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Case Report

Iatrogenic type A aortic dissection complicating percutaneous coronary intervention: a case report

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ABSTRACT

Iatrogenic type A dissection caused by percutaneous coronary intervention is a rare but life-threatening condition. Computed tomographic angiography is an excellent diagnostic tool commonly utilized if such procedural complication is clinically suspected. There are, however, potential diagnostic challenges. Herein, we present an illustrative case of iatrogenic type A dissection successfully diagnosed on computed tomography angiography along with a potential diagnostic pitfall.

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Presentation

A 58-year-old male with remote history of coronary artery disease, distal right coronary artery angioplasty, and stenting, presented in April 2016 with acute onset shortness of breath. Sequential troponin levels and clinical examination is consistent with non-ST (period between the S wave and the T wave of the cardiac cycle) elevation of myocardial infarction. Echocardiography (ECG) demonstrated inferolateral wall hypokinesis with left ventricular ejection fraction >60%. Cardiac perfusion study confirmed reversible ischemia of the inferolateral wall. The patient subsequently underwent percutaneous coronary angiography using a right femoral approach, which demonstrated right dominant coronary anatomy, with occlusion of the mid segment of the right

coronary artery (RCA) as well as occlusion of the in situ stent. There was 50% stenosis of the mid left anterior descending artery and 90% stenosis of the distal circumflex artery. The patient underwent stenting of the circumflex artery with planned staged recanalization of the RCA on an elective basis.

The patient underwent elective recanalization of the chronically occluded RCA in August 2016. The PCI was performed using femoral approach. Rotablation was not performed. Intraoperatively, a dissection of the right coronary sinus was suspected after recanalization of the RCA, due to new accumulation of contrast material at the coronary cusp after contrast injection. Although the exact etiology of the dissection cannot be determined, it is favored to be related to catheter trauma. The interventional cardiologist placed a drug eluting stent at the ostium of the RCA. Contrast-enhanced

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computed tomography (CT) of the chest was requested immediately after the percutaneous coronary intervention (PCI) for further assessment.

The suspected dissection was confirmed on CT angiography. In consultation with cardiac surgery, the patient was medically managed and was closely observed in hospital. A follow-up CT angiography was performed 2 days after the PCI, which showed no ongoing dissection and complete resolution of extraluminal contrast. The patient was discharged on the same day.

Imaging technique

The patient underwent nongated unenhanced CT chest and subsequently ECG-gated CT angiography of the thoracic aorta. The same protocol was used for the index study and the follow-up study.

Findings

On the index unenhanced CT and CT angiography immediately following the PCI, there was an 8-mm thick plaque-like hyperattenuating lesion at the right coronary sinus extending just above the sinotubular junction (Figs 1A and B). This lesion mimicked a calcified atherosclerotic plaque on both the unenhanced and the contrast-enhanced images. The attenuation coefficient of the material was approximately 2500 Hounsfield units on the unenhanced study and did not change following contrast administration. In mediastinal window, the lesion was indistinguishable from surrounding enhanced aorta (Fig. 1C). The extent of the dissection was best demonstrated on the maximum intensity projection (Fig. 1D).

The follow-up study 2 days later showed no residual extraluminal contrast, and no ongoing dissection (Figs 2A and B).

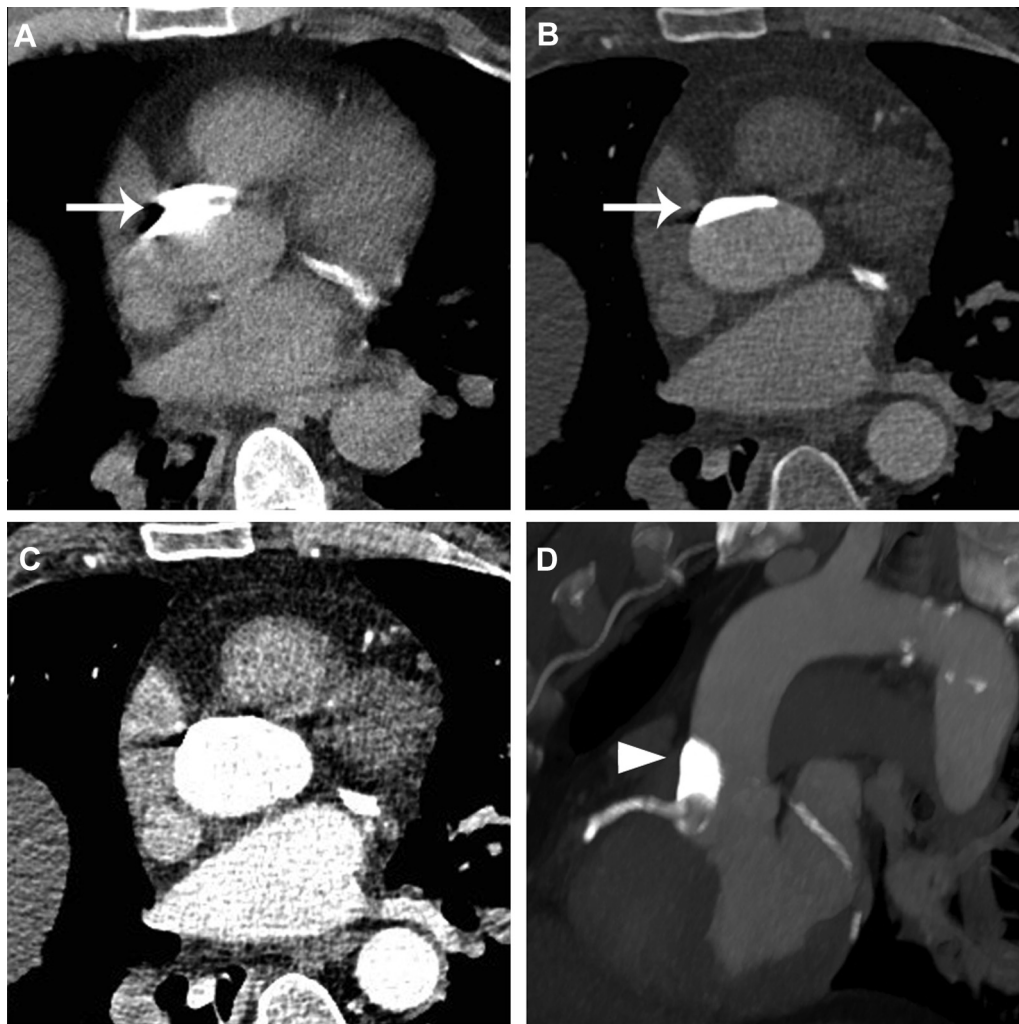


Fig. 1 – Postprocedure CT. (A) Unenhanced, nongated and (B) enhanced, electrocardiography-gated thoracic CT immediately after coronary angiography and stenting. The plaque-like density (arrows) at the right coronary sinus measured 2500 Hounsfield units and did not change in attenuation after contrast enhancement, consistent with pooled extraluminal contrast from the preceding coronary angiography. (C) In mediastinal window, the contrast pool was indistinguishable from surrounding enhanced aorta. (D) The extent of the dissection at the right coronary sinus was well demonstrated on double obliques maximum-intensity projection (arrowhead).

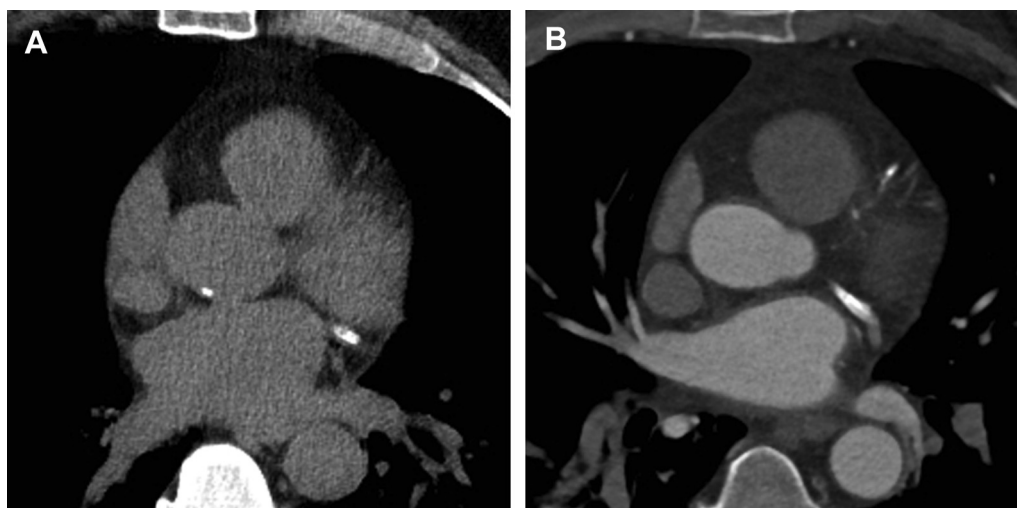


Fig. 2 – Follow up CT. (A) On postprocedure day 2, the follow-up unenhanced thoracic CT study demonstrated resolution of the extraluminal pooled contrast. (B) The follow-up enhanced study did not demonstrate any ongoing dissection.

Discussion

In a large retrospective review of 43,143 cases, there were only 9 cases of iatrogenic aortic dissection after diagnostic coronary angiography and PCI, or an overall incidence of 0.02% [1]. This confirmed earlier large retrospective reviews of 21,000 patients by Vega [2], which also showed the incidence to be about 0.02%. The incidence of aortocoronary dissection after PCI for chronic total occlusion is higher; however, this entity remains exceedingly rare [3]. The increased incidence of dissection after treatment of chronic total occlusion is thought to be due to more aggressive techniques and dedicated equipments, which result in greater chances of intimal injury [3]. Iatrogenic dissection predominantly involves the ostium of the RCA and aortic root and may extend variably in an antegrade and retrograde fashion [3]. The most common treatment strategy has been aortoostial stenting to occlude the dissection flap, although surgical repair have also been used [2,3].

This was an illustrative case of iatrogenic aortic dissection after PCI. Based on the criteria proposed by Dunning et al [1], this was a type 2 dissection. In our case, the extraluminal contrast material mimicked aortic calcification, which could have potentially led to a misdiagnosis. For patients with coronary disease, calcified aortic atherosclerotic plaque is common. Calcification and contrast are indistinguishable on standard soft tissue or mediastinal windows. Therefore, higher windowing level is required for accurate diagnosis. ECG gating is very helpful to improve visualization of the aortic root and diagnostic confidence. An unenhanced examination is required to differentiate between enhancement and pre-existing extraluminal contrast or surgical material. In our experience, ECG gating for the unenhanced examination is ideal, although not required. Furthermore, the attenuation of the extraluminal material was 2500 Hounsfield units,

significantly higher than that expected for vascular calcifications and, thus, should raise concern for being pooled contrast. In this case, the density of extraluminal material did not change significantly after infusion of intravenous contrast, as the contrast was sequestered within the dissected aortic wall, and the dissection defect was occluded by the aortoostial stent. In cases where there is alteration in the attenuation values of the false lumen after intravenous contrast administration, ongoing communication between the true and false lumen should be suspected. In our case, review of preprocedural PCI images provided further evidence as to the lack of any significant calcified plaque at the location of the right coronary cusp, thus, raising our level of diagnostic confidence for an iatrogenic aortic dissection. This case further highlights the importance of review of the PCI images for correlation and consideration of iatrogenic dissection.

Iatrogenic coronary artery and aortic root dissection is a rare but serious complication of coronary angiography, and radiologists should maintain a high index of suspicion in these patients.

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