

DISEASES OF THE AORTA

PERIOPERATIVE MANAGEMENT

Coronary Thrombosis and Type A Aortic Dissection

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ABSTRACT A 74-year-old female had urgent surgery with replacement of the ascending aorta for acute type A dissection. Postprocedure, the electrocardiogram showed an ST-segment elevation myocardial infarction in the antero-lateral leads. Angiography revealed a thrombotic occlusion of the left anterior descending artery, treated successfully with bivalirudin administration, thrombus aspiration and a balloon angioplasty. This case involves the rare coexistence of acute type A aortic dissection and myocardial infarction due to coronary plaque thrombosis. doi: 10.1111/jocs.12577 (*J Card Surg* 2015;30:583–585)

PATIENT PROFILE

A 74-year-old female with arterial hypertension and type 2 diabetes was admitted with severe chest pain radiating to her back, during a hypertensive crisis (blood pressure 200/100 mmHg). Her electrocardiogram (ECG) was unremarkable.

Computed angiography (angio-CT) of the chest revealed a type A acute aortic dissection (AAD) with an intimal flap which extended from the aortic root to the iliac bifurcation, involving the brachiocephalic artery (Fig. 1).

The left coronary ostium originated in the true lumen, whereas the right coronary ostium seemed to arise from both the true and false lumens. Extensive calcifications of the coronary vessels were also observed, without any detectable significant stenosis (Fig. 2).

The patient underwent urgent replacement of the ascending aorta, from the sino-tubular junction to the hemiarch, with a Hemashield 30 mm prosthetic tube (Maquet, Getinge Group, Rastatt, Germany) and aortic valvuloplasty with cusp resuspension. The true and false lumen were also fixed with biogluce at the level of the aortic root, aortic arch, and brachiocephalic trunk. The coronary ostia were not involved in the dissection and therefore they were preserved.

After the chest was closed, there was a sudden, complete atrioventricular block and cardiac arrest. Spontaneous circulation was restored by temporary ventricular pacing and the use of an inotropic agent. Emergency re sternotomy and reexploration revealed no cardiac tamponade or other explanation for the cardiac arrest; the patient was, therefore, transferred to the intensive care unit (ICU).

The ECG performed in the ICU revealed elevated ST-segments in the antero-lateral leads and so the patient was transferred to the catheterization laboratory for an urgent angiographic study. The catheterization was performed via the right femoral artery.

The aortic angiogram showed no residual dissection at the level of the aortic root and only mild residual aortic regurgitation (Fig. 3).

The coronary angiogram revealed a thrombotic occlusion of the left anterior descending artery, immediately beyond the diagonal branch (Fig. 4).

Antithrombotic therapy with bivalirudin bolus at 0.75 mg/kg and a four-hour infusion at a rate of 1.4 mg/kg/h was started, together with 250 mg of i.v. lysine acetylsalicylate.

A thrombus aspiration with an export device (Export Medtronic Inc., Minneapolis, MN, USA), followed by a balloon angioplasty (POBA), with 2.5 and 3.0 mm balloons, was successfully performed and restored TIMI 3 flow (Fig. 5) along with resolution of the ST-segment elevation.

The patient's postoperative course was complicated by progressive renal failure, due to the involvement of the renal arteries in the dissection, requiring permanent hemodialysis. Predischarge echocardiogram showed

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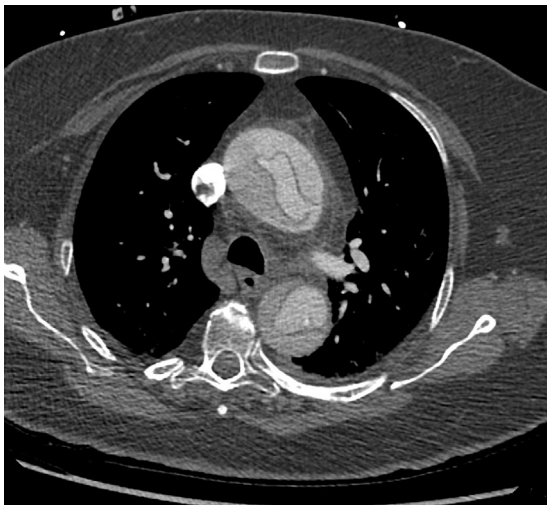


Figure 1. Angio-CT scan at admission with the intimal flap at the level of the ascending aorta.

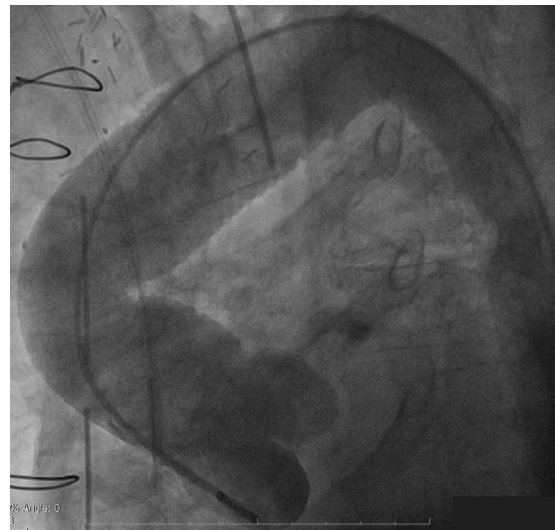


Figure 3. Aortic angiogram showing the prosthetic tube in the ascending aorta. Note also the patent left main stem.

normal left ventricular global and regional contraction with an ejection fraction of 55% and only mild aortic regurgitation, moderate mitral regurgitation, and a slight increase in systolic pulmonary arterial pressure (PAPs 45 mmHg). The patient was asymptomatic and in good clinical health at 51 postsurgical days and was transferred to a rehabilitation facility with single antiplatelet therapy with acetylsalicylic acid 100 mg per day.

DISCUSSION

Several distinct underlying mechanisms have been reported for coronary malperfusion following AAD,¹ including: the intimal flap in the ascending aorta tearing the remaining intimal layer leading to a circumferential

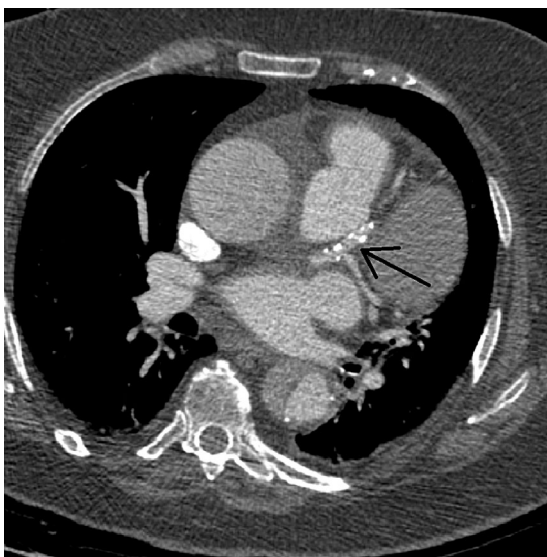


Figure 2. Angio-CT scan showing extensive calcifications of the left coronary artery (arrow).

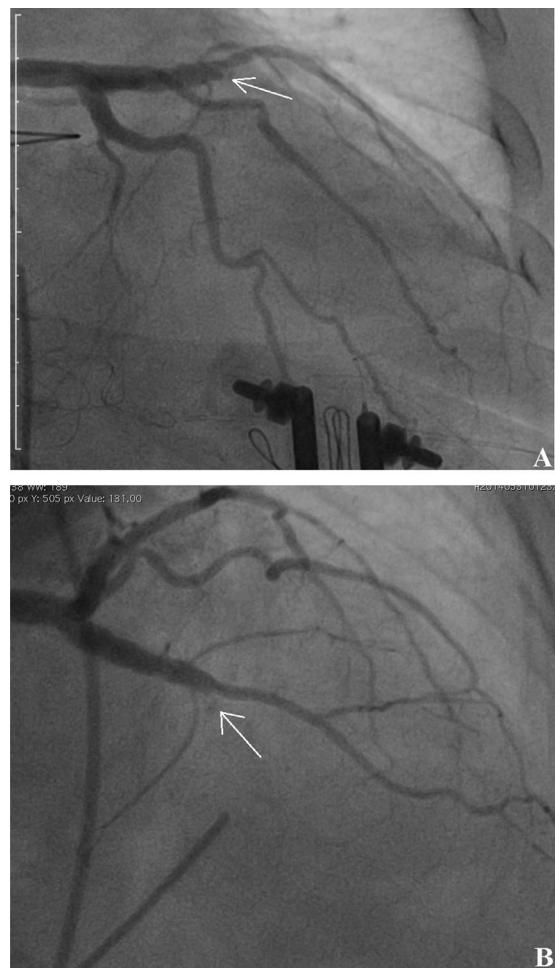


Figure 4. Coronary angiography showing the occluded descending artery (arrows) from the caudal (A) and cranial (B) right anterior view.

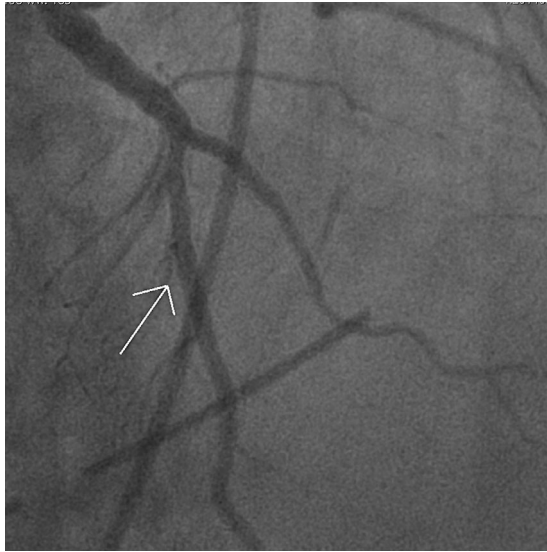


Figure 5. The patent descending artery after angioplasty (arrow).

flap that may be dislodged by the *to-and-fro* movement of the blood. This, in turn, may lead to both distal and proximal invagination of the flap, that is, the so-called “intimointimal intussusception.”² The circumferentially dissected flap prolapses into the left ventricle during diastole, occluding the coronary ostia. Another mechanism is a proximal extension of the false lumen towards the aortic root; the dissection may involve only the coronary ostia, or may extend for several centimeters along the length of the artery. In some cases the coronary artery is completely detached from the aortic root and the dissection encircles the coronary vessel.^{3,4}

The “gold standard” treatment for this condition is surgery with ascending aorta replacement and coronary artery bypass grafting or, whenever possible, coronary repair. Several authors have proposed treating dissected coronary arteries with stents or the use of a coronary perfusion catheter as a *bridge-approach* to gain time for critical unstable patients before definitive surgery.⁵

In the case reported herein the occlusion of the left anterior descending artery resulted from an atherosclerotic plaque thrombosis immediately after replacement of the ascending aorta for acute aortic dissection.

Having to face two concomitant conflicting diseases presents a real challenge, as the patient runs both a high

thrombotic and hemorrhagic risk. The balloon angioplasty avoids the need for a dual antiplatelet therapy and, when performed with a “stent-like result”, is efficacious in the setting of acute myocardial infarction.⁶ Moreover, it has been demonstrated that thrombus aspiration during primary percutaneous coronary intervention improves procedural outcomes limiting no-reflow and reduces distal embolization and mortality.⁷

In conclusion, a careful balance between hemorrhagic and thrombotic risk and the rational use of currently available therapeutic options are mandatory when facing the rare coexistence of aortic dissection and coronary thrombosis.

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