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SUPRAVENTRICULAR TACHYCARDIA IN A YOUNG ADULT WITH CONGENITAL HEART DISEASE: SUCCESSFUL ABLATION AFTER PATENT DUCTUS ARTERIOSUS CLOSURE

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ABSTRACT

Arrhythmias in adults with congenital heart disease (ACHD) can occur due to remodelling and post-intervention injury. As ablation is the gold standard for arrhythmia intervention, finding the electrical source from ACHD can be challenging, especially after surgical repair. We present an ACHD with patent ductus arteriosus (PDA) without risk factor cardiovascular disease, with post-closure complaints of frequent palpitations and a history of supraventricular tachycardia (SVT) after transcatheter PDA closure; electrocardiography (ECG) shows SVT. Mean pulmonary arterial pressure (MPAP) was 81 mmHg before PDA closure and 64 mmHg after closure. The patient was planned for 2D ablation, and concentric coronary sinus activation was found. VAV activation showed PPI-TCL >115 ms, ventriculoatrial interval <70 ms, SVT AVNRT/AVRT with excluded AT (V-A stimulus 202 ms). ECG and electrogram after radiofrequency ablation show successful ablation with paroxysmal atrial tachycardia. Detailed examination of ECG and echocardiography with manoeuvres is important to distinguish concomitant arrhythmia or correlation in ACHD.

KEYWORDS

Adult with congenital heart disease, atrial arrhythmia, patent ductus arteriosus closure, pulmonary hypertension

LEARNING POINTS

- Managing adults with congenital heart disease (ACHD) can be challenging because of its various complication risks, with arrhythmias being common complication.
- An ablation technique for arrhythmia intervention in ACHD can be complex for the mapping of electric conduction based on the complex structural anatomy from congenital heart disease.
- Monitoring cardiac function after cardiac repair surgery among ACHDs is needed to reduce complication after surgery.

INTRODUCTION

Adult patients with congenital heart disease (ACHD) have a higher risk of atrial fibrillation compared to the population without congenital heart disease (CHD)[1]. Age is known to be a predisposing factor where those aged under 50 years have a 20-fold risk of atrial fibrillation and those aged 50–59

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years have a 10-fold risk[1]. The risk of atrial arrhythmia is also higher in ACHD patients with 7% aged 20 years increasing to 38% at the age of 50[2]. ACHD also has a higher risk of developing heart failure and ischaemic stroke compared to the general population[1]. The risk of mortality in the ACHD population is up to three times higher in those over 68 years of age compared to the general population[1]. ACHD also has a more than a three-fold higher risk of developing arrhythmias and almost two-fold increased risk of pulmonary hypertension due to ventricular dysfunction associated with impaired systemic/pulmonary systolic function, which is a risk factor for heart failure[2].

Supraventricular tachycardia (SVT) is one of the most common types of arrhythmia in ACHD, with 50% of the ACHD population experiencing it during their lifetime[3]. Rhythm disturbances can be various between CHD anatomy, conduction system malformations and previous CHD post-operative injuries[4]. Recurrent arrhythmias can also develop despite defect correction. We present a case of ACHD with arrhythmia following defect correction surgery and our strategy for analysing the underlying cardiac sinus disorder.

CASE DESCRIPTION

We present a 20-year-old woman who was diagnosed with patent ductus arteriosus (PDA) from one month of age, with the defect becoming clinically significant by the age of 5 years. Surgical intervention was recommended but declined by the family. She experienced recurrent respiratory infections throughout childhood, consistent with volume overload from the left-to-right shunt. An echocardiography in October 2023 revealed an 8 mm bidirectional PDA with dominant left-to-right shunting, accompanied by right ventricular dilatation and pulmonary hypertension. Right heart catheterisation in November 2023 confirmed severe pulmonary hypertension with a mean pulmonary arterial pressure (MPAP) of 81 mmHg and elevated pulmonary vascular resistance of 17.6 WU·m^2. Importantly, oxygen testing demonstrated vascular reactivity with significant reduction in pulmonary vascular resistance to 7.9 WU·m^2, indicating reversible pulmonary hypertension suitable for closure. Transcatheter PDA closure was performed in June 2024 using a PDA occluder device (Lepu Medical MemoPart™, size 22/24 mm) *(Fig. 1)*. The procedure was successful with complete elimination of residual shunting. Post-closure haemodynamic assessment showed a reduction in MPAP to 64 mmHg, although pulmonary hypertension persisted.

Four months post PDA closure, the patient developed symptomatic palpitations. In October 2024, she presented to the emergency department. An electrocardiography (ECG) showed SVT *(Fig. 2A)* that was refractory to vagal manoeuvres but responded to intravenous diltiazem. An electrophysiology study was planned for definitive management, which confirmed typical atrioventricular nodal re-entrant tachycardia (AVNRT) (slow-fast type) based on characteristic diagnostic criteria. This showed the

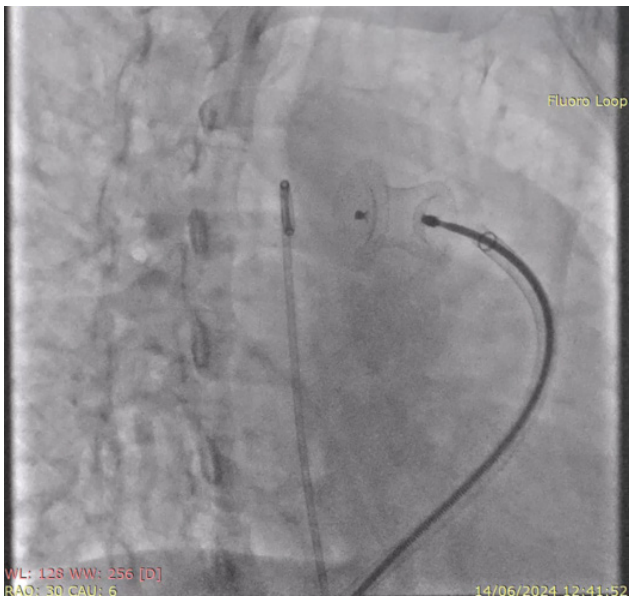


Figure 1. Patent ductus arteriosus device.

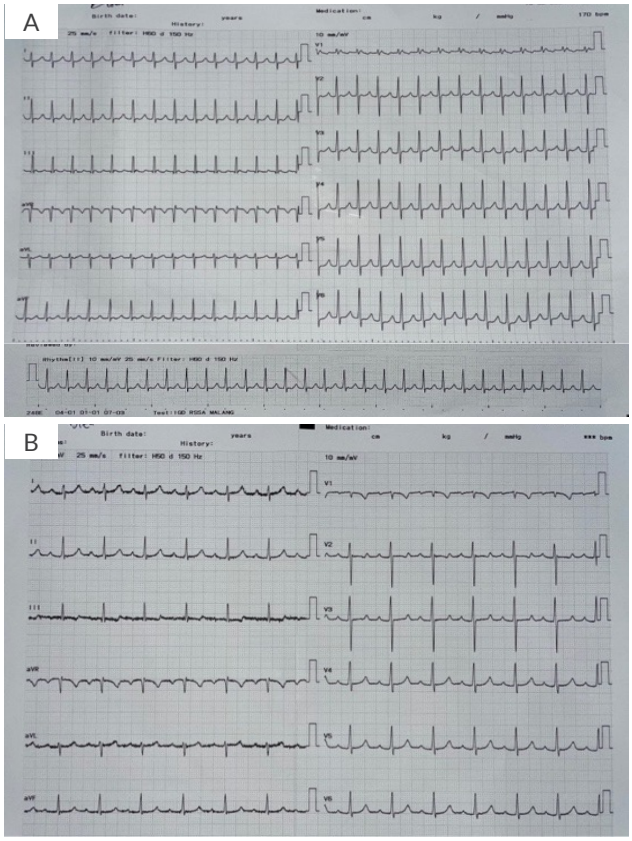


Figure 2. ECG from post-closure PDA patient A) before and B) after ablation.

ventriculoatrial interval <70 ms during tachycardia, post-pacing interval minus tachycardia cycle length (PPI-TCL) >115 ms from the right ventricle, a concentric coronary sinus activation pattern, and VAV activation sequence consistent with AVNRT mechanism. Slow pathway ablation was performed using radiofrequency energy (30 watts, 65°C for 150 seconds) delivered to the posteroseptal region near the coronary sinus ostium. Post-ablation testing confirmed elimination of AVNRT inducibility and dual AV nodal physiology, with no evidence of atrio-Hisian (AH) jump.

The ablation procedure was successful, with no acute complications. Post-ablation, the patient did not experience recurrent AVNRT episodes, although occasional episodes of paroxysmal atrial tachycardia were observed on the ECG *(Fig. 2B)* and electrogram *(Fig. 3)*. Follow-up echocardiography showed stable device position without residual shunt and a slight decrease in tricuspid regurgitation velocity from 4.1 m/s to 3.8 m/s. Eight months later, follow-up echocardiography performed after 2D ablation showed PDA closure accompanied by mild tricuspid regurgitation with a high probability of pulmonary hypertension (tricuspid regurgitation velocity 3.8 m/s, tricuspid valve gradient 59 mmHg) *(Fig. 4)*.

DISCUSSION

This case presents a rare presentation of arrhythmias occurring in post-closure PDA. Here, we present arrhythmias occurring in a patient with a history of PDA closure who, after 2D ablation, was found to have no access pathway for AVNT. Reconstruction and the addition of an atrial substrate provide a pathway for re-entrant arrhythmias, with a higher risk of complex CHD that allows for the recurrence of cardiac electrical disturbances[5]. Intra-atrial re-entrant arrhythmia is the most common type of arrhythmia in the elderly CHD population, accounting for approximately 15% of all ACHD[4]. However, there is limited evidence of arrhythmias occurring in post-closure PDA cases, so it is important to understand the origin of the electrical conduction disturbance.



Figure 3. SVT AVNRT post successful ablation with paroxysmal atrial tachycardia after radiofrequency ablation with burst pacing.

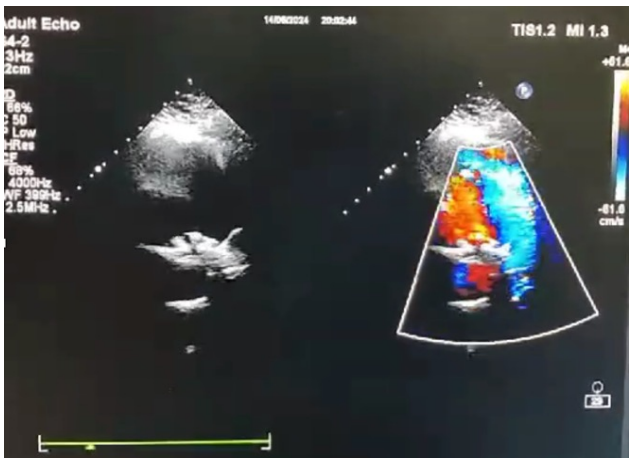


Figure 4. PDA device from echocardiography after PDA closure.

PDA incidence contributed for 5–10% of all congenital heart defects and represents one of the most successfully treated conditions through transcatheter closure[6]. While transcatheter PDA closure has become the gold standard treatment with success rates exceeding 95%, the haemodynamic changes following closure can lead to cardiac remodelling and subsequent arrhythmia after the procedure. This may be prevented by avoiding prolonged intracardiac surgery and adding an occluder that is slightly larger (±1–2 mm) than the defect, to prevent arrhythmia[7]. SVT, particularly AVNRT, can emerge as a complication following structural cardiac interventions in young adults with CHD[8]. In this case, the risk of arrhythmias may arise from post-closure PDA scarring combined with pulmonary hypertension. Pulmonary hypertension may develop as a result of shear stress from endothelial dysfunction and smooth muscle cell proliferation in the defect structure, resulting in a remodelling process similar to that seen in idiopathic disease[9]. In the adult population, surgical intervention is not only to repair the defect but also to reduce the risk of arrhythmias due to damage to the electrical pathways of the heart related to structural defects[10].

One of the challenges in arrhythmia management in ACHD is the primary source of cardiac rhythm disturbances, where in cases of normal structure, the primary source comes from the pulmonary veins, but in ACHD the findings are often in extrapulmonary triggers such as the right atrium or ventricle[11,12]. Structural changes (hypertrophy and remodelling in the atrium or ventricle) followed by structural changes (fibrosis) disrupt electrical conduction and increase the risk of re-entry[11]. Catheter ablation is the first line of arrhythmia management in CHD, but a procedure that is appropriate to the conduction system in the anatomy of each CHD individual is needed, especially in individuals who have a history of surgical correction of defects that require removing fibrosis or clinical scar areas[13]. Several studies suggest that radiofrequency ablation is a safe option for catheter ablation of complex anatomical structures[12].

CONCLUSION

Arrhythmias are inseparable in the management of CHD. The anatomical complexity of CHD contributes to cardiac rhythm disturbances and increases recurrence compared to non-CHD patients, and reduces the effectiveness required for intervention.

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