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Atrial Septal Defect, Diagnosis, and Treatment Options

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Abstract: **We aimed by this review to discuss the diagnostic procedures and treatment options for Atrial septal defect (ASD). We also aimed to review the incident, pathophysiology and causes of ASD.**
Narrative review of the literature was performed using medical databases; PubMed, and Embase, searching relevant studies reporting data on the treatment and diagnosis of Atrial septal defect (ASD), searched was performed up to January 2017, we restricted our search to only English language and human subject, furthermore, we search the references included in each study to be able to find more relative articles. Atrial septal defect (ASD) are the 3rd most common type of congenital heart disease. Consisted of in this group of malformations are a number of kinds of atrial communications that permit shunting of blood in between the systemic and the pulmonary blood circulations. Most children with separated atrial septal flaws are free of symptoms, however the rates of workout intolerance, atrial tachyarrhythmias, right ventricular dysfunction, and pulmonary hypertension increase with advancing age and life expectancy is decreased in adults with untreated defects. The risk of development of pulmonary vascular disease, a potentially deadly complication, is higher in female patients and in older adults with untreated problems. When done before age 25 years is associated with regular life expectancy, Surgical closure is safe and reliable and.

Keywords: **Atrial septal defect (ASD), English language and human subject.**

1. INTRODUCTION

Atrial septal defect (ASD) is a form of congenital heart flaw that enables blood flow in between 2 compartments of the heart called the left and right atria. Typically, the right and left atria are separated by a septum called the interatrial septum. If this septum is malfunctioning or absent, then oxygen-rich blood can flow straight from the left side of the heart to combine with the oxygen-poor blood in the best side of the heart, or vice versa [(1)]. ASD is among the most typical congenital heart defects and accounts for approximately 6-- 10% of all congenital cardiac flaws [(2)]. Young children with ASD are typically asymptomatic and might wait for optional surgical or catheter-based closure for 3 years or more [(3,4)]. Shunts from ASD are typically identified in childhood or young their adult years. The defect is generally well endured in infants and young children, however symptoms of mild tiredness, bad growth or dyspnoea on exertion may exist [(5)]. Early closure might be beneficial for such infants. If the ASD is little, it may not need any intervention, or it may close on its own. ASD, and for that reason the shunt, tends to increase with age, and repair normally happens when ASD is detected [(5)]. In asymptomatic patients with a large left-to-right shunt, surgical closure needs to be performed in between 2 and 5 years of age [(6)]. ASD is not without morbidity. About 40% of such patients may provide with cryptogenic stroke at their adult years; paradoxical embolism has actually also been reported [(7)]. Surgical repair of ASD is a reputable treatment and is very safe, with a minimal mortality rate [(8)]. Transcatheter occlusion of an ASD was first described by King and associates in 1976 [(9)].

We aimed by this review to discuss the diagnostic procedures and treatment options for Atrial septal defect (ASD). We also aimed to review the incident, pathophysiology and causes of ASD.

2. METHODOLOGY

Narrative review of the literature was performed using medical databases; PubMed, and Embase, searching relevant studies reporting data on the treatment and diagnosis of Atrial septal defect (ASD), searched was performed up to January 2017, we restricted our search to only English language and human subject, furthermore, we search the references included in each study to be able to find more relative articles.

3. RESULTS

- **Incidence and causes and pathophysiology:**

Flaws of the atrial septum are the 3rd most common kind of congenital heart disease, with an approximated occurrence of 56 per 100 000 livebirths [(11)]. With improved acknowledgment of medically quiet flaws by echocardiography, current quotes are about 100 per 100 000 livebirths [(12)]. About 65 - 70% of patients with a secundum defect, approximately 50% of those with a primum atrial septal problem, and 40-- 50% of those with a sinus venosus problem are female. Many atrial septal flaws are sporadic with no identifiable cause. Reports of familial clusters of secundum problems have noted different modes of inheritance, most significantly autosomal dominant [(13,14)]. Irregularities in genes necessary to heart septation have actually been associated with atrial septal problems, including anomalies in the heart transcription factor gene GATA4, nkx2-5 and tbx5, MYH6 situated on chromosome 14q12 and other anomalies [(15,16,17)]. The association between secundum problems and conduction problems, specifically atrioventricular block, has actually been connected to anomalies in NKX2-5 [(17)]. The risk of a secundum problem is increased in families with history of congenital heart disease, specifically when an atrial septal problem exists in a sibling. Secundum problems are often experienced in hereditary syndromes such as Holt-Oram, Ellis van Creveld, Noonan, Down, Budd-Chiari, and Jarcho-Levine, to mention only a few [(14,16)]. In Holt Oram syndrome (NKX2-5 mutation), an atrial septal flaw is seen in 66% of cases. In patients with trisomy [(18,19)], secundum and primum defects are the most regular sores, accounting for 42% and 39% of major congenital heart disease, respectively [(20)]. Direct exposure to several compounds has actually been associated with atrial septal defects, including fetal alcohol syndrome, very first trimester maternal cigarette intake, and some antidepressants [(21)]. Other maternal risk factors include diabetes, increased dietary glycaemic index in females without diabetes, and advanced maternal age (≥ 35 years) [(22)].

The magnitude of and direction of circulation through any ASD depend on the size of the problem and the relative diastolic filling properties of the ideal and left ventricles [(23)]. Conditions that cause decreased left ventricular compliance (eg, left ventricular hypertrophy or scarring) and mitral stenosis will increase left-to-right shunting. Conditions that trigger decreased best ventricular compliance (eg, lung hypertension or pulmonary stenosis) and tricuspid stenosis will have the opposite result of lowering a left-to-right shunt and/or triggering a right-to-left shunt. As a rule, an ASD must be at least 10 mm in size to carry a substantial left-to-right shunt, although many ASDs are not circular, and maximum size may be challenging to determine properly. A left-to-right atrial shunt is considered significant when the Qp/Qs ratio is greater than 1.5/ 1.0, or if it causes dilation of the right heart chambers [(23)]. Although there are restrictions in approximating Qp/Qs with any method, this level of shunting is typically connected with right heart dilation and has actually been associated with unfavorable long-term outcomes [(23)].

- **Anatomy overview:**

Patent foramen ovale:

Patent foramen ovale is the space between a strong (valve-competent) septum primum and a normally formed septum secundum (**Figure 1**). It is a regular interatrial interaction throughout fetal life, characterized by streaming of oxygen-rich circulation from the ductus venosus and, to a lesser degree, from the inferior vena cava, through the foramen ovale to the left atrium. After birth, left atrial pressure generally surpasses ideal atrial pressure and, septum primum opposes septum secundum, and the foramen ovale narrows. A patent foramen ovale is seen in almost all newborns, but its frequency decreases with advancing age [(24,25)]. Complete physiological closure of the foramen ovale takes place in 70 - 75% of grownups [(26)].

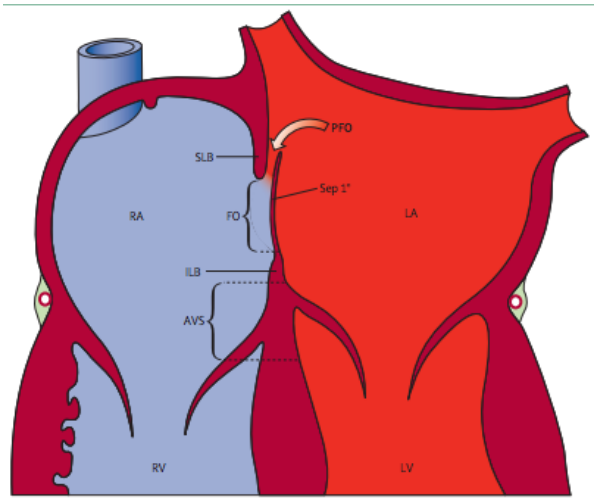

Figure 1: Atrial septal components and patent foramen ovale

Secundum atrial septal defect:

Secundum atrial septal problem is a defect within the fossa ovalis normally due to one or a number of defects within septum primum (**Figure 2**). Septum secundum is well-formed in many patients. Most secundum flaws are not confluent with the vena cavae, right pulmonary veins, coronary sinus, or the atrioventricular valves. With the exception of patent foramen ovale, secundum atrial septal defect is the most common cause of an atrial-level shunt. The size of secundum flaws differs from numerous millimeters to 2 - 3 cm. Big flaws are usually related to significant deficiency, or even complete lack, of septum primum [(25)].

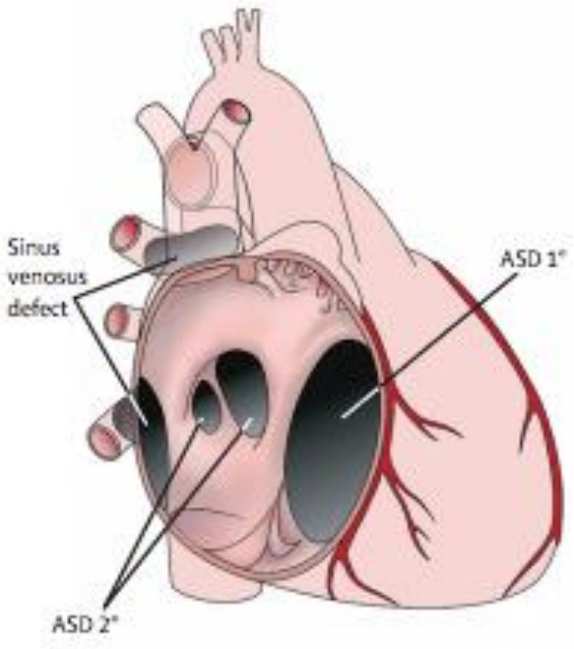

Figure 2: Anatomy of the atrial septum and neighbouring structures, Types of interatrial communications.

Primum atrial septal defect:

Primum atrial septal problem is among numerous variations of typical atrioventricular canal defects (likewise described atrioventricular septal problem) with an interatrial interaction situated in between the anterior-inferior margin of the fossa ovalis and the atrioventricular valves. The defect is defined by a common atrioventricular orifice with two unique atrioventricular valve annuli finished by valve tissue adhering to the crest of the ventricular septum. The atrioventricular tissue occludes the space that accounts for the ventricular septal flaw component in the complete form of the malformation (**Figure 2**). In addition to the septal flaw, the atrioventricular valves in this abnormality are usually abnormal, including a cleft in the anterior mitral brochure [(25,26)].

- **Diagnosis of ASD:**

The remarkable form of the sinus venosus ASD constitutes 5% to 10% of all ASDs. Its posterior aspect is the right atrial complimentary wall, and its remarkable border is typically missing because of an overriding superior vena cava (**Figure 3 A**). Anomalous connection of some or all the best pulmonary veins to the SVC or the best atrium is very common. Diagnosis is often harder than for other forms of ASD and might require unique imaging, such as transesophageal echocardiography, magnetic resonance imaging (MRI), and computed tomographic scanning, and the possibility of a sinus venosus ASD should be thought about for any patient with inexplicable right atrial and right ventricular dilation. Catheter closure is not possible, and the treatment is surgical [16,19].

Chest Radiograph:

The chest x-ray movie is frequently, however not always, unusual in patients with significant ASDs [27,28]. Cardiomegaly may exist from best heart dilation and occasionally from left heart dilation if substantial mitral regurgitation is present in the patient with an ostium primum ASD. Right heart dilation is better appreciated in lateral movies. The central pulmonary arteries are characteristically bigger, with pulmonary variety showing increased pulmonary flow. A little aortic knuckle is characteristic, which shows a chronically low systemic heart output state, due to the fact that increased pulmonary flow in these patients occurs at the expense of reduced systemic flow [27,28].

Echocardiography:

Transthoracic echocardiography documents the type(s) and size of the ASD(s), the instructions(s) of the shunt, and, in knowledgeable hands, the existence of anomalous lung venous return. The functional importance of the defect can be estimated by the size of the right atrium and ventricle, the presence/absence of paradoxical septal motion (best ventricular volume overload), ventricular septal orientation in diastole (volume overload) and systole (pressure overload), and an estimation of the shunt ratio (based on aortic and pulmonary flows). Lung artery systolic pressures might be approximated from the Doppler velocity of tricuspid regurgitation. In a patient with a primum ASD, the left AV valve is trileaflet (**Figure 3B**) and almost always demonstrates some regurgitation. Transesophageal echocardiography may be useful to confirm the type of ASD and to delineate the pulmonary venous return. It is likewise typically utilized in support of device closure of ASDs [13,19].

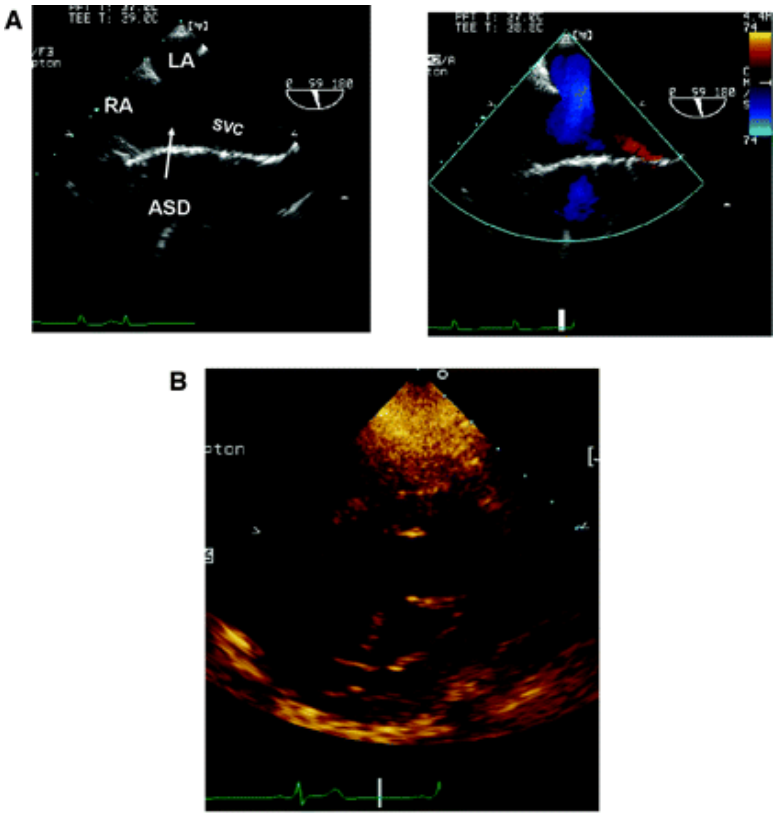

Figure 3: (A) Sinus venosus ASD (superior); vertical section from a transesophageal echocardiogram. Note the SVC overriding the ASD. (B) Trileaflet left AV valve in a patient with a primum ASD or partial AV septal defect (short axis).

MRI&CT scans for diagnosis of ASD:

Cardiac MRI may be useful and may give the same type of information that echocardiography can provide. It is seen as providing the "gold standard" for the assessment of right ventricular size and function, and it may help define whether the right heart chambers are in fact enlarged (**Figure 4**). MRI is also excellent at assessing pulmonary venous return (29). In patients who cannot have an MRI, computed tomographic scanning and angiography can offer similar information (29).

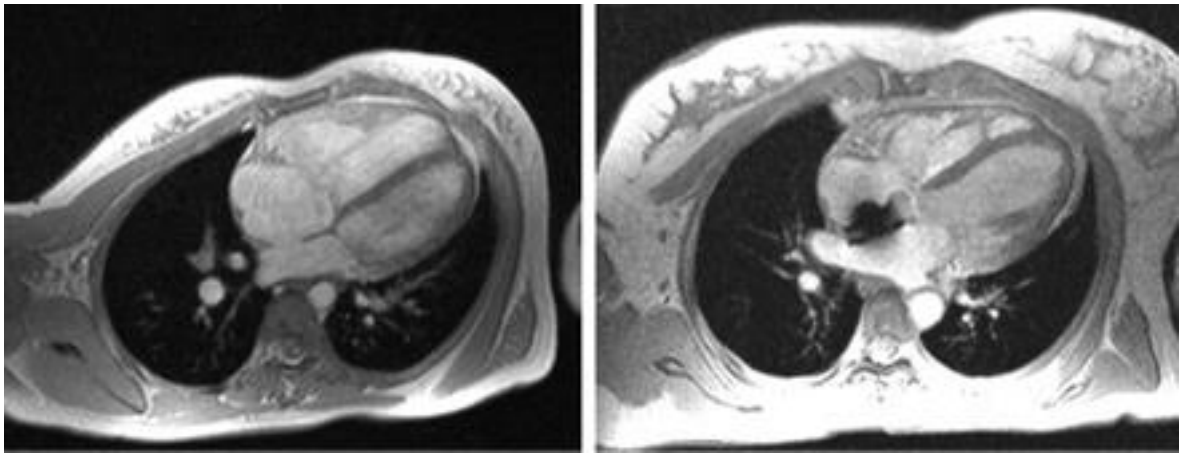

Figure 4: Cardiac MRI showing right heart remodeling after closure of a secundum ASD with an Amplatzer device.

- **Management approaches of ASD:**

As a basic rule, patients with a considerable ASD as specified above (with indications of right heart dilation) ought to be offered optional closure soon after the medical diagnosis is developed, irrespective of age (**Table 1**) [(30)]. There can be, however, numerous factors for not closing an ASD:

Table 1: Management of Atrial Septal Defects in Adults

CT indicates computed tomography.
Indications for ASD closure
Right atrial and right ventricular dilation by echocardiography, MRI, or CT (in the presence of an ASD and in the absence of advanced pulmonary arterial hypertension) manifested with 1 or more of the following:
ASD minimum diameter >10 mm on echocardiography
Qp:Qs >1.5:1 by echocardiographic or cardiac MRI flow assessment, or from oxygen saturation runs, when cardiac catheterization is performed (for other reasons)
Anticipated benefits from ASD closure
Improved functional class, dyspnea index, and exercise capacity (irrespective of age). Improvement occurs earlier after device closure than with surgical closure. Physical reconditioning is recommended.
In addition, the following long-term prognostic benefits can be anticipated:
Improved survival after youthful repair[22]
Improved quality of life
Prevention of right heart failure

Prevention of pulmonary arterial hypertension
Potential midterm/long-term complications after ASD closure in adulthood
Tachyarrhythmia (atrial flutter or atrial fibrillation) may persist or develop in the older patient; tachyarrhythmia should be better tolerated and easier to manage after ASD closure. Consider arrhythmia-targeting intervention (surgical or catheter) either before or at the time of ASD closure for high-risk patients and those with preexisting sustained tachyarrhythmia.
Bradyarrhythmia, potentially leading to permanent pacing:
caused by sinus node dysfunction, secondary to longstanding right atrial dilation and stretch (among patients who underwent late ASD closure)
caused by complete heart block, to which patients with AV septal defects (any AV septal defect including primum ASD) are predisposed.
Stroke risk higher in older patients. Consider empiric thromboprophylaxis for patients >40 years of age and those who required complex repair.
Residual ASDs
Small: relatively common after catheter device closure (most are hemodynamically insignificant and usually close spontaneously over a period of 12 months from intervention)
Large: may be caused by a dehisced ASD patch (good practice to review all patients at least once, in the year after ASD closure, to confirm the absence of residual atrial communications; ASD dehiscence leading to hemodynamically important atrial communication is unlikely to occur thereafter)
Right heart failure or progressive pulmonary arterial hypertension; overall risk is small and inversely related to age of patient at time of ASD closure
Left AV valve regurgitation and subaortic stenosis (seen primarily in patients with primum ASDs)
Device migration or erosion (the latter when very large devices are used); both rare
Left atrial hypertension and pulmonary venous congestion; a very uncommon complication that can be seen soon after ASD closure in the occasional older patient with poor left ventricular compliance (which in itself can be difficult to delineate before ASD closure)

Surgical intervention for ASD:

Surgical closure is required for patients with ostium primum and sinus venosus ASDs, in addition to for patients with secundum ASDs whose anatomy disagrees for device closure. In some settings, surgical closure of secundum defects is still preferred or needed [(31)]. A secundum ASD may be closed with direct stitches ("primary closure") or with a patch utilizing pericardium or artificial material. Ostium primum problems require spot closure and repair of the "cleft" AV valve. The repair work of sinus venosus flaws with anomalous lung venous return can be technically difficult, and a number of techniques are utilized to accomplish this [(32,33)]. Care must be required to see that the lower end of the SVC is large enough to accommodate both the SVC and the lung venous return being baffled to the left atrium. 2 separate channels may be produced to make sure these 2 sources of venous return are unblocked.

In many centers in the industrialized world, device closure has actually ended up being the treatment of option for secundum ASDs. The procedure is supported by transesophageal or intracardiac echocardiography [(34)]. Catheter closure decreases medical facility stay and healing, avoids surgical injuries and their prospective complications, and

communicates the exact same hemodynamic advantages as does surgical treatment. Indications for catheter closure are the same as for surgical closure, however patient choice requirements are more narrowly specified. Patients with an extended secundum ASD > 36 mm, those with inadequate atrial septal rims to permit steady device release, or those with proximity of the defect to the AV valves, the coronary sinus, or the vena cavae are generally referred for surgical repair. Device closure is a safe and reliable procedure in skilled hands, with significant issues such as cardiac perforation or gadget embolization happening in less than 1% of patients [35,36]. Successful closure is achieved in approximately 95% of patients, [37] although little recurring shunts are frequently seen on echocardiography at the end of the treatment; these are not hemodynamically important, and the majority of will close spontaneously within 1 year. Device closure of secundum ASDs can produce fast and favorable cardiac improvement, as described below. There is no consensus regarding exactly what constitutes proper follow-up of patients after ASD device closure. Late problems appear unusual, there is the capacity for mitral valve dysfunction, obstruction to lung and systemic venous pathways, and erosion or perforation of the atrial wall or aorta [33,37].

4. CONCLUSION

Atrial septal defects are the 3rd most common type of congenital heart disease. Consisted of in this group of malformations are a number of kinds of atrial communications that permit shunting of blood in between the systemic and the pulmonary blood circulations. Most children with separated atrial septal flaws are free of symptoms, however the rates of workout intolerance, atrial tachyarrhythmias, right ventricular dysfunction, and pulmonary hypertension increase with advancing age and life expectancy is decreased in adults with untreated defects. The risk of development of pulmonary vascular disease, a potentially deadly complication, is higher in female patients and in older adults with untreated problems. When done before age 25 years is associated with regular life expectancy, Surgical closure is safe and reliable.

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