

Managed successfully: Iatrogenic aortic dissection during primary percutaneous coronary intervention

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Abstract

Iatrogenic aortic dissection caused by primary percutaneous coronary intervention (PCI) is a rare but potentially fatal complication; therefore prompt recognition of this life-threatening condition is crucial. We present herein a case of a 70-year-old lady who underwent primary PCI for transmural myocardial infarction of left anterior descending artery territory. Manipulation of the extra backup (EBU) guiding catheter during an attempt to cannulate the left system resulted in an aortic dissection. The patient was managed conservatively with strict monitoring in the coronary care unit (CCU), and underwent serial evaluation with non-invasive imaging studies including a computed tomography angiography (CTA). On the 3rd post-procedure day, she developed cerebrovascular accident from which she recovered completely. Repeat CT angiogram showed complete resolution of the ascending aortic dissection. Initial follow-up was conducted at 2 weeks and the patient was doing well.

Keywords: Aortic dissection, Percutaneous coronary intervention

Introduction

Incidence of acute aortic dissection during cardiac catheterisation is estimated at about 0.02 to 0.04%.¹ Iatrogenic aortic dissection occurs more commonly in the setting of acute myocardial infarction (AMI) (0.19%) than for non-AMI intervention (<0.01%).¹ The incidence is generally higher during percutaneous coronary intervention (PCI) (overall 0.03%) than during diagnostic procedure.² Iatrogenic aortic dissection is a relatively rare but potentially catastrophic complication of diagnostic and therapeutic vascular procedures.³ Therefore awareness of this complication and its prompt recognition is important. New classification of aortic dissection by the European Society of Cardiology is as follows: classical aortic dissection (Class 1); intramural haematoma/haemorrhage (Class 2); subtle aortic dissection (Class 3); plaque rupture/ulceration (Class 4);

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iatrogenic/traumatic aortic dissection (Class 5).⁴ The overall mortality among patients with iatrogenic dissection is not significantly different from that observed for patients with spontaneous aortic dissection.⁵ Aortic dissection may become immediately fatal, via number of mechanisms, including haemorrhage into pericardium resulting in cardiac tamponade and haemodynamic collapse; occlusion of the contralateral coronary ostium (for example, occlusion of the left main coronary artery during PCI); occlusion of other aortic arch vessels, resulting in cerebrovascular accident (CVA); and propagation of dissection into descending aorta.⁶ Heavy calcification in the vessel wall often requires aggressive catheter manipulation and vigorous injection of contrast dye into subintimal space which results in dissection.⁷ However, treatment of iatrogenic dissection of ascending aorta is challenging and may present as a dilemma. We report herein a case of iatrogenic aortic dissection during percutaneous transluminal coronary angioplasty (PTCA) after taking informed consent from the patient and approval from institutional ethical review committee. The iatrogenic injury was successfully managed with a conservative approach.

Case Report

A 70-year-old female with history of hypertension and diabetes mellitus presented to the emergency department of our hospital on 18th November 2015, complaining of typical chest pain that began three hours prior to presentation in the emergency department. Her diabetes and hypertension were well-controlled on oral medications. She had no past history of coronary artery disease (CAD). Physical examination revealed an acutely ill, diaphoretic lady with blood pressure measuring 140/90 mmHg and pulse rate of 94 beats per minute with regular normal volume and character. Rest of the cardiovascular examination was unremarkable. Electrocardiogram (ECG) revealed signs of acute anterior wall infarction. Coronary angiography was performed with the intent to carry out primary angioplasty if need arose, and revealed significant obstructive three-vessel CAD. Left anterior descending (LAD) artery was identified as the culprit vessel and it was therefore decided to perform primary angioplasty of LAD. An extra backup

(EBU) 3.0 6F guide catheter was used for the procedure. As it was a bit difficult to cannulate the left coronary artery, the operator did a few manipulations. Contrast staining was noted at the root of aorta during this course, as shown in Figure-1. Patient was clinically and haemodynamically stable at the time. Thereafter the left main coronary artery was cannulated with careful manoeuvre and primary angioplasty of LAD was performed quickly with bare metal stent of size 3.0 x 15mm. The patient was shifted to the coronary care unit (CCU) in stable condition. Post-PCI ECG showed >50% resolution of ST-segment elevation and no new ST-segment changes in inferior or any other leads. The procedure had been conducted at 3:00 A.M, and the patient remained stable. Computed tomography angiography (CTA) of the chest was performed early in the morning to confirm the diagnosis and to note the extension of the aortic dissection and any other complication. The CTA revealed a small circumferential pericardial effusion, more so at the aortic recess, and a small dissection at the non-coronary cusp. Left ventricle (LV) and right ventricle (RV) were of normal size, the stent was seen in the proximal LAD, and a mild calcified plaque was noted in the mid LAD and right coronary artery (RCA). The pulmonary arteries were normal. Transthoracic echocardiography (TTE) showed a large hypokinetic area of LV apex, mid intraventricular septum and anterior wall. The LV was of normal size with moderate LV dysfunction. Ejection fraction was 30-35%. Mild pericardial effusion (<5mm) was noted anteriorly. Routine labs were within normal limits and there was no drop in haemoglobin. The patient kept on strict monitoring in the CCU and standard medical therapy was continued. She was clinically and haemodynamically stable till 72-hours after the procedure when she developed left-sided CVA. CT scan of the brain was performed immediately which ruled out intracranial bleeding. Left-sided weakness of the body improved in the next 24-hours and the Glasgow Coma Scale (GCS) was 15/15. CTA of the chest repeated 5 days after the procedure showed resolving pericardial effusion and completely healed aortic dissection of the non-coronary cusp. The patient was referred to a neurologist for CVA. Literature of patients with iatrogenic aortic dissection suggest early follow-up within 2 weeks,⁸ which we did. She was doing well and was vitally stable. ECG revealed no new changes. Follow-up TTE showed complete resolution of pericardial effusion.

Discussion

Aortic dissection is a rare but potentially life-threatening complication of PCI. Clear guidelines regarding its management are still lacking, likely due to variations in severity, patient factors including medical history, age and

vascular anatomy, and institution variables including procedure volume, operator experience and high- versus low-risk procedure. Dissection involving the aortic arch and ascending aorta are generally considered life-threatening requiring immediate surgical intervention while dissection distal to the right brachiocephalic artery and descending aorta can be managed medically. Although not standardised, Dunning et al have proposed a classification system for coronary artery-provoked aortic dissection based on the extent of involvement: class 1, where the contrast staining is limited to the coronary cusp; class 2, where the contrast extends within 40 mm up the aortic wall; and class 3, where the contrast extends to more than 40 mm up the aortic wall.¹ While classes 1 and 2 dissections are typically medically managed, class 3 dissections may necessitate immediate surgical intervention and are associated with higher mortality. In our case, the dye stain was confined to the non-coronary cusp on fluoroscopic view which is consistent with Dunning class 1 without haemodynamic compromise. Predisposing factors for catheter-induced aortic dissection include hypertension, increased age, calcification of aortic root, recent myocardial infarction and intra-aortic balloon pump support.⁹ In case of iatrogenic aortic dissection several factors come into play when deciding what management approach should be employed. Factors which have impact on management decision include haemodynamic stability of the patient, mechanism of aortic injury, size, and severity, propagation of the dissection, presence of intimal flap, and pre-existing atherosclerotic disease. The mechanism of aortic injury during PCI is different from that of spontaneous dissection, assuming the patient has age-related atherosclerotic disease or hypertension, in the absence of collagen vascular disease such as Marfan's and Ehlers-Danlos syndrome.¹⁰ Aortic injury during PCI describes an externally applied mechanical force with trauma to an otherwise potentially normal vessel. In contrast, spontaneous dissection describes a qualitative defect at cellular level in the infrastructure of the vessel. Because propagation of dissection may be halted by more robust and healthy intrinsic architecture, it is not unreasonable to assume that patient with PCI-induced dissection may benefit less from surgical intervention than a patient presenting with spontaneous aortic dissection due to intrinsic vascular disease. Conservative management is a reasonable option only in haemodynamically stable patients with localised aortic dissection. Sinus of Valsalva dissections that are localised during catheterisation tend to resolve spontaneously in the first month.¹¹ In our case, the trauma was caused by manipulation of guiding catheter for an attempt to engage the left system. Aortic

dissection was limited to aortic cusp and did not propagate antegrade or retrograde, and our patient was clinically and haemodynamically stable at that time. TTE demonstrated small pericardial effusion and small amount of fluid in the aortic recess but no echocardiographic sign of cardiac tamponade was seen. CTA showed mild pericardial fluid and small aortic dissection that was confined to non-coronary cusp, therefore we treated this patient conservatively. CTA repeated after five days showed complete resolution of aortic dissection. Our case report along with other published cases suggests that conservative treatment of iatrogenic aortic dissection can result in successful outcome.

Conclusion

Catheter-induced dissection of ascending aorta is a recognised complication of angioplasty. In presence of low-risk aortic dissection which is confined to the aortic cusp, conservative management with serial haemodynamic monitoring, imaging and follow-up is appropriate.

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Conflict of Interest: None.

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