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# TRANSTHORACIC COLOR ECHOCARDIOGRAPHIC EVALUATION OF OPERATED ATRIAL SEPTAL DEFECT IN A PATIENT OF LUTEMBACHER'S SYNDROME: CASE REPORT AND REVIEW OF LITERATURE

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**ABSTRACT**
Lutembacher syndrome (LS) is an uncommon cardiac condition involving the co-occurrence of an atrial septal defect (ASD) and mitral stenosis. While some individuals may not exhibit symptoms for extended periods, timely intervention significantly improves outcomes. Delayed diagnosis, however, often leads to severe complications such as heart failure and pulmonary hypertension, resulting in a worse prognosis. We are reporting a 47-year-old female who underwent surgical repair for a large secundum ASD in March 2025. A subsequent transthoracic echocardiogram (TTE) revealed an enlarged Coronary sinus (CS) and a residual ASD patch without a detectable shunt.

**KEYWORDS**: *Atrial septal defect, Echocardiography, Lutembacher syndrome, Mitral stenosis, Pulmonary hypertension.*

## INTRODUCTION

The term "Lutembacher syndrome" was initially documented by anatomist Johann Friedrich Meckel.[1][2] Corvisart later noted the association between ASD and mitral stenosis (MS) in 1811. LS was named after the French physician Rene Lutembacher, since he described the first case of LS in 1916.

Over time, the criteria for LS have evolved. While traditionally defined as MS combined with ASD, some researchers include cases with ASD and mitral regurgitation (MR). Current guidelines, however, specify LS as any combination of ASD (congenital or surgically induced) and MS (congenital or acquired)[1] (Figure 1).

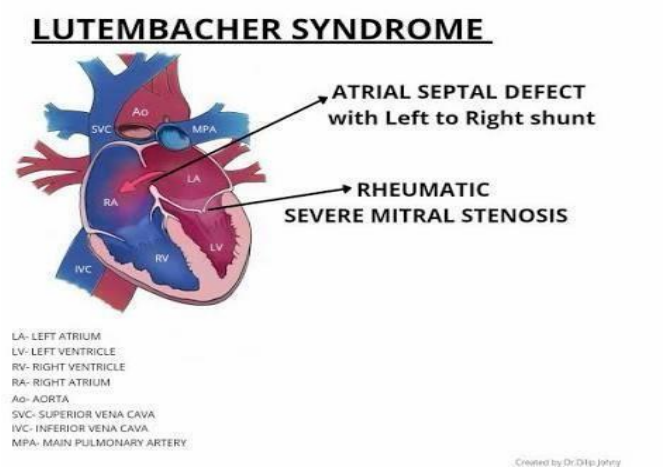


**Figure 1: Diagrammatic portrayal of Lutembacher syndrome.**

In most LS cases, the ASD is typically larger than 15 mm. Yet, with the rise of percutaneous balloon mitral valvuloplasty (BMV) for acquired MS, iatrogenic ASD caused by trans-septal puncture has become more frequent than congenital ASD. This variant is termed "iatrogenic LS".[3]

**Etiology**

Both ASD and MS in LS can arise from congenital or acquired causes. The prevalence of congenital ASD among MS patients is approximately 0.6–0.7%. As BMV procedures for MS increase, so does the incidence of residual ASD, contributing to a rise in iatrogenic LS cases. Congenital MS is exceptionally rare, comprising only 0.6% of congenital heart defects.

Mitral stenosis in LS is almost always linked to rheumatic heart disease (RHD) (Figure 2). Given the global variability in ASD prevalence, the occurrence of rheumatic MS in LS depends on regional RHD rates.[4]

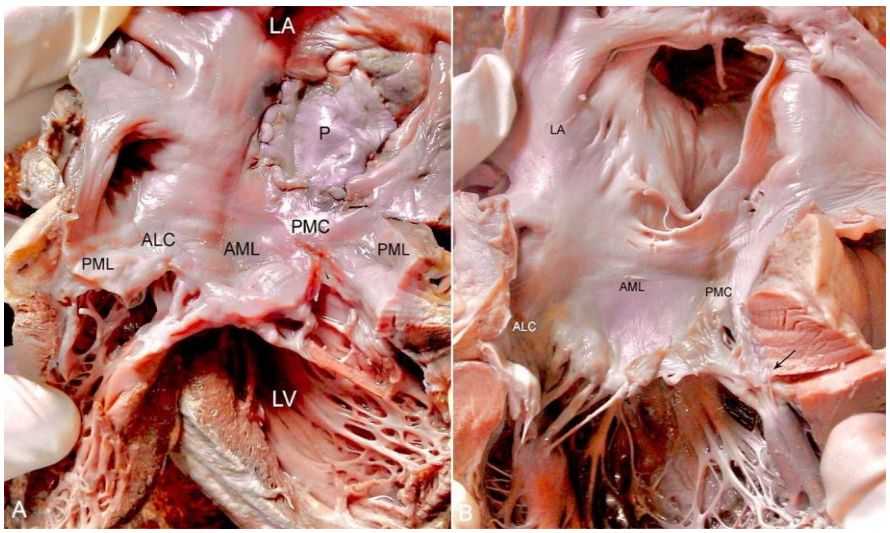


**Fig. 2. (A) Opened out left ventricular inflow tract in another patient of LS showing a patch P covering a large secundum atrial septal defect. There was rheumatic mitral stenosis with characteristic leaflet thickening, commissural fusion and accompanying sub-valvular chordal pathology and (B) a large secundum atrial septal defect with a non- rheumatic mitral valvular affliction. Note plastering (arrow) of the posterior leaflet PML to the underlying left ventricular LV endocardium. Both images show predominant involvement of valvular components at its postero-medial region (ALC, antero-lateral commissure; AML, anterior mitral leaflet; LA, left atrium; PMC, postero-medial commissure).**

**Epidemiology**

The exact prevalence of LS remains undetermined.[5] It is more frequently observed in regions with high RHD rates, such as Southeast Asia. In these areas, 40% of LS patients report a history of rheumatic fever.

The syndrome can manifest at any age but is most common in young adults, with a higher incidence in women due to the greater prevalence of ASD and rheumatic MS in females.

**Pathophysiology[6]**

The clinical manifestations of LS stem from the interaction between ASD and MS. Factors like ASD size,

 

MS severity, right ventricular compliance, and pulmonary vascular resistance influence the hemodynamic profile. When MS is severe and ASD is non-restrictive, the left atrium (LA) may decompress via the septal defect alongside the mitral valve, delaying elevated LA pressure despite severe MS.

This delay prolongs the development of pulmonary venous hypertension. However, it leads to a left-to-right shunt across the ASD, causing gradual enlargement of the right atrium (RA) and right ventricle (RV) due to increased pulmonary blood flow. Untreated cases may progress to right ventricular failure. Pulmonary artery hypertension (PAH) in LS is typically hyperkinetic, driven by increased shunting, unlike isolated severe MS, where PAH results from direct backpressure, vasoconstriction, and arterial changes. Concurrently, reduced left ventricular filling and stroke volume occur.

If the ASD is restrictive, the shunt diminishes, and the clinical course mirrors isolated MS. Additionally, LS increases susceptibility to infective endocarditis compared to isolated ASD. However, mitral valve calcification is less common in LS than in standalone MS cases.

**Transthoracic Echocardiography (TTE)**

TTE is widely regarded as the primary diagnostic method for identifying Lutembacher syndrome (LS). This approach is non-surgical, accessible in diverse healthcare environments, and reliable for diagnosing both atrial septal defects (ASD) and mitral stenosis (MS). It also plays a key role in evaluating valve leakage, blood flow dynamics, and changes in volume or pressure.[7] At different stages of LS, 2D TTE can identify left atrial enlargement, right-sided cavity expansion, ASD, pulmonary artery dilation, and mitral valve narrowing. Techniques like color Doppler and spectral imaging further aid in confirming and grading the severity of ASD, mitral valve stenosis or regurgitation, as well as tricuspid regurgitation and pulmonary pressure fluctuations[8] (Figure 3).

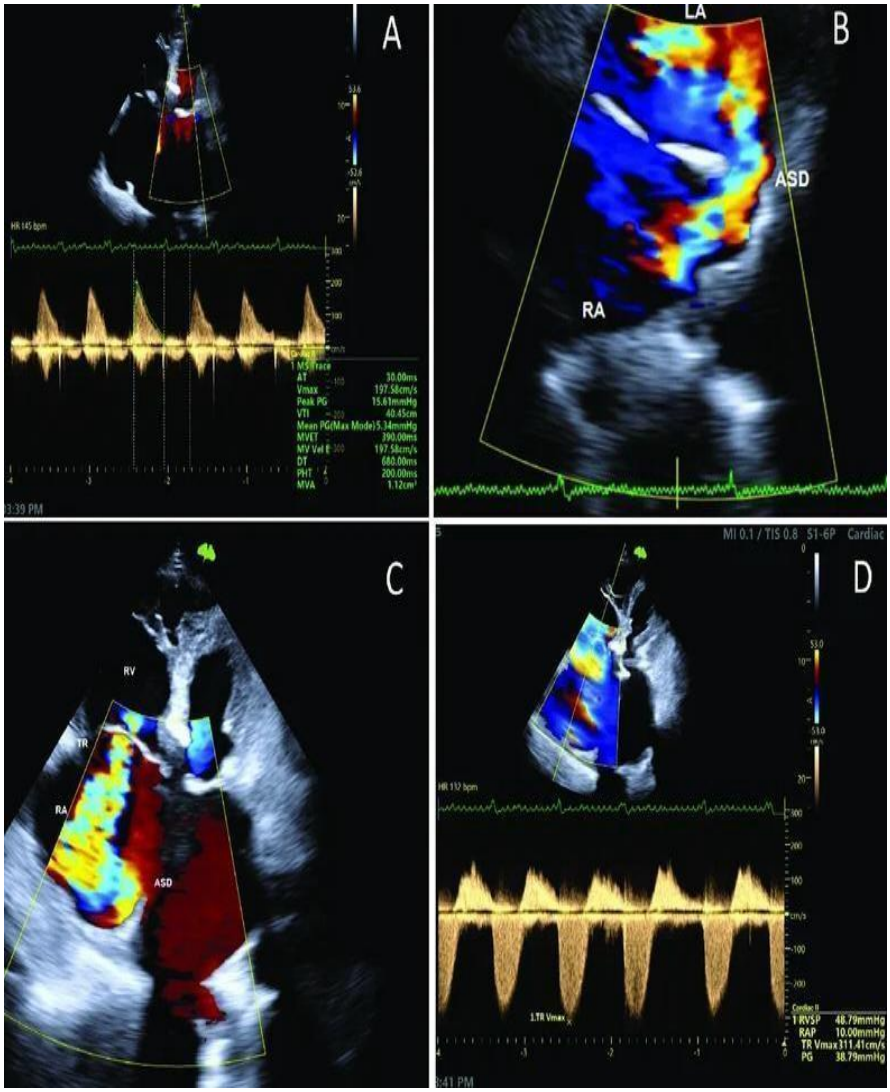


**Figure 3: Transthoracic echocardiogram. A) Mitral stenosis gradient; B) ASD color doppler flow; C) Severe tricuspid regurgitation color doppler; D) Tricuspid valve continuous wave doppler signal.**

 

**Transesophageal Echocardiography (TEE)**

TEE is particularly valuable when TTE results are unclear, aiding in the detection of associated complications such as left atrial clots before or after balloon mitral commissurotomy or post-thromboembolic events.[8] Some studies suggest TEE may offer precise insights into valve structure and the extent of commissural fusion, though further validation is needed.[9,10] Stress or exercise echocardiography tracks pressure and gradient changes during physical exertion. This method is especially critical for patients whose symptoms do not align with the apparent severity of MS[10,11] (Figure 4a,b).

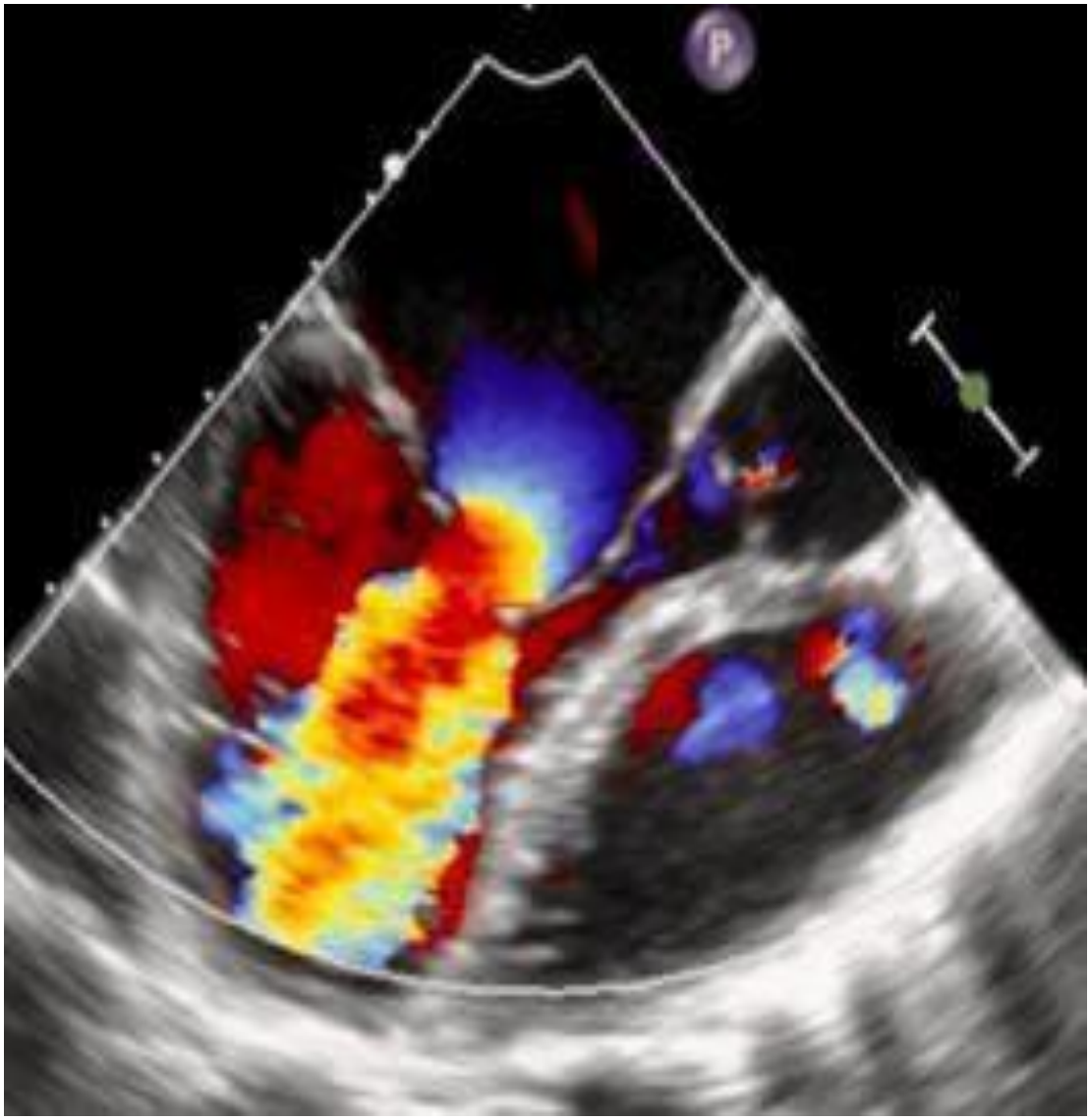

**Figure 4a: Transesophageal echocardiography (TEE) with color Doppler showing turbulent, high-velocity diastolic flow across a stenotic mitral valve (aliasing at the valve orifice and increased trans-mitral flow acceleration) consistent with significant mitral valve stenosis.**

 

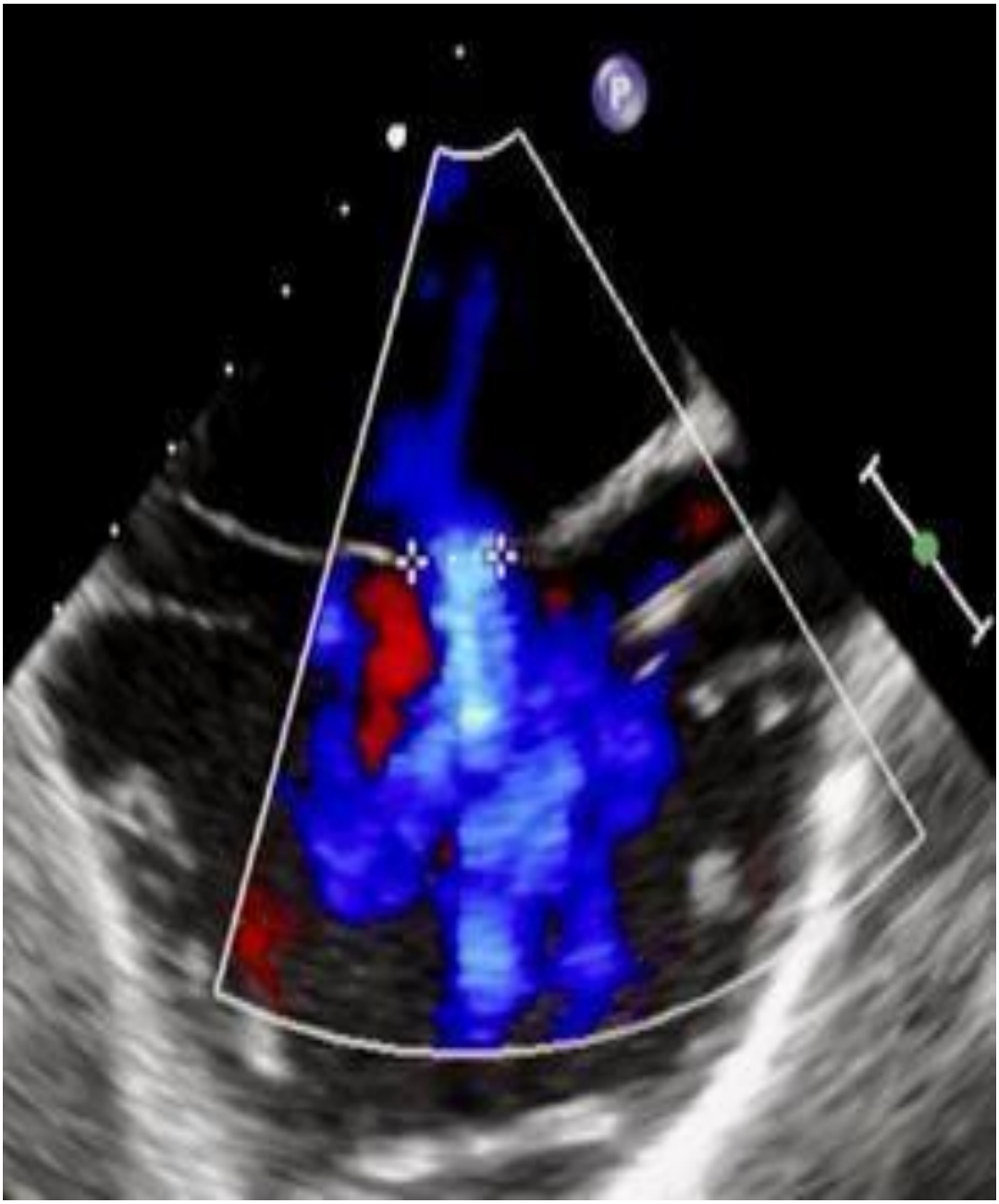

**Figure 4b: Transesophageal echocardiography (TEE) image, showing large ASD (1.09 cm) with left-to-right shunt.**

**CASE REPORT**

A 47 year old female presented to our cardiac OPD with complaints of breathlessness on minimal effort, palpitations and difficulty in climbing one flight of stairs. She informed that she was a follow up case of operated large ostium secundum atrial septal defect in March 2025. Additionally, she apprized us about her history of heavy vaginal bleeding, as she was advised oral anticoagulant due to the presence of atrial fibrillation for a prolonged period of time (currently oral anticoagulant has been stopped for last 2-3 month). There was no history of chest pain, cyanosis or syncope.

On clinical examination she was a healthy looking, over weight woman (Figure 5) with a weight of 69 kg, height 162 cm, Bp 120/80 mmHg, SPO2 96% at room air, respiratory rate of 22/min and Pulse rate was irregularly regular with a rate of 94/min. There was no cyanosis, clubbing, or any signs of congestive heart failure, On cardiovascular examination there was Grade II/VI ejection systolic murmur in the pulmonary area with normal Ist and IInd heart sounds There were **no gallop sound or clicks. Rest of the systemic examination was non contributory.**

 

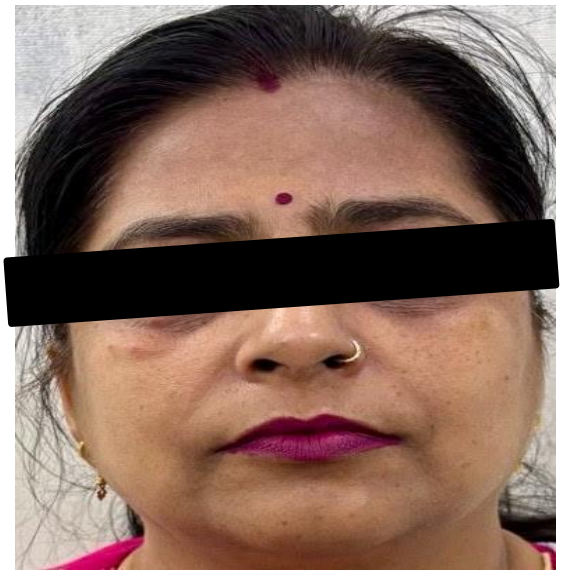

**Figure 5: Facies of our index patient.**

Xray chest PA view displayed marked cardiomegaly with distinctive bronchovascular marking consistent with restrictive lung disease (Figure 6).

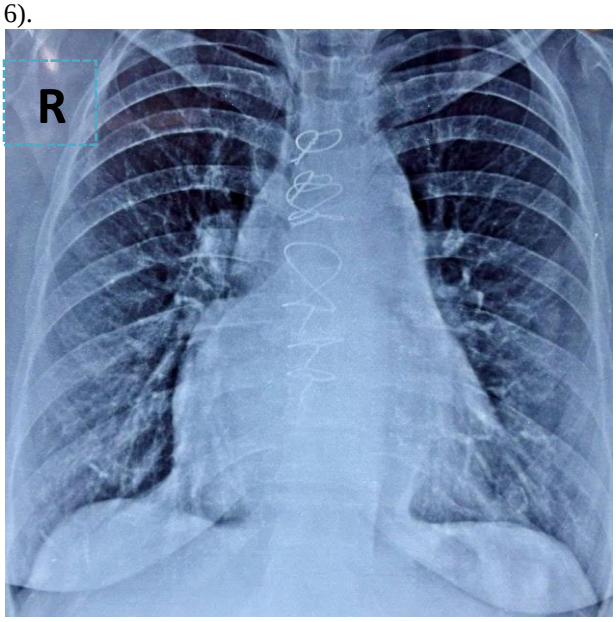


**Figure 6: X-Ray Chest PA view.**

Resting ECG showed presence of atrial fibrillation with a ventricular rate of 90/mm normal QRS axis. There was no evidence of any ventricular enlargement (Figure 7).

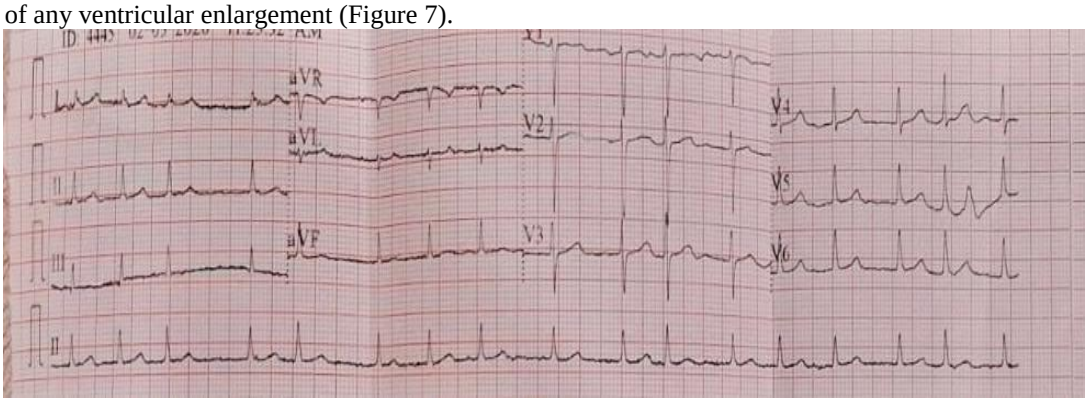

**Figure 7: Resting ECG.**

 

**Transthoracic Color Echocardiography**

The author performed all echocardiographic assessments using an Esaote My Lab X7 4D XStrain system from Italy. Images were obtained with an adult probe featuring a harmonic variable frequency electronic single-crystal array transducer while the subject lay supine and in the left lateral decubitus position.

Standard echocardiographic assessments, including M-mode, 2D, and both pulse and continuous wave and color Doppler, were conducted using subcostal, parasternal long and short axis, four- and five-chamber, and suprasternal perspectives.

**M-Mode Echocardiography**

The estimations of LV volumes, systolic functions etc was accomplished by M-mode echocardiography detailed in Table 1.

**Table 1: Calculations of M-mode echocardiography.**

| Variables | LV |
|---|---|
| IVS d | 12.4 mm |
| LVID d | 42.2 mm |
| LVPW d | 11 mm |
| IVS s | 14.2 mm |
| LVID s | 32.1 mm |
| LVPW s | 22 mm |
| HR (ECG) | 82 bpm |
| EF | 48 % |
| % LVFS | 24 % |
| LVEDV | 79.6 ml |
| LVESV | 41.4 ml |
| SV | 38.2 ml |
| CO | 3.14 l/min |
| %IVS | 15 % |
| % PW | 100 % |
| LV Mass | 173 g |
| RWT | 0.52 |

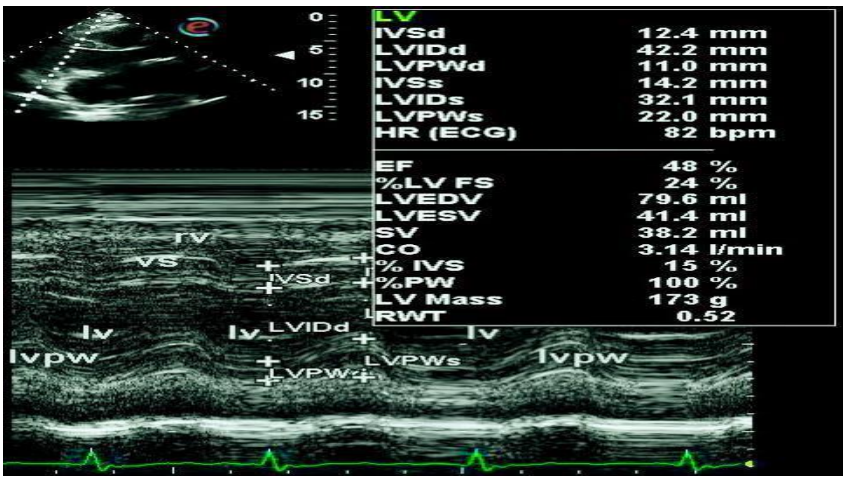


**Figure 8a**

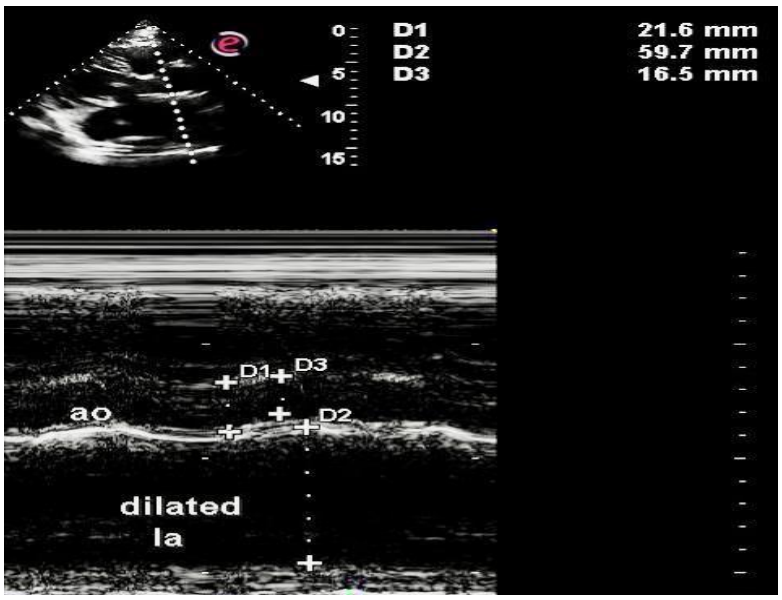


**Figure 8b**

**Figure 8: M-mode echocardiography measurements of left ventricle (Fig 8a); Left atrium was significantly dilated (Fig. 8b).**

 

**Summary of M-Mode Echocardiography:** M-Mode echocardiography showed concentric hypertrophy of left ventricle with mildly reduced LVEF = 48 %. LV mass was elevated – 173 gm. Moreover there was significantly dilated left atrium detected.

**2 Dimensional Color Echocardiography**

A detailed 2-Dimensional transthoracic echocardiography was carried out and the charecteristic features are outlined in Table 2.

**Table 2: 2-Dimensional transthoracic echocardiography.**

| | |
|---|---|
| LVADd A4C | 19.12 $cm^2$ |
| LVAd A2C | 23.14 $cm^2$ |
| LVEDV (MOD A4C) | 49.3 ml |
| LVEDV (MOD A2C) | 72.5 ml |
| LVEDV (MOD BP) | 60.3 |
| EF (MOD A4C) | 53 % |
| EF (MOD BP) | 51 % |
| SV (MOD A2C) | 36 ml |
| LVAs A4C | 11.73 $cm^2$ |
| LVAs A2C | 15.02 $cm^2$ |
| LVESV (MOD A4C) | 23.1 ml |
| LVESV (MOD A2C) | 36.6 ml |
| LSESV (MOD BP) | 29.3 ml |
| EF (MOD A2C) | 50 % |
| SV (MOD A2C) | 26.2 ml |
| SV (MOD BP) | 31.1 ml |

Other distinctive features were; **FTC OPERATED ASD**

1. ASD patch 'INSITU' & functioning normally **(Fig. 9).**

No evidence of residual Lt to Rt. shunt.

2. Dilated coronary sinus. **(Fig. 10).**
3. MITRAL STENOSIS (MILD) **(Fig. 11).**

MV domed, non calcified

MVA (PHT) = 2.92 sqcm **(Fig. 12).**

Peak/mean gradient across MV = 10.2/4.2 mmHg.

4. MITRAL REGURGITATION (MODERATE)

Thickened, large AML & PML.**(Fig.13)** MR velocity = 4.49 m/sec **(Fig. 14)**

On color flow mapping MR JET area 3.81 sq.cm; 15 % of LA area; eccentric posterior jet (Fig. 15).

5. Concentric hypertrophy of LV.
6. Normal LVEF = 48 % (M-Mode)

= 51 % (Biplane Simpson's Method) **(Fig. 16)**

Normal LV dimension.

No regional wall motion abnormality seen.

7. Mild PAH (RVSP/PAP = 35 mmHg).
8. Contrast Echocardiography (**Fig. 17**). In the 4C view contrast echo elucidated mitral stenosis, dilated right atrium, left atrium and coronary sinus dimensions were :

Height- 22.4 mm, Width - 22.5 mm and area of 5.15 sqcm.

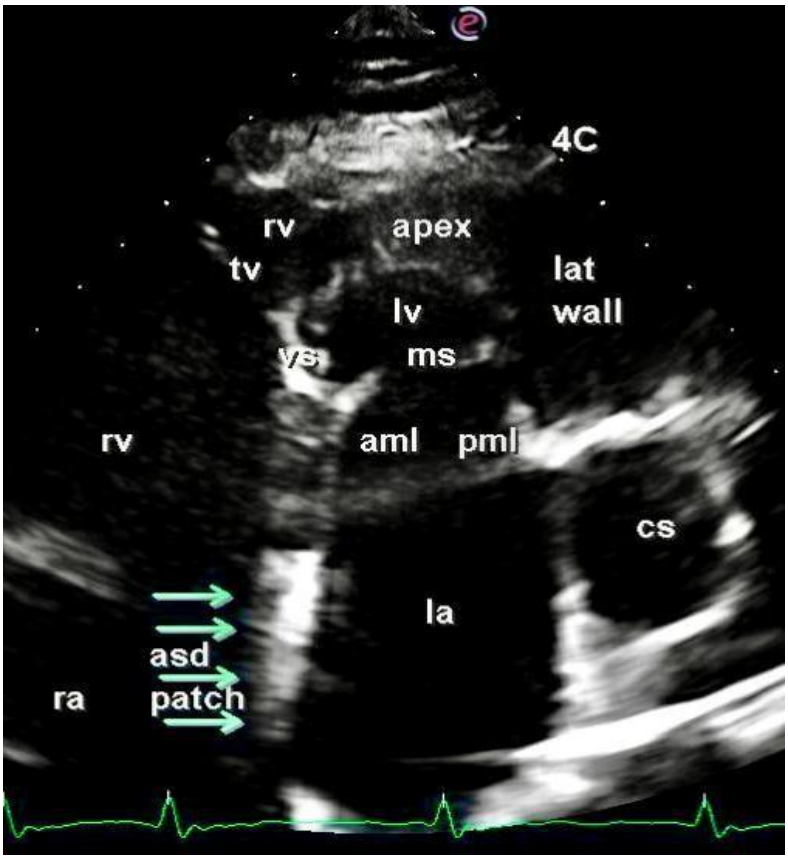


**Figure 9: 4C view depicting operated ASD patch, dilated la, right atrium (ra) and coronary sinus(cs).**

 

Domed anterior mitral leaflet (aml) and posterior mitral leaflet (pml) is displayed, consistent with rheumatic mitral stenosis (ms).

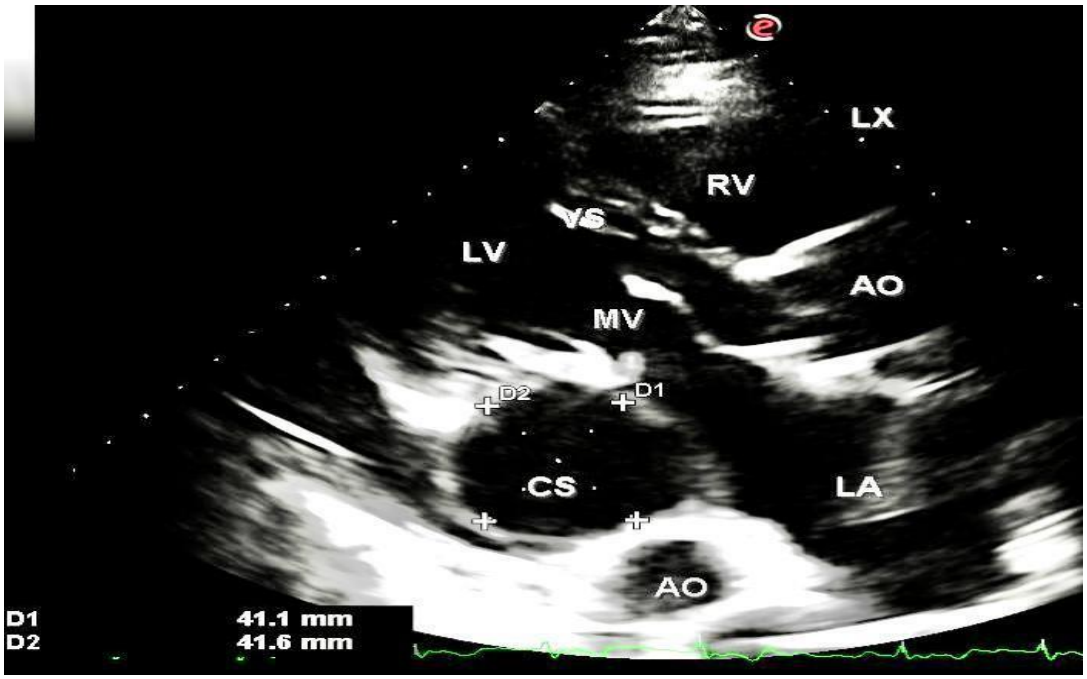


**Figure 10a**

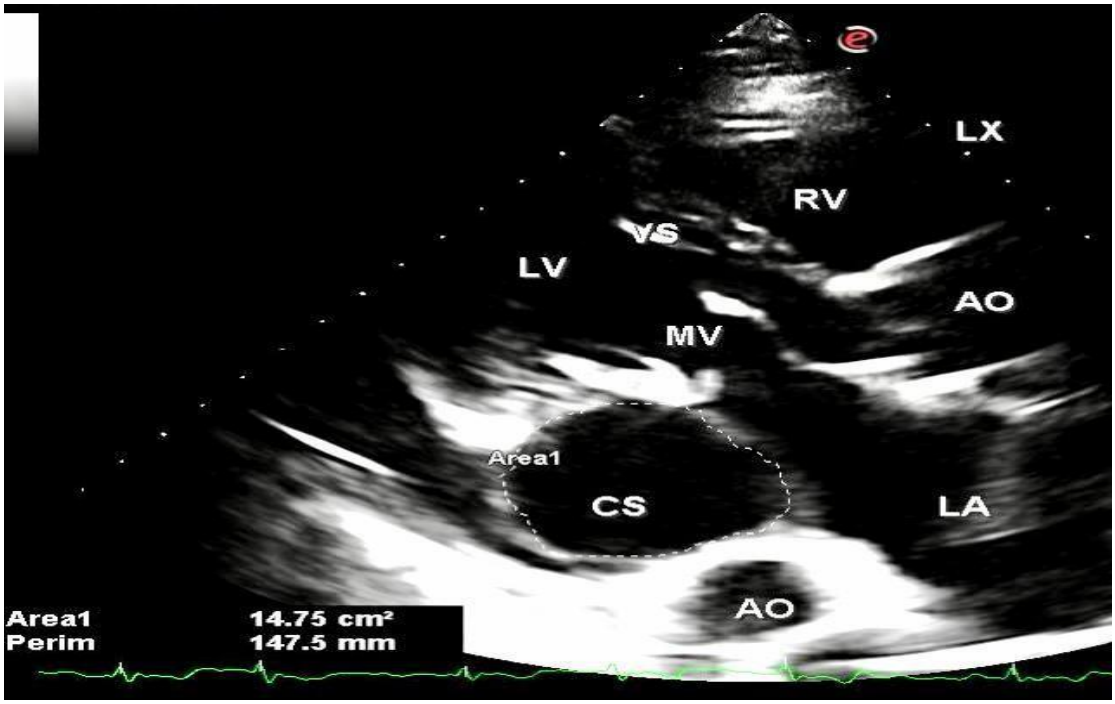


**Figure 10b**

**Figure 10: LX view portrays dilated coronary sinus (CS) with a dimension of : height - 41.1 mm, width -41.6 mm and area of 14.75 sqcm.**

 

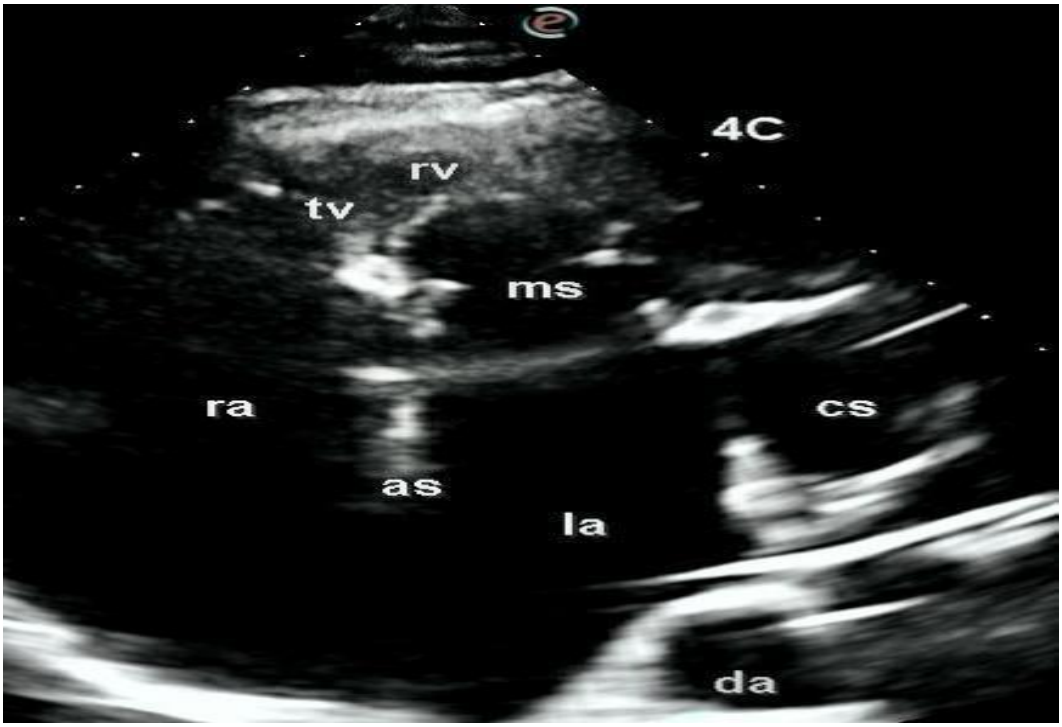


**Figure 11: 4C view shows mitral stenosis (ms) with dilated RA, LA, and CS.**

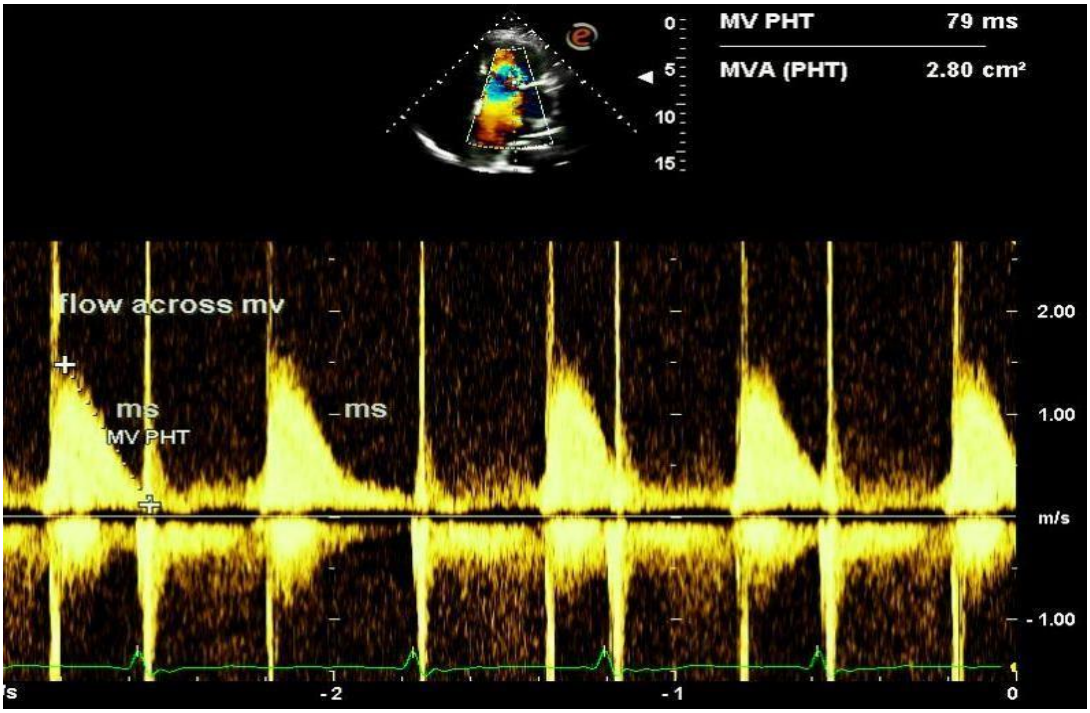


**Figure 12: Continuous wave Doppler analysis across the mitral valve reveals mild mitral stenosis with MVA (PHT) - 2.80 sqcm with peak/ mean gradient of 12/5 mmHg.**

 

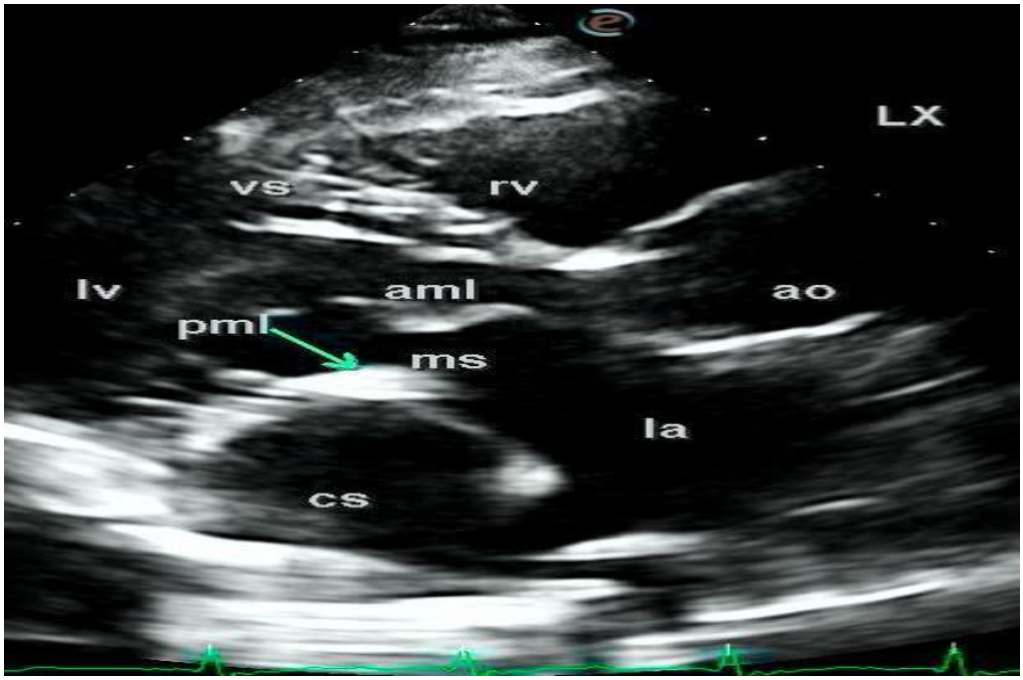


**Figure 13: LX view demonstrates mitral stenosis with large domed anterior and posterior mitral leaflet.**

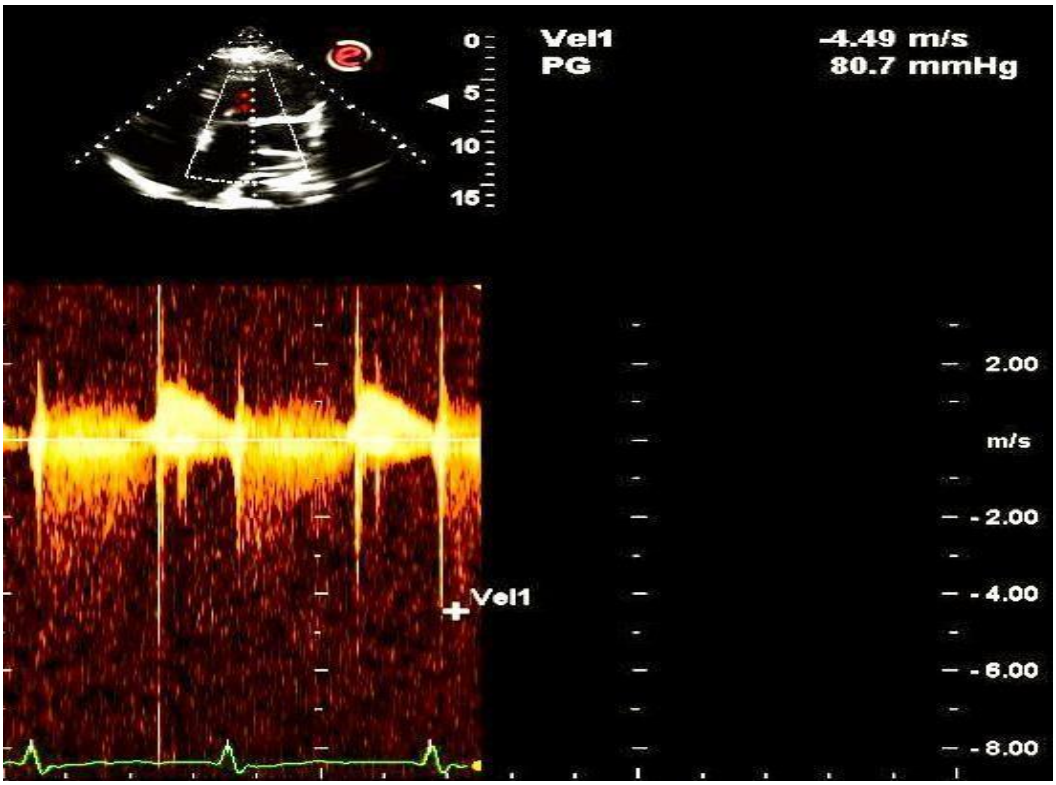


**Figure 14: Continuous wave Doppler signal of peak Mitral regurgitation velocity of 4.49 m/sec was observed.**

 

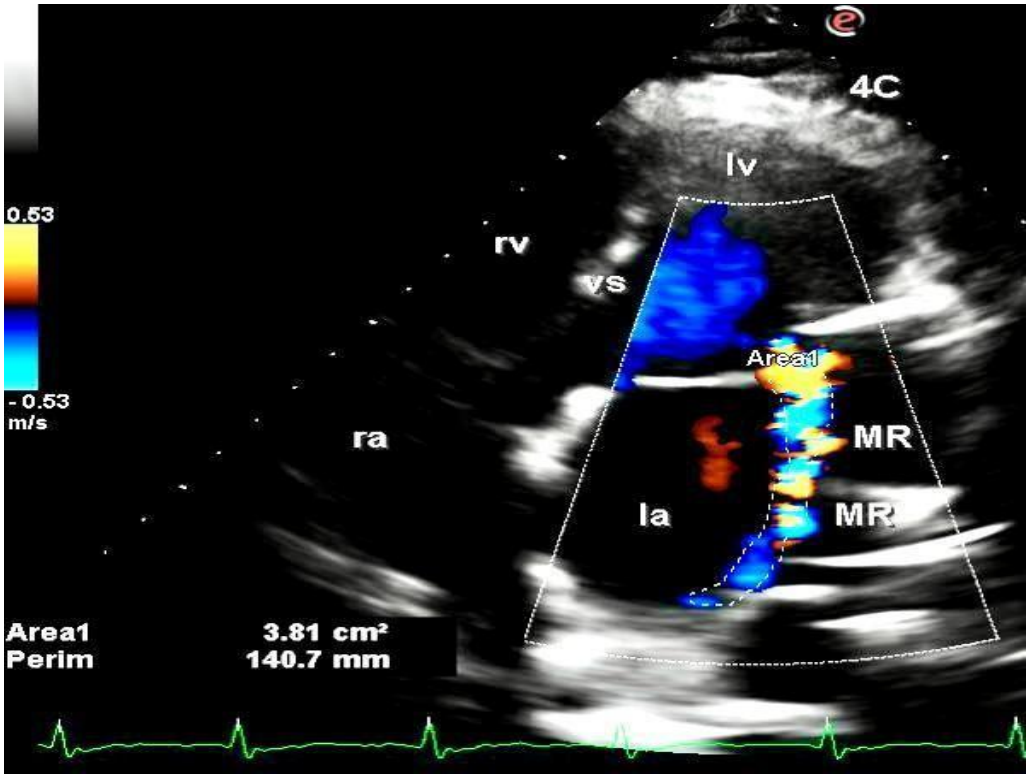


**Figure 15: Moderate sized mitral regurgitation central jet with an area of 3.81 sqcm was revealed in the 4C view.**

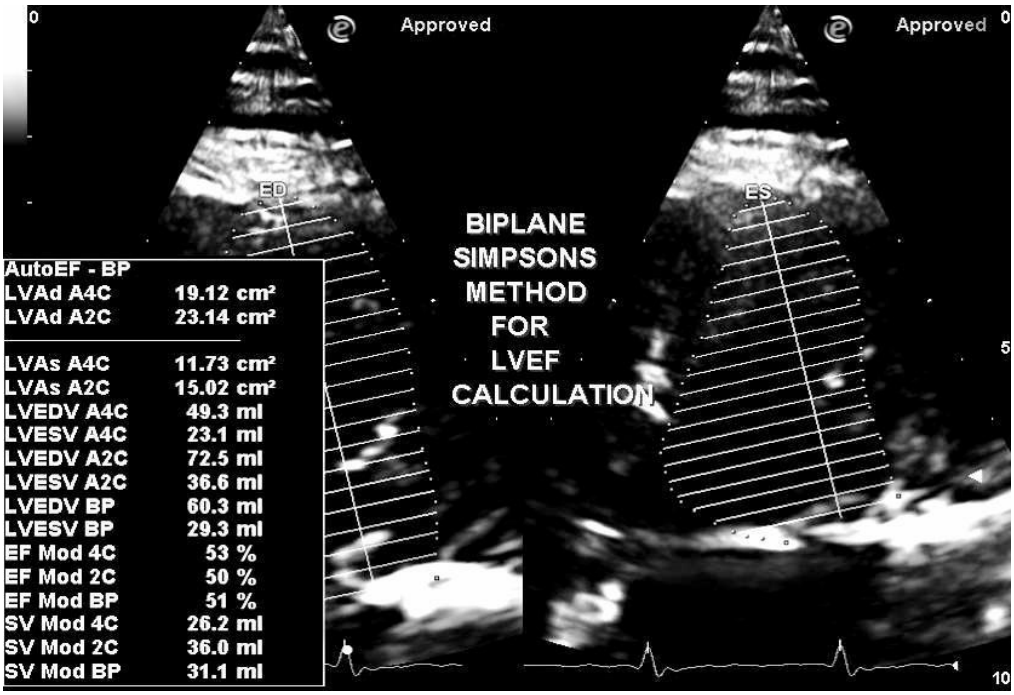


**Figure 16: LVEF estimation by biplane Simpson's method.**

 

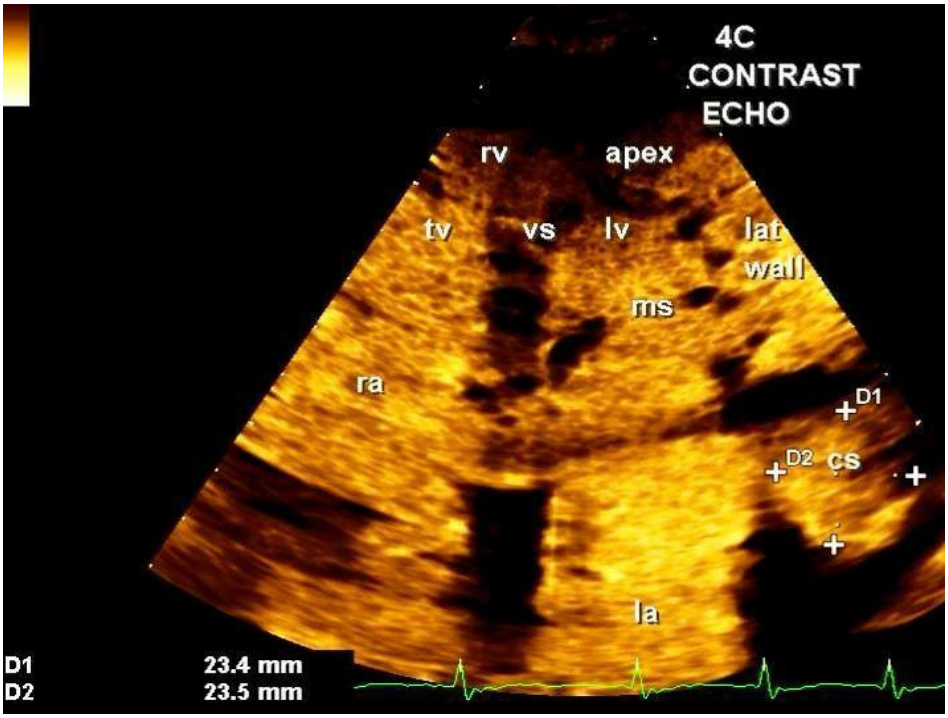


**Figure 17a**

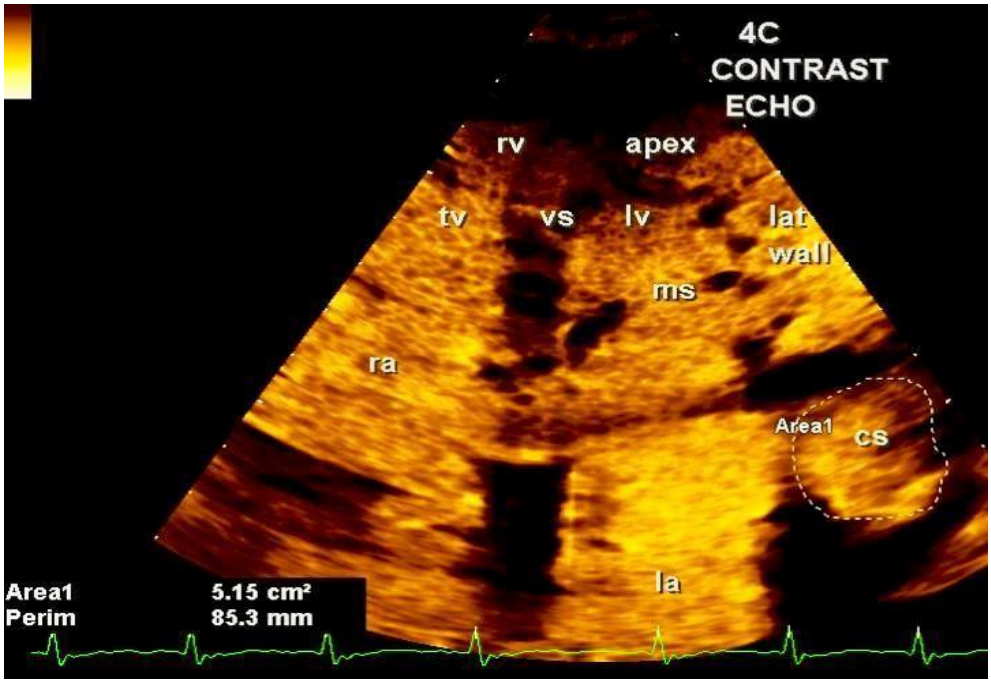


**Figure 17b**

**Figure 17: Contrast echocardiography in 4C view clearly demonstrates mitral stenosis, dilated right, left atrium and CS dimensions were:**
Height- 22.4 mm, width of 22.5 mm and area of 5.15 sqcm. The operated ASD patch is also discerned.

**Summary of Transthoracic Color Echocardiography**
In our index patient we demonstrated the presence of dilated CS in an operated case of ASD, Importantly, we also exhibited existence of mitral valve disease in the form of mild mitral stenosis (MS) and moderate mitral regurgitation (MR). Furthermore, both the left and right atrium were dilated with concentric hypertrophy of LV. LVEF was 51% by Biplane-Simpson's method and LV mass was 173 gm.

**DISCUSSION**
Cardiovascular diseases (CVD) remain a top global cause of mortality and a pressing public health issue.[12] The rising incidence of congenital heart conditions, alongside acquired CVD, is driving increased demand for both surgical and non-surgical treatments.[13] This

 

trend carries significant economic and societal burdens. Low- and middle-income countries, face exacerbated challenges due to the high prevalence of rheumatic heart disease.[14] In rare cases, congenital and acquired heart disorders may coexist.

Lutembacher syndrome (LS) is a rare condition defined by the coexistence of a congenital secundum ASD and acquired mitral stenosis (often due to rheumatic fever).[15] LS typically follows a progressive and adverse natural history.[16] The ASD in LS is often larger than 1.5 cm, leading to left-to-right shunting, worsening pulmonary hypertension (PH), and potential Eisenmenger syndrome.[15] A variant termed "reverse LS" involves right-to-left shunting alongside severe tricuspid stenosis.[17] Some researchers argue LS could encompass any mitral valve issue (stenosis, regurgitation, or mixed). The definition of LS has evolved over time, but it now commonly includes any combination of congenital or acquired ASD and MS.[18]

LS is more prevalent in females, as both ASD and MS are more common in women.[19] While isolated familial cases exist, they remain singular reports.[20]

Earlier theories suggested ASD in LS arose from a patent foramen ovale (PFO) expanding due to high left atrial pressure caused by mitral stenosis. However, modern understanding indicates ASD may be either congenital or acquired.[21] A 2001 case study by Anantha Subramaniam et al. reinforced this, showing that mitral valve replacement eliminated the left-to-right shunt, suggesting elevated left atrial pressure alone could trigger ASD.[22]

The coexistence of MS and ASD creates complex hemodynamic interactions. Factors like ASD size, MS severity, and right ventricular compliance influence clinical outcomes. In LS, blood shunting from the left atrium to the right atrium via ASD alleviates pressure buildup caused by mitral stenosis. A larger ASD can reduce harmful effects of high left atrial pressure. Without ASD, elevated atrial pressure from MS often leads to rapid pulmonary congestion. ASD in LS mitigates this by redirecting blood flow, lessening pulmonary strain. Similarly, symptoms of lung congestion may be less severe or absent due to this shunting mechanism. The presence of ASD helps reduce congestion and pressure buildup in the pulmonary blood vessels among affected individuals. As a result, symptoms linked to lung congestion such as coughing up blood (hemoptysis), fluid accumulation in the lungs (pulmonary edema), difficulty breathing when lying flat (orthopnea), and sudden shortness of breath at night (paroxysmal nocturnal dyspnea) may appear. Conversely, a left-to-right shunt caused by MS can lead to excessive blood flow into the right side of the heart, causing enlargement of the right heart chambers, pulmonary hypertension, and eventual right heart failure.[23]

Diagnosing and assessing the severity of ASD and MS relies heavily on echocardiography. Advanced imaging techniques like transesophageal echocardiography (TEE) and cardiac catheterization provide detailed anatomical and functional data critical for treatment planning. Managing Lutembacher syndrome requires a tailored strategy, considering factors like the extent of MS, the size and position of ASD, and symptom severity. Closing the ASD—either through surgery or catheter-based procedures—is often necessary to prevent complications like paradoxical embolism or worsening pulmonary hypertension. In severe MS cases, interventions like balloon valvuloplasty or valve repair/replacement may be explored. Minimally invasive catheter-based techniques are increasingly favored over traditional open-heart surgery for treating this condition.[24]

Recent advancements, including percutaneous mitral commissurotomy (PTMC) and ASD closure using devices like the Amplatzer septal occluder, offer less invasive alternatives to major surgeries.[25,26] These methods reduce the risks associated with traditional surgical approaches.

Further research is needed to develop new therapies, refine diagnostic tools, and evaluate long-term treatment effects in Lutembacher syndrome patients. Collaboration among healthcare providers, researchers, and industry partners is vital to enhance understanding of this condition and improve patient outcomes.

## CONCLUSION

Lutembacher's syndrome is a rare, intricate congenital heart disorder. Early detection and surgical intervention yield favorable results, whereas delayed diagnosis and progression to heart failure lead to poor outcomes. Many patients succumb to heart failure, irregular heart rhythms, or blood clots affecting the brain. Timely management can lower complications and death rates. Transthoracic echocardiography is the primary and extremely valuable tool for early detection and assessment of severity to guide treatment. Expanding access to echocardiography in areas prone to rheumatic heart disease could prevent late diagnosis of related heart issues. Doppler echocardiography is the gold standard for evaluating mitral valve area (MVA). While open-heart surgery is often used for combined heart defects, percutaneous catheter-based therapies now serve as exceptions. These include combining balloon valvuloplasty for MS with Amplatzer devices for ASD closure.

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