Iatrogenic Acute Aortic Dissection during Percutaneous Coronary Intervention for Acute Myocardial Infarction

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Iatrogenic acute aortic dissection during percutaneous coronary intervention is an extremely rare, but critical complication. Localized aortic dissections have been treated by sealing the entry with a coronary stent. Extensive dissections may require a surgical intervention. We present a case of type A extensive aortic dissection occurring during angioplasty of the left circumflex artery for acute myocardial infarction. This iatrogenic aortic dissection required emergent surgical repair with supracoronary replacement of the ascending aorta.

Keywords: iatrogenic acute aortic dissection, percutaneous coronary intervention, acute myocardial infarction

INTRODUCTION

Acute aortic dissection is a life-threatening event that requires early recognition and frequently requires emergent surgical treatment. It usually occurs spontaneously in patients with long-standing hypertension and cystic medial necrosis of the aortic wall. The occurrence of dissection of the ascending aorta as a complication of coronary angiography or coronary angioplasty is exceedingly rare. In contrast, coronary dissection is a well-known complication of coronary angioplasty that nowadays can be successfully treated with intracoronary stenting.

The options for treatment are determined on the basis of patient stability, nature of dissection of the coronary artery, and extent of aortic dissection. Surgical intervention is associated with particularly high risk in unstable patients with acute myocardial infarction (AMI). We present a case of a patient who developed an iatrogenic acute aortic dissection during percutaneous coronary intervention (PCI) following an AMI.

CASE REPORT

A 66-year-old man with untreated hypertension was admitted to our hospital because of AMI. The patient’s blood pressure was 140/60 mmHg on admission. An electrocardiogram showed elevation of the ST segment in II, III, and aVf leads. Serial creatine kinase levels were elevated at 3065 U/L (normal range, 25–195 U/L). A coronary angiography (CAG) using a MP 3.75 4Fr guide catheter, performed through the right radial artery approach, revealed single vessel disease with a 100% proximal left circumflex artery (LCX) lesion (Fig. 1). When we carried a Launcher EBU 3.5 6Fr guide catheter, performed through the right radial artery approach, revealed single vessel disease with a 100% proximal left circumflex artery (LCX) lesion (Fig. 1). When we carried a Launcher EBU 3.5 6Fr guide catheter, there was resistance with the brachiocephalic artery and the patient complained of the right shoulder pain. However, reperfusion was achieved by passing a guidewire (Rinato) through the affected artery, followed by aspiration thrombectomy. After balloon predilatation of the LCX lesion, a bare metal stent (3.5–18 mm DRIVER, Medtronic) was implanted. An angiogram showed antegrade thrombolysis in myocardial infarction (TIMI) 3 flow in the LCX. During coronary angioplasty, the patient’s blood pressure was normal range. After coronary intervention, the patient developed retrosternal pain and complained of a migrating pain starting in the neck to the back. At this point, we noticed this critical complication. In preparations for an emergent CT scan, the patient’s blood pressure dropped (60/40 mmHg), and he required rapid fluid replacement.
Transthoracic echocardiography showed a large quantity of pericardial effusion. An emergent CT scan revealed an extended aortic dissection from the ascending aorta to the abdominal aorta (Fig. 2a).

An emergency operation was performed because of hemodynamic deterioration. Cardiopulmonary bypass was immediately established by right femoral artery and bicaval cannulation, and the patient’s body was cooled. The ascending aorta was dilated to as much as 4.5 cm in diameter and was a dark reddish color along its entire length. Because the CT-Scan revealed intimal tear around the brachiocephalic artery, the aortic cross-clamp was not performed. Hypothermic circulatory arrest was induced at 22°C, and the ascending aorta was opened. For myocardial protection, cold blood cardioplegic solution was administered retrogradely through the coronary sinus. Careful observation revealed no intimal tear in the proximal or around the left coronary orifice. The initial entry of dissection was located at the front of the ascending aorta near the orifice of the brachiocephalic artery. Moreover, the size of entry was approximately 3 cm. After the entry abscission, the ascending aorta was reconstructed with a 26-mm woven Dacron graft (Fig. 2b). The patient was weaned from cardiopulmonary bypass without difficulty. He recovered uneventfully and was discharged on the 16th postoperative day.

**DISCUSSION**

Acute aortic dissection during cardiac catheterization is a very rare complication. (overall incidence, 0.02%) and occurs more frequently in the emergency setting of AMI (0.19%) than in an elective setting (0.01%).

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*Iatrogenic Acute Aortic Dissection*
is generally higher during PCI (overall 0.03%) than during diagnostic procedures (<0.01% in elective settings).²

In most previously reported cases, the entry point was within a dissected coronary artery with progressive retrograde dispersion within the subintimal space in the aortic root. Few studies have reported isolated dissection of the aorta without involvement of coronary arteries.³ In our case, the entry point was located around the brachiocephalic artery, and the coronary artery was not dissected.

The etiology of this condition is most likely multifactorial. Several possible mechanisms have been proposed: retrograde propagation of a coronary dissection resulting from mechanical trauma due to a wire, catheter, inflated balloon, or other device and aortic trauma resulting directly from the guide catheter.⁴ The nature of the coronary lesion to be treated may have the greatest impact on the probability of aortic dissection occurring because of catheterization. Heavily calcified vessels, which often require more aggressive guiding catheter manipulation in order to deliver coronary balloons and stents, are inherently more prone to dissection.⁵

In recent years, a transradial approach has gained widespread acceptance among interventional cardiologists, aiming to minimize bleeding complications and increase patient comfort. However, technical failure has been reported up to 5% of cases even if performed by skilled operators. The main reason of failure is represented by anatomic abnormalities of upper limb arteries and subclavian-brachiocephalic axis.⁶ We postulate that the cause of the aortic dissection in our patient was an aggressive manipulation of the guiding catheter through the right radial artery approach, given that the intimal tear was located around the brachiocephalic artery. Moreover, we assume that an aggressive manipulation probably occurred because of presence of anatomic abnormalities of subclavian-brachiocephalic axis. Next, we consider the management of catheter-induced aortocoronary dissection. Dunning and colleagues⁷ have classified aortocoronary dissection based upon the extent of aortic involvement: Class I, contrast staining involves only the coronary cusp; Class II, contrast extends up the aortic wall to <40 mm; and Class III, contrast extends to >40 mm up the aortic wall. In their published case series, the extent of propagation of aortic dissection yielded prognostic information, with Class III dissections having uniformly poor outcomes. This classification may be useful for risk stratification. The optimal treatment for this complication has not been established, but it had been recommended that cases of localized aortocoronary dissection not complicated by ischemia or hemodynamic instability can be treated with intracoronary stenting or conservative management. However, if ischemia of any of the aortic branches occurs, or if there is extensive dissection or hemodynamic instability, urgent surgery is the treatment of choice.⁸

In our patient, the iatrogenic aortic dissection occurred during PCI for AMI and was very extensive. Moreover, the patient also experienced hemodynamic deterioration due to cardiac tamponade, and emergent surgery was required in spite of a high risk. Lentini and Perrotta⁹ suggest that despite considerable myocardial protection and improved surgical techniques, the mortality rate for patients with aortic dissection complicated by extensive myocardial damage remain high. The patient’s serial creatine kinase levels increased to 3065 U/L before emergent surgery. It was therefore assumed that the operative mortality of replacement of the ascending aorta was very high because of the poor cardiac condition and severe inflammatory response. In conclusion, this case demonstrates an iatrogenic acute aortic dissection with a supracoronary entry without involvement of the coronary arteries, which most likely occurred as a result of rather aggressive use of a guide catheter. Fortunately, this patient recovered well after emergency replacement of the supracoronary ascending aorta. However, this complication is extremely dangerous and is a life-threatening event; therefore, it is crucial to prevent a catheter-induced aortic dissection during PCI. In particular, the operator should be aware of the possibility of dissection during aggressive manipulation of guide catheters.

REFERENCES

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