, frequently associated with small pleural effusion. At the beginning, it is ill-defined, but with time, it becomes sharp, the so-called Hampton's hump in the peripherally wedge-shaped shade with curvy peak headed to the hilus. The infarct healing, or the so-called »melting«, demonstrates as keeping its shape and, with time, reducing in size. Pneumonia and oedema usually »disappear – fade away« gradually.[1,2,10]

In big branch embolism, the whole lobe of the lung can »fall out« of function, so, the pulmonary-blood vessel bed or pulmonary tissue cannot be seen with chest X-ray.[1]

The lung scintigraphy with technetium 99 (V/Q) has been preferred for a long time as screening test for detecting clinically significant pulmonary embolism was. It shows »fall out« of lobe of a lung or part of it. A segmental or greater perfusion defect is present with normal ventilation in that zone (V/Q discord) indicates a high possibility of pulmonary embolism.[1,10]

V/Q scanning findings are indirect indicators of a clot, not visualized directly, so it has a high sensitivity, but low method specificity, especially in the patients with other lung diseases. Because of that, the interpretation of perfusion scintigraphy results is difficult.[2,3,10]

PIOPED study results show that only 41% PE can be confirmed with this method, also the accordance in the interpretation of findings among different examiners is poor (30%).[2,3]

Until recently, the most reliable method has been diagnostic pulmonary angiography, sometimes it is therapeutical as well (local thrombolysis, fragmentation, embolectomy).[1,11]

Angiography has been considered the most precise examination, but it is invasive (morbidity 6%, mortality 0.5%), and it is not available everywhere.[3]

The pulmonary angiography findings were considered a gold standard; however, they show 25% false negative results for small subsegmental emboli, and the accordance in interpretation of findings among different examiners is poor (<30%). This investigation is rarely performed in clinical practice.[2]

The technology revolution in diagnostic approach to suspicious pulmonary embolism has been happening in the last 10 years by introducing spiral computed tomography (SCT).[2,12]

In 1978, Sinner was the first who described PE diagnosed with CT. In 1980, Godwin and co-operators showed directly endovascular emboli. In 1992, the first comparative analysis of SCT and PA was made, and in the next years, Teigen and co-operators used electron beam CT (EBCT).[7]

In the last 10 years, CT reached a high accuracy in the pulmonary embolism evaluation.[7]

Spiral or electron beam CT findings have revolutionized the pulmonary embolism diag-

nosis and made possible the direct visualization of a clot in the central pulmonary artery.[2] CT provides, with high sensitivity and specificity (>90%), a direct visualization of obstructing emboli together with their vascular and pleuroparenchymal sequels (cardio pulmonary status). An insufficient contrast bolus, hilar limphadenopathy and hilar calcifications, respiration artefacts can cause diagnostic problems, e.g. subsegmental emboli can be overlooked, oblique arteries may demand oblique reconstructions for better visualization, etc. It can be combined with the pelvis and extremities scanning for analyzing sources of thromboembolism (CT flebography). The consequences of a negative CT angiogram are favourable, with DVT or PE in 0.5%, and fatal embolism occurs in 0 to 0.7% cases.[2,13]

The comparisons of SCT pulmonary angiographies (SCTPA) with V/Q scans proved higher punctuality of SCTPA than V/Q; SCT was correct in 92%. CT, in comparison with V/Q scan, shows a reliability of 90% to 54%, respectively. SCT has a higher sensitivity (77-81%) than V/Q (41%), and a similar proportion has been observed in specificity. In total, SCT is more punctuate than V/Q. Various studies in several European centres proved a higher specificity of CT than that of V/Q, and better accordance in the interpretation of CT findings between different examiners.

In that way, SCT has put into question the role of ventilation-perfusion scintighraphy and has thrown suspicion on pulmonary angiographies as a gold standard.[8]

Moreover, CT allows the visualization of other changes in the thorax, which may be the cause of patient's condition and symptoms. In 65% of patients, various changes are discovered by CT, upon which an alternative diagnosis was made in the patients suspicious for PE. Neither scintigraphy nor angiography has these possibilities.

The introduction of the multislice CT (MSCT) has brought significant advantages, such as the possibility to examine dyspnoeic patient in an emergency situation in a few seconds, covering broad volumes with a low collimation, the possibility of a precise analysis of peripheral pulmonary arteries and of a detailed whole lung exploration. The combined CT venography and pulmonary CT angiography, using one injection of contrast medium, reduces the examination time and excludes additional examinations. Finally, by the evaluation of the right heart, the load and distension of the right-side cavities can be estimated. In that way, the multislice CT exposed one of its most important applications.[6,12]

Risky and symptomatic patients are often exposed to ascendant venography, which is considered as a gold standard fort he detection of small deep thrombi in the vein system, but this technique is invasive, so radiologists are looking for a replacement.

Ultrasound (US) B mod and Colour Doppler are fast and reliable methods, but need experienced examiners. MRI is also a promising method, but so far, it has not been used widely in urgent situations and in seriously ill patients, mostly because of long-term examination, monitoring problems, high costs and limited availability.[7,11]

The time of flight and phase contrast imaging proved to be highly accurate in some research studies on imaging of the blood circulation in the proximal vein system, and also provides a direct visualization of a clot in the pulmonary artery or extremities veins. In pregnant women and the patients with plaster cast, the acute clot can be differentiated from the chronic clot and from imitating pathology. MRI is an expensive screening method. It has some deficiencies, e.g. every patient cannot fit in the machine (overweight), and for the time being, the clot visualization below the knee is not satisfactory.[6]

The treatment of PE presumes usage of anticoagulants and fibrinolitics.[2]

The therapy of blood clotting lasts usually

6-12 months and is accompanied by hemorrhagic complications in 2-15% cases; a thrombotic vein can be surgically separated from the thrombosis place, or a special device is inserted (IVC filter) in which the clots are kept not to go in the right heart, more exactly, in the pulmonary circulation.[1]

They are used when the contraindication for anticoagulation therapy is present. Pulmonary thromboendarterecotmy can be used in vitally endangered patients.[13]

The prognosis is good with adequate therapy. However, there is a high level of suspicion on fatal result (about 20%) in non-treated subjects. Hardly noticed subsegmental emboli present a problem. The consequences for non-treated subsegmental emboli are unknown, while the consequences of following up the negative pulmonary angiograms or SCTPA are favourable.[2]

The goal of this study is to analyze the contribution of MSCT in the detection of pulmonary artery embolism in our material, as well as to review the frequency of findings spectrum that follows it, and which can be diagnostically important.

Methods

In the period of one year and a half, we found PA embolism in 25 patients (15 male and 10 female) during MSCT scanning. The youngest patient was 25 and the oldest one was 74. Average age of patients was 54.4 years.

After the native CT serial, which included the whole thorax and upper abdomen, the contrast serial was made in the area from the arcus of the aorta to 2 cm below the mouth of the lower pulmonary veins.

Scanning was performed on the »Somatom Volume Zoom« Siemens device, MSCT with 4 rows of detectors, with retrospective ECG-gating, thick layer of 3 x 2.5 mm and section width of 0.8 mm.

The contrast medium (CM) 130 ml and 10 ml of physiological solution were injected by automatic syringe in the cubital vein with a flow rate of 3.5 ml/s and with a delayed time, determined mostly empirically and ranging from 22 to 25 sec, depending on the cardiac status and patient's age.

The analysis of the following findings was made: embolism frequency and dilatation of central and segmental branches, frequency of pulmonary circulation redistribution, infarct, contrast enhanced consolidation of pulmonary parenchyma, zone opacification type ground glass, olighemy mosaic, septal bumps and reticular drawing, pleural reaction and pericardial effusion, the right heart dilatation, heart failure signs, frequency of clots in the heart, haemoptysis, appearing of enhanced mediastenal lymph nodes, and frequency of malignant process coincidence and pulmonary embolism.

Results

During the examination we found pulmonary artery embolism in 25 (100%) patients. Among them, 15 (60%) were male and 10 (40%) female. The spectrum and frequency of CT findings in examined patients are shown in Table 1.

From the given chart, we can see that PE was more often followed by pleural effusion, which was found in 21 (84%) patients, zone opacification ground glass found in 15 (60%) patients, central pulmonary artery branches embolism in 14 (56%) patients, pulmonary infarct in 12 (48%) patients, bilateral PE, ipsilateral pulmonary artery enhancement, and striped and reticular pulmonary drawing in 11 (44%) patients, contrast enhanced pulmonary parenchyma consolidation and PE of the right branch of pulmonary artery in 10 (40%) patients. Other CT findings were rare, or harder to notice.

Table1. Patient data: spectrum of findings in pulmonary embolism

Features	Number of patients
Bilateral PE	11 (44%)
Central branches embolism	14 (56%)
PE R.Branch	10 (40%)
PE L.Branch	4 (16%)
Subsegmental PE	8 (32%)
Ipsilateral pulmonary artery enhancement	11 (44%)
Dilation R.Branch	8 (32%)
Dilation L.Branch	3 (12%)
Circulation redistribution and branch dilation in infarct zone	9 (36%)
Sudden failure of peripheral pulmonary artery leading to infarct apex	1 (4%)
Infarct	12 (48%)
Contrast enhanced pulmonary parenchyma	10 (40%)
United Infarct and contrast enhanced consolidation pp	6 (24%)
Rag zone of ground glass opacification	15 (60%)
Olighemy mosaic	3 (12%)
Striped and reticular pulmonary drawing	11 (44%)
Effusion and adjacent pleura reaction	21 (84%)
Presence of previous heart failure indications	7 (28%)
Clot in R/L atrium	2 (8%)
Malignance (1 Ca. Recti,2 Ca pulmo)	3 (12%)
Haemoptysis	1 (4%)
Pericardial effusion	1 (4%)
Boundary lymph nodes in mediastinum	1 (4%)

Discussion

Obstruction of any pulmonary artery branch, mostly with blood clot, is called pulmonary embolism.

The pulmonary embolism is the final result of thrombosis in the peripheral veins of lower extremities and is considered the third most frequent cause of death.

The risk factors are prolonged staying in bad, varices in lower extremities, trauma, recent surgical treatment, obesity, pregnancy, deficiency of antithrombin III, deficiency of S protein, increased blood clotting in the patients with malignant disease, migrant thrombophlebithis, deep vein thrombosis in pelvis, acute heart attack, serious heart impairment, central vein catheters, congestive heart disease, arrhythmias, atrial fibrillation, etc.[1,2,10,11,14]

Sudden death occurs most frequently when the obstruction happens at the bifurcation of the pulmonary artery. When the distant branches are occluded, the patient is breathless, becomes blue in the face and body, the veins of the neck are swollen, breathing is superficial and accelerated (>21/min), the blood flow rate through the lung is decreased, all organs suffer from ischemia, especially the brain, so, in 1/6 of patients, syncope develops. If the patient stays alive, acute pulmonary heart will develop, with strong chest pain bellow the sternum. The prognosis is dependent on the heart condition and quick intervention.

The embolus in the intermediate artery causes the deterioration of the patient's condition, chest pain, cough, blood-stained cough up, feeling of choking, tachypnea and superficial breathing.

Symptomatology can be divided in few

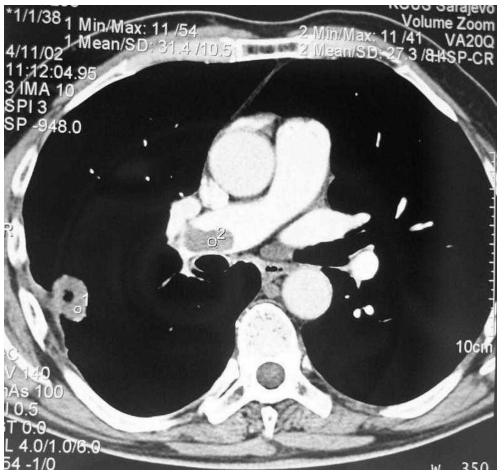

Figure 1. Pulmonary embolus in the right central branch.

phenomena, such as the pulmonary infarct syndrome (pleural pain or haemoptysis), isolated dyspnoea syndrome (dyspnoea in absence of pleural pain, haemoptysis or circulatory collapse) and circulatory collapse syndrome (losing consciousness or blood pressure <80mmHg) and can be met in 65%; 22% and 8% patients, separately.

The emboli in the central branches are most often and most easily detected (Figure 1).

In this study, it was found in 14(56%) cases; PE of the right branch in 10(40%), in the left branch in 4(16%), and bilateral PE in 11(%). The average age of the patients was 54.4 years. It was more frequent in men (60%) than in women (40%).

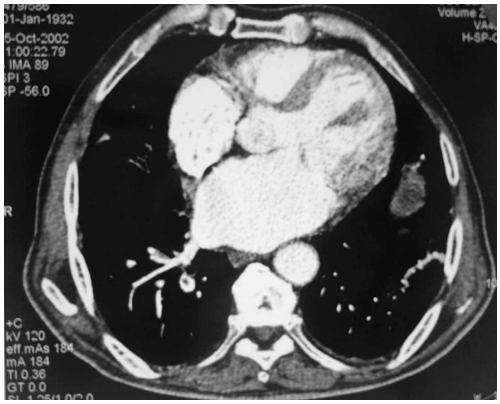

Figure 2. Pulmonary emboli of subsegmental artery.

As shown in Table 1, PE in the subsegmental artery were found in 8 (32%) examined patients (Figure 2).

PE divides in 3-11 parts, on average, when it comes in the heart. One or few fragments are big enough to be detected.[3]

One third of PE causes or contributes to the patient's death where clinical diagnosis of suspected PE is unreliable; so, over 70% of cases are not clinically suspected. These results have not changed for 3 decades in spite of the progresses in medicine and prophylaxis. Approximately 10% of the patients do not survive the initial stage of PE. PE is fatal if it is not treated in 30%, and this can be reduced to 2-10% if diagnostic and treatment with anticoagulants are quick enough. This therapy is accompanied with complications in 10-30%.

The estimation frequency of isolated subsegmenal PE is very significant because they can be indicator of a silent deep vein thrombosis (DVT), which potentially indicates harder embolic accidents. The detection of a small embolus can be relevant for a chronic pulmonary hypertension diagnosis in the patients with thromboembolism disease, and may represents a »tip of the iceberg«. This problem may not be solved for a long time.

The only solution to solve the problem of these small missed or potentially missed clots is to evaluate consequences of patients with negative SCT pulmonary angiographies (SCT-PA), in other terms, to determine the subsequent PE rate (negative predicting value).

Different authors cite different results for the main, lobar, segmental and subsegmental branches: sensitivity 88-91%, specify 81.5-86%, positive 75.81.5% and negative predicting value 91-94%.[20]

Some authors reported the sensitivity of 90%; specificity 94%, positive and negative predicting value 90 and 94%.[4]

According to certain authors, the sensitivity at segment level is 91-96% and specifity 78-100%, the subsegment sensitivity 63% and specificity 89% and, recently, the sensitivity

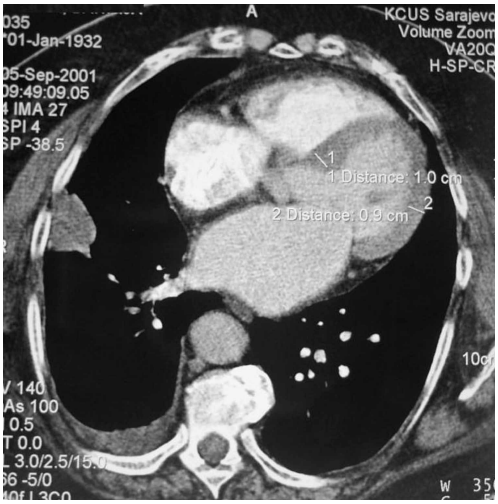

Figure 3. Pulmonary infarct (Humptons hump).

and specifity ranges of 88-96% and 94-100%, repsectively. In that way, CT is kept busy with angiographies on subsegmental level.

According to these authors, angiographies have some lower results at subsegmental level, so it cannot be used as a reference method for success evaluation.[3]

A one-mm breath hold collimation results with significant greater percentage of embolism detection in subsegmental pulmonary arteries, and greater harmony between different readers than using thicker cross sections. Using one-mm cross vs. opposite 3mm ones, the number of indefinite cases reduces to 70%.[21]

Also 54% subsegmental arteries are identified after using pulmonary window, and they are correctly visualised using mediastinal window.[18]

The prevalence of isolated subsegmental embolism, according to different studies, is between 5 and 30%. If satisfied, the subsegmental branches don't show during examination. The pulmonary diagnosis can be predicted in 5-30%. This is especially important in the patients with accompanying lung and heart diseases, in whom they may be warning signs for developing recurrent potentially mortal embolism.[3]

Only relatively specific PE finding is wedge shaped pulmonary consolidation, which most probably represents pulmonary infarct, although in PE patients, this finding is very rare and, according to some authors, varies between 10%, 25% and 62%.[3,13]

A similar image may appear in 5% of patients who have not PE (It is often seen in pneumonia, tumours, pulmonary fibrosis, haemorrhage, oedema).[3]

In this investigation, pulmonary infarct was found in 12 (48%) patients. We believe that it is more frequent than reported in literature and that the advance of diagnostic methods will contribute to a more frequent revealing (Figure 3).

Pulmonary infarcts can be any shape or size, like peripheral opacification, lobular or wedge-shaped image, irregular polyhedral, depending on the number and location of affected secondary pulmonary lobules with cut apex, when the lobules lay just right opposite embolus and they are divided adequately from bronchial collateral vessels.

The infarct can include central regions of low attenuation, which show the combination of opaque glass shade and reticulation below and that represent no infarct secondary pulmonary lobules, which in time of embolism cannot be per funded. Alternatively, they can be supplied from nonembolised pulmonary arteries, retrograde circulation from pulmonary veins, or from bronchial collateral vessels.

The contrast enhancement of lesion after the fourth injection of contrast medium is connected with pulmonary haemorrhage (76%), and non-enhanced lesion suites pulmonary infarct. However, a drop in enhancement in the collapsed lung is not a specific sign of pulmonary infarct, because it can be seen in some kinds of pneumonia.[7]

The contrast enhanced consolidation of pulmonary parenchyma was, in this investigation, found in 10 (40%) cases. In 6 (24%) cases it was related to the infarct (Figure 4). In

radiology literature, these two categories are not adequately distinguished.

The presence of vascular sign, associated with the vessel bump leading to shade apex, increases the possibility that the lesion represents infarct, but this sign is not frequent and therefore hard to recognize.

The enhancement of ipsilateral pulmonary artery in this investigation was found in 11 (44%) cases, 8 (32%) on the right and 3 (12%) on the left branch. Circulation redistribution and branch dilation in the infarct region are found in 9 (36%) cases, and a sudden break of the peripheral pulmonary artery leading to infarct apex only in 1 (4%) case. This sign is difficult to recognize and requires a meticulous analysis.[10,13,15]

The obstruction exceeding 30% of pulmonary circulation causes a sufficient increase in pulmonary vascular resistance to produce significant pulmonary hypertension which results in overloading the right ventricle (RV), which increases and dilates. The interventricular septum moves to the left, compressing the left ventricle (LV). It was found that small acute pulmonary embolism was related to short axis (RV/LV) under 1.1/1. In all serious acute PE cases, the relation was greater than 1.5/1. Straightening or left movement interventricular septum and contrast reflux in the vena cava inferior (VCI) can be seen, but it is more difficult to quantify. An acute dilation of RV can be a useful sign to evaluate physiology effect PE difficulty, because revealing vasoactive agents can increase pulmonary vascular resistance to a reflexive pulmonary vasoconstriction followed by mechanical obstruction with intravascular clots.[3]

Chemodynamic consequences are the reduction >50% vascular trough leads to pulmonary hypertension and failure right-site heart, and 1% ill patients with acute pulmonary embolism will become chronically ill.[2,15]

An earlier diagnosed heart failure as infarct source is often present, and in our study, it was found in 7 (28%) cases, of which the clot was found in left/right atrium in 2 (8%) cases. A malignant process as infarct source, found in 3 (12%) cases, was not significant in this small serial.

Accompanying effusions develop suddenly and usually are small and unilateral, reaching the maximum length in the first three days. These effusions are often hemorrhagic and connected with inflammatory response, following pulmonary necrosis. High effusion frequency was also confirmed in this investigation. It was found in 21 (84%) patients; in only one patient (4%), pericardial effusion was found. Incremental mediastinal lymph nodes were rare, too, only 1 (4%) case.

Pleural effusion, as it has been reported, is the most frequent in the group with pulmonary embolism, and segment consolidation which morphology easily can represent pulmonary infarct, until mosaic sample and enhanced mediastinal lymph nodes can meet rarely.

Except the mentioned parenchymal and pleural changes, pulmonary thromboembolism can result in haemorrhage without infarct, and on CT, the haemorrhage is viewed as a ground glass opacification, or as an air ways consolidation, not differing from pneumonia or oedema.[13]

Vascular occlusion of small arteries, which supply secondary pulmonary lobules, makes inhomogeneous pulmonary parenchyma attenuation on CT. This is called mosaic oligohemy. These limited regions of changed pulmonary parenchyma attenuation (mosaic sample) are a specific sign of perfusion obstacle and are helpful in diagnosing pulmonary embolism.[3,13]

Our investigation showed the existing ground glass opacification in 15 (60%) cases, and in 3 (12%), mosaic oligohemy. The detection of this sign takes a lot of patience.

When we talk about diagnostic approach, a relatively great number of indefinite investigations stand out, especially in the patients

with chronic pulmonary diseases or parenchyma abnormalities on chest radiography. Chest X-Ray (RTG) is mostly non-specific and normal only in 10%.[2,10]

Although CT is more sensitive for additional signs than RTG, the absence of abnormalities on CT does not exclude PE, because 29% of patients with PE had no pleuropulmonary abnormalities described on CT.[7]

The majority of late PE occurs in the first weeks after treating or excluding PE: 50% of PE have relapse, and 90% of PE are fatal within the first 1-2 weeks. So, an average follow-up of 3 months is acceptable to differentiate the missed PE. A vein fatal PE occurs in 0-0.9%. In CT examinations with the 3mm collimation, the vein thromboembolism frequency (VTE) was 0.5% and fatal PE 0.3%.[3]

The lower extremities investigation on DVT can be used as an alternative method in some patients with adequate cardiopulmonary reserve or low or moderate clinical suspicion on VTE.

Pulmonary vessels analysis is based on different algorithms depending on accessible equipment quality.

As it is mentioned in the introduction part, beside chest x ray in PE diagnostic, other methods are used, such as SCT angiographies (SCTA), which came into the first imaging line in PE studies, followed echocardiography and ventilation/perfusion (VQ) scinthigraphy, pulmonary angiographies (PA), venography and often D-dimmer test.[16]

Normal V/Q scanning excludes PE, and consequently, V/Q scanning diagnoses of PE with possibility over 90%. However, investigations show that 60-70% V/Q scanning are not diagnostic and ask for additional tests. Studies, which compare V/Q scanning and SCTPA, show that, in scintigraphy, the diagnosis has been made in 74% and on CT in 92% samples.[7,8]

It has been published that unsuccessful or indefinite SCTPA rate is between 2 and 13%. This is in contrast to V/Q scanning rate without diagnosis, which vary a lot (30-80%), and in the same range, it is not diagnostic for PA (0-17%).[3]

CT showed itself superior to V/Q scintigraphy in the estimation of embolus maturity.[7]

The duration of investigation in SCT is approximately 10min, and in V/Q scanning 45 min, which can be extremely important in seriously ill patients who need special care and monitoring.

CT has better results than scintigraphy, and in many things, it is equal to angiographies. It is non-invasive, fast, widely accessible, especially in the institutions where pulmonary scintigraphy and angiographies are not accessible, and presents imaging method of choice, if it is carefully composed as a whole diagnostic procedure. When CT is optimal to subsegmental branches, angiographies are not necessary.[3]

Studies reported on 2002 RSNA meeting found a significant decrease in using ventilation-perfusion (V/Q) pulmonary scintigraphy and pulmonary angiographies during the last decade. The usage of CT pulmonary arteriography did not only increase during the same period, but it replaced V/Q scanning as a standard test.[9,12]

Negative SCT can exclude clinically suspected pulmonary embolism as precisely as normal pulmonary scintigraphy or negative pulmonary angiographies.[17]

Pulmonary angiography was a method of choice for a long time, high sensitive and specific (gold standard) for pulmonary embolism detection, such as intraarterial defect of filling or sudden break (totally obstruction) of pulmonary vessels.

It is indicated when the radionucleid scan is indefinite or indirectly possible when the patient is candidate for operation (for embolectomy) or in extremely high risk of using anticoagulants.[2,10]

Angiographies are indicated in case of high PE suspicion, in normal CT and negative

extremity US, and in low-quality CT image of peripheral pulmonary circulation.[3]

In recent studies SCTPA found more subsegmental PE than PA (92 to56%). In this role, SCTPA proved to be better than PA.[7]

Depending on slice thickness, the literature cites different results about SCTPA effect, from 53 to 100%. The comparison of SCTPA with the 1 mm collimation and pulmonary angiographies showed that both techniques were comparative for discovering subsegmental size emboli. Thinner slice has an advantage in CT. The 1mm-thick reconstructive scan allows detection 14 to 40% additional subsegmental PE comparing with 2 and 3mm-thick reconstructive cross sections.[7]

Spiral CT gives possibility for making alternative diagnosis; its findings percentage varies from 11-85%.

Alternative diagnoses in the patients without PE included pneumonia, atelectasis, pneumothorax, pneumomediastinum, pericardial or pleural haemorrhage, aortal dissection, cardiovascular disease, interstitial pulmonary disease, traumatic changes, postoperative changes, abscess, esophagitis, mycosis cork, bronchial infection, chronic obstructive pulmonary disease, bronchopleural fistula, mediastinitis, pulmonary artery hypertension, aspirate pneumonia, septical embolism, diaphragm hernia, oesophageal rupture and malignant tumours. Alternative finding can be met with or without PE.

Other SCTPA advantages are that it is a strategy with the lowest rate of mortality, with the lowest total price per saved life, usually in combination with leg examination by US-Doppler. The chipper approach was using leg US, followed with SCTPA.[7,8]

SCTPA is a routine widely spread 24-hour technique, more accessible than nuclear studies, which is one of the main reasons that SCT is the first choice in diagnosing PE at many institutions.[3]

Researches and technology progress made multislice SCT (MSCT) de facto a gold standard for imaging pulmonary embolism. CT has become accepted as the first-line method for imaging pulmonary circulation in the patients with suspective pulmonary embolism in daily clinical practice.

However CT has not accepted yet universally as gold standard, especially in internists and pulmologists, although these CT critics prefer to send patients regularly on CT than on V/Q.

However, many clinicians do not accept SCTPA as definitive method for excluding PE because of some interpretation mistakes. But, minimum experience and knowledge in interpretation mistakes (technical, anatomical, connected with patients, inadequate parameters of injection, flow rate, concentration and time delay, or insufficient apnoea lasting, what could have result as pseudo defects of loading) are needed.

The best compromise must be found among high longitudinal space resolution and short time apnoea. Breath hold artefacts can result in an inhomogeneous opacification of pulmonary arteries, with hypodense doubling or steaming up of vessel contours. Artefacts beams from contrast in the vena cava superior (VCS) can create defects of pseudo-loading in pulmonary arteries of right upper slice. These artefacts can be reduced with the saline thrust right after the injection of contrast medium, which rinses the contrast into VCS. Anatomical limiting factors and variants should be known for accurate interpretation.

VCS obstruction, cardiomiophaty, focal or global enhancement pulmonary and cardiac resistance, or pulmonary shunts can request longer delay time, until circumferential per vascular oedema or mycosis corks of small bronchi can simulate PE. Changing windows and levels can increase the confidence in interpretation of suspect loading defect, but that also can increase obvious artefacts caused by image noise, solid beam and moving.

An additional follow-up of the patient's

condition is also possible with CT, usually with perfusion pulmonary scans. The changing of perfusion image can be unnoticeable in some patients with central clots. Dissolving and fragmentation of central embolus with peripheral migration can lead to obvious deterioration of perfusion defects with chest pain and missing repeated PE diagnosis. With CT venography, stratifying contrast medium advances from slow blood circulation in varicose veins, proceeds to the obstacles and hardening artefacts, which can be made from bones, orthopaedic material and calcifications, thus averting accurate diagnosis.[6]

Contrast extravasation can appear on the injection place, as undesired reactions to contrast; teophilinum can be given as prophylactic therapy.[3]

Technical problems, which could lead to investigation without conclusion, were met in less than 3% of patients, and usually they are connected with breathing artefacts (hard dyspnoeic patients), bad relation signal-noise, and insufficient opacification of pulmonary arteries, which may occur in 1-10% cases.[7]

CT provides a quantitative estimation of PE effects on tissue perfusion, with an additional direct embolus visualization, which has a significant influence on treatment planning.[9]

As it has been seen in the last years, CT has become a method of choice for central pulmonary embolism (PE) diagnosis to the level of segmental arteries. Multiplanar and 3D reconstruction and colour delineation of perfusion defect, ROI measuring of arterial proceeds for pulmonary blood circulation estimation, 1 mm collimations, axial and 3D SSD image reconstruction and analysis of sub segmental arteries by mediastinal and pulmonary window, contribute those.[13,14,18,19]

On the basis of these experiences, the patients with negative CTPA made with 3 mm collimation, can be without anticoagulant therapy if they are not seriously ill and if they have no limited cardiopulmonary reserve and/or if there is no high clinical suspect on PE.[22]

For those who can't keep breathing is suggested to breath low; however, the survey of segmental and subsegmental emboli is not optimal.[3]

Using monitoring reading became important for pulmonary arteries analysis, using cine-mod scanning, resulted in an enhanced detection PE rate. Monitors are also helpful for Multiplanar reconstruction (MPR) to differentiate infra and extra vascular structures and to improve diagnostic reliability. Double-screen monitors show simultaneously the mediastinal and parenchymal window, thus providing a more precise diagnosis; preventing false-positive may cause respiratory or vascular artefacts. Windows sets also should be adapted according to vascular reinforcement, intending not to miss small emboli. Large number of images is a problem, because it slows down the work. The total number of SCTPA images is in range from 100-200 for single SCT (SSCT) and 500-1000 for MSCT.[7]

With reference to irradiation quantity, it must be said that SCTPA is responsible for high effective radiation dose, larger than the radiation dose in chest X ray and venography.

Today, SCTPA is the most used primary method in suspected PE in Austrian hospitals.[16]

It is a diagnostic procedure of choice in clinical practice with high accordance among different examiners for main, lobar and segmental zones.[11,20]

For exclusion of pulmonary embolism, SCT is used as a transitive step, leaving pulmonary angiographies for cases with no reliable results. The following protocol was suggested: when CT is positive, stop; when it is negative, lower than subsegmental branches, other examinations are not needed.[8]

In the arsenal of diagnostic examinations, except the mentioned ones, echocardiography in bed is obviously appropriate initial di-

agnostic test to evidence the overloading of RV, which is frequently united with massive PE, and to show clots in the heart cavities or central pulmonary arteries or other disorders, as pericardial tamponade, acute valvular disease, infarct of myocardium or aorta dissection. US examination is also priceless with classical flexography for examining the vein system of lower extremities.[7]

MRI has significant role in diagnostic arsenal, too.[23]

Which of these diagnostic procedures will triumph at the end, will be seen, taking into consideration a permanent technological innovation.

Conclusions

MSCT is easily accessible and excellent non-invasive method for the clot visualization in pulmonary artery. As the investigations show, it has become effective at the subsegmental level, and in many respects, surpassed pulmonary angiographies. It provides information about the whole spectrum of findings which can be seen in pulmonary embolism; so far, no other method could provide it. Moreover, it also allows alternative diagnosis. Pulmonary artery embolism is, in most cases, associated with the changes in the pulmonary parenchyma and with indications for heart failure. In this investigation, pulmonary embolism was mostly followed by the findings of pleural effusion, enhanced attenuation ground glass type, pulmonary infarct, contrast enhanced consolidation pulmonary parenchyma, and striped and reticular septal bumps. The right pulmonary artery embolism was found more often than the left branches embolism. It is harder to detect other signs described in literature or they are very rare. An investigation on greater serials with new MSCT devices with 16, 32 and 64 detector lines will probably bring new significant information.

References

1. Duraković Z. Plućna embolija. *Medix* 1998; **4**(17/18): 77-80.
2. Gurney JW, Winwr-Muram HT. Chest.pulmonary embolism. In: Amirsys, editor. *Pocket Radiologist.* Salt Lake City: W.B. Saunders Company; 2003. p. 273-5.
3. Rainer S, Benko D. Spiralna CT preiskava pljučnih arterij v diagnostiki pljučne embolije. *Zdrav Vestn* 2000; **69:** 317-23.
4. Qanadli SD, Hajjam ME, MesurolleB, Barre O, Bruckert F, Joseph T, et al. Pulmonary embolism detection: prospective evaluation of dual-section helical CT versus selective pulmonary arteriography in 157 patients. *Radiology* 2000; **217:** 447-55.
5. Raptopoulos V, Boiselle PM. Multi-detector row spiral CT pulmonary angiography: comparison with single-detector row spiral CT. *Radiology* 2001; **221:** 606-13.
6. Gould P. MRI gains favor for early, accurate DVT diagnosis. *Diagnost Imaging Europe* 2003; **19:** 7-8.
7. Ghaye B, Remy J, Remy-Jardin M. Non-traumatic thoracic emergencies: CT diagnosis of acute pulmnary embolism: the first 10 years. *Eur Radiol* 2002; **12**(8): 1886-905.
8. Marchant J. Spiral CT advances in pulmonary embolism. *Diagnost Imaging Europe.*2000; **2:** 9-10.
9. Abella H. CT gains favor for imaging pulmonary embolism. *Diagnostic Imaging* 2003; **25:** 16-7.
10. Eisenberg RL, Margulis AR. Pulmonary embolism. In: *Radiology pocket reference.* Philadelphia: Lippincott-Raven; 1996. p. 51-2.
11. Thieme T, Kleber FD, Mutze S. Pulmonary embolism. In: *Schering diagnostics.* Ultravist; 2001. p. 12-3.
12. Laurant F. Pulmonary embolism. *Eur Clin Radiol* 2002; **12(Suppl 1):** 58.
13. Greaves SM, Hart EM, Brown K, Young DA, Batra P, Aberle DR. Pulmonary thromboembolism: spectrum of findings on CT. *AJR Am J Roentgenol* 1995; **165:** 1359-63.
14. Schoepf UJ, Bruening R, Konschitzky H, Becker ChR, Knez A, Weber J, et al. Pulmonary embolism: comprehensive diagnosis by using electron-beam CT for detection of emboli and assessment of pulmonary blood flow. *Radiology* 2000; **217:** 693-700.

15. Gurney JW, Winer-Muram HT. Pulmonary hypertension. In: Amirsys, editor. *Pocket radiologist, chest.* Salt Lake City: W.B. Saunders Company; 2003. 276-8.

16. Schibany N, Fleischmann D, Thallinger C, Schibany A, Hahne J, Ba-Ssalamah A, et al. Equipment availability and diagnostic strategies for suspected pulmonary embolism in Austria. *Eur Radiol* 2001; **11:** 2287-94.

17. Gottsater A, Berg A, Centergard J, Frennby B, Nirhov N, Nyman U. Clinically suspected pulmonary embolism: it is safe to withhold anticoagulation after a negative spiral CT? *Eur Radiol* 2001; **11:** 65-72.

18. Coche E, Pawlak S, Dechambre S, Maldague B. Peripheral pulmonary arteries: identification at multi-slice spiral CT with 3D reconstruction. *Eur Radiol* 2003; **13:** 815-22.

19. Schoepf UJ, Kessler MA, Rieger CT, Herzog P, Klotz E, Wiesgigl S, et al. Multislice CT imaging of pulmonary embolism. *Eur Radiol* 2001; **11:** 2278-86.

20. Ruiz Y, Caballero P, Caniego JL, Friera A, Olivera MJ, Tagarro D, et al. Prospective comparison of helical CT with angiography in pulmonary embolism: global and selective vascular territory analysis. Interobserver agreement. *Eur Radiol* 2003; **13:** 823-9.

21. Schoepf UJ, Holzknecht N, Helmberger TK, Crispin A, Hong C, Becker CR, et al. Subsegmental pulmonary emboli: improved detection with thin-collimation multi-detector row spiral CT. *Radiology* 2002; **222:** 483-90.

22. Nilsson T, Olausson A, JohnssonH, Nyman U, Aspelin A. Negative spiral CT in acute pulmonary embolism. *Acta Radiologica* 2002; **43:** 486-91.

23. Coche EE, Hammer FD, Goffette PP. Demonstration of pulmonary embolism with gadolinium-enhanced spiral CT. *Eur Radiol* 2001; **11:** 2306-9.