CASE REPORT

ASD closure under pressure

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SUMMARY

Transcatheter atrial septal defect device implantation in elderly patients may cause acute pulmonary oedema when impaired left ventricular diastolic function causes an abrupt increase in left atrial pressure. Though left atrial pressure is often monitored during test occlusion of a defect, it is not clear at what cut-off value device implantation is contraindicated. We report successful closure of an atrial septal defect in a 73-year-old patient, even though the mean left atrial pressure increased from 18 to 25 mm Hg with device implantation. Although a fenestrated device was used, this did not prevent the rise in left atrial pressure. The patient was supported with mechanical ventilation, milrinone and intravenous diuretics following the procedure and did not develop pulmonary oedema. Her dyspnoea improved and her functional status increased from New York Heart Association (NYHA) III to NYHA II. In conclusion, successful device closure can be accomplished even with high left atrial pressure.

BACKGROUND

Transcatheter atrial septal defect (ASD) device occlusion improves dyspnoea and exercise ability and achieves right ventricular remodelling, even in elderly patients.[1–7] However, a small number of elderly patients with poor left ventricular diastolic function develop acute pulmonary oedema following ASD closure.[1 8] The acute increase in left ventricular filling that occurs when the atrial septum is closed elevates left ventricular end diastolic pressure and, in turn, drives up left atrial pressure.[9] It is unclear how long left ventricular compliance takes to adjust to septal closure. It is also unclear how high left atrial pressure can be before septal closure is contraindicated. There is potentially, therefore, a group of patients who may benefit from device closure, despite being deemed unsuitable due to high left atrial pressure.

CASE PRESENTATION

A 73-year-old woman with atrial fibrillation (AF) was referred for transcatheter ASD occlusion. She had progressive dyspnoea and reduced exercise ability—New York Heart Association (NYHA) class III. There was no history of ischaemic heart disease, valvular heart disease or systemic hypertension. A previous attempt at ASD closure at another hospital had been abandoned when she was found to have high left atrial pressure on test occlusion of the defect. On examination, there were no signs of heart failure. Before attempting device closure, she underwent unsuccessful pulmonary vein isolation, with recurrence of her AF on 3-month follow-up.

INVESTIGATIONS

Her chest X-ray showed cardiomegaly with a cardiothoracic ratio of 0.55. Transthoracic and transoesophageal echocardiography demonstrated a moderate-sized anterosuperior ASD with a colour flow diameter of 18 mm. The rims were adequate for device occlusion, with an attenuated aortic rim measuring 9 mm.

Cardiac catheterisation was carried out under general anaesthesia, with simultaneous transoesophageal echocardiography (TOE). A four French catheter was introduced into the left atrium (LA) via a second venous access, to monitor left atrial pressure during test balloon occlusion of the ASD and a catheter was placed in the left ventricle (LV) to measure left ventricular end-diastolic pressure (LVEDP). Test occlusion of the ASD was carried out using a 30 mm PTSX sizing balloon (NuMed, Hopkinton, USA), taking care not to occlude the pulmonary veins or the orifice of the mitral valve. The diameter of the defect on stop-flow balloon sizing was 18 mm. Haemodynamic measurements were made in AF, initially with the ASD open, then after a 10-min period of balloon occlusion (table 1). DC cardioversion was carried out. There was some hope of maintaining sinus rhythm as amiodarone had been started 16 weeks prior to the procedure. After cardioversion, in sinus rhythm, the haemodynamic measurements were repeated with the ASD open and occluded (table 1). Device implantation was abandoned because the LA pressure increased to 25 mm Hg when the balloon was inflated. TOE functional assessment demonstrated LV diastolic dysfunction (figure 1). There was concern that closing the septum would result in acute pulmonary oedema.

TREATMENT

The patient was treated with an ACE inhibitor and diuretic therapy for 18 months. She returned to the catheter laboratory for a further attempt at ASD closure in the hope that preconditioning the left ventricle would have favourably influenced her haemodynamics. She was in permanent AF and, again, had a catheter introduced into the LA to measure pressures during balloon occlusion. We speculated that any increase in LV filling pressure during test occlusion would be transient, the left ventricle would quickly adapt to the increased volume load and the LA pressure would return to its initial level if test occlusion was continued for 30 min. Fenestrated Occlutech ASD occluders (Occlutech, Helsingborg, Sweden) had been preordered to use in the event of unfavourable haemodynamics. The Occlutech device has a single 6 mm fenestration (figure 2).

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Table 1 Haemodynamics at first catheterisation

ASD status	Condition	LVEDP	a	v	Mean
Open	Precardioversion	12–14	4	19	11
Balloon occluded	Precardioversion		7	33	18
Open	Postcardioversion	14	5	22	13
Balloon occluded	Postcardioversion	22	12	43	25

ASD, atrial septal defect; LVEDP, left ventricular end-diastolic pressure.

Occluding the ASD for a long period of time proved to be technically difficult. The PTSX balloon was supported by an Amplatzer superstiff guidewire (Boston Scientific, Natick, USA) placed in the distal left upper pulmonary vein. The balloon repeatedly prolapsed into the right atrium 10 min after inflation. A decision was therefore made to test occlude the defect using the ASD device itself.

A 12 French Mullins sheath was placed in the left upper pulmonary vein and a 0.014″ Choice PT extrasupport (Boston Scientific, Natick, USA) coronary wire was introduced through the sheath into the distal left upper pulmonary vein. A 21 mm fenestrated Occlutech occluder was selected and the proximal end of the coronary wire was passed through its fenestration. The device was then introduced into the Mullins sheath, tracking it up to the LA over the coronary wire. The device was deployed in a stable position across the ASD without difficulty, with no residual shunt at the margins. After the device had been implanted, a 6 mm coronary balloon was passed over the coronary wire until it was positioned across the fenestration. The balloon was then inflated to temporarily occlude the fenestration using TOE to confirm that there was no residual shunt (figure 3).

LA pressure measurements were made with the ASD open, with the ASD closed (10 and 30 min after device implantation) and with the fenestration open and closed (table 2).

The mean LA pressure increased from 18 to 32 mm Hg after 5 min of balloon occlusion then dropped to a pressure between 24 to 26 mm Hg after device implantation. The pressure did not change after 15 then 30 min and was not much altered by opening and closing the fenestration.

A decision was made to leave the device in place, despite the high LA pressure, with a contingency plan to snare and remove the device the following day if there was severe pulmonary oedema. Forty milligrams of intravenous furosemide was given.

Figure 2 Picture of Occlutech fenestrated atrial septal defect device.

At that point, fresh bright red blood was observed coming up the endotracheal tube. The aspirates from the airway did not appear to be pink and frothy as would be expected with pulmonary oedema. We concluded that the bleed was caused by an injury to the left-sided pulmonary vein by the superstiff guidewire, as there had been considerable balloon and wire movement during the earlier attempts at balloon occlusion. The patient was transferred, intubated, to the intensive care unit and the bleeding stopped after 30 min. A chest radiogram showed complete collapse of the left lung. Bronchoscopy demonstrated that the left bronchus was blocked by thrombus. The thrombus was aspirated, bronchial patency was restored and the left lung completely re-expanded, with no further bleeding.

Although there were no clinical or radiographic signs of pulmonary oedema, twice daily intravenous furosemide 0.5 mg/kg intravenous (40 mg twice daily) and intravenous milrinone 0.5 µg/kg/min were started to improve left ventricular filling pressures, owing to concerns that pulmonary oedema would occur when positive pressure ventilation was stopped. The patient was successfully extubated 24 h after device implantation. Milrinone was stopped 48 h later and bumetanide 2.5 mg once daily and spirinolactone 50 mg twice daily were restarted

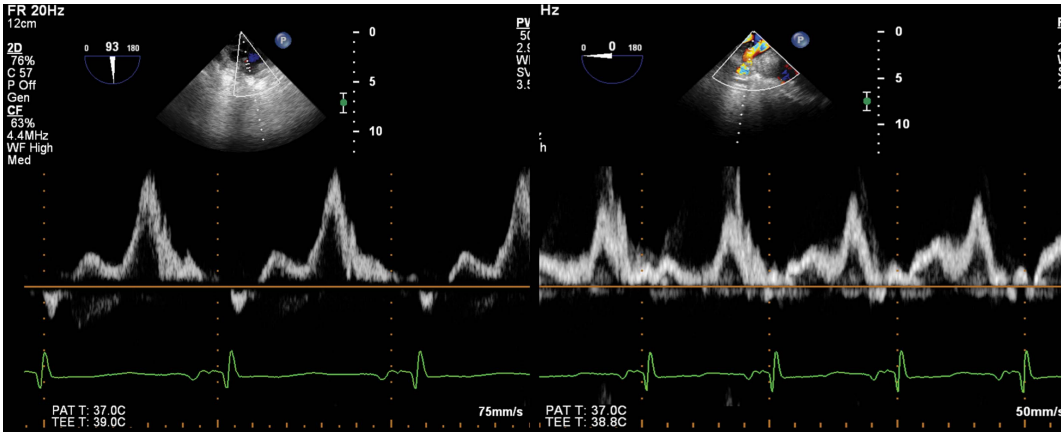

Figure 1 Deterioration in diastolic function on balloon occlusion of atrial septal defect (left—mitral valve inflow Doppler trace, right—pulmonary vein Doppler trace).

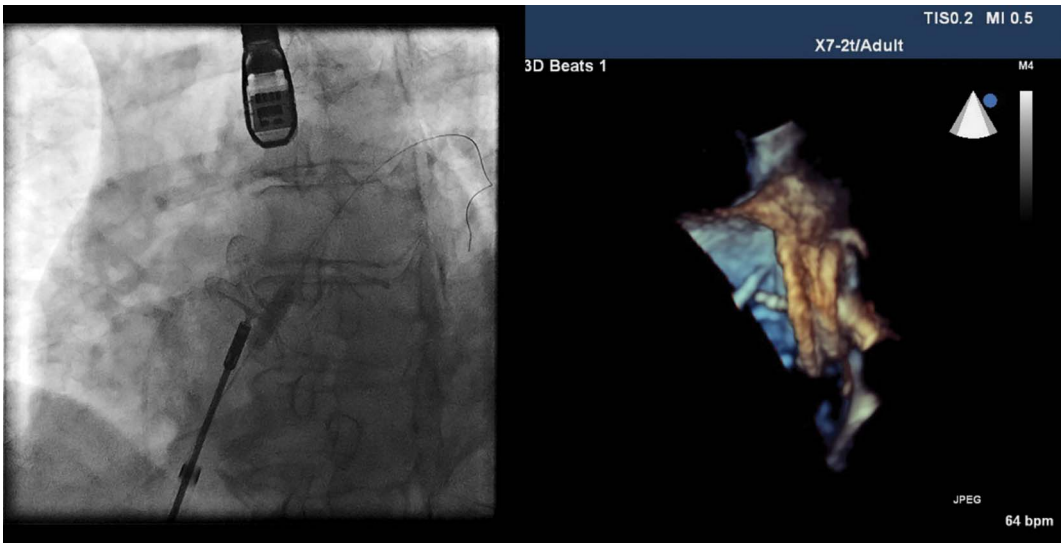

Figure 3 Three-dimensional transoesophageal echocardiography and matching fluoroscopy image demonstrating test occlusion of the fenestration with a coronary balloon.

along with ramipril 2.5 mg once daily. There was no clinical evidence of pulmonary oedema at any stage. The patient was well on discharge from hospital 4 days after device implantation.

OUTCOME AND FOLLOW-UP

At outpatient follow-up 6 weeks later the patient reported a significant improvement in her dyspnoea and exercise ability (NYHA II). The Occlutech device position was unchanged and the fenestration remained open, shunting left-to-right.

DISCUSSION

Since the first case of pulmonary oedema following ASD closure in an elderly patient,[8] operators have tried to estimate the upper safety limit for left atrial pressure, beyond which closure should not be attempted. Different operators have estimated this limit to be 10, 20 or even 25 mm Hg by.[10–12] When test occlusion of the ASD has resulted in mean atrial pressures above this value, the procedure has been stopped and medical preconditioning undertaken before a further attempt at device implantation. Using these estimated limits, more than 25% of patients over 60 have been deemed unsuitable for ASD closure.[9 10 12] However, in a series of 50 patients over 60 years old in whom left atrial pressure was not reported, only one patient developed pulmonary oedema.[1] It therefore seems that pulmonary oedema remains a rare complication and that concerns about left atrial pressure may be overemphasised.

Table 2 Haemodynamics at second catheterisation

ASD status	Fenestration	a	v	Mean LA_P	Mean RA_P
Open		17	23	18	14
Balloon occluded 5 min		31	52	32	
Device occluded 15 min	Fenestration open	22	35	24	
Device occluded 15 min	Fenestration closed	25	42	26	
Device occluded 30 min	Fenestration open	25	37	25	
Device occluded 30 min	Fenestration closed	25	42	26	
Final pressures	Device deployed, fenstration open	24	37	25	16

ASD, atrial septal defect; LA, left atrium; RA, right atrium.

Preconditioning, using either 4 weeks of oral diuretics with an ACE inhibitor or an angiotensin receptor blocker, or intravenous dopamine, milrinone and furosemide for 3 days before the procedure, has been shown to reduce left atrial pressure to acceptable levels in most patients with a significant LA pressure increase on balloon occlusion.[10 12] However, what an 'acceptable' pressure and a 'significant' increment are remains open for debate. It is possible that many patients put forward for preconditioning based on arbitrary cut-off values could have immediate closure without developing pulmonary oedema.

Not all patients respond to preconditioning. In some reports, such patients have undergone successful ASD closure with a fenestrated device.[3 10 13] In four cases, either a single or double 6 mm fenestration allowed ASD closure without the elevation in LA pressure observed during balloon occlusion.[10 13 14] This is contrary to our observation that the 6 mm fenestration in the Occlutech device did not provide a large enough communication to prevent the pressure increase. This is the first reported use of the device. In one other reported case, a single 6–8 mm fenestration in an Amplatzer device also failed to prevent LA pressure elevation.[14] It therefore remains unclear whether a single 6 mm fenestration is adequate. The question: 'who should receive a fenestrated device?' clearly depends on knowing what level of LA pressure is problematic.

From this case, we observed that ASD closure could still be carried out even though the LA mean pressure increased by 7 mm Hg to a final level of 25 mm Hg. In a few reported cases, successful closure has also been demonstrated with mean LA pressures ranging from 16 to 24 mm Hg.[5 11 13 14] In those rare cases where pulmonary oedema has occurred, patients have been successfully treated with either diuretics alone or a combination of mechanical ventilation, catecholamines and diuretics.[1 8 9]

It is unclear in our case whether recovery would have been uneventful without the assistance of a period of positive pressure ventilation, milrinone and furosemide. It may be that the LA pressure returns to its former value after a number of hours, though it clearly did not do so after 30 min.

Learning points

- There is no clearly established level of or rise in left atrium (LA) pressure that has been proven to increase the risk of pulmonary oedema in patients undergoing atrial septal defect (ASD) device closure.
- Previous perceived wisdom may be denying the benefits of device closure to a significant proportion of elderly patients with ASDs.
- Preconditioning prior to the procedure may improve the haemodynamics and make ASD closure safer.
- Patients with high LA pressure postdevice closure should be carefully monitored and may require pharmacological intervention to allow time for left ventricular remodelling to occur.

Competing interests CD is a consultant and proctor for Occlutech.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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