"SURGICAL CLOSURE OF ATRIAL SEPTAL DEFECT (ASD) – A COMPARATIVE STUDY OF DIRECT SUTURE CLOSURE AND PERICARDIAL PATCH CLOSURE TECHNIQUE"

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CARDIO VASCULAR AND THORACIC SURGERY

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CHENNAI – 600 003.

CERTIFICATE

This is to certify that the dissertation entitled **"SURGICAL CLOSURE OF ATRIAL SEPTAL DEFECT (ASD) – A COMPARATIVE STUDY OF DIRECT SUTURE CLOSURE AND PERICARDIAL PATCH CLOSURE TECHNIQUE"** presented here is the original work done by **Dr. SHEGU G,** in the department of Cardio Thoracic Surgery, Rajiv Gandhi Government General Hospital, Madras Medical college, Chennai 600003, in partial fulfillment of the University rules and regulations for the award of **Branch I *M.Ch Cardio Vascular and Thoracic Surgery*** degree under our guidance and supervision during the academic period from 2011 - 2013.

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DECLARATION

I, **Dr.SHEGU.G,** hereby solemnly declare that this dissertation titled **"SURGICAL CLOSURE OF ATRIAL SEPTAL DEFECT (ASD) – A COMPARATIVE STUDY OF DIRECT SUTURE CLOSURE AND PERICARDIAL PATCH CLOSURE TECHNIQUE"** was done by me in the Department of Cardio Thoracic Surgery, Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai-3 during the period from Jan 2011 to Dec 2012 under the guidance and supervision of **Prof.Dr.T.S.MANOHARAN, MS., M.Ch.,** This dissertation is submitted to the Tamil Nadu Dr.M.G.R.Medical University towards the partial fulfillment of requirement for the award of M.Ch Degree in Cardio Thoracic Surgery.

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INDEX

ABBREVIATION

ASD	:	Atrial Septal Defect
PPC	:	Pericardial Patch Closure
DC	:	Direct Closure
MR	:	Mitral Regurgitation
TR	:	Tricuspid Regurgitation
PAP	:	Pulmonary Artery Pressure
AR	:	Aortic Regurgitation
PHT	:	Pulmonary Hypertension
LVD	:	Left Ventricular Dimension
RVD	:	Right Ventricular Dimension
MVP	:	Mitral Valve Prolapse

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Dear Dr.Shegu G,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Surgical closure of Atrial Septal Defect (ASD) – A Comparative study of direct suture and pericardial patch closure technique" No.08122012.

The following members of Ethics Committee were present in the meeting held on 11.12.2012 conducted at Madras Medical College, Chennai -3.

1. Dr.S.K.Rajan, M.D.FRCP, DSc --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
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We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini 21/12/12
Member Secretary, Ethics Committee

INTRODUCTION

Atrial septal defect (ASD) accounts for one third of congenital heart disease in adults. 90% of the ASDs are fossa ovalis defect. Natural History is death by fourth or fifth decade when not repaired and when repaired at an earlier stage the survival approaches the rate for that of the normal population. Surgical closure of the ASD has a low morbidity and mortality. Surgical closure is usually accomplished by two methods, direct suture closure and pericardial patch closure. Cardiac surgeons preference to choose either of these two techniques to close the ASDs are very much variable. Small to moderate sized ASD were closed using direct suture technique and large ASDs were closed using pericardial patch technique.

In the present study, we tried to identify those characteristics of the ASD and the surgical outcomes when either of these two techniques were involved. It is a retrospective study of two groups of patients, the direct closure group and pericardial patch closure group.

A comparison is made between these two groups in terms of –

1) The mortality and morbidity associated with each group
2) Associated anomalies in both the groups
3) The amount of blood transfusion needed between the groups

4) To compare the effectiveness of symptom relief between each group
5) To compare the effect of these repairs on associated conditions like mitral regurgitation (MR), mitral valve prolapse (MVP) and tricuspid regurgitation (TR)
6) To compare the effects of these techniques on right ventricle, left ventricle and mitral valve geometry
7) To compare the cardiopulmonary bypass time, aortic cross clamp time and duration of surgery
8) To compare the differences in the duration of mechanical ventilation time and duration of hospital stay
9) To evaluate the postoperative complications like residual shunt and thromboembolism between the two groups

Atrial septal defect ostium secundum (ASD OS) accounts for 5- 10% of all congenital heart disease. The gold standard for ASD is surgical closure of the ASD. The surgeon has the advantage of closing the ASD invariable of its location, size and proximity to vital structures. The criterion standard in the treatment of atrial septal defect (ASD) is direct closure of the defect by using an open approach with extracorporeal support. [9]John Gibbon performed the first successful ASD closure by applying this method in 1953. Surgical

techniques and equipment have since improved to the point that the mortality rate from this repair approaches zero.

In the usual procedure, a median sternotomy incision is made, and the sternum is split in the midline. Direct arterial and double venous (superior vena cava and inferior vena cava) cannulation are performed. By applying cardiopulmonary bypass, the aorta is clamped, and the heart is arrested with a cardioplegia solution. The caval snares are tightened, and the right atrium is opened. Most secundum defects can be closed by using a direct continuous suture of 3-0 or 4-0 polypropylene (Prolene).

Caution must be taken when large defects are directly closed because this closure can distort the atrium. Large defects that rise superiorly can distort the aortic annulus if closed directly. These ASDs are best closed by using autologous pericardium or synthetic patches made of polyester polymer (Dacron) or polytetrafluoroethylene (PTFE). Care must be taken to completely remove any air or debris from the left atrium and

ventricle before cardiopulmonary bypass is discontinued. Temporary pacing wires are left in place on the right ventricle before the chest is closed over the drains.

In this present study, comparison between the outcomes of direct suture and pericardial patch closure of ASD were analysed. The Comparison of outcomes of both the surgical techniques- duration of the surgery, cardio pulmonary by pass time, changes in chamber geometry, aortic cross clamp time, need for blood products, thromboembolism, infections, other complications, duration of hospital stay and relief from symptoms will be studied.

AIM AND OBJECTIVES

AIM

- To compare the outcomes following direct suture and pericardial patch closure in Atrial septal defect

OBJECTIVE OF THE STUDY

- To compare the mortality and morbidity
- To identify associated anomalies in both groups
- To compare the effect of these techniques in those patients with Mitral valve prolapse (MVP) and Mitral regurgitation (MR) and Tricuspid regurgitation (TR)
- To compare the effects of these techniques in right ventricle and left ventricle chamber geometry
- To compare the complications (Residual shunt and Thromboembolism)
- To compare the effectiveness of the symptom relief
- To compare the duration of cardio pulmonary bypass time and surgery between the two groups
- To compare the differences in duration of ventilator time and Hospital stay

- To compare the amount of blood transfusion needed in the two groups

REVIEW OF LITERATURE

An ASD is a hole of variable size in the atrial septum. Recognition of ASD has been possible only in the last 60 years. By 1941, Bedford and colleagues had started to diagnose ASDs clinically. With the introduction of catheterization studies in 1950s a correct diagnosis was possible. With the invention of echocardiogram, one could easily visualize an ASD by the bedside without causing much difficulty for the patient.

In the initial period many ingenious methods were discovered for the closure of ASD. They were atrioseptopexy, external suturing, Sondergrads external suture closure and inflow stasis method.

Another interesting method discovered by Grass was the atrial well technique. With the invention of cardiopulmonary bypass machine by John gibbon in 1953, a golden era for cardiac surgery had started. Open heart surgery had become possible. He first used this technique and closed the ASD in a young woman using a pump oxygenator.

Embroyology:

Formation of the atrial septum starts in the 4th week of gestation. Two septae develop parallel to each other. The septum primum starts to develop as a crescentric septum at the postero superior aspect of the left of the atrial heart field. It grows towards the endocardial cushions which are separating the ventricles. The ostium primum is the gap between the endocardial cushion and the septum primum. The ostium secondum (OS) is formed when there is resorption of the septum primum's superior aspect. During this time the septum secondum starts to develop immediately rightward of the septum primum, it closes the ostium primum and circumscribes the fossa ovalis. The septum secondum's free edge acts as a flap valve for the fossa ovalis allowing only the right to left shunt.

Conditions that affect the formation of the valve or impairment of the valve results in persistent inter atrial communication. The secondum defect lies within the perimeter of the fossa ovalis and its morphology varies from a slit like patent foramen ovale (PFO) to a confluent defect involving a part or all of the fossae. ASD-OS can also form due to maldevelopment of the septum secondum or septum primum that results in inadequate closure of the ostium secondum.

Natural History:

PFO is present in 60% of newborns. At one year of life it is present in 92-99% of babies. Secondum ASD in children accounts for 10% of congenital heart disease and 20-40% of adult ASDs (**Sabiston et al**).

Spontaneous closure of ASD occurs only when the size of ASD is less than 4millimeters at infancy[10]. Spontaneous closure does not occur after three to four years of age. When the size is more than 10 millimeters at diagnosis then the ASD does not close spontaneously. Adults with ASD are symptomatic mostly and they would have already developed pulmonary hypertension. Even when symptoms are absent there is reduced exercise capacity. When adult ASDs are not repaired the life expectancy is reduced to 40 to 50 years. The incidence of pulmonary hypertension increases after 30 years of age in those patients who have an ASD.

In most adult patients with RV dysfunction, right ventricular volume overload is the best predictor of exercise capacity[8]. The shunt through the defect increases with age. Hemodynamically small ASDs need not be closed. Subacute endocarditis may occur in ASD patients but the incidence as such is very low. Atrial septal endocarditis has

occurred in patients with ASDs and also by septal involvement of endocarditis by endocarditis involving the other structures of the heart.

Associated defects:

ASDs can occur isolated. But, it is found many times to be associated with defects like ventricular septal defect (VSD), patent ductus arteriosis (PDA) and mitral valve prolapse (MVP). VSD was present in 18% of ASD patients. Left sided obstruction like mitral stenosis (MS) and aortic stenosis (AS) are present in 29% of patients with ASD. Right sided obstruction like pulmonary stenosis was present in 31% of ASD patients. ASD can be associated with mitral stenosis and dilated pulmonary artery, a condition termed Lutembacher's syndrome. This was more common in the era when rheumatic heart disease was common. Non rheumatic mitral stenosis can also be associated with ASD. Some patients with ostium primum ASD present with cleft anterior mitral leaflet. MR is associated with ASD and it is present 2.5- 10% of patients with large ASD. MVP is present in 20% of patients. MR in ASD patients occurs due to MVP. MVP is due to volume over load of right ventricle due to septal distortion which affects the mitral valve geometry. As an evidence of this, MVP and MR regress significantly after repair of the ASD.

Tricuspid regurgitation (TR) is present in most of the patients with large ASD. This is due to annular dilatation. This condition reverses after repair of the ASD. Other associated anomalies with ASD are VSD and patent ductus arteriosis (PDA) (1%), valvular pulmonary stenosis (VPS) (4%), left superior venacava (LSVC) (5%), peripheral pulmonary artery stenosis (1%) and azygoz extension of IVC (1%)[1].

'P' wave abnormalities usually accompany patients with ASD. P wave duration is increased. The P wave duration reduces after the repair of ASD in young patients implying that problem is due to chronic stretching of the atrial wall. Although in elderly patients and those who have prior paroxysmal atrial fibrillation this reversal does not occur. ASD is associated with syndromes like Noonan's syndrome where there is associated VPS also, Holt Oram syndrome and in trisomy 21 as the part of the endocardial cushion defect.

Pathophysiology

During the early neonatal period pulmonary vascular resistance is very high and the RV and LV chamber pressures are equal, hence only slight shunting of blood between the two chambers. With growth of the neonate the pulmonary vascular resistance starts falling and the left ventricle starts maturing and the left atrial pressure increases and the

shunting, hence there is shunting of blood from left to right. Increase in the shunt results in volume overload and hypertrophy of the right ventrticle and the flow through the pulmonary valve also increases. Due to volume overload of the RV the interventricular septum (IVS) bulges into the LV. This can impede LV filling and also causes systolic anterior motion (SAM) of the mitral valve. This results in LV dysfunction. RV hypertrophy causes reduced coronary reserve. LV dysfunction becomes more prominent with exercise and it is more often a diastolic dysfunction. This dysfunction reverts to normal 6 months after surgery.

Pulmonary vascular disease:

Pulmonary hypertension (PHT) develops in 35 – 40% of patients with ASD by the age of forty years. Development of pulmonary hypertension disease is not uniformly related to the age and size of the shunt. PHT can develop earlier in patients with trisomy 21 and premature infants. Histological examination of some ASD patients revealed intra-acinar and pre-acinar pulmonary vascular disease which suggests that this could have been a primary one or that the ASD could have been an incidental finding in patients with PHT disease.

In patients with ASD, due to increased pulmonary flow the pulmonary artery and arterioles dilate. As time progresses these

vessels significantly dilate to accommodate the increased flow in the pulmonary vascular system. This causes reduced flow velocity and apparently stagnation leading to pulmonary artery thrombosis. These thrombi can embolise to different parts of the lung and cause multiple infarctions. This leads to pulmonary vascular occlusive disease.

Clinical presentation:

Most ASD patients are asymptomatic. Symptoms are more common in the later stages of life. Symptoms of ASD are breathlessness, recurrent respiratory tract infections, syncope and palpitations. Breathlessness and palpitations are more common in the elderly, while in children, breathlessness is present only on severe exertion. Rarely chylothorax was the presenting feature of the ASD. Some ASD patients are cyanosed as in the case of Eisenmengar's syndrome where there reversal of the shunt. Some patients without pulmonary vascular disease also presents with cyanosis. Cyanosis may also be due to preferential flow of IVC blood into the large ASD, baffled by the Eustachian valve.

Physical examination:

Signs of RV volume overload and left to right shunt are present in patients with ASD. RV parasternal left, precordial bulge and shift of the apex to the left are also seen. Auscultatory findings are an ejection systolic flow murmur in the pulmonary area, an apical mid-diastolic murmur on inspiration due to increased flow across the tricuspid valve. A loud P2 component is present and a split S2 fixed throughout the respiratory cycle is prominent. The chest X ray shows an increased cardiothoracic ratio with prominent pulmonary vascular markings. Electrocardiography (ECG) shows RV hypertrophy, prolonged PR interval, RSR in V1 and incomplete right bundle branch block (RBBB). RV enlargement in ECG is seen in children. RBBB with right axis deviation is seen in ASD OS and RBBB with left axis deviation is seen in ostium primum ASD. Catheterisation studies are routinely performed for ASD patients. This study shows an oxygen step up at atrial level due to left to right shunt. A gradient of less than 25 mm Hg across the pulmonary valve is noted due to the increased flow.

Transthoracic echocardiogram (TTE) is sensitive, specific and widely available. The morphology and characteristics of the ASD can be evaluated. ASD is seen as an ECHO drop out of variable size in the

interatrial septum. Colour Doppler can be used study the direction of the shunt. Bubble contrast echocardiography is standardly used to diagnose smaller intra-cardiac shunts. High resolution computed tomography (HRCT) and magnetic resonance imaging (MRI) are also used to diagnose ASDs when it is difficult to diagnose ASD by ECHO. MRI helps in visualizing PAPVC lying adjacent to the lung and airways.

Clinical presentation of patients with ASD is dependent upon the type of defect, the magnitude of intracardiac shunt, and presence or absence of associated anomalies. The volume overload from interatrial shunting is generally well tolerated for long periods of time. Asymptomatic patients with small intracardiac shunt (Q_P:Q_S < 1.5:1), no cardiomegaly on chest X-ray, and only minimal enlargement of the right ventricle have little to no risk of developing symptoms or pulmonary vascular complications and repair is unnecessary. For patients with larger shunts (Q_P:Q_S > 1.5:1), onset of symptoms may be expected beyond the second decade of life. The magnitude of intracardiac shunting may increase with age as left ventricular compliance decreases. The most common presenting symptom is reduced exercise tolerance with dyspnea and fatigue resulting from chronic volume overload of the right ventricular. Beyond 30 years of

age, there is increasing risk of palpitations and arrhythmia resulting from atrial dilation. The onset of arrhythmia, either atrial flutter or fibrillation, usually causes a rapid deterioration in functional status because of loss of coordinated atrial contraction in the setting of right ventricular dysfunction. Patients who develop SVT will probably have persistent arrhythmia after successful ASD closure, especially if beyond 40 years of age. These patients should be considered for arrhythmia ablation during ASD repair.

Older patients with an unrepaired hemodynamically important ASD may develop pulmonary vascular occlusive disease, although this is far less common and delayed in comparison to patients with VSD. Pulmonary vascular disease leads to a reversal of intracardiac shunting and systemic desaturation. Overall, patients with unrepaired ASD with Q_P:Q_S > 1.5:1 have a decreased life expectancy with an average life expectancy of 45 to 50 years.

Associated Lesions

Frequently older patients with an ASD develop tricuspid insufficiency and subsequent atrial arrhythmia. Other long-term complications of ASD such as mitral valve incompetence, pulmonary valve complications, and systemic arterial hypertension may be

observed. Because of the potential for bidirectional shunting at the atrial level, patients with ASD are at increased risk of paradoxic embolization. Emboli within the systemic venous circulation that are normally cleared by the lungs may cross the ASD and enter the systemic arterial circulation. Cryptogenic stroke (i.e., a stroke with no source other than a paradoxic embolus) may occur in patients with patent foramen ovale (PFO).

SURGICAL INTERVENTION

Indications

The patients benefiting most from ASD closure are those for whom pulmonary hypertension will develop, but once pulmonary hypertension is present, surgical risk increases. This principle is the basis for the recommendation to close all significant ASDs. Elective closure of ASD is generally recommended when the Qp:Qs is 1.5:1 or greater, ideally performed at age 2 to 5 years, before exercise capacity changes, while chest wall compliance is optimal, and before school age[2]. An echo diagnosis of a significant defect with right ventricular volume overload is common and sufficient indication to close an ASD. Long-term follow-up data after surgical ASD closure show survival equal to the normal population when repair is performed early

in life, with age-related diminution in survival. Twenty-seven-year survival for those operated on after 40 years of age is only 40%.

Irreversible pulmonary hypertension is the only contraindication to ASD closure. It is important to consider that, in a high flow state, with a large Qp:Qs, high pulmonary artery pressure may not represent fixed pulmonary hypertension. Generally, irreversible pulmonary hypertension is characterized by a pulmonary vascular resistance (PVR) 8–12 wood units/m^2, with Qp:Qs <1.2:1, despite a vasodilator challenge.

Moderate pulmonary hypertension with a reactive component is not a contraindication to ASD closure, though pulmonary hypertension may progress in these patients regardless of closure. Guidelines for inoperability are largely based on VSD data. Generally, the PVR must fall below 7 U/m^2 with vasodilator therapy at cardiac catheterization for ASD closure risk to be less than prohibitive. Vasodilators used at cardiac catheterization to determine the reversible component of pulmonary hypertension include hyperoxia, inhaled nitric oxide, and isoproterenol.

Device versus Surgical Closure

A majority of ASDs today are closed by a variety of catheter-based devices, although the Amplatzer ASD occluder is the only Food and Drug Administration (FDA)-approved device at this time. The success rate and morbidity are nearly equal with the two approaches. Current published studies comparing device closure versus surgical closure with anatomically similar defects show a device success rate of 80% to 95.7%, compared with 95% to 100% success of surgical closure, though the success of device closures continues to evolve. Complications requiring treatment occur in 0% to 8% of device closures and 23% to 24% of surgical closures, and mean length of hospital stay is 1 day in the device group versus 3.4 days in the surgical group. Continual advances in the hardware and experience with device closures are improving the success rate of these catheter-based approaches.

Conflicting cost data currently fail to definitively favor device or surgical closure as the more cost-effective approach. The major cost at present for the surgically closed ASD is intensive care unit cost, whereas the major cost of the device closure is the device itself. Cost advantage may or may not favor device closure, particularly in light of

strategies of early extubation and accelerated postoperative management protocols.

Anatomical determinants that prohibit device closure remain the major indications for surgical ASD closure in the current era. Defects unsuitable for device closure include those that have failed attempted device closure, common atria or those without sufficient septal rim to engage the device, and sinus venosus defects for which device closure would threaten obstruction of pulmonary veins, IVC, or SVC. Anterior-inferior septal deficiency can be prohibitive of device closure, as the device can interfere with the tricuspid valve, mitral valve, or coronary sinus. Individual deficient septal rims, while originally constituting contraindication to device closure, no longer are absolute contraindications but may reduce success rates. The largest Amplatzer septal occlusion device presently available in the United States is 38 mm, and defects exceeding this size would require surgical closure. Multiple defects can be closed with multiple devices, though the cost of multiple device closures may exceed the cost of surgery[3]. Determinants of the limitations to device closure are under evolution as devices and their delivery systems continue to undergo refinements.

OPERATIVE TECHNIQUE:

Secundum ASD

The standard surgical incision for the repair of ASD is the median sternotomy. A portion of the anterior pericardium is preserved for use as a patch. Although other materials can be used, we prefer an autologous pericardial patch, treated with glutaraldehyde. Bicaval venous cannulation, mild hypothermia, and antegrade cardioplegia are employed to provide a still, blood-free field through which to expose the interatrial septum via right atriotomy made in parallel to the atrioventricular groove. A careful examination of the interatrial septum is carried out to ensure the correct identification of the margins of the defect. The SVC and IVC are identified, with special attention to any structures that might represent partial anomalous pulmonary venous return to the right atrium or vena cavae. The Eustachian valve is carefully identified to avoid the error of baffling the IVC to the left atrium. The coronary sinus is identified and protected from inclusion in the suture line, as impaired venous effluent from the coronary circulation can result in precipitous cardiac edema and heart failure after separation from cardiopulmonary bypass. A determination is made to close the defect primarily where there is sufficient septum primum tissue, or with a patch. Care is exercised to place sutures

firmly into surrounding tissue but without interfering with the adjacent non-coronary sinus of the aorta superiorly, the tricuspid or mitral valves anteriorly, the coronary sinus and atrioventricular AV node inferoanteriorly, the IVC and right lower pulmonary vein orifice inferiorly and posteriorly, or the right upper pulmonary vein and SVC superoposteriorly

Minimally Invasive Approaches

A variety of alternatives to the median sternotomy have been described for the repair of numerous cardiac defects, most notably the ASD. An inframammary incision with right anterolateral thoracotomy, bilateral anterior thoracotomy, or median sternotomy provides exposure of the right atrium with a scar that is more easily concealed than the full median sternotomy, though these approaches may risk phrenic nerve palsy, lung herniation, scoliosis, and breast or chest muscle deformity[4]. The subxiphoid "ministernotomy" or partial lower sternotomy can be performed safely through incisions as small as 3.5 cm, with cannulation through the incision. Smaller incisions still, without any sternotomy, have been described, with video-assistance and femoral cannulation. Video-assist technology also permits ASD closure through a small right thoracotomy. Though these alternative incisions may confer a cosmetic advantage over the standard midline

incision and median sternotomy, it has been difficult to demonstrate objective advantages in chest wall stability, pulmonary physiology, pain, or length of hospital stay. Robotic-assisted closure of ASD is not widespread, but advances in the field of robotics promise to introduce newer approaches to ASD repair as well as other cardiac procedures through diminishing invasive incisions.

Complications of Surgery

Complications following the surgical closure of ASD include early- or late-patch dehiscence, thromboembolism, and arrhythmias such as heart block, sinus node dysfunction, and atrial fibrillation or flutter. In the rare context of ASD closure with pulmonary hypertension, systemic venous hypertension, right ventricular failure, and low cardiac output can result acutely, necessitating a return to cardiopulmonary bypass to fenestrate the closure.

Though early sinus node dysfunction occurs in 9% of patients undergoing repair of superior sinus venosus defects by either Warden procedure or baffle and SVC patch, 8-year follow-up data show no persistent late sinus node dysfunction following these procedures.

At examination of outcome 27 to 32 years after surgical repair of ASD, age at the time of repair is an independent risk factor for late

complications, including late cardiac failure, stroke, and atrial fibrillation, all of which are more frequent when the age at repair is older than 25 years. Independent risk factors for the development of atrial fibrillation with ASD, repaired or not, include age over 25 years, left atrial enlargement, and mitral or tricuspid regurgitation. Thirty to forty percent of patients over 40 who exhibit atrial fibrillation after ASD repair may have an embolic event within 10 years of ASD repair, and systemic anticoagulation is recommended in this group.

Catheter-Based Treatment

King and Mills reported the first catheter-delivered ASD closure in 1976, using a double umbrella device and a 23-Fr delivery catheter. The large-delivery catheter size precluded its use in children. The clamshell occlusion device, reported in 1990, could be delivered through an 11-Fr sheath, bringing device closures to the pediatric population. Device arm fracture resulted in its redesign, and a variety of other devices appeared and remain in use[5]. Current devices include the CardioSEAL (Nitenol Medical, Boston, MA), the Amplatzer (AGA Medical Corp., Golden Valley, MN), the Sideris buttoned occluder (Custom Medical devices, Amarillo, TX), the Das Angel

Wings device (Microventa Corp., White Bear Lake, MN),[23] the ASDOS device (Osypka Corp., Rheinfeldon, Germany), the Helix septal occluder (W.L. Gore & associates, Inc., Flagstaff, AZ), and a transcatheter polyurethane foam patch.

Complications of Device Closure

The reported overall complication rate following catheter-deployed ASD closure devices is about 8%. Included among cardiac complications are device malposition or dislocation, early or late embolization, arrhythmia, pericardial effusion, left or right atrial thrombus, atrial or ventricular perforation, mitral or tricuspid regurgitation, aortoatrial fistula, eustachian valve entrapment, and sudden death[6]. Mitral or tricuspid regurgitation can result from device entrapment within chordae, chordal rupture, or leaflet perforation by the device or the delivery system. Reported noncardiac complications include iliac vein dissection, retroperitoneal or groin hematoma, and leg ischemia.

OUTCOME

Physiology of ASD Closure

Subtle changes in exercise performance in ASD patients can be measured even in childhood[7]. An abnormal ventilatory threshold during submaximal exercise returns to normal by 6 months after repair of ASD in patients under 5 years of age, but remains subnormal for patients repaired older than 5 years. Most patients older than 5 years old at the time of repair have at least some residual RV dilation and abnormal septal wall motion after ASD closure, not predicted by preoperative shunt or ASD size. The clinical significance of this finding is unclear. These data may further support a strategy of ASD closure before school age.

Adult

Though the adult with ASD clearly benefits by improvement in exercise physiology and reduction of RV dilation after closure of ASD, the improvements are less pronounced with advancing age. There is a clear survival advantage and a reduction in the incidence of cardiovascular events for ASD closure to the age of 40, compared to

medical management. Some controversy remains as to the best treatment strategy in the older adult population[11].

Regardless of the presence of symptoms, RV and RA enlargement in adults decreases after surgical or device closure of ASD, though right atrial enlargement persists proportional to the age at repair. Though younger adults demonstrate an improved VO2 max within months of ASD closure, patients older than 40 years who undergo repair may take several years to show exercise improvement. The incidence of atrial fibrillation is similar for the patient older than 40 years, whether surgically or medically treated[12]. Though its onset is sooner after surgical closure of ASD, the arrhythmia has a higher relation to long-term mortality in the medically treated group. The significant incidence of sustained postoperative atrial fibrillation suggests that a Maze procedure concurrent with ASD closure may be advisable for the patient older than 40.

Prior to ASD closure, >60% of patients older than 40 are NYHA class III to IV, whereas after ASD closure, >80% are NYHA class I to II. Patients older than 60 show functional class improvement, immediate and late reduction in pulmonary artery pressure, and improved 5- and 10-year survival after ASD closure, by comparison to medical management. These data support a strategy of ASD closure

regardless of age for the symptomatic patient, though some controversy remains over the closure of the asymptomatic ASD after age 40, as functional class deterioration and arrhythmia has been observed in this group after ASD closure.

In a study done by **Kambet et al** on cross sectional ECHO on mitral valve prolapse associated with ostium secondum ASD, a preoperative and postoperative comparative study on 71 patients, the incidence of MVP was 53.2%. Anterior MVP was found in 36% patients. Posterior MVP was found in 3 patients and 2 patients had both. Anterior MVP decreased in incidence from 28 patients to 17 following ASD closure, whereas posterior MVP remained unchanged even after surgery.

Speechly et al. has done a study on long term surgical outcome and the problem of late MR in secondum ASD on 52 patients with a mean age of 33 years with ASD. 52 patients underwent direct suture closure and 3 patients underwent pericardial patch closure. In this series surgical mortality was zero. Late postoperative morbidity happened in 2 patients, one of them required mitral and tricuspid wall replacement and the other patient developed cardiac failure. Both these patients were above 60 years of age. 2 patients developed sinus node dysfunction and required permanent pacing. Atrial fibrillation

(AF) persisted even after surgery in 6 patients. New AF developed in two elderly patients. MVP was present pre operatively in 9 (16%) elderly patients ($p < 0.001$). These patients had high pulmonary artery pressure ($p<0.001$). MVP persisted in these 9 patients postoperatively. MVP and MR developed newly in 5 patients postoperatively of which one patient needed MVR and TVR.

Takahashi H et al. in a study on MVP in patients with postoperative ASD involving 90 patients reported that the preoperative incidence of MVP in their study was 78% and postoperative incidence was 56%. Improvement of MVP was noted only in 24% of patients. No case of MVP newly developed after the surgery. In those patients with persistent MVP after surgery was associated with a large QP/QS, high pulmonary artery pressure and right ventricular dilatation. In those patients with MVP after surgery the resultant MR was not significant.

In a study done by **Gatoulis M A et al.** on atrial arrhythmias after ASD closure in adults on 213 patients of which 82 were men and 131 were women. The preoperative incidence of AF was 19% (40 patients). AF was not present in the age group between 37±13 years ($p< 0.01$) and the pulmonary arterial pressure (PAP) was 19.7+8.2 mm Hg. AF was prevalent in the age group of 59±11 years. The PAP in

this group of patients was 29±9.7 mmHg. There was no perioperative deaths reported in this study. After a mean follow up of 3.8±2.5 years, 24 out of 40 patients (60%) continued to have AF. Mean age of these patients was greater than that of 16 patients whose rhythm converted to sinus rhythm ($p<0.02$)[19]. Late events (one month after surgery) included stroke in 6 patients and death from noncardiac cause in 2 patients. The risk of AF in adults with ASD was related age at the time of surgery and the pulmonary arterial pressure.

Horer J et al. did a study on surgical closure of ASD in patients older than 30 years and evaluated the risk factors for late death from arrhythmias and heart failure. The long term follow up of 281 adult patients who underwent ASD closure was reviewed. The mean age at surgery was 43.8 ±10 years (30-76 years). There were 2 early deaths. Death from arrhythmia or heart failure was present in 9 patients at a mean time of 8.5± 6.6 years after operation. Patients more than 43 years exhibited a high PAP. Preoperative pulmonary artery systolic pressure (PASP) more than 36 mmHg and mean PAP more than 21 mm Hg were predictive of late death due to arrhythmia or heart failure.

Jose M et al in their study on surgical closure of ASD before or after the age of 25 years compared the natural history of operated and

unoperated patients. In this study the age group of patients were between 31 ± 19 years. 70% of the patients were of female sex, age at surgery was 31± 20. The diastolic RV size was 27± 7 mm and PAP was 34 ± 14 mm Hg. Degree of mitral insufficiency was 0.7± 0.8. Degree of tricuspid insufficiency was 1.2 ± 0.8. The LV diameter was 42 ± 11 mm. The incidence of AF was 17% and 9% (25 of 280 patients) had tricuspid regurgitation (TR). 2 % of the patients who had surgery before the age of 25 years presented with serious TR, whereas 18% of patients who were operated after the age of 25 years had TR. The left atrial size was more than 40 mm in 54% of patients and more than 50 mm in 20% of patients.

Attie F et al. did a study on 521 patients on surgical treatment of secundum ASD in patients more than 40 years old, a randomized control trial study. It was a prospective study measuring the long term clinical outcome. Of these 521 patients 232 underwent surgery and 241 underwent medical management. The primary and secondary end points were 1) Major cardiovascular events (functional class detoriation or heart failure, pulmonary embolism, major arrhythmic event, cerebro vascular event, recurrent pulmonary infection and death) 2) Overall mortality. Risk of mortality was high in the medical management group. Anatomic closure of ASDs should always be

attempted as a first treatment of ASD in adults older than 40 years of age with PASP less than 70mm Hg and QP:QS ≥ 1.7. The operation must be done earlier when symptoms of hemodynamic effects seem to be minimal.

Kodo T et al. in their study titled surgery of ASD in patients aged over 40 years: comparative study of direct suture closure and pericardial patch closure on 39 patients, evaluated the postoperative complications. Patients who underwent patch closure (13 patients) had a larger defect. In the group of patients who underwent direct closure (27 patients), there was one patient with pulmonary stenosis. In the other group 2 patients had mitral valve disease and 6 patients had pulmonary hypertension (PHT). In the direct closure group, incidence of heart failure was 29% and arrhythmia was 47% whereas in the other group incidence of heart failure was 61% and arrhythmia was 69%. In 33% of patients aged over 40 years with ASD heart failure developed after ASD closure.

In a study that evaluated left ventricular adaptation after ASD closure by increased concentration of N-terminal pro-brain natriuretic peptide (BNP) and cardiac MRI in adult patients showed that NT-pro BNP increased after interventional closure of the ASD. This increase was associated with increase left ventricular dimension as assessed by

MRI. Later after surgery NT-pro BNP returned to baseline[20]. This suggested that the presence of transient hemodynamic stress on left ventricle leading to acute heart failure that explains the late improvement that occurs in exercise capacity after ASD closure.

In a study done by **Beyer J at al**. on 716 patients evaluating acute left heart failure following repair of ASD and its treatment by ASD. 15 of these patients developed acute left heart failure requiring partial reopening. Some patients had severe PHT which led to right ventricular hypertrophy (RVH). There was marked difference in stroke work between the right and left ventricle. High incidence of left heart failure as a cause of death after repair of an ASD indicates the importance of the complications.

In a study of ASD over 60 years of age and older evaluated the operative results and long term postoperative follow up. 56 patients were divided into 3 groups according to the PASP. In these groups there was no significant difference in QP/QS, RA, LA pressures, right and left ventricular end diastolic pressure but pulmonary vascular resistance index was more significant when the PASP was more than 60 mm Hg. Operative mortality was 6%. All of these patients underwent appropriate additional procedures. The operative mortality was unrelated to the preoperative functional class, PAP are pulmonary

vascular resistance (PVR). The patients improved symptomatically regardless of the functional class, PAP or PVR. There was significant longevity at 5 and 10 years after surgery when compared to the medical treatment.

Felix et al. studied the incidence of atrial fibrillation and atrial flutter in adults with ASD before and after surgery. The study was done on 211 adult patients with ASD. Holter monitoring was done preoperatively and early and late postoperatively. These patients were divided into 3 groups according to their age groups. Atrial flutter was significant in the age group of 54±12 years whereas atrial fibrillation was significant in the age group of 59± 8 years. Atrial flutter converted to sinus rhythm in 10 out of 18 patients postoperatively and atrial fibrillation converted into sinus rhythm in 21 out of 28 patients. The surgical repair of ASD to treat supraventricular tacchyarrhythmias (SVT) is warranted in adults but when atrial fibrillation is present, sometimes it may have to be combined with Maze operation.

Shah et al[13]. published a prospective study on the natural history of OS-ASD in adults after medical and surgical treatment. In the nonsurgical group 13 patients (43%) had TR, mild TR was present in 7 patients and moderate TR in 6 patients. Average TR velocity was 3.1 m/sec and peak gradient was 39.04 mm Hg. In the surgical group 8

patients (17%) had mild TR with TR velocity was 2.7 m/sec and the peak gradient was 29.5mm Hg. In the medically managed group atrial fibrillation was present in 45% of patients above the age of forty years, which was comparable to 51% of patients as reported by **Murpy et al.** in the same age group.

Sutton et al[18]. reported an incidence of atrial fibrillation to be 58% in his series. The percentage of atrial fibrillation in surgical group was 40% in the age group of 40 years and 58% for patients in the age group of 60 years. All systemic embolic episodes occurred in patients who had atrial fibrillation and their incidence was equal in both groups. Warfarin was the treatment of choice to prevent embolism in patients with atrial fibrillation. 45% of the medically managed group and 50% of the surgically treated patients was receiving diuretics to treat congestive cardiac failure. All these patients had atrial fibrillation.

In the above mentioned study the authors reported that the LV dysfunction could not be detected by ECHO. RV was dilated in both the operated and unoperated group of patients but their function was good in both the groups. RV thickness was essentially the same in both groups. Cardiac failure was due to TR and atrial fibrillation. The frequency of raised venous pressure was not influenced by surgical

treatment though TR was detected more significantly by Doppler ECHO in the medical group.

M Vogel et al[14]. studied the incidence of secondary PHT in adults with ASD and sinus venosus defect. This study involved 31 patients with SVC type ASD and 138 patients with ASD-OS. In the SVC defect group there was significantly higher systolic, diastolic and mean PAP and PVRI than the ASD-OS group. PHT was present when mean PAP was more than 30 mmHg. Incidence of PHT was 26 % in SVC ASD and 9% in ASD-OS group. Raised PVRI (Rp/Rs > 0.3) was present in 60% of SVC group and 4% with ASD-OS group. Raised PAP and PVRI was more apparent in the age group of more than 40 years than the 18-40 years group and there was no progression of pulmonary vascular disease (PVD) in patients more than 60 years[21]. Significant correlation was present between age and New York heart association (NYHA) class in both the groups however the correlation was more significant for the SVC group. 2 patients in each group were listed for heart lung transplant. 1 patient with SVC ASD died. He was 58 years old. In this patient the PAP was 68 mm and Rp/Rs was 0.4. He had a left to right shunt with a QP/QS of 1.8: 1. The left to right shunt increased with oxygen and so surgery was done. Postoperatively

patient developed PHT crisis six hours after surgery and the patient died

C A Boucher et al[15]. did a retrospective analysis of 235 adults with ASD-OS and evaluated the incidence of MR, its management and morphologic basis. In this study 4% (10) of patients had significant MR. 3 of them required MVR along with the ASD surgery. 4 patients had ASD closure alone, in which one of them required MVR after 5 years. 3 patients did not undergo surgery due to coexisting comorbid illness. 6 patients had their mitral valve studied pathologically and it revealed that 3 had myxomatous disease and the other 3 where of rheumatic origin. Their data shows that the association between MR and ASD was not significant. The morphology of the mitral valve were mainly chordal and leaflet thickening, fibrosis and deformity and not thinning and ballooning as in MVP.

All the patients in the above mentioned study presented with symptoms and were on digoxin and diuretics.one patient had congestive cardiac failure (CCF). 7 had atrial fibrillation and left atrial enlargement in ECG. 3 patients had LVH on ECG. All of them had generalised cardiomegaly in their chest X-ray right ventricle and pulmonary artery was prominent. 8 patients had radiological evidence of left atrial enlargement. In 3 patients who underwent MVR 2

patients are in NYHA class I after 2 and 6 years. Third patient died of severe right and left heart failure. In the 4 patients with isolated ASD closure postoperative LA pressure was elevated and all of them continued to demonstrate significant clinical MR and one of them subsequently required MVR after 5 years but he died one year after the surgery. The fifth patient developed grade II symptoms and left atrial enlargement over 5 years. Sixth and seventh patients were in grade I after 5 years. Incidence of MR was uncommon in young patients with ASD and more common in elderly patients. The residual MR is significant after ASD closure. Pulmonary venous and left atrial pressure increased and caused symptoms in 2 patients.

MVR at the time of ASD repair should be waited against the long term risk of anticoagulation. The decision to replace the valve should be done after inspecting the valve elements, partially occluding the ascending aorta to increase the afterload and by volume overloading. These maneuvers unmasks significant MR. When MR is severe MVR is done in the same setting. When MR is less significant MVR is delayed but it may be required subsequently. Hence surgery in patients with ASD and MR were delayed until significant symptoms developed, so that at the same setting both defects are repaired.

MJ Davis et al[16]. in the study of 16 adult hearts with ASD studied the morphology of the mitral valve. 15 of these hearts showed thickening of medial half of the anterior cusps and fusion of chordae, fibrosis and neovascularization and myxomatous degeneration. The function abnormality in mitral valve was due the changes caused by increased flow and altered LV geometry.

TL Schreiber et al[17]. in their study on effect of atrial septa defect repair on LV geometry and degree of MVP in 14 patients reported that cross sectional ECHO showed MVP in 7 patients (50%) preoperatively. The degree of prolapse was studied by measuring the net algebraic area subtended by the area of apposed surface of the leaflet in systole with respect to mitral ring. In patients with MVP the area was 0.3 ± 3.1 Units and in patient with no MVP the area was 12.5 ± 3.1 Units. Post-operative prolapse was reduced or abolished in 6 of 7 patients (86%) and the mitral valve apposing surface area was 14.7 ± 4.4 Units after ASD closure. RV internal dimension index (RVidi) reduced from a preoperative value of 2.69 ± 0.16 cm /m^2 to 2.19 ± 0.17 cm /m^2 post operatively. Mean LV internal dimension (LVidi) reduced from 3.16 ± 0.21 cm /m^2 preoperatively to 3.64 ± 0.19 cm / m^2 but it was not significant. In 1 patient RVidi remained in the upper limit even after surgery because of the chronic changes due to left to

right shunting. No significant changes noted in the LA dimension. Septa motion normalized in 86% of the patients.

MG St John Sutton et al[18]. did a study in 53 patients with ASD assessing the left ventricular function by complete analysis of the M-mode ECHO. LV function was assessed by computer assessed analysis of LV Echo and cardiac catheterization. There were two groups of patients one above 60 years and other was below 60 years. Cavity size and septal motion were abnormal in 86% but cardiac index left ventricular end diastolic pressure, fractional shortening, rapid filling and LV filling were grossly normal. They concluded that in patients with ASD the LV function is normal irrespective of increased age RV failure and PHT. The abnormal septal motion is compensated by enhanced septal and posterior wall percentage thickening.

MATERIALS AND METHODS

Study design

Retrospective study

Methodology

Subject Selection: 100 patients

Inclusion Criteria: All patients with Atrial septal defect of ostium secundum type between 12 to 60 years of age with a significant left to right shunt with Qp /Qs of 1.5: 1 or greater and patients with ASD OS with symptoms

Exclusion Criteria: ASD Ostium Secundum (OS) type associated with complex congenital malformations, ASD OS with severe Mitral or Tricuspid regurgitation, angiographically confirmed acquired coronary artery disease, ASD associated with Partially Anomalous Pulmonary Venous Connections (PAPVC)

Analysis Plan: Comparison of outcomes of both the surgical techniques- duration of the surgery, cardio pulmonary by pass time , changes in chamber geometry, aortic cross clamp time, need for blood products, thromboembolism, infections, other

complications, duration of hospital stay and relief from symptoms

Screening Procedures / Visits: Postoperative

Transthoracic echocardiography

Follow up Procedures / Visits: Transthoracic

echocardiography

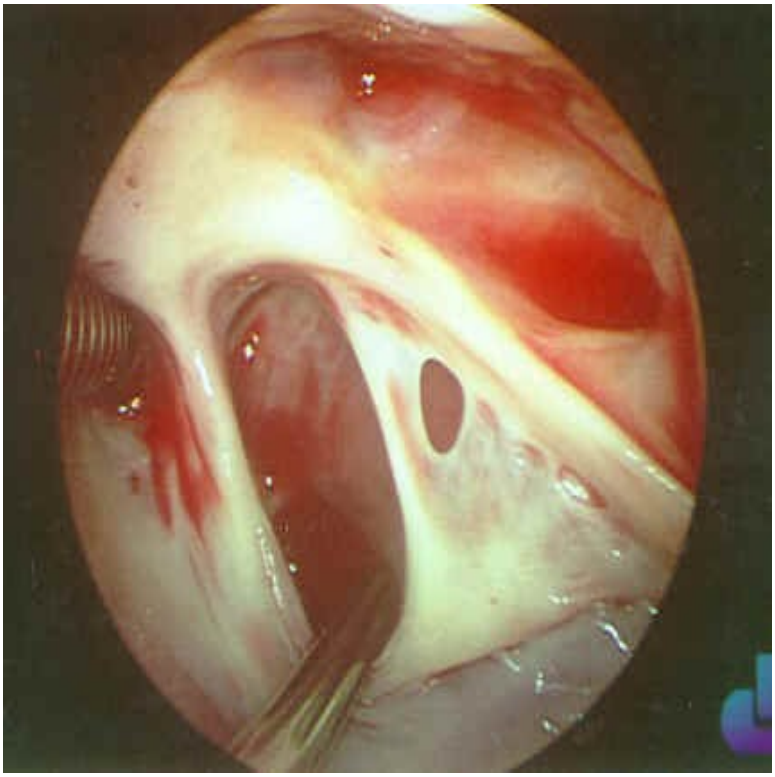
Assessments of Parameters:

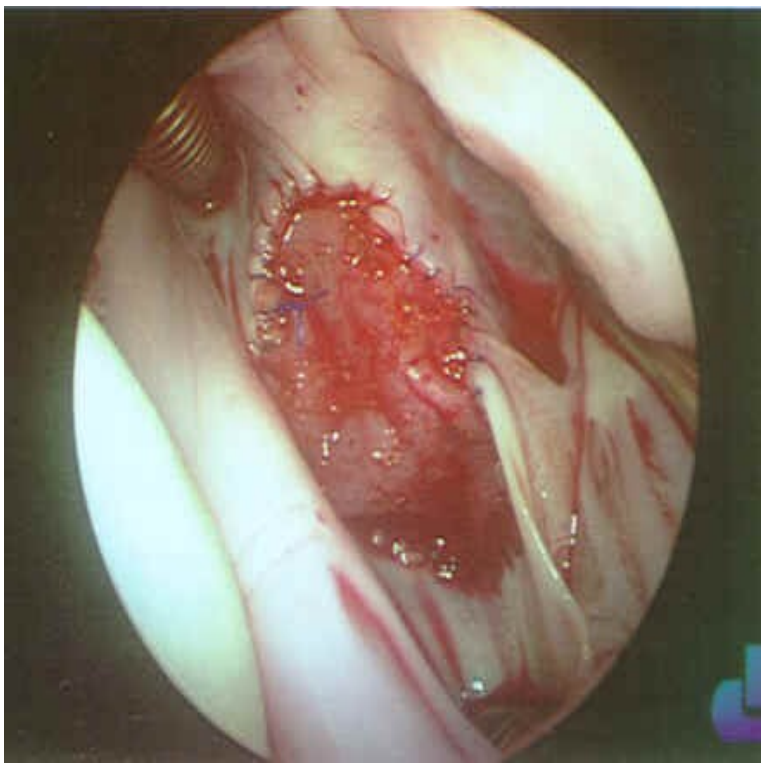
ECG- Reduction in QRS duration

ECHO- Left ventricle internal diastolic dimension (LVID), Right ventrical internal diastolic dimension (RVID), Mitral regurgitation (MR), shunt fraction, residual shunt. In this study transthoracic ECHO was done in all patients preoperatively, postoperatively and on follow up. It was a comprehensive study that included M mode, two dimensional ECHO, continuous wave, pulsed wave and colour Doppler. In the apical four chamber view, the transverse diameter measured was taken as the right ventricle size. The tricuspid regurgitant jet velocity was used to calculate pulmonary artery pressure. The shunt ratio was measured by taking into account the velocity time integral (VTI) and the cross sectional area at the corresponding sites in the left ventricular outflow tract and the pulmonary artery. MVP

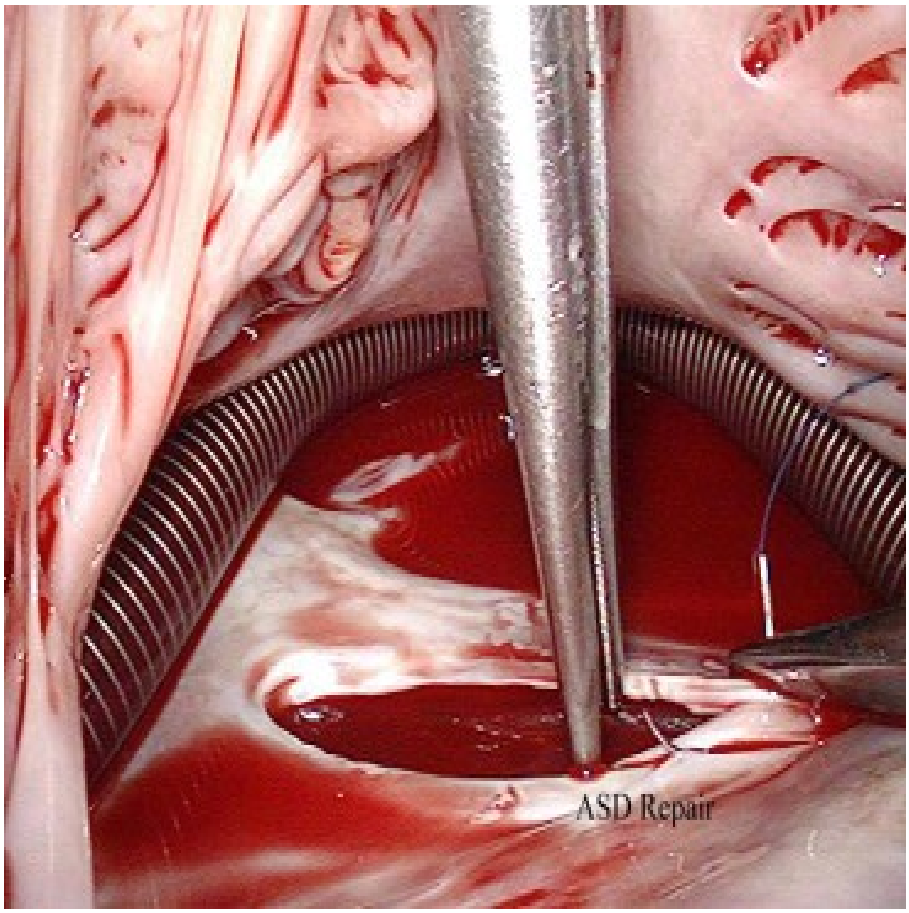
was diagnosed when there was a sudden mid to late systolic posterior displacement of a part of the mitral valve to atleast 2mm below the line that joins the point of closure of the mitral valve in systole to the point of opening of the mitral valve in diastole, with leaflet displacement superiorly, confirmed by cross sectional ECHO. MVP was also diagnosed when there was pan systolic prolapse with displacement 3mm below the line joining the closure and opening of the mitral valve, with the peak in the mid systole confirmed by bulging of the mitral annulus during systole on ECHO.

ASD OSTIUM SECUNDUM TYPE PERICARDIAL PATCH CLOSURE





DIRECT SUTURE CLOSURE OF ASD



RESULTS

Clinical data

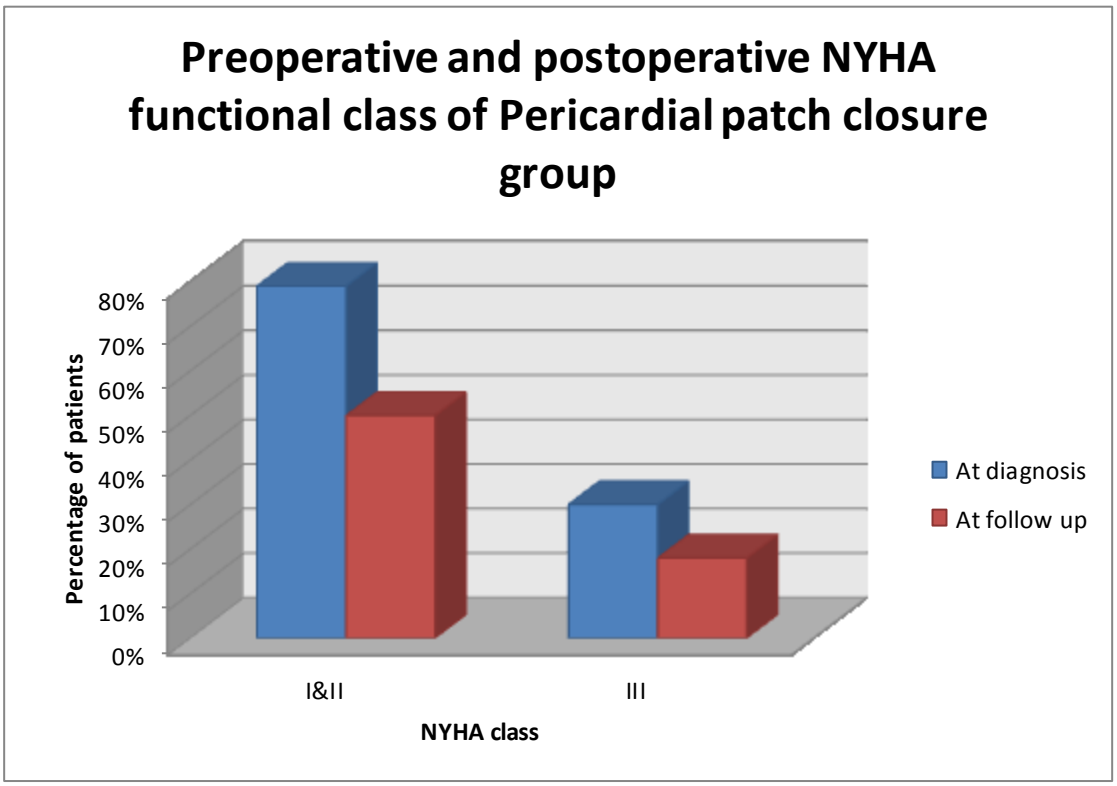
In the study of 100 patients, 54 patients presented with symptoms while the remaining had no symptoms. Patients presented with symptoms like breathlessness on exertion, palpitations, fainting attacks, tiredness, recurrent respiratory tract infections and atypical chest pain. The mean age of patients in the pericardial patch closure group was 38 ± 6 years and in that of the direct closure group mean age was 41 ± 7 years (Table 1).In the first group 62% were females and in the second group 50% were females. 27 % of patients in the first group and 30% in the second group were in NYHA class II-III respectively. Atrial fibrillation was present in 24 of the 100 patients at the time of diagnosis. 65% of the patients were females with the median age of 26 years and 35% were males with the median age of 33 years.

Comparison between the two study groups

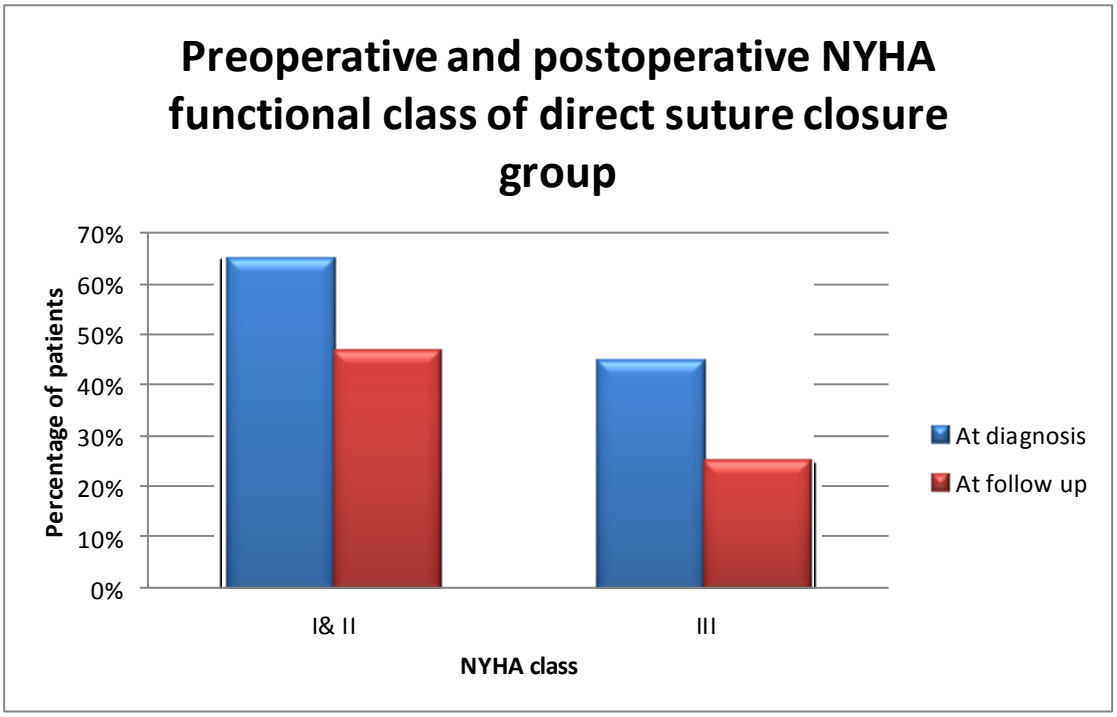
Table 1

Variables	Pericardial patch closure group	Direct suture closure group
Age (years)	38±6	41±7
Female	31	28
Male	19	22
NYHA class II & III	27%	30%
ASD average size	26±8 mm	24±7 mm
Total cross clamp time	16±3 mins	8±3 mins
Cardiopulmonary bypass time	35±2 mins	25±3 mins
Residual shunt	None	None
Duration of hospital stay	6±2 days	6±3 days

Graph 1



Graph 2


Preoperative and postoperative NYHA functional class of direct suture closure group
Percentage of patients
70%
60%
50%
40%
30%
20%
10%
0%
I& II
III
NYHA class
At diagnosis
At follow up

Echocardiographic (ECHO) data:

Average size of the ASD was 26±8mm, 24±7mm in the pericardial patch closure and direct suture closure group respectively. Mitral valve prolapse (MVP) was noted in 16 patients (30%) in the pericardial patch closure group (Table 2) and 12 patients (24%) in the direct suture closure group (Table 3). In the patients with MVP the pulmonary artery mean pressures were higher and these patients were older, most of them being above the age of forty years. Mitral regurgitation (MR) was present in 9 patients in the pericardial patch closure group, 7 of them had mild MR and 2 had moderate MR. In the direct suture closure group 11 patients had mild MR. None of the patients in both groups had a severe MR. 34 patients in the pericardial patch closure group and 38% in the direct suture closure had no MVP and their mean age was 25 years. We did not note any significant correlation between the prolapse and the shunt size of the ASD. All patients underwent prospective follow up ECHO at a mean period of four months from the time of surgery. 4 patients in the pericardial patch closure group and 3 patients in the direct suture closure group developed MR in the follow up period. All these patients were elderly patients with the mean age of 49±6 years.

Pericardial patch closure- Preoperative ECHO findings

Table 2

ECHO findings	No. of Patients	Percentage
Mild MR	7	14%
Moderate MR	2	4%
MVP	16	30%
Mild TR	11	22%
Moderate TR	12	24%

Direct closure group- Preoperative ECHO findings

Table 3

ECHO findings	No. of Patients	Percentage
Mild MR	8	16%
Moderate MR	3	6%
MVP	12	24%
Mild TR	10	20%
Moderate TR	6	12%

MR-Mitral regurgitation, MVP-Mitral valve prolapse, TR-Tricuspid regurgitation, PHT-Pulmonary hypertension

Echocardiographic parameters

Table 4

Pericardial patch closure group			Direct suture closure group	
Parameters	**Preoperative**	**Postoperative**	**Preoperative**	**Postoperative**
RV size (mm)	45±8	37±5	43±6	39±3
LVID (mm)	31±3	36±2	34±3	37±4
LA (cm)	2.9±0.5	2.7±0.3	2.5±0.5	2.3±0.6
MVASI(U/m^2)	8.0±3.3	18.3±3	11±3.6	20±5
PAP	32±8	34±6	29±6	25±9

RV- Right ventricle at end diastole, **LVID-**Left ventricular internal dimension at end diastole**, LA-**Left atrium**, MVASI-** Area subtended by the opposed mitral valve in systole in the long axis, cross sectional ECHO with respect to the mitral ring

Surgical data:

In this study 50 patients underwent pericardial patch closure and another 50 patients underwent direct suture closure for ASD. We observed that in those patients who underwent pericardial patch closure the defect size was not significantly larger than in those who had direct repair of their defects. There was no mortality in both the groups. Surgical morbidity was present in 2 patients in the pericardial patch closure group in the form of bradyarrythmia but they asymptomatic and they were treated with orciprenaline. In the follow

up period after 2 months the drug was slowly withdrawn and bradyarrhythmia did not recur in these patients. None of them needed a permanent pace maker implantation. Atrial fibrillation was present postoperatively in all the patients in both groups who had developed it preoperatively (Table 5). 2 patients in the pericardial patch closure group and 1 patient in the direct suture closure group developed new atrial fibrillation the follow up. 3 patients in the pericardial patch closure group and 4 patients in the direct suture closure group had transient tachyarrhythmia their mean ages were 53±5. In the pericardial patch closure group 1 patient aged 60 years developed cardiac failure and required treatment with diuretics. 2 patients in the direct suture closure group and 1 patient in the pericardial patch closure group developed acute pericarditis in the postoperative period and needed treatment with aspirin and steroids. 46% in the pericardial patch closure group and 32% in the direct closure group had tricuspid regurgitation. The mean pulmonary artery pressure in the first group was 32±8 mm Hg preoperatively and in the postoperative follow up period it was 34±6 mm Hg .In the direct closure group the mean pulmonary artery pressure was 29±6 mm Hg preoperatively and reduced to 25±9 mm Hg in the postoperative follow up period (Table 4). Two patients in the PPC group and one patient in the DC group developed new atrial fibrillation. Four patients in the PPC group and

five patients in the DC group with preoperative atrial fibrillation reverted to sinus rhythm. The right ventricular size in the PPC group reduced from 45±8mm in the preop period to 37±5mm in the postop follow up period. In the DC group it reduced from 43±6mm before surgery to 39±3mm in the follow up period after surgery. Mitral valve apposition area in systole (MVAS) improved from 8±3.3 to 18.3±3 in the post op follow up in the PPC group. In the DC group the MVAS improved from 11± 3.6 to 20±5 in the post op follow up.

Preoperative atrial flutter of fibrillation

Table 5

Variables	Pericardial patch closure group	Direct suture closure group
Number of patients	14	10
Qp:Qs	2.3±4	1.9±7
PAP(mm Hg)	31±8	30±6

Qp:Qs- Pulmonary to systemic flow ratio, **PAP**-Pulmonary artery pressure

Electrocardiogram and chest X-ray findings

Table 6

	Pericardial patch closure group		Direct suture closure group	
Variables	**Pre operative**	**Post operative**	**Pre operative**	**Post operative**
Axis	68±30	54±20	70±20	57±30
PR interval (m sec.)	184±6	152±5	179±8	140±3
QRS duration (m sec.)	123±2	107±2	128±3	111±5
R wave in lead V1	2.4±2	2.2±3	2.6±5	2.3±3
CT ratio in chest X-ray (%)	46±3	41±5	49±3	42±7

Cardiac events and clinical outcome after surgery

Table 7

Event	Pericardial patch closure group	Direct suture closure group
Stroke	4%	2%
Cardiac failure	16%	10%
New onset AF	4%	2%
Acute pericarditis	2%	4%

DISCUSSION

Kambet et al in their study on incidence of MVP associated with atrial septal defect mentioned that the incidence of MVP was 53.2% whereas in our study the incidence of MVP was 54 %. 30 % of patients in the PPC group had MVP and 24% in the DC group had MVP. Postoperatively in the follow up ECHO the MVP reduced to 24% in the PPC group and to 13% in the DC group.

Comparing our results with the study done by **Speechly et al**, surgical mortality in both study were zero. Late postoperative morbidity was present in 7 patients in the PPC group and 6 patients in the DC group in the form of atrial fibrillation, failure and acute pericarditis whereas in the Speechly's study group one patient required mitral valve replacement and tricuspid valve replacement and the other one had developed cardiac failure, two patients had developed sinus node dysfunction, new AF developed in two elderly patients. In our study, 54% of patients who had MVP also had high pulmonary artery pressures. MVP improved after surgery in 10 of patients in the PPC group and 12% of patients in the DC group. While in Speechly's study nine preoperative patients had high PAP and MVP persisted in all of them post operatively and new mitral regurgitation

and MVP developed in five patients. In the PPC group four patients and three patients in the DC group had developed new mitral regurgitation. These results were also comparable to the study done by **Tahahashi H et al**. In his study improvement in MVP was noted in 24% of patients.

The preoperative incidence of atrial fibrillation in the PPC group was 28% and in the DC group was 20%. Atrial fibrillation was prevalent in the age group of 53±9 (PAP 31±8mmhg) in the PPC group and 50±7 (PAP30±6mmhg) in the DC group. On comparing these results with the study by **Gaoulis MA et al** the incidence of Atrial fibrillation was 19% and it was prevalent in the age group of 59±11(PAP 29±9.7).In our patients 8% in the PPC group and 10% in the DC group AF reverted to sinus rhythm post operatively. 82% of the patients continued to have atrial fibrillation after surgery. In the Gaoulis study 60% continued to have atrial fibrillation after a follow up of 3.8±2.5 years. Three patients in the PPC group two patients in the DC group developed stroke in the postoperative period.

In our study patients who underwent pericardial patch closure had a bigger defect. 14% and 10% of patients in the PPC and DC group respectively had pulmonary hypertension. **Kodo et al** in their study noted that one patient had pulmonary stenosis in the DC group

and six patients in the PPC group had PHT. In the PPC group incidence of heart failure was 16% and 10% in the DC group and incidence of arrhythmia was 34% in PPC and 20% in the DC group while in Kodo's study incidence of cardiac failure and arrhythmia was 61% and 69% in the PPC group and DC group respectively.

The incidence of tricuspid regurgitation in the PPC group was 46% (23 patients), mild TR was present in 11 patients and moderate TR was present in 12 patients. The incidence of TR in the DC group was 32% (16 patients) 10 had mild TR and 6 had moderate TR. These results were comparable to that of the study done by Shah et al. Pulmonary artery pressure in the PPC group was 32±8mmhg and in the DC group was 29±6 in the preoperative period and on follow up it was 34±6 and 25±9 in the PPC group and DC group respectively. The incidence of heart failure was 16% in the PPC group and 10% in the DC group. Cardiac failure was a result of arrhythmias and TR. The frequency of raised PAP was not influenced by the different modes of treatment. Incidence of PHT was 14% in the PPC group and 10% in the DC group. Postoperatively 9 of the 16 patients in the PPC group and 10 of the 12 patients in the DC group experienced a reduction in the MVAS and degree of prolapse. MVAS in the PPC group was 8±3.3 and improved to 18.3±3 and in the DC group it improved from

11±3.6 to 20±5 post operatively. Right ventricular size in the PPC group reduced from 45±8mm in the pre-op period to 37±5mm post op and in the DC group it reduced from a mean of 43±6mm to 39±3mm.LVID reduced from 31±3mm pre-op to 36±2 and from 34±3 to 37±4 in the PPC group and DC group respectively. Left atrial size reduced from 2.9±0.5 pre op to 2.9±0.3 and 2.5±0.5 to 2.3±0.6 in the PPC and DC group respectively.

SUMMARY

This is a retrospective study of two groups of patients who underwent pericardial patch closure and direct suture closure for ASD. The average size of the ASDs in the pericardial patch closure (PPC) group was larger (26±8 mm). MVP was present in 30% and 24% in the PPC group and direct closure (DC) group respectively. In the postoperative follow up period the MVP reduced in 9 patients in PPC group and ten patients in the DC group. MR was present in 18% and 22% of patients in PPC group and the DC group respectively. 4 patients in the PPC and 3 DC group developed postoperative new mitral regurgitation. Preoperative AF was present in 28% and 20% of PPC and DC groups respectively. Of these 8% and 10% of patients reverted to sinus rhythm postoperatively in both groups respectively. The total cross clamp time and cardiopulmonary bypass time was longer in the PPC group. None of the patients in both groups had residual shunt in the postoperative ECHO. There was no difference in the duration of hospital stay between the two groups.

CONCLUSION

Cardiac surgeons vary in their frequency with which they select the pericardial patch technique or direct suture closure. A patch can be used when the defect is significantly larger or when the tissues are friable and there appears to be no significant difference in the results, early and late thromboembolic complications. When circumstances are ideal the ease and simplicity of direct closure supports its use in many patients.

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INFORMEDCONSENTFORM

Title of the study: "Surgical closure of Atrial Septal Defect (ASD) – A Comparative study of direct suture and pericardial patch closure technique"

Name of the Participant: _____

Name of the Principal (Co-Investigator): Dr. Shegu G

Name of the Institution: Rajiv Gandhi Government General Hospital & Madras Medical College,

Chennai-3

Name and address of the sponsor / agency (ies) (if any): _____

_____

Documentation of the informed consent

I _____have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am exercising my free power of choice

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past 12 months including any native (alternative) treatment.
6. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital. *****
7. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
8. I have understood that my identity will be kept confidential if my data are publicly presented.
9. I have had my questions answered to my satisfaction.
10. I have decided to be in the research study.
11. I have the right to refuse to give blood sample in case I don't want to, for any reason.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name _____Signature_____Date_____

Name and Signature of impartial witness (required for illiterate patients):

Name _____Signature_____Date_____

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

Name _____Signature_____Date_____

For Children being enrolled in research:

Whether child's assent was asked: Yes / No (Tick one)
[If the answer to be above question is yes, write the following phrase:
You agree with the manner in which assent was asked for from your child and given by your child. You agree to have your child take part in this study].
[If answer to be above question No, give reason (s) :

_____

.
Although your child did not or could not give his or her assent, you agree to your child's participation in this study.

Name and Signature of / thumb impression of the participant's parent(s)
(or legal representative)

Name _____Signature_____Date_____
Name _____Signature_____Date_____
Name and Signature of impartial witness (required for parents of participant child illiterate):
Name _____Signature_____Date_____

Address and contact number of the impartial witness: _____

Name and Signature of the investigator or his representative obtaining consent
Name _____Signature_____Date ___

MASTER CHART

1	Amul	27/m	80438	PPC	7	II	I
2	Devaraj	48/m	63650	PPC	7	III	II
3	Singadurai	46/m	67085	PPC	8	III	I
4	Jeyaraman	35/m	23456	PPC	10	III	I
5	Rekha	21/m	71934	PPC	8	II	I
6	Selvarani	28/f	82694	PPC	7	III	I
7	Kalyani	45/f	4567	PPC	8	II	I
8	Prabhu	26/m	3427	PPC	8	II	I
9	Chitra	19/f	82706	PPC	9	II	I
10	Banu	40/f	3427	PPC	7	III	I
11	Riyaz	50/m	65207	PPC	7	II	II
12	Rajasekar	16/m	78057	PPC	8	II	I
13	Venkataramanammal	30/f	54028	PPC	7	II	I
14	Krishnan	46/m	62732	PPC	7	III	I
15	Devandran	30/M	60503	PPC	7	II	I
16	Sahul hameed	47/M	60503	PPC	7	III	II
17	Ramar	34/M	74204	PPC	8	II	I
18	Ellamal	40/F	75462	PPC	9	II	I
19	Amsa	42/F	71432	PPC	8	III	II
20	Govindasamy	45/M	75476	PPC	7	II	I
21	Leela	37/F	85423	PPC	7	III	I
22	Chinnapan	26/M	90714	PPC	8	III	II
23	Kalpana	22/F	99308	PPC	8	III	I
24	Chandrasekar	49/M	33083	PPC	9	III	I
25	Kanchana	24/F	33272	PPC	7	II	I
26	Deenadayalan	45/M	27223	PPC	8	II	I
27	Prabhu	25/M	31210	PPC	7	II	I
28	Durairaj	45/M	94287	PPC	8	II	I
29	Babu	37/M	71210	PPC	8	II	II
30	Chellammal	40/F	90524	PPC	7	III	I
31	Nirosha	19/F	85065	PPC	7	II	I
32	Shantha kumar	25/M	25396	PPC	9	II	I
33	Naveesa Begam	28/F	101648	PPC	8	II	I
34	Aadhikesavan	46/M	98488	PPC	10	III	II
35	Solaiammal	43/F	78035	PPC	9	III	I
36	Senthil	31/M	97535	PPC	8	II	I

37	Devi	42/F	87452	PPC	9	II	I
38	Renu	17/F	97542	PPC	8	II	I
39	Pradeep	26/F	31380	PPC	8	II	I
40	Anjali Devi	27/F	92968	PPC	7	III	I
41	Boopathy	36/M	10162	PPC	8	II	I
42	Sakaya Mary	36/F	106731	PPC	7	II	I
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46	Rani	40/F	18123	PPC	8	III	I
47	Eswari	20/F	93416	PPC	7	II	I
48	Veerammal	32/F	103760	PPC	8	III	I
49	Saranya	14/F	1960	PPC	8	II	I
50	Maniammal	27/F	1861	PPC	7	III	I
51	Vembu	38/F	105384	DC	7	II	I
52	Chitra	31/F	103766	DC	8	II	I
53	Pandiyan	41/M	5389	DC	9	III	I
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56	Sevvanthi	16/F	5653	DC	8	III	II
57	Kannappan	35/M	39162	DC	9	III	I
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72	Ranjani	36/F	10572	DC	8	II	I
73	Krishnamoorthy	37/M	97559	DC	9	III	II
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75	Karthik	21/M	17351	DC	8	II	I
76	Suguna	37/F	8411	DC	7	II	I
77	Sathyamoorthy	34/M	24335	DC	8	III	I
78	Raja	38/M	10524	DC	7	II	I
79	Mona	17/F	19301	DC	7	III	I
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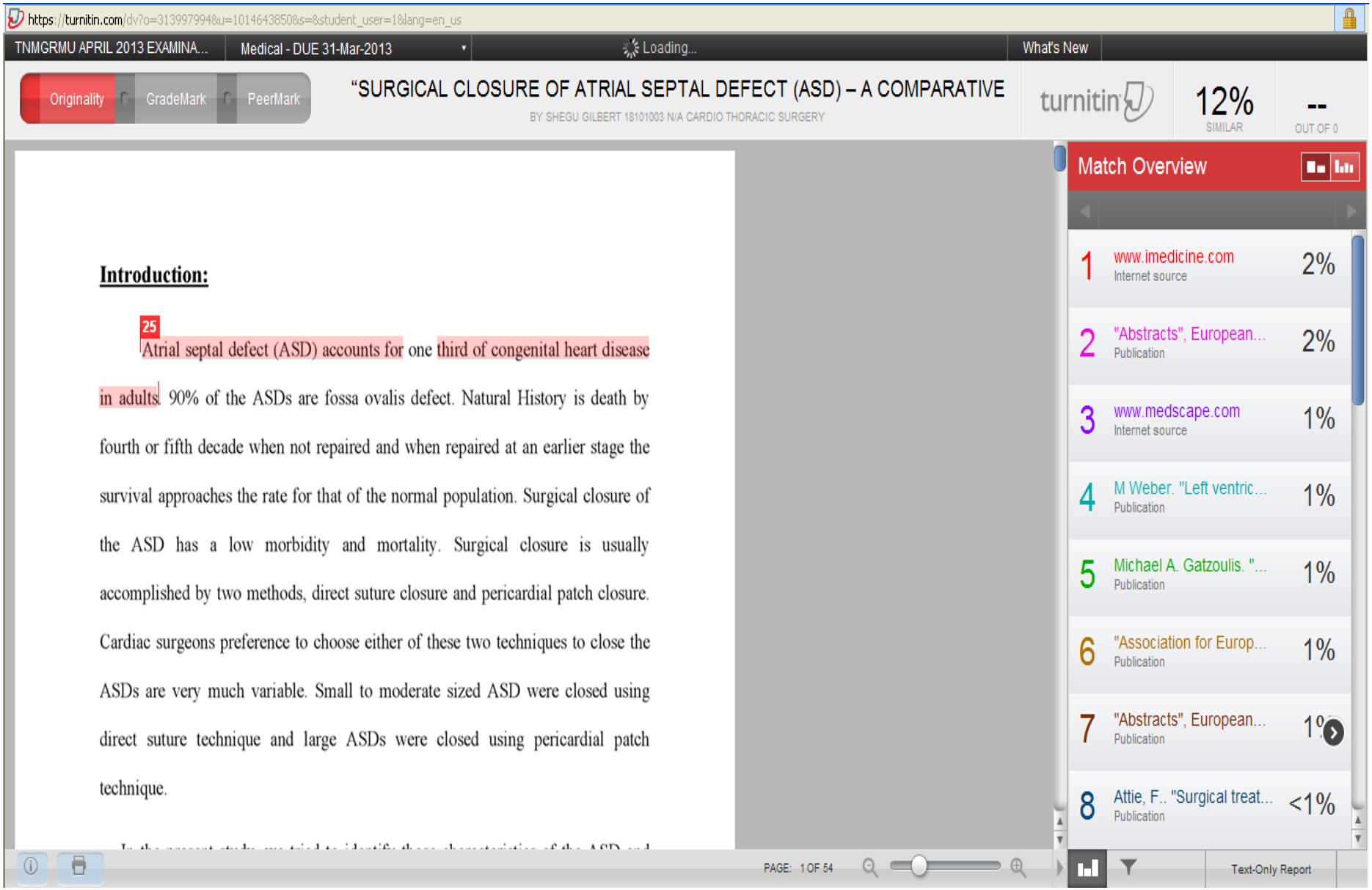
81	Selvi	31/F	17297	DC	8	II	I
82	Valli	36/F	22776	DC	9	III	I
83	Ganesan	41/M	26505	DC	8	III	I
84	Kuppu	20/F	24268	DC	9	II	I
85	Lakshmi	45/F	12788	DC	7	II	I
86	Ambujam	35/F	12798	DC	7	II	I
87	Ammu	21/F	5966	DC	8	III	I
88	Henry	17/M	5305	DC	7	II	I
89	Ravi	30/M	10370	DC	7	III	II
90	Nandha	27/M	14952	DC	8	II	I
91	Varun	25/M	14951	DC	9	II	I
92	Nagammal	35/F	1324	DC	7	III	I
93	Ramesh	24/M	1232	DC	8	II	I
94	Imthias	13/M	34845	DC	7	II	I
95	Poovi	29/M	16307	DC	9	III	I
96	Chitra	25/F	13803	DC	8	II	I
97	Meenakshi	39/F	24905	DC	7	II	I
98	Samuthiram	60/M	19551	DC	8	II	I
99	Manikala	54/F	21281	DC	8	II	I
100	Ravisekar	43/M	32572	DC	7	II	I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