

ACUTE PULMONARY EMBOLISM

The incidence of pulmonary embolism (PE) in Sweden is 20-60/100 000 and causes approximately 1000 deaths/year. The probability for PE increases for example with a history of previous deep vein thrombosis, malignancy or immobilisation. 95% of the patients with PE have dyspnoea, pleuritic pain/haemoptysis or right ventricular failure with or without chock. Chest X-ray can sometimes give the suspicion of PE but never confirm or exclude it. The workup of patients with suspicion of PE depends on the probability for PE. Wells score can be used to determine the pretest probability of PE. MDCTPA is the first-line test in patients with a positive d-dimer or high pretest probability of PE.

MDCTPA offers direct visualization of emboli, both parenchymal and mediastinal structures can be evaluated and in 2/3 of cases with a negative scan it offers differential diagnosis. Apart from visualization of emboli, CT can detect signs of right ventricular (RV) dysfunction which increases the risk of death. RV dilates and the interventricular septum deviates to the left. With increase of pressure in the right atrium there is a risk of paradoxical embolism if the patient has a patent foramen ovale.

ACUTE AORTIC SYNDROME

The term acute aortic syndrome (AAS) is applied to the combination of chest pain and hypertension and can be the result of aortic dissection (AD), intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU) or aneurysm leak or rupture.

There has been an increase in the incidence of thoracic aortic disease in Sweden between 1987-2002 but the mortality has decreased.

MDCT is the diagnostic method of choice. The diagnostic goals are:

- Describe localisation according to the Stanford classification.
- Assess involvement of branches
- Detect thoracic complications: pericardial and pleural effusion
- Differentiate true and false lumen
- Detect aneurysm rupture or impending rupture

Typical aortic dissection is produced by an intimal tear that allows blood to enter the medial layer, giving rise to two lumina, one true and one false. Although hypertension is the most frequent predisposing factor other conditions like Marfan's syndrome, Turner syndrome, other connective tissue diseases, aortic coarctation and pregnancy are associated with dissection. Aortic dissection is the most common catastrophe of the aorta, 2-3 times more common than rupture of the abdominal aorta.

The most common site of dissection is the first few centimetres of the ascending aorta, with 90% occurring within 10 centimetres of the aortic valve. The second most common site is just distal to the left subclavian artery.

8-30% of AAS are IMH. The most common recognized risk factors for aortic IMH include hypertension, advanced age, and atherosclerosis (the latter especially in cases of PAU). IMH is usually a disease of the elderly. IMH is caused by rupture of the vasa vasorum in the media without an intimal tear. It is recognized on an unenhanced CT scan as a crescent of high attenuation in the aortic wall. IMH in the ascending aorta has the same severity as acute dissection. The risk increases with the diameter of the aorta and the thickness of the hematoma.

Acute aortic symptomatic penetrating atherosclerotic ulcer has a risk equal to or higher than that of AD or IMH. Penetrating ulcer is normally diagnosed in patients over 60 years

old with atherosclerosis in other areas and related cardiovascular risk factors. Like intramural hematoma, it is located much more frequently in the descending aorta. The aortic diameter varies with age and gender. Maximum diameter in ascending aorta is 4 cm, descending aorta 3 cm and in abdominal aorta 2 cm. A true aneurysm contains all three wall-layers. The wall consists of mostly the adventitia and parts of the media in a pseudo aneurysm. Potential sites of postoperative ascending aortic pseudo aneurysms are clamping site, cannulation site, graft, graft anastomosis, needle puncture site and valvulotomy site. Aneurysms can cause rupture, dissection, aortic regurgitation or compression of adjacent structures. Signs of rupture or impending rupture are Haemorrhage (mediastinal or retroperitoneal), high attenuation crescent of blood within a mural thrombus or the "draped aorta sign".