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Case Report

Transcatheter closure of left ventricle to right atrial communication using cera duct occluder



IHJ

Gnanavelu Ganesan^{a,*}, G. Justin Paul^a, Vaikom S. Mahadevan^b

^a Madras Medical College, Chennai, India

^b University of California, San Francisco, USA

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ABSTRACT

Left ventricle—right atrial communication could be congenital (Gerbode defect) or acquired as a complication of surgery or infective endocarditis and leads to volume overloading of pulmonary circulation. Two types, direct and indirect types are known depending on the involvement of septal tricuspid leaflet. Transcatheter closure of this defect is feasible and appears an attractive alternative to surgical management. Various devices like Amplatzer duct occluder I, II, Muscular ventricular septal defect device etc. have been used to close this defect. We report two patients, a preteen boy with direct left ventricle-right atrial communication as post operative complication and an adult female with indirect communication who underwent transcatheter closure with Cera duct occluder (Lifetech Scientific (Shenzhen), China).

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1. Introduction

Left ventricle (LV) to right atrial (RA) communication can be congenital or acquired. Congenital communication is named after Gerbode, who described it in 1958.¹ Gerbode defect could be direct or indirect communication depending on the involvement of tricuspid septal leaflet and presence of additional communication between LV and right ventricle (RV). Acquired LV-RA shunt results as a complication of surgical closure of ventricular septal defect, after mitral or aortic valve replacement or as a result of infective endocarditis.² LV-RA communication was managed surgically till the first report of device closure of acquired LV-RA communication in 2006.³

Various devices like ADO I, ADO II, Muscular VSD device etc. have been used to close this defect. We present two patients with LV-RA shunt, a preteen boy with acquired defect and an adult female with congenital indirect type, successfully closed by Cera duct occluder (Lifetech Scientific, Shenzhen, China).

2. Case 1

9 year old boy presented with shortness of breath and palpitation on moderate exertion since his childhood. He was separated from a conjoined parasitic thoracophagus twin in his early infancy. At 3 years of age he underwent ligation of small patent ductus arteriosus (PDA) and fenestrated gortex patch closure of large inlet ventricular septal defect (VSD). The VSD was closed through right atrial approach and patch fenestration was possibly performed in view of severe pulmonary artery hypertension (PAH) preoperatively. LV- RA communication was recognized during the postoperative period but was not intervened at that time.

At the time of presentation, examination revealed thin built child with normal heart sounds and harsh 4/6 holosystolic murmur at left sternal border. Trans-thoracic and trans-esophageal echocardiography revealed 8 mm LV-RA communication with left to right shunt. The pulmonary artery pressure during catheterization was 40/16/25 mmHg. Device closure was initially attempted with 8 mm Amplatzer Vascular Plug II. This device was chosen because of its low profile, ease of deployment and presumably inert effect on AV node. But vascular plug slipped through the defect. Finally the defect was closed with 8–10 Cera duct occluder. Trans-thoracic echocardiography revealed no residual shunt and mild tricuspid regurgitation. Post device closure period was uneventful except for a brief period of sinus bradycardia and junctional escape rhythm. He was empirically given, oral

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 $^{^{\}ast}$ Correspondence to: New No.7 Kanakkar Street Tiruvottiyur, Chennai 600 019, India.

E-mail addresses: gnanaveluganesan61@gmail.com, velunesan@yahoo.co.in (G. Ganesan).

prednisolone 10 mg and orciprenaline 5 mg twice a day for a week. His left and right ventricular function remained normal. On follow up at one year he was asymptomatic with good weight gain and no rhythm abnormality. Trans-thoracic echocardiography revealed no residual shunt, normal biventricular function and estimated right ventricular systolic pressure of 24 mmHg.

3. Case 2

26 year old thin built female presented with shortness of breath, fatigue and dizziness on mild exertion. Clinical examination revealed pectus carinatum, sinus tachycardia and harsh 4/6 holosystolic murmur over left sternal border with normal heart sounds. Investigations revealed severe iron deficiency anemia (Hb 7 gms/dl) due to menorrhagia. Trans-thoracic and trans-esophageal echocardiography revealed left to right shunt across indirect Gerbode defect measuring 12 mm and moderate tricuspid regurgitation. She also had mitral valve prolapse with mild mitral regurgitation and normal LV function (EF 65%). Anemia was

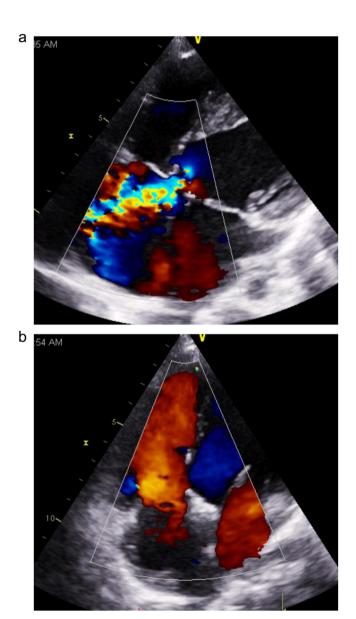


Fig. 1. (a) Case 1: Echocardiogram: Apical 4 chamber view showing LV-RA shunt. (b) Case 1: Post-procedure echocardiogram: Apical 4 chamber view showing device in situ with no residual shunt.

corrected with iron supplements. She underwent device closure of Gerbode defect once her hemoglobin improved to 11 gms/dl, with a 14–16 Cera duct occluder. Cath data revealed aortic pressure of 83/ 32/54 mmHg. Pulmonary artery pressures were 34/17/22 mmHg. After device closure aortic pressures measured 94/64/80 mmHg. Preprocedurally she had first degree atrioventricular (AV) block. During the procedure she had transient 2:1 AV block, which resolved at the end of the procedure. First degree AV block persisted even after the procedure. She was empirically treated with oral prednisolone 10 mg tid for 5 days. Soon after the procedure her left ventricular ejection fraction was 54% suggesting mild dysfunction probably due to afterload mismatch.⁴ She was prescribed spironolactone 25 mg once daily and enalapril 2.5 mg twice a day at discharge. On follow up at 6 months she was asymptomatic with faint systolic murmur of mitral regurgitation due to mitral valve prolapse. Trans-thoracic echocardiography

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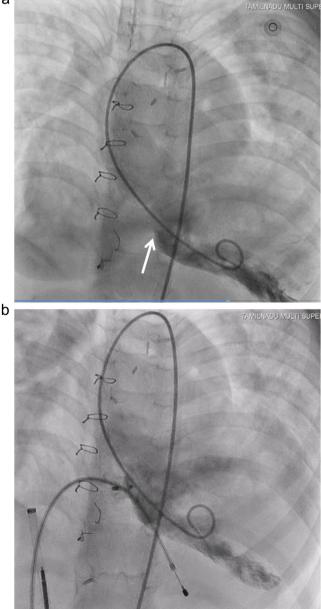


Fig. 2. (a) Case 1: LV angiogram- LAO 30° cranial 10° view showing Gerbode defect. (b) Case 1: LV angiogram- LAO 30° cranial 10° view showing device in situ without residual shunt.

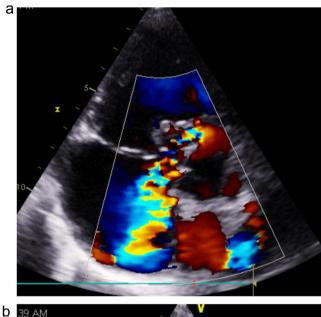




Fig. 3. (a) Case 2: Echocardiogram: Modified apical 4 chamber view showing Indirect LV-RA communication with left to right shunt. (b) Case 2: Post-procedure echocardiogram: Apical 4 chamber view showing device in situ with no residual shunt.

revealed mitral valve prolapse with mild mitral regurgitation and mild tricuspid regurgitation with peak gradient of 23 mmHg. Her Left ventricular ejection fraction was 54%. Holter monitoring revealed atrial ectopics with no evidence of heart block.

4. Procedure description

Under general anesthesia, right femoral vein, right femoral artery and left femoral vein were accessed. Left venous access was used for temporary pacing lead insertion. Heparin 100 U/kg was used. LV angiography was done to delineate the defect. The defect was crossed with 4 Fr MPA catheter from the left ventricle using 0.035" angled TerumoTM (Terumo medical corporation) guidewire and exchanged for 0.035" extrastiff Amplatz (St Jude's Medical, MN, USA) 260 cm guide wire. This wire was snared from the IVC using a 15 mm One Snare [®] (Merit Medical Systems Inc.) and

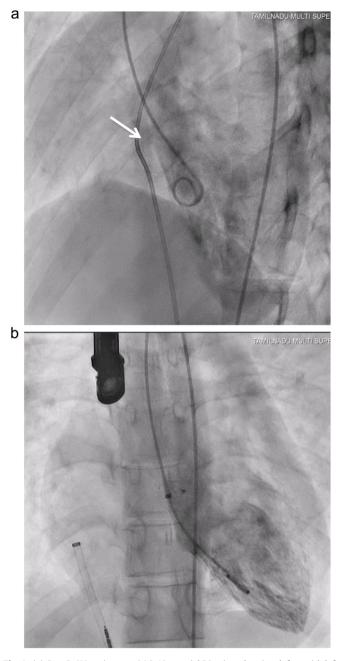


Fig. 4. (a) Case 2: LV angiogram- LAO 40° cranial 20° view showing defect with left to right shunt. (b) Case 2: LV angiogram—AP view showing device in situ with no residual shunt.

arteriovenous loop was formed. Appropriate size Cook delivery sheath was introduced into ascending aorta antegradely without exposing the stiff guide wire across the defect to avoid damage to the AV node and AV valves. In the first case 8 mm Amplatzer Vascular plug II prolapsed through the defect on testing and hence it was withdrawn and a 8–10 Cera Duct occluder (Figs. 1 and 2) was used to close the defect. In the second case, the defect was closed with 14–16 Cera duct occluder successfully in the first attempt (Figs. 3 and 4). There was only mild tricuspid regurgitation post procedure in both the cases.

5. Discussion

LV-RA communication leads to volume overloading of pulmonary circulation. Spontaneous closure of congenital defect by aneurysm transformation of septal tricuspid leaflet is known (7%), but the incidence of infective endocarditis is high (8.5%).⁵ However spontaneous closure of postoperative LV-RA communication has not been reported. Acquired defect could be due to direct injury to the membranous septum, inappropriate suture technique, infective endocarditis, following transcatheter aortic valve replacement⁶ and iatrogenic as in our first patient.

In our first patient, patch fenestration was possibly performed in view of severe PAH preoperatively. Unfortunately when the septal tricuspid leaflet was reattached to the annulus, the fenestration led to LV-RA communication.

Both congenital and acquired LV-RA communication have been traditionally closed by surgery till the first device closure of an acquired Gerbode defect by Amplatzer muscular VSD device by Trehan in 2006.³

Since then LV-RA communication has been routinely closed by various devices like ADO II, VSD device and ADO I depending on the operator's choice, proximity of the defect to the conduction tissue⁷ and size of the defect. The choice of device depends on previous surgery and the underlying heart defect in case of acquired defects. The size of the device is chosen 1–2 mm more than the maximum diameter of the defect estimated by trans-esophageal echocardiography avoiding oversizing. Amplatzer duct occluder has been found to be better than muscular VSD device because of its low radial force.⁸ Cera duct occluder (Lifetech Scientific, Schenzhen, China) has a superelastic metal frame coated with titanium nitride and its design is similar to Amplatzer duct occluder I. Cera duct occluder was used in both of our patients successfully without any significant adverse effect on the AV node and tricuspid valve.

The major concerns of this technique include proper alignment of the device across the defect, intra and post procedural conduction disturbance⁹ and worsening of tricuspid regurgitation. We noted only transient rhythm disturbances in our cases, without any long term effects.

6. Conclusion

LV-RA communication both congenital and acquired can be effectively closed by transcatheter technique. CeraTM (Life tech scientific, Shenzhen, China) duct occluder can be used in this situation without any adverse effect.

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