A Case Study of Immediate Pulmonary Edema Due to Small Left Atrium after ASD Device Closure and its anesthesia management

Dr. Himanshu Shah, Dr. Mrugank Bhavsar, Dr. Jaishree S.B., Dr. Bhavesh Roy

Abstract

Percutaneous atrial septal defect (ASD) closure is getting safer in expert hands and popular amongst patients. But it has certain drawbacks which includes rare complication like pulmonary edema after device closure of atrial septal defect that we encountered in a 45 years old post menopausal female. Chest x-ray images, 2D-echo findings in post operative period and intra operative images of fluoroscopy used for to arrive at confirmed diagnosis of the cause of pulmonary edema in this patient.

Keywords: Atrial Septal Defect (ASD), pulmonary edema, 2D-Echo.

1Associate Professor, Consultant Cardiac Anesthetist, 2MBBS, 3Assistant Professor, 4Assistant Professor, Consultant Cardiologist
SBKS MI & RC, Sumandeep Vidyapeeth, Piparia, Vadodara, India
Corresponding author mail: mb_gmcs_1987@sify.com
Source of Support: Nil
Conflict of Interest: Nil

Introduction

Atrial septal defect (ASD) is common form of congenital heart disease. Its occurrence is 10% of all congenital heart defects. Surgical treatment for ASD is the gold standard treatment for Secundum ASD. In 1976, King and Mills were the first to practice transcatheter closure of secundum ASD and now it has evolved over past three decades and nowadays preferred over surgical closure in selected cases. The Amplatzer Septal Occcluder (ASO) is the commonly used device and previous studies demonstrate that it is safe and easy to use with high success rate. ASD device closure procedure was done under general anesthesia with intubation for to prevent obstructed respiration due to Transesophageal probe. As an anesthesiologist working in cardiac field, we need to know about complications arising out of the interventional or surgical procedures.
Pulmonary edema is a rare complication immediately after device closure. Here we report a case of 45 years old post menopausal female posted for ASD closure that developed pulmonary edema immediately after deploying the device.

**Case Report**

A 45 years old post menopausal female referred to us from gynecologist with complain of shortness of breath, fatigue and soft diastolic and harsh systolic murmur across tricuspid area. She had shortness of breath and fatigability which was progressive with exertion and relieved on rest. There was no complains of chest pain, fever, cough etc. She was normotensive and non diabetic. The two dimensional echocardiography revealed moderate size secundum ASD with moderate TR and no MR, AR. Ejection fraction was 55-60% with grade I diastolic dysfunction. The defect size on Transthoracic echocardiography (TTE) was 25 mm with good margin superoinferiorly and anteroposteriorly. It was case of left to right shunt with hyperdynamic right sided circulation. All investigations regarding anesthesia check up were normal. ECG showed RBBB. X-ray chest was showing increased pulmonary plethora.

After written informed consent, patient was taken inside Cathlab. Patient’s pulse was 54/min and so we gave Inj. Glycopyrrolate after that pulse reached to 94/min and blood pressure shoot up to 180/110. Gradually pressure came back to normal in 2-3 minutes. After that we induced the patient with Fentanyl, Midazolam and Vecuronium and Intubated. Patient was maintained with ventilator support O2-Air 50-50%, PEEP -0, Tidal Volume -600 ml, Rate was 14/min and Inspiratory and Expiratory (TI:TE) ratio was 1:3. Peak Inspiratory Pressure was 15. We used ECG, SpO₂, and Arterial BP as monitoring tool for the procedure.

During cardiac catheterization, PA pressure was 40/13 mm Hg. Coronary angiography was normal. By a 6F JR catheter, the guide wire was positioned in RUPV and a 12F ASD device delivery sheath loaded with 28 mm device (Cera™ ASD Device, Lifetech Scientific Ltd., Banglore, India) was taken over the wire. After sheath opening LA retention disc in LA, the device was positioned across the ASD under fluoroscopic and TEE guidance. After confirming the optimum position and
unobstructed PV SV return, device was released. Post release no residual flow, no MR, TR no flow obstruction to SVC and PV. Complete occlusion of ASD was confirmed by TTE and TEE.

Immediately after deploying the device patient’s peak pressure reached to 49 cm H$_2$O and saturation gradually falls to 80%. We did not find any mechanical cause of desaturation. On auscultation, bilateral rales were present. O$_2$ increased to 100% at that time, pinkish froth was coming out from oral ET tube. Saturation further falls to 60% and Blood pressure was 56/34 mmHg. So we changed ventilator parameters (O$_2$ – 100%, PEEP – 5, Inspiratory: Expiratory ratio- 1:1.5, Tidal Volume 600) started and Inj. Lasix 20 mg stat along with Inj. Dopamine 10 µg/kg/min and Inj. Dobutamine 5 µg/kg/min started. 12 lead ECG was taken, it was isoelectric with no sign of ST-T changes due to air embolism. TEE probe was there but we also took in to account a possibility of compression of pulmonary vein resulting in pulmonary edema so first of all we removed TEE and TTE was done (Figure 2, 3, 4). Normal LA volume is around 20ml/m$^2$ of BSA and the dimensions in this case showed LA volume around 10ml. Patient was shifted to ICCU. Dopamine and Dobutamine infusion continued. Inj Lasix 40 mg intravenous repeated. Triple lumen CVP line and urine catheter inserted. Inj. Atrcurium + Inj Midazolam infusion started and Patient was kept on ventilator with 100% O$_2$, PEEP -5.

We have used following formula to calculate LA volume by using TTE which is 10ml/m$^2$ just after deploying device.

$$\text{LA Volume} = 0.523 \times D1 \times D2 \times D3$$

where D1, D2 and D3 means diameters of LA.
A Case Study of Immediate Pulmonary Edema Due to Small Left Atrium after ASD Device Closure and its anesthesia management

Dr. Himanshu Shah et al. 2013

Figure 1: Dimension of LA (D1)

Figure 2: Dimension of LA (D2)

Figure 3: Dimension of LA (D3)

Figure 4: Chest X-ray after Extubation

We did ABG after 3 hours of shifting, PO2 was 68 mmHg and peak airway pressure was 44 cm H2O. We continued same strategy. Again did ABG after 4 hours, PO2 was 88 mmHg and peak pressure coming down to 41 cm H2O. We gradually tapered O2 concentration to 80%. Inj. Lasix 20 mg I.V given. Total 1.5 litre of urine was there in the urine bag over period of time. After 3 hours again ABG was done and it showed PO2 98 mmHg and peak pressure 38 cm H2O. With the same ventilator parameters we have ventilated for almost 18 hours ABG was done. ABG was showing PO2 140 mmHg and Peak pressure 23 cm H2O. After that she was extubated after reversing with Inj. Neostigmine 0.05 mg/kg and Glyco 0.008 mg/kg. Patient was kept on Non invasive ventilator support (Bipap of 10:5, O2-35%) for another 4 hours. ABG was showing PO2 130 mmHg and peak pressure was 20 cm H2O. Thereafter patient was put on O2 mask.

Discussion

A procedure like ASD device closure is a boon for patients in terms of avoiding cardiac surgery. It has various complications like injury to the heart and vascular system, pericardial tamponade, accidental release of device in right or left heart, air embolism etc.

Pulmonary edema in ASD device closure usually happens due to the pulmonary venous or coronary sinus obstruction or due to MR that develops due to device interfering into the mitral valve.
closure. Pulmonary edema was due to small LA which is very and develops very gradually. It is very difficult to presume occurrence of pulmonary edema due to small LA immediately after deploying of the ASD device3. Diastolic dysfunction can be the reason behind pulmonary edema after device deployment. But in this case, diastolic function was normal. A case report presented by AK SInghi et al7. described occurrence of late pulmonary edema due to diastolic dysfunction. Restrictive left ventricular dysfunction in elderly may be masked by the presence of an ASD. Deterioration of left ventricular diastolic function can occur with acute hemodynamic change, following interventional closure of ASD, leading to acute lung edema8,9. The only independent risk factor to identify these patients is elevated left atrial pressure, which increases significantly during balloon test occlusion, indicating underlying restrictive left ventricular dysfunction.9

One more reason for left ventricular dysfunction is coronary air embolism. But in our study, post procedure ST analysis and Echo findings for left ventricular function was absolutely normal10.

Our presentation is to bring into notice that small LA size can be the reason for immediate pulmonary edema following device deployment. And even in the case of ASD it is still possible to measure LA dimension by following formula:

\[
\text{LA Volume} = 0.523 \times D1 \times D2 \times D3/\text{BSA},
\]

where D1, D2 and D3 means diameters of LA and Distance AB/2 is considered as one dimension of LA into above mention equation. Schematic presentation is shown below:

![Rough Estimation of LA Dimension](image.png)

Figure 5: Rough Estimation of LA Dimension
Conclusion

Pulmonary edema due to small LA gradually resolves once LA starts accommodating volume. For immediately treating pulmonary edema inotropic support, diuretics and ventilator support plays crucial part. For the cardiac anesthetist, pre-operative evaluation of LA volume is important to avoid such complication leading to emergency surgery to remove the deployed device and surgical closure of ASD OR before deploying the device measurement of LA pressure, PAP and LVEDP would be very much helpful in anticipating this event.

References

5. William F. Armstrong MD. Feigenbaum's Echocardiography, Lippincott Williams & Wilkins. 2009
7. C. Angel, MD, J. Losay, MD, J. Petit, MD, V. Lambert, MD, G. Esna, MD, X. Berthaux, MD, P. Brenot, MD, Percutaneous closure with Amplatzer device is a safe and efficient alternative to surgery in adults with large atrial septal defects American Heart Journal Volume
A Case Study of Immediate Pulmonary Edema Due to Small Left Atrium after ASD Device Closure and its anesthesia management

Dr. Himanshu Shah et al.


Acknowledgement

We are thankful to the whole team of Cathlab and cardiac recovery for their support. Patient has given written informed consent to send this case report as a publication.