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REVIEW

Adult congenital heart disease: a review of the simple lesions

Milos Prica[1]

Stephe Kamalathasan[1]

Karina Gopaul[1]

David Warriner[1,2]

Author details can be found at the end of this article

Correspondence to:
David Warriner;
david.warriner@nhs.net

Abstract

There has been a dramatic improvement in mortality rates among children with congenital heart disease with advances in neonatal screening and surgical techniques, resulting in a significant increase in the prevalence of adults living with congenital heart disease. The most common simple lesions of congenital heart disease include atrial and ventricular septal defects, patent ductus arteriosus and coarctation of the aorta, which are typically detected and treated in childhood. However, they may also present in adulthood with non-specific symptoms or incidental findings, such as refractory hypertension. As the adult population of those living with congenital heart disease grows, it is imperative that all clinicians remain abreast of these common cardiac conditions, irrespective of their specialty, as patients may present with sequelae of their congenital heart disease or other non-cardiac conditions.

Key words: Arteriosus; Atrial septal defect; Coarctation of aorta; Congenital heart disease; Patent ductus; Ventricular septal defect

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Introduction

As a result of the advances in neonatal screening and surgical techniques, there has been a dramatic improvement in mortality rates among children with congenital heart disease, resulting in a significant increase in the prevalence of adults living with congenital heart disease. It is evident that with these advances, more physicians across different specialties will encounter patients with congenital heart disease in their clinical practice, many of whom will not necessarily present with a primary cardiac complaint. This article reviews and outlines the pathophysiology, clinical manifestations and updates in the management of some of the common defects, with a focus on simple lesions in adult congenital heart disease.

Atrial septal defect

Atrial septal defects are the most common congenital heart defects diagnosed in adulthood (Marelli et al, 2007), and include secundum atrial septal defect (80% of diagnosed atrial septal defect cases), primum atrial septal defect (15%), superior sinus venosus defect (5%), inferior sinus venosus defect (<1%) and unroofed coronary sinus (<1%) (Baumgartner et al, 2021). The classification of atrial septal defects depends on the location of the defect on the interatrial septum.

Pathophysiology

Secundum atrial septal defects are caused by an excessive reabsorption of the septum primum, or the interruption of the formation of the septum secundum (Figure 1). Primum atrial septal defects result from the incomplete fusion of the septum primum endocardial cushions. Sinus venosus defects occur from an error during the incorporation of the sinus venosus chamber into the right atrium, and are also commonly associated with anomalous pulmonary venous drainage (Naqvi et al, 2018). Irrespective of their location, atrial septal defects cause a shunt at the atrial level, resulting in a shunt from the left atrium to the right atrium. This can lead to right ventricular and right atrial volume overload. The volume of the shunt depends on the size of the defect, the compliance of

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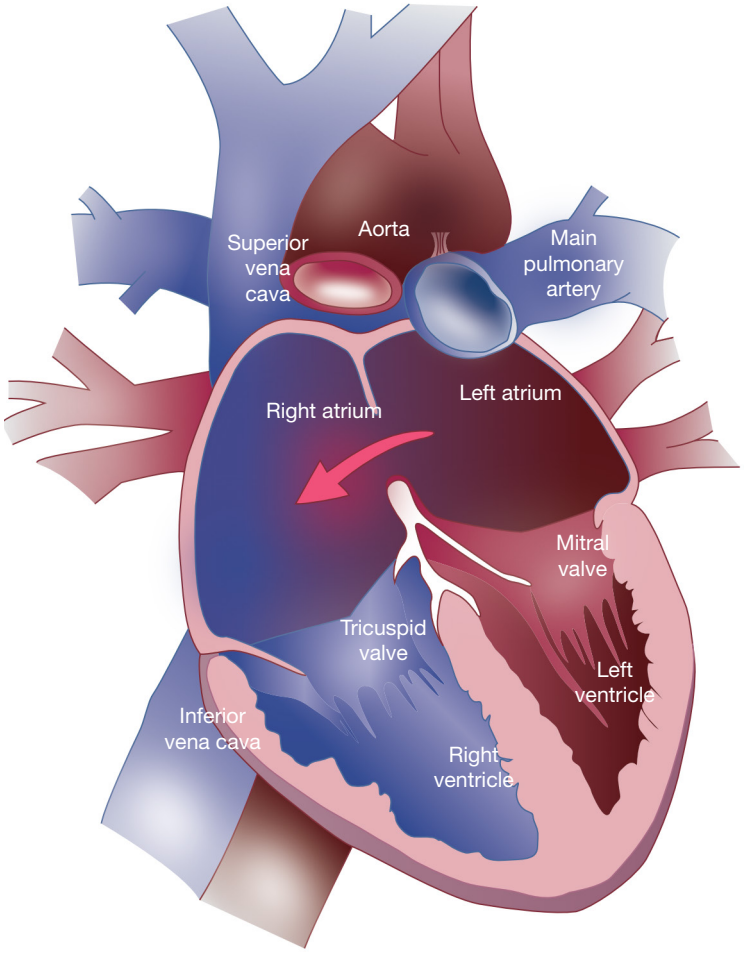

Figure 1. An illustration of an atrial septal defect (secundum type).

the right and left ventricles to expand when filled with blood, and the pressure within the atria. Pulmonary hypertension can be seen independently in patients with an atrial septal defect, or may occur as a complication in a small number of patients, particularly those diagnosed later in life. High pulmonary vascular resistance can eventually cause the reversal of the shunt from right to left and cyanosis, which is termed 'Eisenmenger's syndrome' (Martin et al, 2015).

Clinical presentation

The size of the atrial septal defect typically dictates the clinical course and presentation (Martin et al, 2015). The most common symptoms include dyspnoea, reduced exercise tolerance, fatigue, palpitations or dizziness. Patients may also present with right heart failure, chest infections or cerebrovascular events as a result of paradoxical embolisation. On examination, the patient may have a right ventricular heave, fixed split second heart sound or systolic flow murmur over the pulmonary valve region.

Diagnosis

In secundum defects, an electrocardiogram will typically show a right bundle–branch block with right axis deviation, or left axis deviation in primum defects. However, when diagnosing atrial septal defects, the gold standard investigation is transthoracic echocardiography (Figure 2). The recently published European Society of Cardiology guidelines (Baumgartner et al, 2021) suggest that right ventricular volume overload is a key finding that characterises the haemodynamic sequelae of the atrial septal defect, irrespective of type. Sinus venosus defects are often challenging to visualise with transthoracic

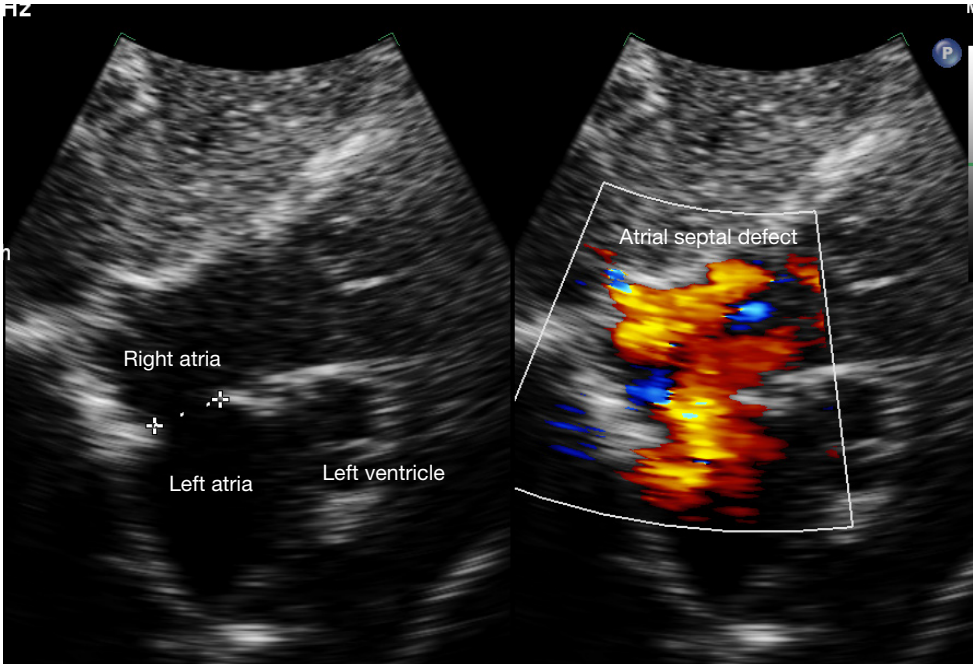

Figure 2. A two-dimensional transthoracic echocardiogram (subcostal four-chamber view) demonstrating a 1.67 cm secundum atrial septal defect (left) with Doppler colour flow (red), showing a left atrial to right atrial shunt (right).

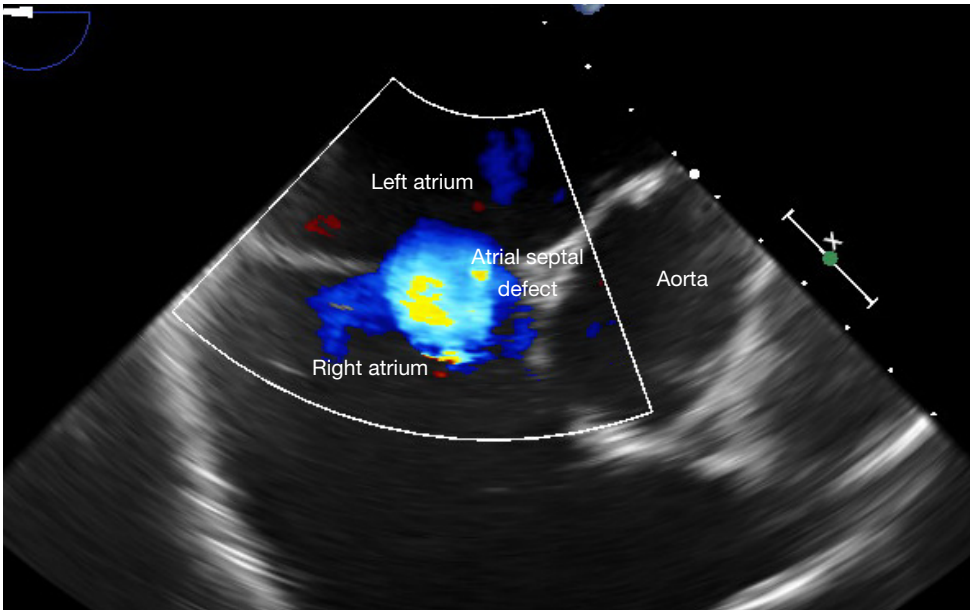

Figure 3. A two-dimensional transoesophageal echocardiogram (mid-oesophageal short axis view) with Doppler colour flow (blue), showing a left atrial to right atrial shunt, across a large secundum atrial septal defect.

echocardiography and may require a transoesophageal echocardiogram (Figure 3) or cross-sectional imaging, such as cardiac magnetic resonance imaging or computed tomography, to accurately delineate the cardiac anatomy. Cross-sectional imaging can also be used to generate three-dimensional models to determine the suitability of the defect for a transcatheter device closure. Cardiac magnetic resonance imaging further provides an estimation of the pulmonary to systemic flow, whereby a pulmonary to systemic flow greater than 1.5:1 is considered to be haemodynamically significant. Cardiac catheterisation can also be used to estimate the pulmonary to systemic flow and provides additional measurements of pulmonary arterial pressure.

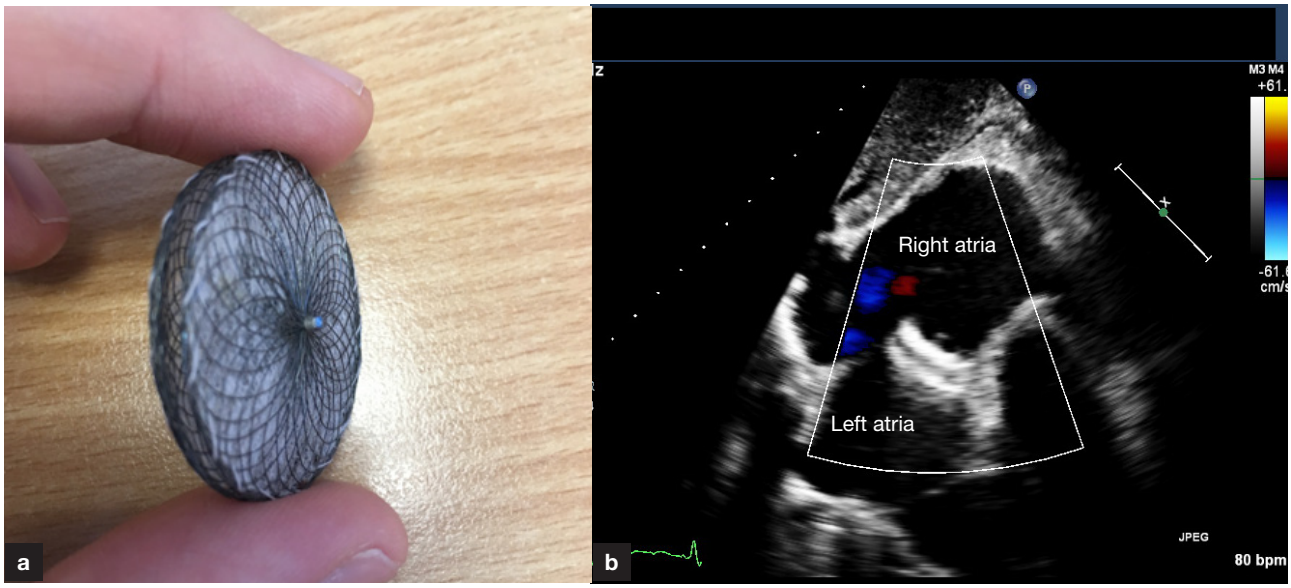

Figure 4. a. A percutaneous atrial septal defect occlude device ex-vivo with (b) a corresponding parasternal short-axis echocardiogram with the device seen in-situ between the atria at the 10 o'clock position.

Management

The presence of an atrial septal defect with right ventricular volume overload, and the absence of pulmonary hypertension or left ventricular dysfunction, fits the criteria for an atrial septal defect closure regardless of the presenting symptoms (Baumgartner et al, 2021). The options for atrial septal defect closure are either open surgical repair or percutaneous closure with a device. At present, a device closure approach is the first line choice for closing a secundum atrial septal defect with favourable anatomy, such as single secundum defect, and morphology, for example with sufficient rim surrounding tissue (Figure 4), and has been the chosen approach in approximately 80% of patients (Baumgartner et al, 2021). The patient should receive antiplatelet therapy for 6 months after the procedure. Device closure is a safe procedure and serious complications are rare (<1% of patients) (Butera et al, 2006). As an alternative, surgical repair of atrial septal defects can be performed on younger patients because of its favourable long-term outcomes and low rate of mortality. Both techniques have similar rates of mortality, while device closure has been associated with lower morbidity and shorter hospital stays, but higher rates of reintervention (Butera et al, 2006).

Follow up

Generally, younger patients with no sequelae would not require a routine follow up, whereas patients with arrhythmia, elevated pulmonary arterial pressures or a residual shunt should have regular follow ups. Patients who have undergone a device closure procedure to repair an atrial septal defect should be followed up periodically, for example every 2 years, because of the rare risk of device embolisation.

Sequelae

The likelihood of atrial tachyarrhythmias, which are common in adults with atrial septal defects, increases with age even after closure (Martin et al, 2015). However, right ventricular dilatation and systolic dysfunction are usually resolved following the closure procedure.

Ventricular septal defect

Ventricular septal defects are the most common congenital heart disease diagnosed at birth (van der Linde et al, 2011), and are commonly treated before adulthood. The spontaneous closure, particularly of muscular ventricular septal defects, is common among ventricular septal defects diagnosed at birth.

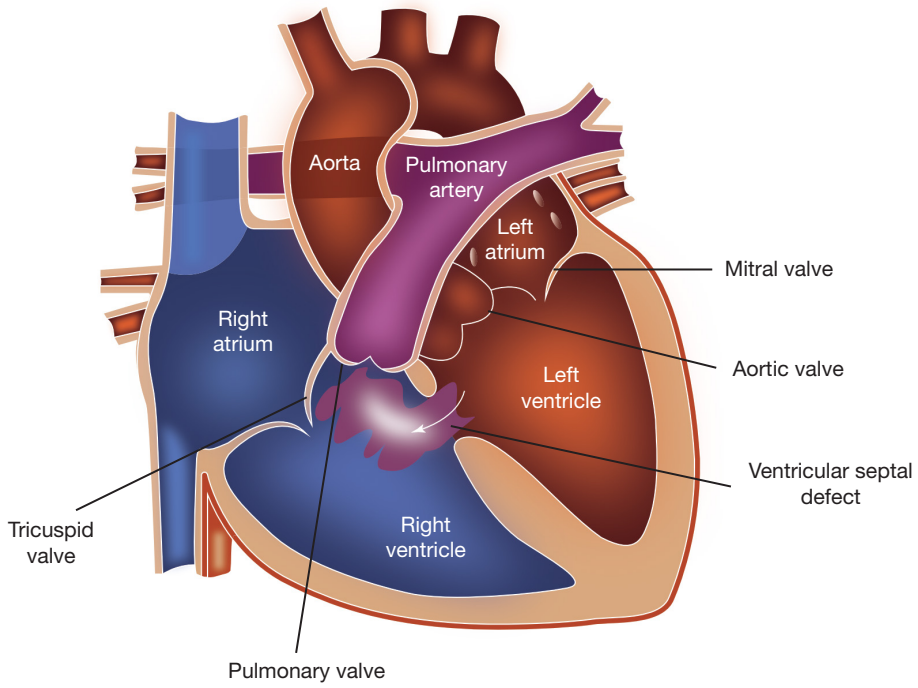

Figure 5. An illustration of a ventricular septal defect (perimembranous type).

Pathophysiology

Ventricular septal defects arise as a result of one or more ventricle septum components failing to fuse during embryological development (Figure 5). Contrary to atrial septal defects, the left to right shunt in a ventricular septal defect occurs at the ventricles and during systole. Thus, the increased volume of blood essentially travels straight through the right ventricle, into the pulmonary circulation and back via the pulmonary veins into the left atria, causing the left ventricle, not right, to become volume loaded. The most common type (approximately 80% of cases) is the perimembranous ventricular septal defect (Baumgartner et al, 2021). These are located in the membranous septum, behind the septal leaflet of the tricuspid valve and below the right and non-coronary leaflets of the aortic valve. The next most common type is the muscular ventricular septal defect, accounting for about 15–20% of ventricular septal defects. These are exclusively surrounded by muscle and can occur in multiple locations simultaneously. The third type is the outlet ventricular septal defect, usually located beneath the semilunar valves. These can be further divided into outlet perimembranous defects, outlet muscular defects or doubly committed juxta-arterial defects. Finally, inlet ventricular septal defects occur in the right ventricle inlet and extend below the septal leaflet of the tricuspid valve.

Clinical presentation

A large proportion of adults with ventricular septal defects may remain asymptomatic, undetected, or not require any surgery or intervention (Gabriel et al, 2002). This group of patients will also include those who underwent spontaneous closure of a ventricular septal defect in childhood, as well as patients with a small ventricular septal defect with no left ventricle volume overload. The shunt size, whether residual or natural, will determine the extent of the left ventricular volume overload and pulmonary hypertension. Therefore, adults may present with small ventricular septal defects with trivial shunts without left ventricular volume overload and pulmonary hypertension, or may present with a left to right shunt and varying grades of left ventricular volume overload and pulmonary hypertension. At one end of the spectrum are the patients with Eisenmenger's syndrome, which is caused by the reversal of the left to right shunt because of high pulmonary vascular resistance. Infective endocarditis, a serious complication of ventricular septal defects, may also be the initial clinical presentation.

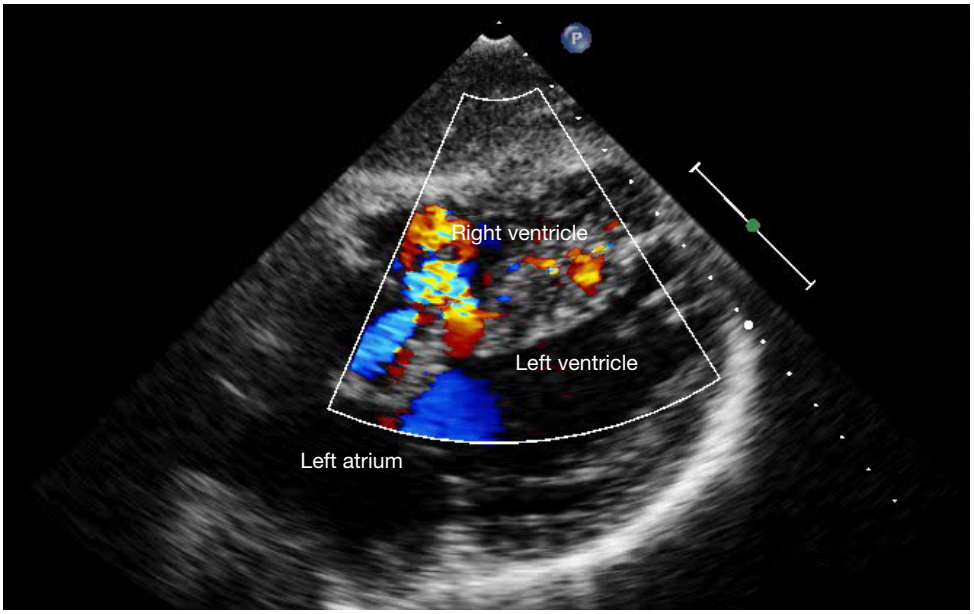

Figure 6. A two-dimensional transthoracic echocardiogram (subcostal four-chamber view) showing multiple small muscular ventricular septal defects with Doppler colour flow (red), demonstrating left to right ventricular systolic shunt across the intraventricular septum with a Swiss cheese type appearance.

Diagnosis

Examination usually reveals a pan-systolic murmur, possibly with a displaced apex beat. The electrocardiogram in patients with a ventricular septal defect may present as normal, but may occasionally show left axis deviation or first degree atrioventricular block. Transthoracic echocardiography is key for the diagnosis of ventricular septal defects (Baumgartner et al, 2021). Transthoracic echocardiography not only provides information on the location, size and number of defects (Figure 6), but can also determine the presence of left ventricular volume overload, estimate the pulmonary arterial pressures and identify any associated aortic incompetence, all of which can occur as a result of the right coronary cusp prolapsing into the ventricular septal defect. Similar to atrial septal defects, cardiac magnetic resonance imaging can be used to measure the pulmonary to systemic flow and quantify the left to right shunt. Cardiac magnetic resonance imaging may also be used if there is diagnostic uncertainty following a transthoracic echocardiography.

Management

The European Society of Cardiology guidelines strongly suggest closure of ventricular septal defects in the presence of left ventricular volume overload and absence of pulmonary hypertension, regardless of the patient's symptoms. However, it is strongly recommended that ventricular septal defects are not closed in patients with Eisenmenger physiology. Progressive aortic incompetence, because of the associated prolapse of the right coronary cusp, should prompt the consideration for surgery. Even in cases of insignificant left to right shunt, ventricular septal defect closure should be considered if there is a presentation of infective endocarditis, particularly if recurrent. In instances where the patient has developed pulmonary hypertension, a significant shunt (pulmonary to systemic flow >1.5 may prompt careful consideration of ventricular septal defect closure). In ventricular septal defects, transcatheter device closure was initially associated with high rates of complete heart block, as a result of the atrioventricular node lying close to the ventricular septal defect, therefore making them liable to injury. However, studies have since shown that this approach is safe and the complication rates are similar to those of surgical closure (Morray, 2019).

Follow up

The follow up of patients with ventricular septal defects should include an assessment via transthoracic echocardiography for the presence of left ventricle dysfunction, as well

as an estimation of pulmonary arterial pressures, the degree of the shunt, development of double chambered right ventricle and the presence of aortic incompetence. The interval for follow up will vary depending on the size of the ventricular septal defect, and the presence of complications. In stable cases, the interval can be as long as 5 years following a surgical closure.

Sequelae

There are a few important complications to note. One is aortic incompetence, which can occur in outlet or perimembranous ventricular septal defects as a result of the right coronary cusp of the atrioventricular node prolapsing into the ventricular septal defect because of a Venturi effect. The second, more commonly seen in perimembranous ventricular septal defects, occurs when the high-velocity jet from the ventricular septal defect leads to focal right ventricle hypertrophy, causing the subdivision of the right ventricle into a proximal high pressure and distal low-pressure chamber, where the mid cavity obstruction protects the lungs from the high-pressure flow. Finally, infective endocarditis is a serious complication and can sometimes be the initial presentation for the patient.

Patent ductus arteriosus

The ductus arteriosus is a vital component of fetal circulation, connecting the proximal descending aorta to the main pulmonary artery, allowing the output of the right ventricle to bypass the non-functioning lungs in-utero. If the duct persists after the first few weeks of life, it is considered abnormal and termed a 'patent' ductus arteriosus (Figure 7). It is thought that patent ductus arteriosus makes up approximately 5–10% of all cases of congenital heart disease (Schneider and Moore, 2006). However, when incidental cases are included, the frequency has been reported to be as high as 1 in 500 cases (Lloyd and Beekman, 1994).

Pathophysiology

Like a ventricular septal defect, a patent ductus arteriosus causes a left to right shunt that leads to left, not right, ventricular volume loading. The size of the shunt is determined by flow resistance, diameter, shape and configuration of the patent ductus arteriosus, which determines whether the shunt causes pulmonary circulation and left ventricular volume load. Over time, progressive increase in pulmonary vascular resistance surpasses systemic vascular resistance and causes ductal shunt reversal from right to left (Eisenmenger's syndrome).

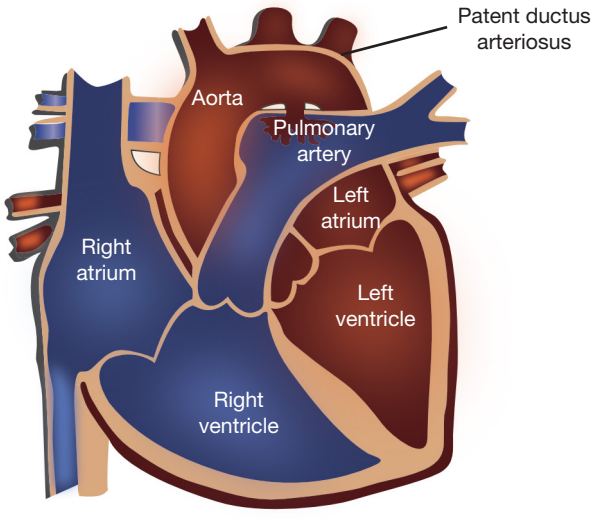

Figure 7. An illustration of a patent ductus arteriosus.

Clinical presentation

Symptoms vary according to the degree of shunting across the patent ductus arteriosus and the patient may present with an incidental murmur or, rarely, infective endocarditis. The classical finding on examination is a continuous 'machinery' murmur at the upper left sternal edge radiating into the back and. Occasionally, a thrill may be palpable (Schneider and Moore, 2006).

Diagnosis

An electrocardiogram is usually normal, or may show left ventricle hypertrophy, whereas a transthoracic echocardiography will ascertain the degree of left ventricular volume overload, pulmonary arterial pressure, pulmonary artery size and the presence of any changes to the right side of the heart (Figure 8) (Baumgartner et al, 2021). Cardiac magnetic resonance imaging can be used to quantify pulmonary to systemic flow and obtain detailed volumetric studies, as well as for precise imaging of anatomy as part of the work up for surgical or catheter intervention (Baumgartner et al, 2021). Cardiac catheterisation can be performed, but it is not an essential investigation as cardiac magnetic resonance imaging can provide enough information about the shunt itself. Angiography is used to study the ductal anatomy in detail and, in combination with assessment of haemodynamics by temporary test occlusion of the shunt with a balloon catheter, guides decision making on the modality of closure.

Management

In adults, device closure using a catheter is the preferred modality. Surgical closure is reserved for rare cases in which the duct is too large and not amenable to device closure, or in the event of aneurysm formation (Baumgartner et al, 2021). Patients with evidence of left ventricular overload but no pulmonary arterial hypertension should be considered for catheter intervention, regardless of the symptom burden. Patients with Eisenmenger physiology are not considered for shunt closure and unfortunately, once the defect reaches this stage, the patient's pathology is irreversible.

Follow up

An echocardiogram should be done 6 months post-intervention. Patients with no residual shunt, normal left ventricle volume load and normal pulmonary arterial pressure do not need regular follow up. Those patients with abnormal left ventricle volume load or residual

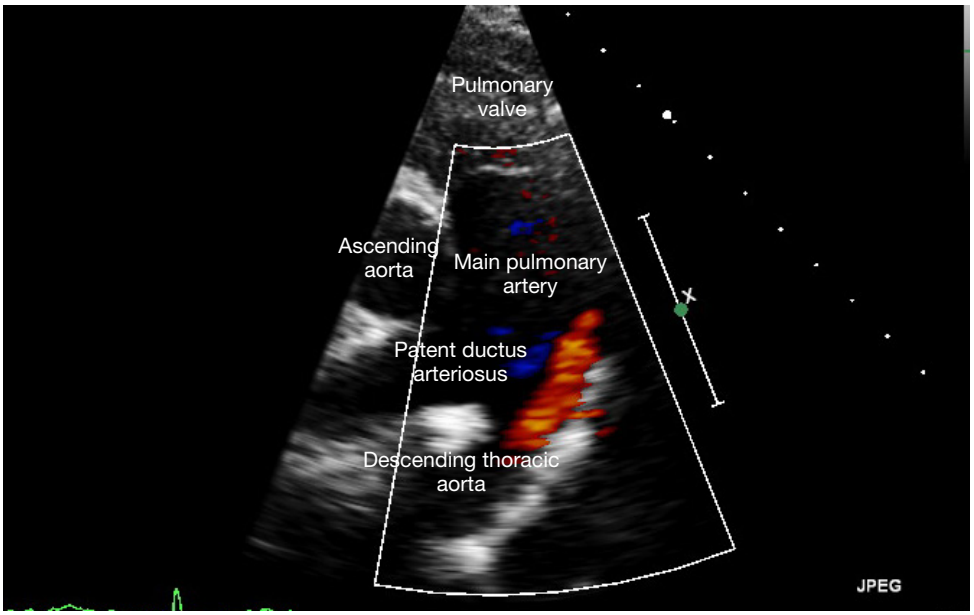

Figure 8. A two-dimensional transthoracic echocardiogram (modified parasternal short-axis right ventricular outflow tract view) with colour compare showing widely patent ductus arteriosus along with pulmonary valve, with Doppler colour flow (red) demonstrating aortic to main pulmonary artery shunt (left to right).

pulmonary arterial hypertension should be followed up on a 1–3-yearly basis depending on the severity of the residual pathology.

Sequelae

Patients may remain completely asymptomatic or, at the other end of the spectrum, develop congestive cardiac failure and eventually Eisenmenger's syndrome.

Coarctation of the aorta

Coarctation of the aorta occurs in 4–6% of infants affected by congenital heart disease (Suradi and Hijazi, 2015). It is generally considered to be an arteriopathy, as abnormal histological changes have been noted proximal and distal to the coarctation of the aorta site (Baumgartner et al, 2021). There is a significant variation in the anatomical presentation of coarctation of the aorta, ranging from a discrete mild narrowing to long hypoplastic segments of the aorta (Figure 9). Coarctation of the aorta is associated with other lesions in the heart, including the bicuspid aortic valve (50–75% of cases), ascending aortic aneurysm, subaortic and supravalvular aortic stenosis (Alkashkari et al, 2019), and inherited diseases such as Turner's, Noonan's, DiGeorge and Williams–Beuren syndrome (Baumgartner et al, 2021).

Pathophysiology

The narrowing of the aorta is typically distal to the left subclavian artery, near the ductus arteriosus. It is hypothesised that coarctation of the aorta originates when ductal tissue blends into the aortic tissue, causing an aortic constriction when the ductus closes shortly after birth. This constriction results in the abnormal distribution of flow, where the proximal segment of the aorta and upper body receives normal blood flow, with diminished flow in the distal segment and the lower body. In turn, this results in significant blood pressure elevation proximal to the site of the coarctation, which if left untreated, leads to the development of arterial collaterals as an alternative pathway for blood flow to bypass the narrowing.

Clinical presentation

The clinical presentation varies from a neonate in critical heart failure to an asymptomatic adult with incidental hypertension. This is largely caused by the variation in anatomy

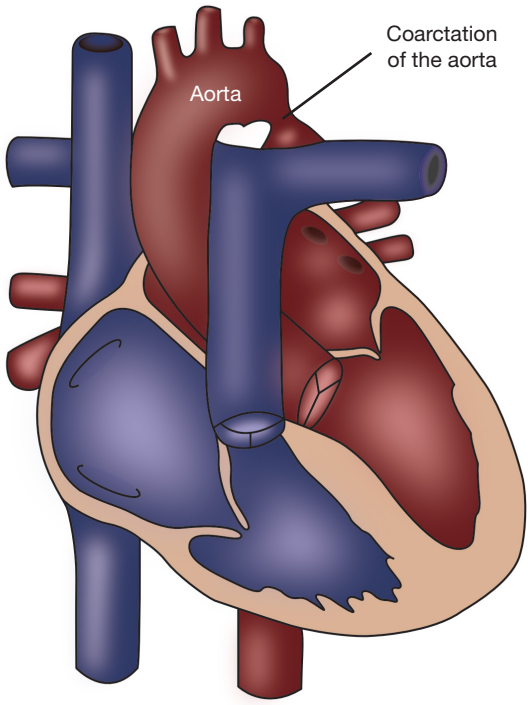

Figure 9. An illustration of an aortic coarctation.

between patients. For adults presenting with coarctation of the aorta, the key symptoms that clinicians should be vigilant for and aware of include headaches, epistaxis, dizziness, cold feet and claudication (Baumgartner et al, 2021). Examination findings may include a radiofemoral delay, suprasternal thrill, and interscapular or continuous murmur because of collaterals (Rhodes et al, 2008).

Diagnosis

Diagnosis may be incidental or, when there is significantly aberrant anatomy, may occur at birth. Blood pressure measurements should be taken in the upper and lower extremities, and a systolic gradient of ≥20 mmHg is indicative of coarctation of the aorta. Ambulatory blood pressure monitoring in the right arm is recommended to confirm systemic arterial hypertension, which is defined as a 24-hour mean systolic reading of >130 mmHg and/or a diastolic reading of >80 mmHg. A chest X-ray can demonstrate rib notching of the third to fourth ribs owing to the formation of collaterals (Rhodes et al, 2008). Echocardiography can be a useful investigation, depending on the site and extent of the coarctation of the aorta, and provides valuable information regarding left ventricular function, left ventricular hypertrophy and associated congenital heart defects.

For the purposes of quantification, cardiac magnetic resonance imaging and cardiac computed tomography are the preferred modality of imaging. These allow three-dimensional reconstruction of the location, length and diameter of coarctation of the aorta, along with the examination of the aortic arch, head and neck vessels, and the detection of collaterals. Other complications can also be readily detected with these imaging modalities, including aneurysms and residual stenosis post-treatment. Cardiac catheterisation is a more invasive investigation, but provides accurate measurements of gradients in the absence of mature collaterals. A peak-to-peak gradient of >20 mmHg is indicative of a significant coarctation of the aorta. Cardiac catheterisation can be useful in ascertaining the need for intervention, assessing results post-intervention and confirmation of restenosis where clinically suspected.

Management

The management of patients with coarctation of the aorta is dependent on three factors: the presence of arterial hypertension, the peak-to-peak gradient and the degree of narrowing of the aorta. In native coarctation of the aorta and re-coarctation after surgery, the treatment of choice is percutaneous stenting with covered stents (**Figure 10**), which have shown better long-term outcomes and have fewer short-term complications (Baumgartner et al, 2021). Balloon angioplasty is only used to re-dilate previously stented areas of the aorta. There have been numerous surgical techniques used, including subclavian flap, Gore-Tex patch and end–end repair, but this is predominantly in paediatric cases, with only interposition grafts and bypass tube grafts typically being used in adults.

Follow up

All patients with coarctation of the aorta should be followed up at least on a yearly basis (Baumgartner et al, 2021). Longer follow-up intervals may be considered for patients whose coarctation of the aorta has been repaired if there are no associated lesions or complications, such as hypertension or dilatation of the ascending aorta. A cardiac magnetic resonance image of the aorta post-intervention should be performed to provide baseline data on the anatomy and to keep a record of any complications. Repeated cross-sectional imaging should be considered on a 3–5-yearly basis depending on the anatomy (Baumgartner et al, 2021).

Sequelae

Adolescents with coarctation of the aorta have good survival outcomes up to 60 years of age. However, the burden of morbidity can be high when complications arise from coarctation of the aorta. These are a consequence of long-standing hypertension and include premature coronary artery disease, stroke, heart failure and aortic dissection. Other complications include left ventricular failure or intracranial haemorrhage caused by a Berry aneurysm. Up to 70% of patients with coarctation of the aorta will also have an associated bicuspid aortic valve, but only 7% of patients with a bicuspid aortic valve will have coarctation of the

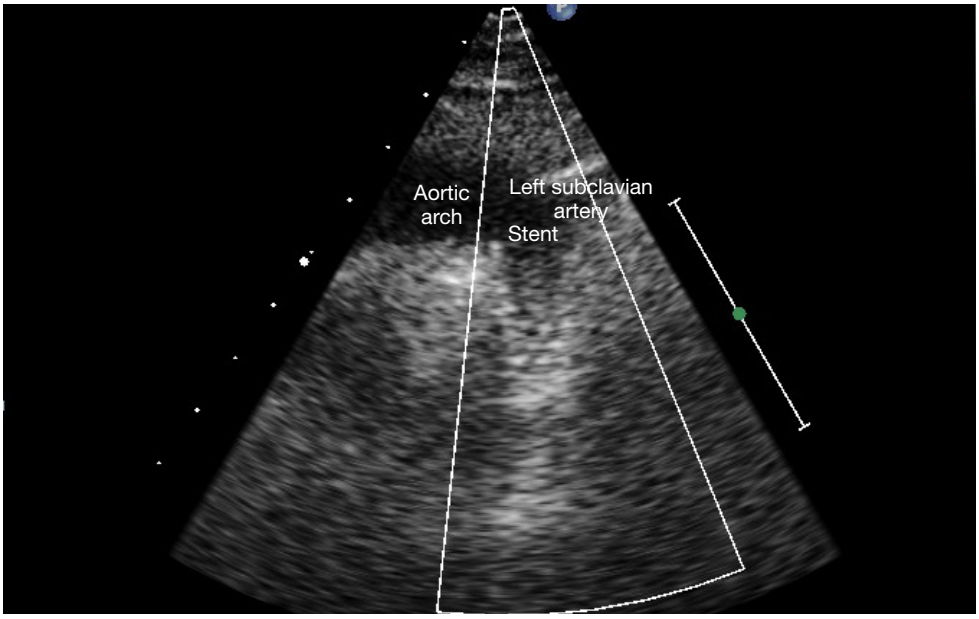

Figure 10. A two-dimensional transthoracic echocardiogram (suprasternal aortic arch view) showing a well deployed covered coarctation stent in the descending thoracic aorta, with prominent horizontal stent struts visible, just distal to the left subclavian artery.

aorta. This is because a bicuspid aortic valve has a high background prevalence compared to coarctation of the aorta (Sinning et al, 2018).

Conclusions

The population of adults living with congenital heart disease is ever expanding. These patients are living longer because of advances in surgical and catheter-based interventions and are thus more likely to present to a general medical clinician with complications from their congenital heart disease or other illnesses commonly seen in the ageing population. A working knowledge of simple congenital cardiac lesions is important for the general physician on the acute take. With this article, the authors hope to instill confidence in clinicians managing patients with simple lesions, provide an understanding of the underpinning pathophysiology and familiarise readers with the nomenclature.

Author details

[1]Department of Adult Congenital Heart Disease, Leeds General Infirmary, Leeds, UK

[2]Department of Cardiology, Doncaster Royal Infirmary, Doncaster, UK

Conflicts of interest

The authors declare that there are no conflicts of interest.

Key points

- Atrial septal defects result in a volume load of the right ventricle, whereas ventricular septal defects lead to a volume load of the left ventricle.
- A patent ductus arteriosus acts just like a ventricular septal defect, with a left to right shunt, leading to volume load of the left ventricle.
- Even after repair, patients with coarctation of the aorta may still be hypertensive and require lifelong medical treatment.
- A significant majority of patients with coarctation of the aorta will also have a bicuspid aortic valve, but only a small minority of patients with a bicuspid aortic valve will have coarctation of the aorta.

References

Alkashkari W, Albumgami S, Hijazi ZM. Management of coarctation of the aorta in adult patients: state of the art. Korean Circ J. 2019;49(4):298–313. https://doi.org/10.4070/kcj.2018.0433

Baumgartner H, De Backer J, Babu-Narayan SV et al. 2020 ESC Guidelines for the management of adult congenital heart disease. Eur Heart J. 2021;42(6):563-645. https://doi.org/10.1093/eurheartj/ehaa554

Butera G, Carminati M, Chessa M et al. Percutaneous versus surgical closure of secundum atrial septal defect: comparison of early results and complications. Am Heart J. 2006;151(1):228–234. https://doi.org/10.1016/j.ahj.2005.02.051

Gabriel HM, Heger M, Innerhofer P et al. Long-term outcome of patients with ventricular septal defect considered not to require surgical closure during childhood. J Am Coll Cardiol. 2002;39(6):1066–1071. https://doi.org/10.1016/s0735-1097(02)01706-0

Lloyd TR, Beekman RH III. Clinically silent patent ductus arteriosus. Am Heart J. 1994;127(6):1664–1665. https://doi.org/10.1016/0002-8703(94)90415-4

Marelli AJ, Mackie AS, Ionescu-Ittu R et al. Congenital heart disease in the general population: changing prevalence and age distribution. Circulation. 2007;115(2):163–172. https://doi.org/10.1161/circulationaha.106.627224

Martin SS, Shapiro EP, Mukherjee M. Atrial septal defects – clinical manifestations, echo assessment and intervention. Clin Med Insights Cardiol. 2015;8(Suppl 1):93–98. https://doi.org/10.4137/CMC.S15715

Morray BH. Ventricular septal defect closure devices, techniques, and outcomes. Interv Cardiol Clin. 2019;8(1):1–10. https://doi.org/10.1016/j.iccl.2018.08.002

Naqvi N, McCarthy KP, Ho SW. Anatomy of the atrial septum and interatrial communications. J Thorac Dis. 2018;10(Suppl 24):S2837–S2847. https://doi.org/10.21037/jtd.2018.02.18

Rhodes JF, Hijazi ZM, Sommer RJ. Pathophysiology of congenital heart disease in the adult, part II: simple obstructive lesions. Circulation. 2008;117(9):1228–1237. https://doi.org/10.1161/CIRCULATIONAHA.107.742072

Schneider M, Moore JW. Patent ductus arteriosus. Circulation. 2006;114(17):1873-1882. https://doi.org/10.1161/circulationaha.105.592063

Sinning C, Zengin E, Kozlik-Feldmann R et al. Bicuspid aortic valve and aortic coarctation in congenital heart disease—Important aspects for treatment with focus on aortic vasculopathy. Cardiovasc Diagn Ther. 2018;8(6):780–788. https://doi.org/10.21037/cdt.2018.09.20

Suradi H, Hijazi ZM. Current management of coarctation of the aorta. Glob Cardiol Sci Pract. 2015;(4):44. http:// doi.org/10.5339/gcsp.2015.44

van der Linde D, Konings EEM, Slager MA et al. Birth prevalence of congenital heart disease worldwide. J Am Coll Cardiol. 2011;58(21):2241–2247. https://doi.org/10.1016/j.jacc.2011.08.025