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A late presentation of acute iatrogenic aortic dissection following percutaneous coronary intervention: A case report

PGN Danushka, R Jayasinghe and M Munsinghe

Abstract

Acute iatrogenic aortic dissection following percutaneous coronary intervention is a rare but sinister cause of post-procedure morbidity and mortality. Delayed diagnosis increases the mortality significantly. We present a case of a 52-year-old male who presented with an iatrogenic aortic dissection following percutaneous coronary intervention for an inferior ST-elevation myocardial infarction. Although the diagnosis was initially missed by conventional imaging for a period of 5 months, it was later diagnosed assisted by cardiac computed tomography. The patient underwent aortic repair with a Dacron graft and had an uneventful recovery. The case highlights the importance of early diagnosis and selection of appropriate imaging for this rare but serious iatrogenic sequel following percutaneous coronary intervention to minimize morbidity.

Keywords

Case report, aortic dissection after PCI, PCI complications, late presentation of aortic dissection

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Background

Aortic dissection (AD) is defined as an aortic intimal tear creating high-pressure flow in the true lumen with seepage into the false lumen. Iatrogenic aortic dissection (IAD) is a rare but sinister sequel following percutaneous coronary intervention (PCI). In both diagnostic and therapeutic procedures, the incidence of catheter-induced AD is as low as 0.02%–0.07%.[1] Although rare, it is a known complication following procedures such as open cardiac surgery, coronary interventions, and complex PCI.[2] The dissection can propagate to the side branches. Complications such as cardiac tamponade (<20%), aortic regurgitation (40%–75%), proximal (10–15%), or distal malperfusion (5–20%) can occur following an AD.[3–7] Depending on the duration following the onset of symptoms, AD is subdivided into acute (< 2 weeks), subacute (2 weeks months), and chronic phases (>3 months).[7] Surgery is the primary mode of treatment, although it carries 25% of perioperative mortality and 18% neurological complications.[7–9]

Case presentation

We present a case of a 52-year-old male, a flight attendant who was apparently well, developed an acute onset, tightening chest pain with associated autonomic symptoms, and dizziness during his work abroad. He was admitted to an emergency treatment unit. Following completion of the emergency medical management, he left against medical advice and flew back to his motherland, the very next day for further medical care. On the fourth day of his illness, he sought medical advice, and he was found to have an inferior ST-elevation myocardial infarction in the electrocardiogram. Troponin I was also elevated to 6.4 ng/ml. He was admitted and treated for the acute event and discharged 4 days later. During the same admission, he was diagnosed with hypertension and was started on an antihypertensive. A 2D echo was performed which showed preserved left ventricular function with an ejection fraction of 60% along with an inferior hypokinetic segment. However, there were no aortic intimal flaps, pericardial effusion, or cardiac tamponade.

Two weeks later he was scheduled for a percutaneous coronary angiogram. The coronary angiogram was attempted through the right femoral artery, failing which the right radial artery was cannulated. The angiogram was performed using

Department of Surgery, National Hospital of Sri Lanka, Colombo, Sri Lanka

Corresponding Author:
PGN Danushka, Department of Surgery, National Hospital of Sri Lanka, Colombo, Western Province 8, Sri Lanka.
Email: danupiti@gmail.com

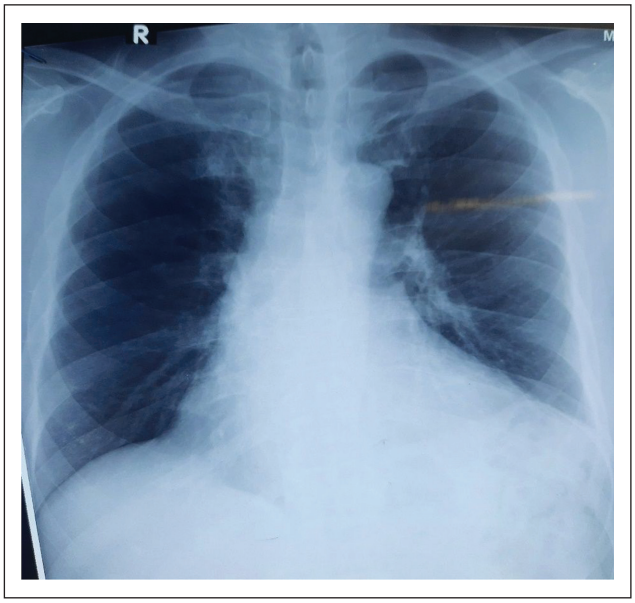

Figure 1. Chest X-ray showing widened mediastinum.

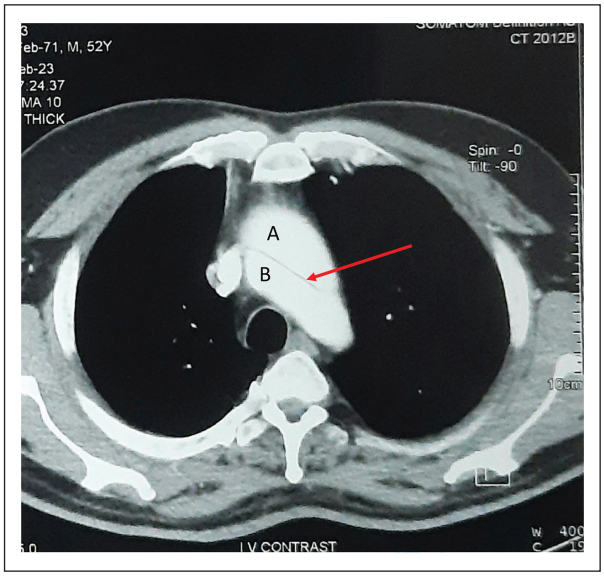

Figure 2. Contrast-enhanced CT showing aortic dissection.
A: false lumen; B: true lumen; red arrow: the intimal flap; CT: computed tomography.

an Amplatz guiding catheter with moderate difficulty. It revealed severe triple vessel disease with 70% proximal plaque in the left anterior descending (LAD) artery, mild plaque disease in the first diagonal artery, 90% mid-vessel plaque in the obtuse marginal (OM) artery, the 70% plaque mid-vessel in the right coronary artery (RCA), and 90% disease in the proximal and mid-vessel of the posterior left ventricular (PLV) artery.

A week later, he underwent percutaneous coronary angioplasty. Size 0.014″ guidewire and 6F guide catheter was used to place the stents in LAD artery (3.5 mm × 33 mm), OM artery (3.5 mm × 18 mm), mid-RCA (2.5 mm × 48 mm) and PLV artery (2.5 mm × 48 mm). After the procedure, Thrombolysis in myocardial infarction III antegrade flow was observed in all the arteries.

Following the procedure, the patient was completely asymptomatic for 3 days, after which he started to feel chest discomfort and excessive tiredness. Since the symptoms were vague, he was treated with analgesics. However, the chest discomfort was progressively worsening over the 4 weeks and he had occasional dizziness with New York heart association class 3 exertional dyspnea. After 4 weeks, a 2D echocardiogram was carried out which showed a large pericardial effusion, apical 2.6 cm, lateral 2.8 cm, and posterior 2.9 cm in size. The ventricular function was normal with no right atrial collapse.

A pericardiocentesis was done 4 weeks later, confirming a hemopericardium, following which the patient's symptoms improved. A repeat echocardiogram was done 2 weeks later which showed a mild residual accumulation of fluid. Despite pericardial aspiration, the patient developed worsening symptoms again.

A chest X-ray was performed which showed a dilated cardiac shadow and widened aortic root (Figure 1). The patient underwent a cardiac computed tomography (CT) on the same day, which revealed an AD in the ascending aorta extending to the aortic arch. The true and false lumens were perfused with neck vessels supplied by the true lumen. Dissection was starting from the infundibulum and was extending up to the end of the arch. This was accompanied by a mild to moderate pericardial effusion.

The patient underwent urgent repair of the AD. Intraoperatively, a hemopericardium of around 80 ml, and a dilated ascending aorta just above its root was revealed. Aortic cross-clamping was done, a hypothermic circulatory arrest was induced at 18 °C, the ascending aorta was opened, and the true lumen and false lumen were identified (Figure 2). Cardioplegia was administered through the coronary ostia. The diseased part of the aorta was resected and repaired with a 26-mm Dacron aortic graft (Figure 3). The patient was weaned from the cardiopulmonary bypass and his postoperative course was uneventful.

Discussion

Recent advances in PCI help manage patients without opting for major surgery, thus improving morbidity.[10] IAD is a known, rare, and fatal complication following complicated cardiovascular lesions and in both diagnostic and therapeutic interventions.[11] During PCI, an IAD can be caused as the catheter is pushed into the aortic wall during the insertion of the guiding catheter. Most commonly, injuries occur at ascending aorta at the ostium of the RCA and can propagate upward. Extension of the aortic intimal flap toward aortic valve may

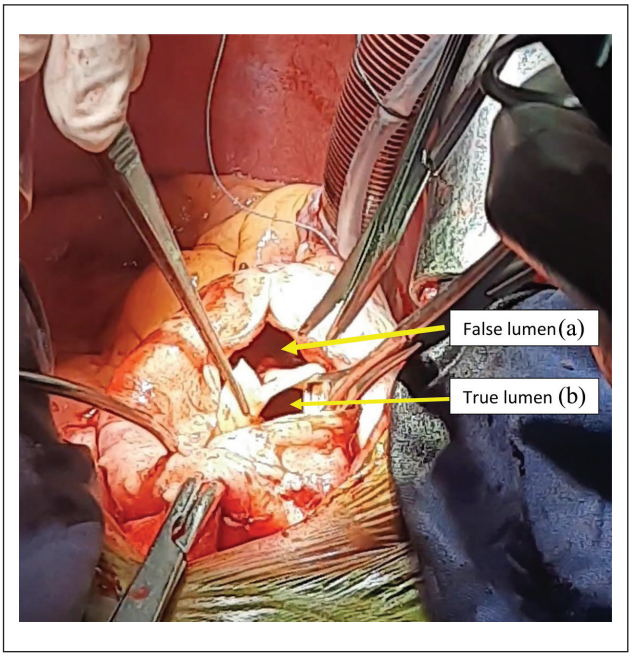

Figure 3. Intraoperative picture depicts the false lumen (a) and the true lumen (b). A clot is inside the false lumen (arrowhead).

lead to aortic regurgitation and haemopericardium.[7] In the index case, the patient developed a hemopericardium. However, the diagnosis was late, and aspiration only took place 3 months after the onset of symptoms. Most published literature reported IAD as an early diagnosis during the coronary angiogram, characterized by the stagnation of contrast medium at the level of the aortic root.[2,7,11,12] In the present case, it was not apparent during the coronary angiogram, and the patient was also asymptomatic during the immediate post-op period. The clinical presentation of AD can range from asymptomatic to extreme back, chest pain.[7]

Cardiac CT is a useful noninvasive imaging modality for the diagnosis and follow-up of the dissection flap which was the investigation of choice, assisting the diagnosis in the index case.[13] In patients managed medically, CT allows visualization of the complete loss of dissection flap.[11]

The current guideline suggests surgery for spontaneous type A AD, although it suggests medical management for IAD.[7] A study conducted among 48 patients who had IAD after a cardiac procedure, observed a 50% perioperative mortality rate following IAD after cardiac catheterization.[14] Moreover, a multicenter retrospective study conducted among 74 patients found that conservative management would result in a better prognosis (>97% survival). Hence the decision to treat medically or surgically depends on the presence of coronary flow limitation, the progression of the dissection flap toward the aortic arch, and the hemodynamic stability of the patient.[11] In contrast, Hung et al. suggested passing the guide wire to the true lumen to maintain the luminal patency of the vessel sealing the dissection flap by stenting the coronary artery up to its origin. Hence, they recommend this approach for patients with Dunning class 3 (Dissection flap extends >40 mm in the ascending aorta).[1,15] An urgent echocardiogram can be used to assess for pericardial effusion, or acute aortic regurgitation which favors surgical management. Undisturbed coronary flow, limited dissection flap, and a stable patient can be managed conservatively with follow-up imaging. In the index case, as the CT showed involvement of the aortic arch, operative measures were undertaken.

Conclusions

IAD is a sinister but rare complication, following PCI. In suspicion, dissection can be identified during the same procedure and stenting can be attempted. Deterioration of the patient's clinical status, pericardial effusion, and acute aortic regurgitation warrants a cardiac surgical input.

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None.

Authors' contributions

N.D. and R.J. contributed to concept and design of study, acquisition of data, analysis, interpretation of data, drafting the article, and final approval of the version to be published. N.D., M.M., and R.J. contributed to concept and design of study, revising it critically for important intellectual content, and final approval of the version to be published.

Data availability

Upon reasonable request from the corresponding author.

Declaration of conflicting interests

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

Consent for publication

Informed written consent was obtained from the patient for publication of the case and related images.

ORCID iD

P G N Danushka https://orcid.org/0000-0001-6068-6081

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