Left Atrial Compression Secondary to Contained Rupture of Type A Aortic Dissection

Robert S. Bonser, MD, FRCS, FACC; H. Abudhaise, MRCSEd; Mohammed Bashir, MRCS; Aaron M. Ranasinghe, MD, MRCS

A 71-year-old man who had undergone coronary artery surgery (coronary artery bypass graft) 9 years previously was admitted to a peripheral hospital with sudden-onset chest pain and progressive dyspnea. Clinical examination revealed reduced basal air entry and crepitations suggestive of heart failure. His ECG demonstrated new-onset atrial fibrillation and T-wave depression in leads I, aVL, and V2 to V5. Cardiac troponin T (1.0 ng \cdot ml^{-1}, reference <0.06 ng \cdot ml^{-1}) and C-reactive protein (209 mg \cdot dL^{-1}, reference 0 to 10 mg \cdot dL^{-1}) were both elevated. Chest radiography showed bilateral pleural effusions and a widened mediastinum (Figure 1A). A presumed diagnosis of acute coronary syndrome was made, and antiplatelet agents and low-molecular-weight heparin were administered. Diagnostic coronary angiography was planned. A transthoracic echocardiogram performed 3 days after presentation due to the features of heart failure demonstrated a type A aortic dissection with evidence of localized left atrial tamponade (Figure 1B and 1C and Movies I and II in the online-only Data Supplement).

He was transferred to our surgical center. Emergency contrast-enhanced computerized tomography revealed a type A aortic dissection extending to the midarch with contained rupture. There was significant hemopericardium in the transverse and oblique pericardial sinuses, with left atrial com-

Figure 1. (A) The admission chest radiograph demonstrating sternal wires from a previous median sternotomy, bilateral pleural effusions, and a widened mediastinum. Note the splaying of the carina indicative of the collection surrounding the left atrium. (B) The transthoracic echocardiogram (4-chamber view) obtained on admission to our institution. The left ventricular (LV) cavity is of normal size, but the left atrium (LA) is compressed by tamponade (Ta). (C) Parasternal long-axis view, again the compression of the LA is noted by Ta. The dissection flap (DF) is visible in the aortic root (AoR). The mitral valve (MV) is labeled for orientation.

From the Department of Cardiac Surgery, UHB NHS FT, Edgbaston, Birmingham, United Kingdom (R.S.B., H.A., M.B., A.M.R.); and the School of Clinical and Experimental Medicine, University of Birmingham, United Kingdom (R.S.B., A.M.R.).

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Correspondence to R.S. Bonser, MD, FRCS, FACC, Department of Cardiac Surgery, UHB NHS FT, Edgbaston Birmingham B15 2TH. E-mail robert.bonser@uhb.nhs.uk

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pression and radiological left atrial tamponade (Figure 2). At emergency operation, a longitudinal intimal tear arising 2 cm distal to the left coronary ostium was identified with rupture posteriorly into the transverse sinus, with further hematoma in the oblique sinus. Free rupture had been prevented by pericardial adhesions from the prior coronary artery bypass graft. The hematoma had extruded above the superior vena cava, rupturing through the parietal pericardium at this point into the right chest. The patient underwent aortic root replacement with a composite biological valve-graft conduit, with button implantation of the native coronary ostia. A left-sided vein graft was also reimplanted, and a pedicled internal thoracic artery graft was preserved. Operative repair was completed by hemiarch replacement, which was undertaken using selective antegrade perfusion, allowing complete extirpation of the dissection with separate reimplantation of the innominate and left common carotid arteries using a multilimbed prosthetic graft (Gelweave Plexus; Vascutek, Renfrewshire, Scotland). The 2 grafts were then anastomosed together. Postoperative recovery was delayed by renal insufficiency that necessitated temporary hemofiltration and a limited left hemiparesis that recovered during rehabilitation. Two years postoperatively, the patient remains well and independent (Figure 3).

Figure 2. A contrast-enhanced computerized tomographic cross-sectional images at the level of aortic root and left atrium obtained on admission. Contrast enhancement is maximal in the left ventricle (LV) leading via the aortic valve into the true lumen (TL). An enlarged aortic root is identified because of dissection with partial thrombosis and enhancement of the false lumen (FL). Hematoma collection is identified in the transverse (TS) and oblique (OS) pericardial sinuses, with no anterior periventricular collection. A pleural effusion (Pe) and pulmonary atelectasis (Pa) are also evident. The inferior vena cava is compressed and not seen; as a consequence, the azygos vein (Az) is hyperenhanced.

Figure 3. The follow-up computerized tomographic scan after discharge. Note the now normal filling pattern of the left atrium (LA) with contrast. The black arrow indicates the buttressed proximal suture line of the aortic replacement.

The dominant cause of death in type A aortic dissection is intrapericardial rupture leading to hemopericardium and tamponade. On occasion, rupture may be contained, in particular if pericardial adhesions are present. In this case, containment occurred serendipitously because of postcardiotomy adhe-

sions, and, in this case, allowed survival to reparative surgery. Tamponade as a presentation of dissection occurs in approximately 19% of cases. It is much less common in patients who have undergone previous surgery.1 This case demonstrates isolated left atrial compression leading to the signs and symptoms of left heart failure. Left atrial tamponade has been reported early after cardiac surgery, and may occur in the absence of a raised jugular venous pressure.2 Recognition of dissection was delayed by several days because a diagnosis of acute coronary syndrome, and left ventricular failure was the forefront differential. Such diagnostic delays may jeopardize patient survival, and are more common when features of the more prevalent acute coronary syndrome with ischemic cardiac biomarker elevation and heart failure are present.3 An index of clinical suspicion, allowing diversion of patients into an appropriate imaging pathway, is currently necessary to secure the right diagnosis and treatment. This underscores the importance of research into point-of-care biomarker testing that may accelerate diagnostic strategies and management.4

Disclosures
None.

References
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