Case Report: A Patient with Acute Cardiac Tamponade Secondary to Complicated Stanford Type A Intramural Haematoma - the role of CT

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SUMMARY

Chest pain is a common presenting complaint in the emergency room of which acute aortic syndrome is a sinister cause associated with high morbidity. A contrast-enhanced CT aortogram is often performed for initial evaluation at the first instance of suspicion. We present a patient with Stanford Type A intramural haematoma complicated by haemopericardium and acute cardiac tamponade and highlight the relevant CT signs that would alert the managing physician to urgent echocardiogram correlation and emergent cardiothoracic intervention.

KEY WORDS:
Cardiac tamponade; aortic intramural haematoma

INTRODUCTION

Acute cardiac tamponade is a known complication of acute aortic intramural haematoma (IMH) and dissection, which come under a spectrum of interrelated conditions known as acute aortic syndrome. In acute IMH, weakening of the aortic wall from spontaneous haemorrhage into the aortic media not uncommonly leads to outward aortic rupture or inward intimal dissection extending to the aortic root, with subsequent progression to haemopericardium and acute cardiac tamponade.

The recognition of the CT signs of acute cardiac tamponade on CT is of paramount importance as early cardiothoracic intervention directly relates to reduced morbidity.

CASE REPORT

An 82-year old female with known history of hypertension was brought to our hospital because of sudden severe chest pain. At the emergency room, she was diaphoretic, tachypneic and hypotensive with a documented blood pressure of 90/70 mm Hg. Physical examination was inconclusive apart from vital signs indicating significant haemodynamic compromise as the patient was in discomfort and was uncooperative. An electrocardiogram did not reveal any ST abnormality. An initial bedside ultrasound performed by the emergency physician did not reveal any pericardial effusion. Chest radiograph showed widened mediastinum that was stable from prior studies. A contrast-enhanced multidetector CT aortogram was performed using 2.5 mm slice thickness reconstructed at 1 mm after administration of 100 ml of nonionic contrast medium (Omnipaque 350) to rule out an aortic dissection.

In the contrast-enhanced arterial phase of the CT aortogram, there is revelation of near- circumferential hypoattenuating intramural lesion extending from the aortic root to the arch proximal to the left subclavian artery consistent with a Stanford Type A IMH.

Moderate pericardial effusion of high attenuation (average HU 60) suspicious for haemopericardium was visualised. In addition, concave flattening of the anterior surface of the heart and bowing of the interventricular septum towards the left ventricle are noted, compatible with tamponade effect (Fig. 1).

Retrograde opacification of the azygos vein with contrast refluxing into the superior vena cava (SVC) was also observed (Fig. 2), indicating significant haemodynamic disturbance. Both the axial and sagittal reconstructed contrast-enhanced CT images in the arterial phase clearly depict the IVC contrast-level sign in the hepatic IVC (Fig. 3). Overall imaging features are in keeping with acute cardiac tamponade secondary to the haemopericardium.

An urgent cardiothoracic consultation was undertaken at the emergency room and a transthoracic echocardiogram (TTE) confirmed an aneurysmal thoracic aorta with IMH. Interestingly, a dissection flap within the proximal aorta, not appreciated on the CT study, was found on the TTE extending to the aortic root. The moderate pericardial effusion demonstrated internal echoes consistent with blood products, measuring up to 3.0 cm with right ventricle (RV) and right atrium (RA) collapse and respiratory variation compatible with tamponade.

An immediate family conference was held and the ominous findings were discussed with the patient and her family. The offer of open surgical repair with risks of death and morbidity as well as the alternative option of medical management was provided. Eventual decision was made for non-surgical intervention in view of the high surgical risk and blood pressure control was initiated once the patient was admitted to the cardiothoracic unit.

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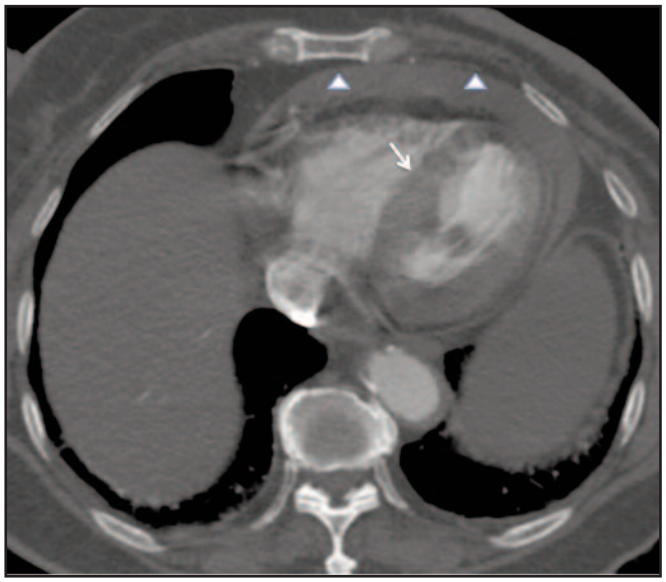

Fig. 1 : Contrast enhanced axial CT image in arterial phase demonstrated moderate pericardial effusion (triangle) with mass effect causing concave flattening of the anterior heart surface and bowing of the interventricular septum (white arrow) towards the left ventricle. Poorly distended left ventricle was seen.

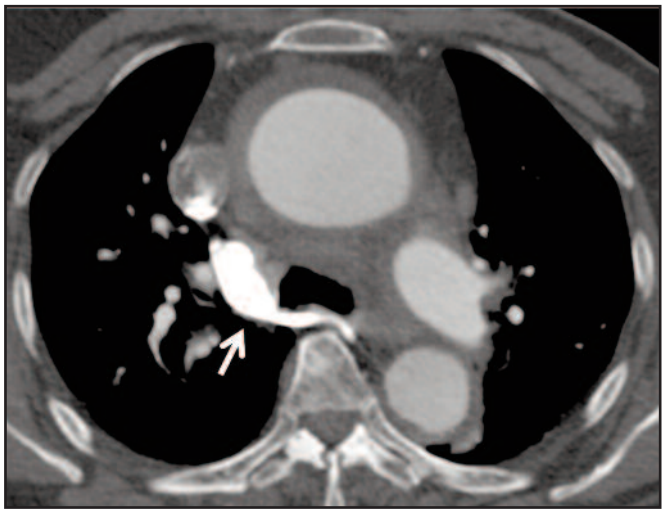

Fig. 2 : Contrast enhanced axial CT image in arterial phase shows reflux of contrast within the azygos vein. Small amount of contrast material was also noted in the SVC. The ascending aorta appears aneurysmal, with hypoattenuating intramural lesion representing acute IMH.

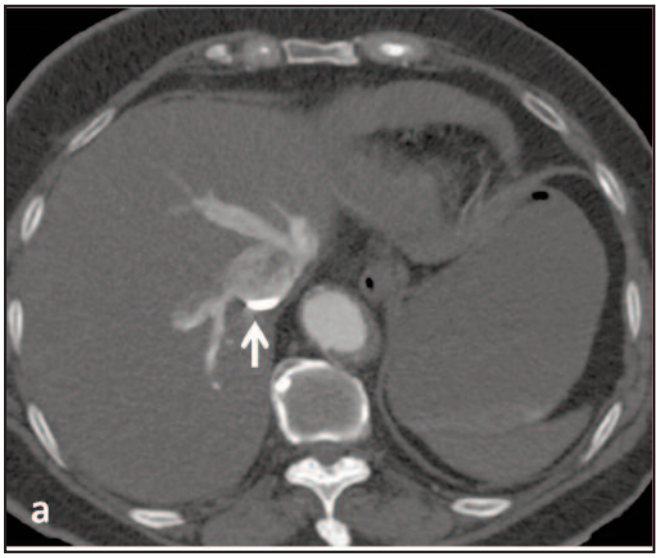

Fig. 3a : Contrast enhanced axial CT image in arterial phase showed retrograde opacification/reflux of contrast within the hepatic IVC and within the hepatic veins, which appear distended. An IVC contrast-fluid level (white arrow) is seen.

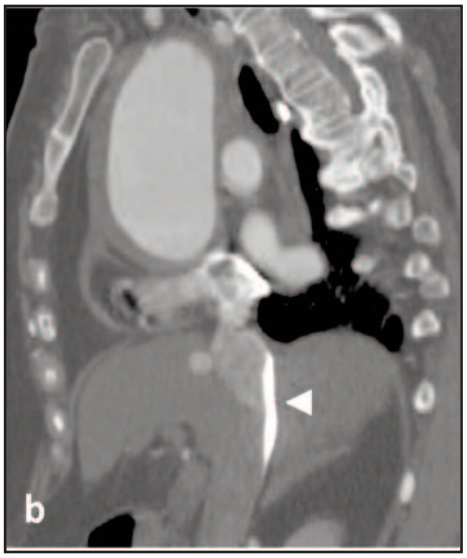

Fig. 3b : Reconstructed sagittal image showed dependent contrast-fluid level within the hepatic IVC (arrowhead). Aneurysmal ascending aorta with hypoattenuating intramural IMH is observed.

Unfortunately, the patient expired from cardio-respiratory arrest 36 hours later.

DISCUSSION

In a meta-analysis reviewing echocardiography, helical CT and MRI in the imaging evaluation of acute aortic syndrome, echocardiography has a reported sensitivity and specificity of 100%, whereas conventional CT is less accurate, having a sensitivity of 83-94% and a specificity of 87-100%[1]. Transesophageal echocardiography is advantageous with regards to the amount of time needed to reach the diagnosis and in the assessment of aortic valve insufficiency associated with aortic dissection. However, suboptimal assessment of the distal ascending aorta and branches of the aortic arch, as well as operator dependence are known limitations. CT hence remains the most widely used imaging technique.

In the diagnostic algorithm of acute aortic syndrome, CT angiogram plays an important role in the confirmation of clinical suspicion, classification of dissection, characterising the extent as well as detection of complications.

Although diagnosis of cardiac tamponade is often clinical with equivocal findings evaluated with echocardiography as the technique of choice, it is often not feasible due to patient habitus or often not performed due to low clinical suspicion from masked symptoms/signs. CT is advantageous in the setting of pericardial effusion over echocardiography with its larger field of view, which offers concurrent assessment of the entire thorax and does not suffer from operator dependence or false-positive findings on echocardiography related to pathological conditions such as pleural effusion or mediastinal lesions that simulate pericardial effusions.

In the context of acute aortic syndrome, the first diagnosis of a cardiac tamponade is often made on a contrast-enhanced CT done at the emergency department. Further in our case, the initial bedside ultrasound has failed to detect a pericardial effusion.

The CT signs of cardiac tamponade can be categorised to that related to direct mass effect from the pericardial effusion and that related to acute haemodynamic change of inter-chamber cardiac pressures with effects on contrast opacification.

Mass effect from the pericardial effusion translates to concave deformity of the anterior heart surface and a reduced anteroposterior diameter. Straightening or bowing of the interventricular septum towards the left ventricle may occur[2]. In severe cases, the cardiac chambers may distend poorly giving a small heart appearance.

Contrast reflux on CT has been shown as a good indicator of cardiac chamber equalization in cardiac tamponade and allows estimation of right atrial (RA) and ventricular (RV) response with findings correlating well with echocardiographic estimations. Reflux of contrast into the azygos veins, IVC and hepatic veins have been shown to be indicative of significant right heart systolic dysfunction, with the phenomenon presumably resulting from equalisation of cardiac chamber pressures due to the tamponade effect[3,4].

Sueyoshi *et al*[5] has retrospectively reviewed and reported a contrast-fluid level sign in the hepatic IVC during an arterial phase CT as a good indicator to differentiate acute cardiac tamponade in acute Stanford type A aortic dissection from patients with chronic pericardial effusions. This sign was also demonstrated in our patient.

As a side note, we observed that the CT had not demonstrate the dissection flap that was appreciated on the TTE which would have classified the case more accurately as an acute Stanford Type A aortic dissection. There was also no demonstration of a contrast-filled false lumen on our CT. We believe that this corresponds to the debate in the literature[6] regarding use of the (more widely accepted) terminology of IMH, versus a thrombosed-type of acute aortic dissection which results from an intimal tear without re-entry and which the false lumen rapidly thromboses off and hence fail to opacify on a contrast enhanced CT, as in our case. This is however beyond the scope of discussion of this case report.

CONCLUSION

This case emphasizes the role of CT in the diagnosis of acute cardiac tamponade in complicated proximal acute aortic syndrome and highlights the CT signs associated with this life-threatening condition.

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