SUBACUTE INTRAMURAL HEMATOMA OF THE AORTA

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Key-word: Hemorrhage

Background: An 82-year-old male was admitted to the emergency department with nausea and severe peri-umbilical and lumbar pain. On physical examination the right peri-umbilical region and right iliac fossa were tender. There was an increased blood pressure of 232/91 mm Hg.

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Work-up

On unenhanced CT scan at the level of the abdominal aorta (Fig. 1), hyperdense crescentic thickening along the aortic wall with inward displacement of calcifications is seen.

The aortic wall was normal on CT scan of the lumbar spine 2 months prior (not shown).

Contrast-enhanced CT scan (Fig. 2) shows on A (axial slice at the level of Fig. 1) typical crescentic density along the aortic wall. Curved MPR image along the aorta (B) confirms the hyperdense crescentic thickening along the aortic wall (arrow). On axial image at the level of the renal arteries (C), the ostium of the left renal artery is seen, but no enhancement is observed in the renal artery and kidney.

MRI at the level of the abdominal aorta (Fig. 3) shows on A (axial T1-weighted image) hyperintense crescent along the aortic wall, and on B (axial T2-weighted image) the crescent along the aortic wall has an intermediate density.

Radiological diagnosis

Based on the CT and MRI findings the diagnosis of a _subacute intramural hematoma of the aorta_ was made. It is our hypothesis that the aortic intramural hematoma was due to hypertension secondary to an embolus in the left renal artery.

Discussion

Intramural hematoma (IMH) is a relatively rare but potentially fatal entity.

Rupture of the vasa vasorum into the media of the aortic wall is the most cited mechanism. The evidence for this theory is thin. IMH cannot be clinically distinguished from aortic dissection. Arterial hypertension is the most frequent predisposing factor for IMH, followed by Marfan and Turner’s syndrome and is associated with blunt or iatrogenic trauma.

In contrast to aortic dissection there is no intimal tear flap visible on imaging.

IMH presents more commonly in the descending aorta (50-80%) and is more frequently found in the elderly. There is an equal male-to-female ratio. IMH can progress to aortic dissection, saccular or fusiform aneurysm or rupture.

The clinical presentation is usually severe back pain. Since intramural hematomas are generally found more superficially (nearer to the adventitia), rupture is more common in IMH than in aortic dissection and occurs in up to 35% of the cases. Patients with uncomplicated IMH are generally treated like patients with uncomplicated aortic dissection in the same aortic location. The Stanford system, classifies dissections that involve the ascending aorta as type A, regardless of the site of the primary intimal tear, and all other dissections as type B. Patients with type A (40%) IMH should undergo immediate surgery and those with type B (60%) IMH may receive initial medical management.

On unenhanced axial CT-images, a crescentic eccentric hyperattenuating region of thickening of the aortic wall is considered pathognomonic of acute intramural hematoma. Intimal calcifications may be displaced inward and are best seen on images with narrow window setting.

On contrast-enhanced axial CT-images the intramural fluid collection appears as a circumferential or crescentic low density along the aortic wall. No intimal flap is seen. The absence of contrast enhancement within the aortic wall helps to differentiate IMH from classic aortic dissection, penetrating aortic ulcers and other aortic wall pathologies.

On MRI, as on CT, IMH presents as a focal, crescent-shaped wall thickening with smooth luminal surface.

The advantage of MRI over CT is the ability to discriminate between acute (< 7 days) and subacute (≥ 7 days) IMH. On gradient-echo T2-weighted images, an acute intramural hematoma is hyperintense, whereas a subacute or chronic intramural hematoma has an intermediate signal intensity on these sequences. On T1-weighted spin-echo images, an acute intramural hematoma appears isointense because of the presence of oxyhemoglobin; however, as the hematoma ages, it becomes hyperintense on T1-weighted images, due to the formation of methemoglobin.

Bibliography