Case report - Cardiac general

Failed closure of a ventricular septal defect with an Amplatzer occluder

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Abstract

A 74-year-old man was diagnosed to have a ventricular septal defect (VSD), which was detected shortly following transvenous pacemaker implantation. Transoesophageal echocardiography suggested the presence of two VSDs, one of which was closed with a device. At surgery, a single large VSD was seen, with the implanted device having embolised into the left ventricle. The defect was successfully closed using a pericardial patch, and the embolised device explanted.

Keywords: Ventricular septum defect; Pacemaker; Transcatheter closure

1. Case report

A 74-year-old man with dyspnoea presented with a symptomatic muscular ventricular septal defect (VSD), which was diagnosed within four weeks following transvenous permanent pacemaker implantation for acquired AV block. Acute myocardial infarction as a cause for ventricular septal rupture was excluded, clinically. Selective coronary angiography did not demonstrate any significant coronary artery pathology which might have accounted for this finding. The most likely cause of the VSD was thought to be septal trauma from lead manipulation during pacemaker lead insertion.

2. Catheterization procedure

As the VSD was located apically, and considering the patient’s age and the potential risks associated with surgical correction, it was decided to undertake transcatheter closure of the defect. Under general anaesthesia, transoesophageal echocardiography was performed. This investigation appeared to demonstrate two separate apical VSDs, separated by a thin rim of septum. The more apically located defect was closed percutaneously using an 8 mm Amplatzer muscular VSD occluder (Aga Corp, USA). The neighbouring larger defect could be easily crossed by a catheter and guidewire combination, and an arteriovenous loop established, using the femoral artery and both the femoral and jugular veins. Multiple attempts at device closure, using a variety of devices [24 mm muscular VSD occluder and 26 mm atrial septum defect (ASD) occluder] proved unsuccessful for a variety of reasons, including device pullthrough and cobra formation of the device. The patient was therefore referred for elective surgical closure of the residual defect, as this was haemodynamically significant.

3. Surgery

Under cardiopulmonary bypass, the VSD was electively approached via a left ventricular incision. The 8 mm VSD device was found to be stably positioned within the left ventricle at the apex, and was not occluding the VSD even partially (Fig. 1). The device was easily explanted, following which a single large apical VSD was seen, which was subsequently closed using an autologous pericardial patch.

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4. Discussion

Transcatheter device closure of acutely acquired VSDs, usually seen in the context of septal rupture following myocardial infarction, is increasingly being applied in clinical practice [1, 2]. Several potential pitfalls associated with closure of such non-congenital defects have been described. One of the major issues with transcatheter closure is related to choosing the appropriate size of device. As the margins of such acute VSDs may be extremely friable, it is advisable to oversize the device to achieve a stable device position. Device embolization is also more common when closing such acquired VSDs. Our patient had an unusual clinical presentation in that his symptoms and the discovery of the VSD were temporally related to transvenous DDD pacemaker implantation for acquired atrioventricular block. The most likely cause of the VSD was therefore rupture of the ventricular septum as a result of pacemaker lead manipulation. Transoesophageal echocardiography was misleading, as it appeared to show two distinct, but adjacent VSDs. It was therefore not surprising that the implanted device, which was meant to close one of the two VSDs, subsequently embolised, albeit without further clinical consequences for the patient. Surgical closure was electively undertaken via the left ventricle, in view of the apical location of the defect. With this approach, the true margins of the defect could be clearly identified, and it was apparent that the patient had a single large defect. The defect was closed uneventfully, following explantation of the embolised device.

References