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OPERATED ATRIAL SEPTAL DEFECT ASSOCIATED WITH DILATED CORONARY SINUS IN AN ADULT FEMALE: CASE REPORT & LITERATURE REVIEW

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ABSTRACT
While uncommon in individuals without heart conditions, the coronary sinus (CS) is frequently observed during echocardiography in those with right-sided cardiac disorders. The frequency of this occurrence and its connection to right-sided heart anatomy and pressure levels remain unclear. A widened coronary sinus in an adult woman is often an accidental discovery. Studies repeatedly highlight Persistent Left Superior Vena Cava (PLSVC) draining into the CS as the most prevalent congenital abnormality. Additional significant causes encompass elevated right atrial pressure and coronary arteriovenous fistulas. Most individuals with an enlarged CS, particularly those with PLSVC, exhibit no symptoms and maintain normal life expectancy. Nevertheless, an exact diagnosis is essential if the patient undergoes invasive treatments. We are detailing a 47-year-old woman who underwent surgery for a large secundum atrial septal defect (ASD) in March 2025. During a standard follow-up transthoracic echocardiogram (TTE), we also identified an enlarged CS alongside an in-situ ASD patch without any residual shunt.

KEYWORDS: *Repaired ASD, Enlarged coronary sinus, Transthoracic echocardiography, Persistent left superior vena cava, Cardiac CT.*

INTRODUCTION

Anatomy and Role of the Coronary Sinus

The coronary sinus, measuring 3–5 cm in length and 1 cm in diameter, is the largest coronary vein. It originates on the posterior side of the heart, between the left atrium and ventricle. It then progresses along the left posterior atrioventricular groove before emptying into the posteroinferior region of the right atrium, located near the inferior vena cava's entrance and above the tricuspid valve's septal leaflet (Figure1, 2).

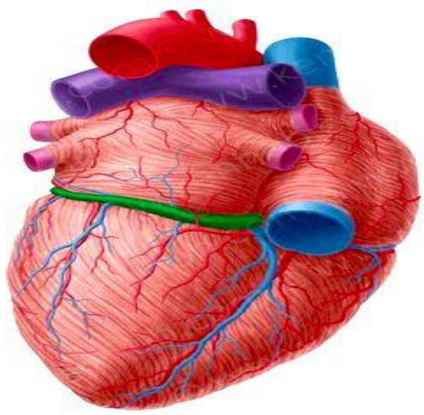

Figure 1: Diagrammatic Illustration of CS.

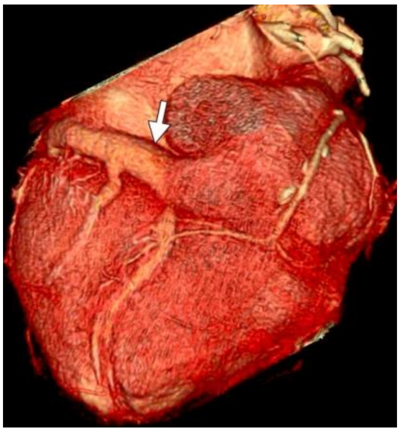

Figure 2: Cardiac CT demonstrating CS (white arrow is pointing to coronary sinus).

The coronary sinus forms from multiple venous connections. Its start is defined by the merging of the great cardiac vein and the oblique vein of the left atrium (Marshall's oblique vein). Downstream, the posterior vein of the left ventricle, middle cardiac vein, and small cardiac vein sequentially drain into the CS before it joins the right atrium.[1,2]

In brief, the following veins empty into the coronary sinus (Figure 3):

- Great cardiac vein
- Oblique vein of the left atrium
- Posterior vein of the left ventricle
- Middle cardiac vein
- Small cardiac vein

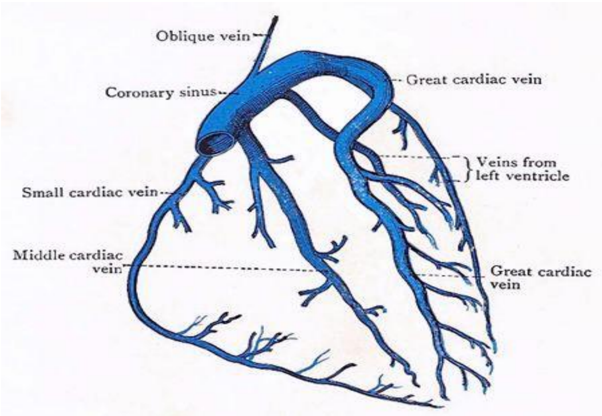

Figure 3: Coronary sinus venous system.

All tributaries, except the oblique vein of the left atrium, have valves at their connection to the CS. The origin of the CS is marked by the Vieussens Valve at the end of the Great Cardiac Vein. The CS extends along the left atrioventricular groove until it reaches the Thebesian valve, situated at the CS's orifice as it empties into the right atrium. This valve prevents backflow of blood from the right atrium into the CS during diastole.[3]

As the primary tributary of the greater cardiac venous system, the CS returns roughly 55% of the coronary blood flow to the right atrium. The anterior cardiac veins, numbering 2–5, account for about 35% of coronary drainage. The remaining 10% is carried by the smallest cardiac veins (Thebesian veins), which drain into any of the four heart chambers.[4]

Embryological Development

During week 3 of embryonic growth, the primitive heart tube splits into two structures. The primordial atrium evolves into the left and right atria, while the sinus venosus forms right and left horns. The primordial IVC and SVC drain into the right horn, which is later absorbed by the expanding right atrium. The left horn persists to form the CS and its associated tributaries.[5] **(Figure 4).**

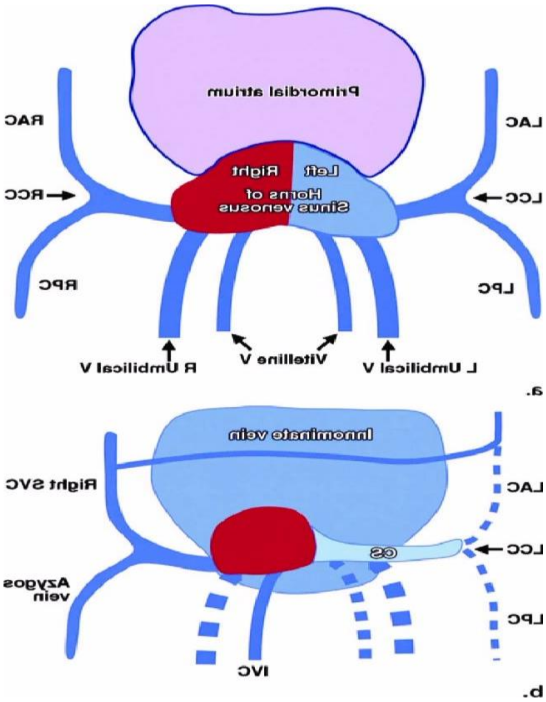

Figure 4: Coronary sinus venous system.

Figure 4: Embryology of the CS. (a) The three main paired venous systems of the embryo, the cardinal, umbilical, and vitelline veins, drain into the ipsilateral horns of the sinus venosus. L = left, LAC = left anterior cardinal vein, LCC = left common cardinal vein, LPC = left posterior cardinal vein, R = right, RAC = right anterior cardinal vein, RCC = right common cardinal vein, RPC = right posterior cardinal vein, V = vein. (b) The right SVC develops from the right common and right anterior cardinal veins. With development of the innominate vein, the LSVC involutes. The left horn of the sinus venosus and adjacent left common cardinal vein (LCC) remain in adult life as the CS. The right horn of the sinus venosus persists as the venous portion of the right atrium. LAC = left anterior cardinal vein, LPC = left posterior cardinal vein.

Normal Coronary Sinus Dimension

An enlarged coronary sinus in an adult woman is frequently an incidental finding on transthoracic echocardiography (TTE). A normal CS diameter typically ranges from 0.5 cm to 0.9 cm; measurements exceeding 1.0 cm are considered abnormal. The most frequent cause is persistent left superior vena cava (PLSVC).[6]

Possible causes of a dilated coronary sinus identified via echocardiography[7] include the following:

1. **Dilated coronary sinus**

a) **Elevated right atrial pressure and pulmonary arterial hypertension**: Increased right atrial pressure (RAP) and systolic pulmonary arterial pressure (sPAP) are often linked to pulmonary hypertension (PH) and may contribute to CS dilation.

b) **Abnormal venous flow into the coronary sinus:** A dilated CS can occur due to excessive blood flow from irregular venous drainage. This may stem from conditions like persistent left superior vena cava (PLSVC), total anomalous pulmonary venous connection (TAPVC), severe tricuspid regurgitation (TR), an unroofed CS, or a coronary artery fistula (CAF) into the CS. In this case, the absence of a left lower sternal murmur and minimal tricuspid regurgitation on imaging reduced suspicion for severe TR.

c) **Persistent left superior vena cava:** PLSVC, a congenital venous anomaly affecting ~0.3–0.5% of people, typically drains into the right atrium via the CS. A dilated CS without elevated RAP might suggest PLSVC. Saline contrast echocardiography was used to confirm or exclude this, revealing a distinct pattern where contrast entered the CS before the right atrium or ventricle if PLSVC was present.

2. **Total anomalous pulmonary venous connection (TAPVC)**

This congenital defect, causing cyanosis, involves pulmonary veins connecting abnormally to the coronary sinus instead of the left atrium. It can lead to CS dilation due to increased pulmonary blood flow. However, the patient's non-cyanotic status, normal left atrium and chamber sizes, and absence of the expected poor clinical course made TAPVC unlikely.

3. **Unroofed coronary sinus**

A defect in the CS-atrium wall causes a left-to-right shunt, dilating the CS. Symptoms often include pulmonary hypertension or heart failure. Echocardiography typically detects this shunt, but none was observed here, lowering the likelihood of this diagnosis.

4. **Coronary artery fistula (CAF)**

An abnormal connection between a coronary artery and low-pressure structures like the CS can cause a left-to-right shunt and CS dilation. Echocardiography showed color aliasing in the CS, hinting at a high-pressure structure connection. Further tests, including CT, angiography, and catheterization, were needed to confirm CAF's presence and shunt size.

CASE REPORT

A 47 year old female presented to our cardiac OPD with complaints of breath with minimal effort, palpitations and difficulty in climbing one flight of stairs. She informed that she is a follow up case of operated large ostium seundum atrial septal defect in March 2025.Additionally, she apprized us about her history of heavy vaginal bleeding, as she was advised Acitrome due to the presence of atrial fibrillation for a prolonged period of time (currently Acitrome 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 94/min. There was no cyanosis, clubbing, or any signs of congestive heart failure, On cardiovascular examination there was Grade II 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).

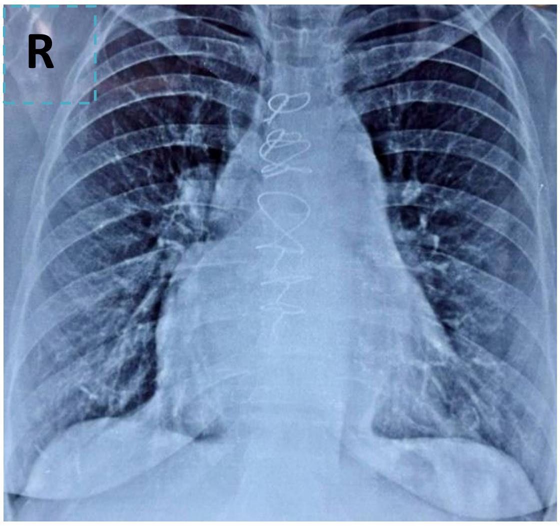

Figure 6: X-Ray Chest PA view.

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).

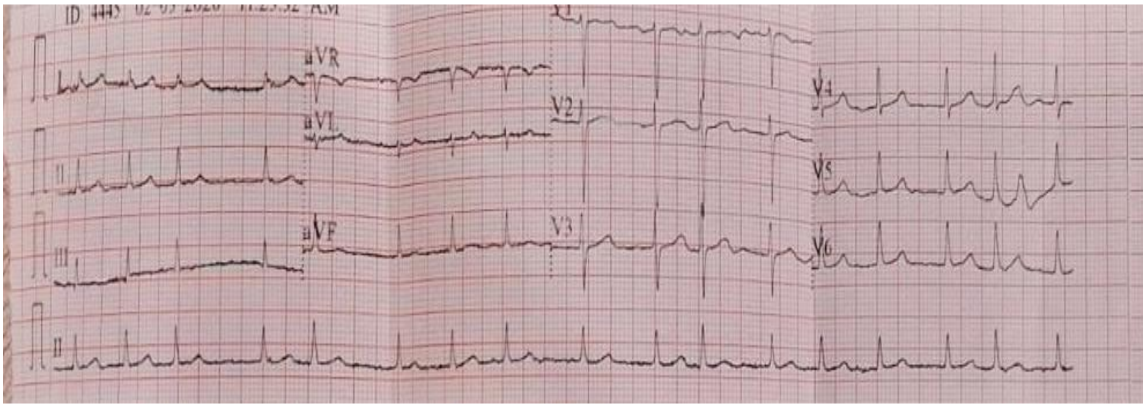

Figure 7: Resting ECG.

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

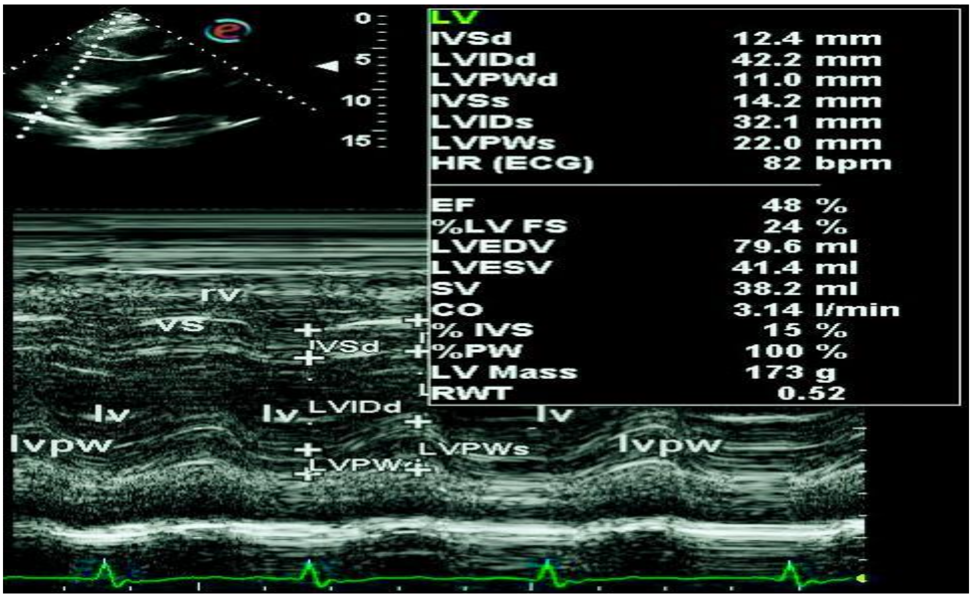

Figure 8a

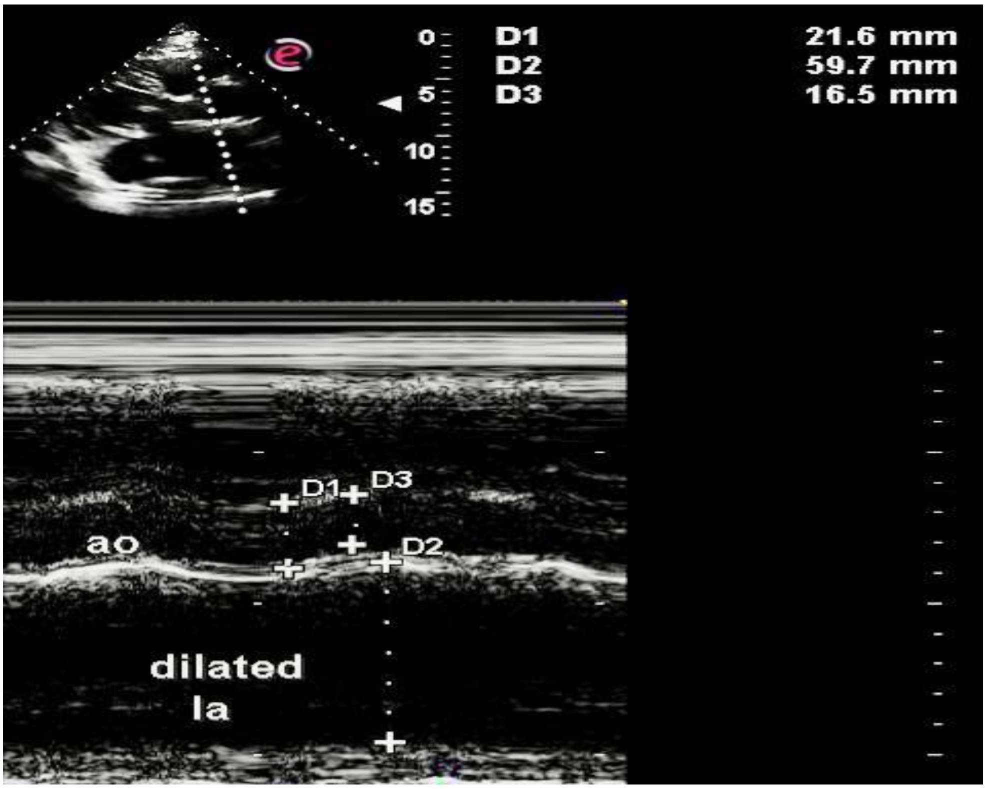

Figure 8b

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 with dimensions :

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

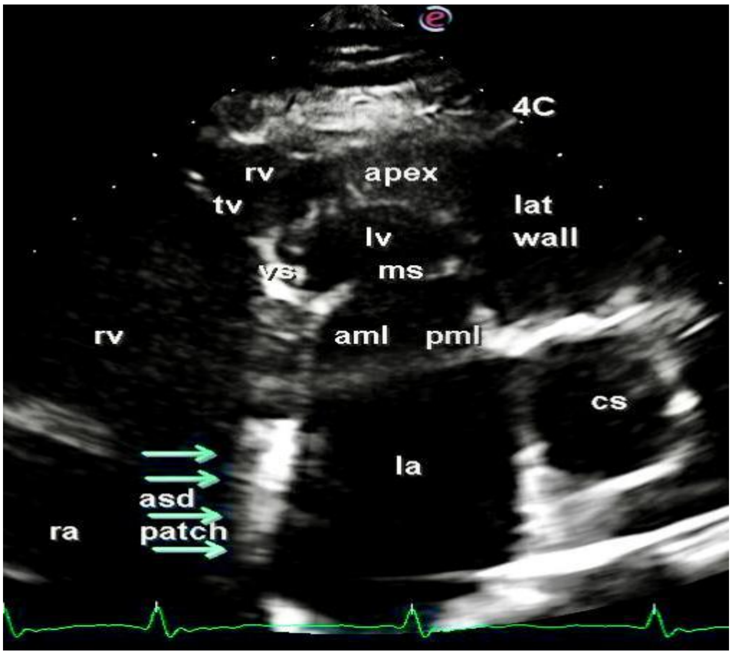

Figure 9: 4C view depicting operated ASD patch, dilated la, right atrium (ra) and coronary sinus (cs). Domed anterior mitral leaflet (aml) and posterior mitral leaflet (pml) is displayed, consistent with rheumatic mitral stenosis (ms).

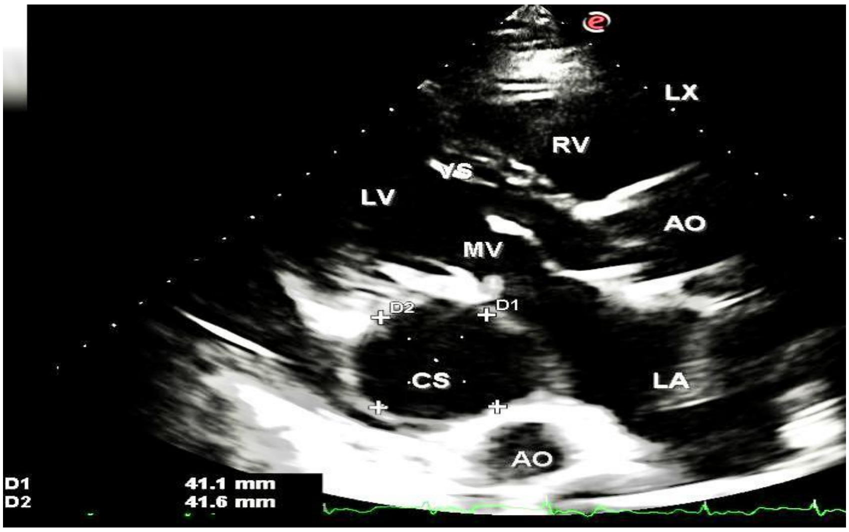

Figure 10a

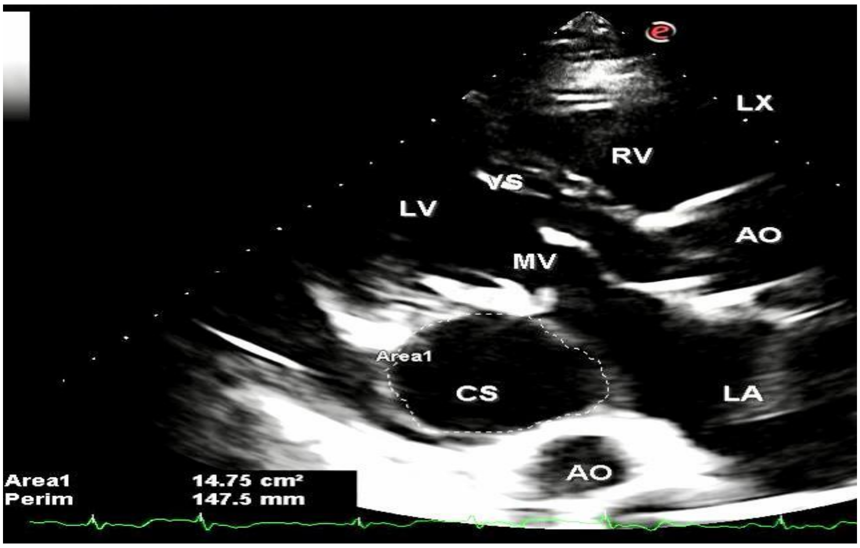

Figure 10b

Figure 10: LX view portrays dilated coronary sinus (CS) with dimensions: 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 coronary sinus (cs).

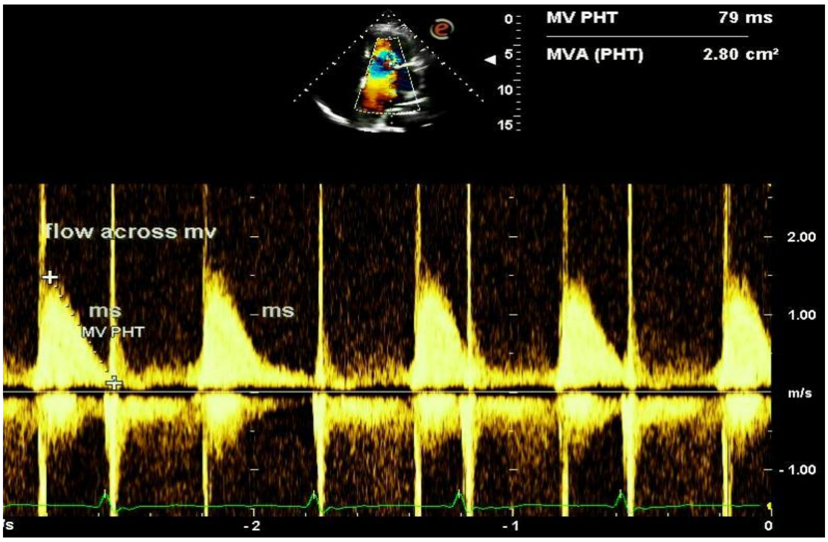

Figure 12: Continuous wave Doppler analysis across the mitral valve reveals mild mitral stenosis with MVA (PHT) - 2.80 sqcm with peak/ mean gradient of 12/5 mmhg.

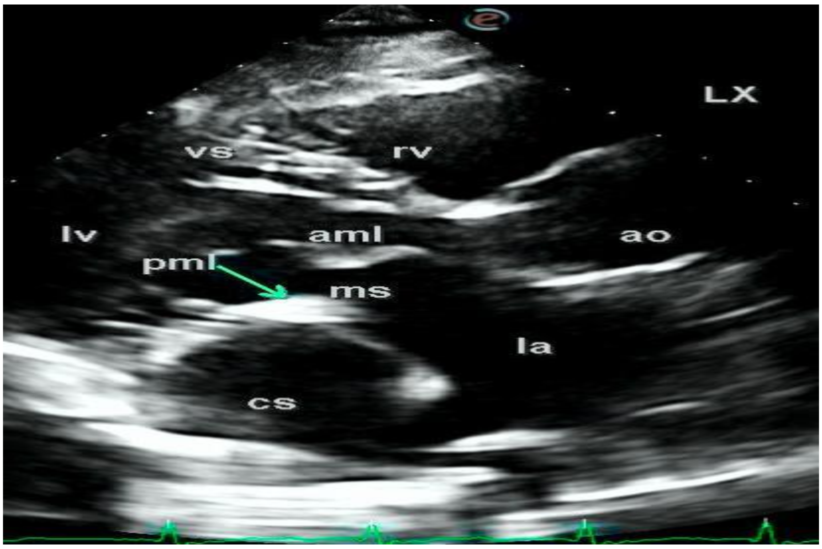

Figure 13: LX view demonstrates mitral stenosis with large domed anterior and posterior mitral leaflet.

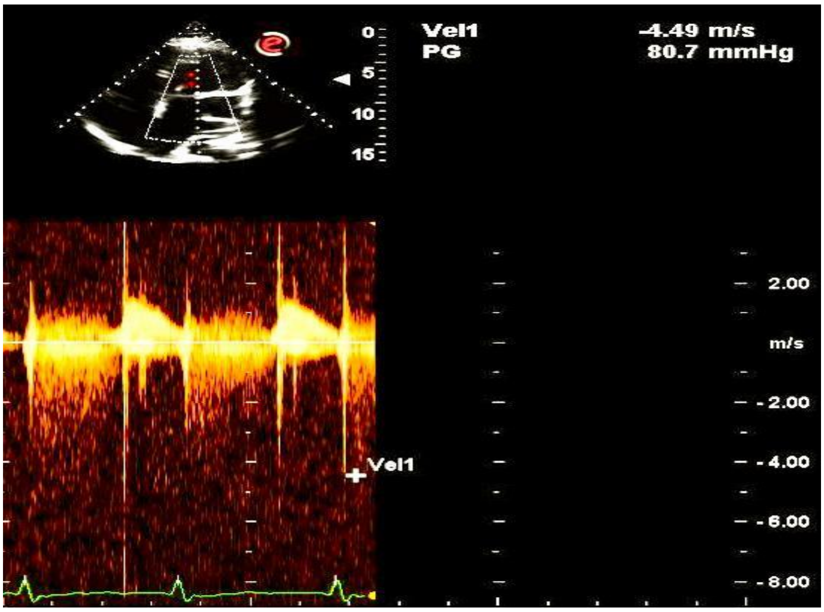

Figure 14: Continuous wave Doppler signal of peak Mitral regurgitation velocity of 4.49 m/sec was observed.

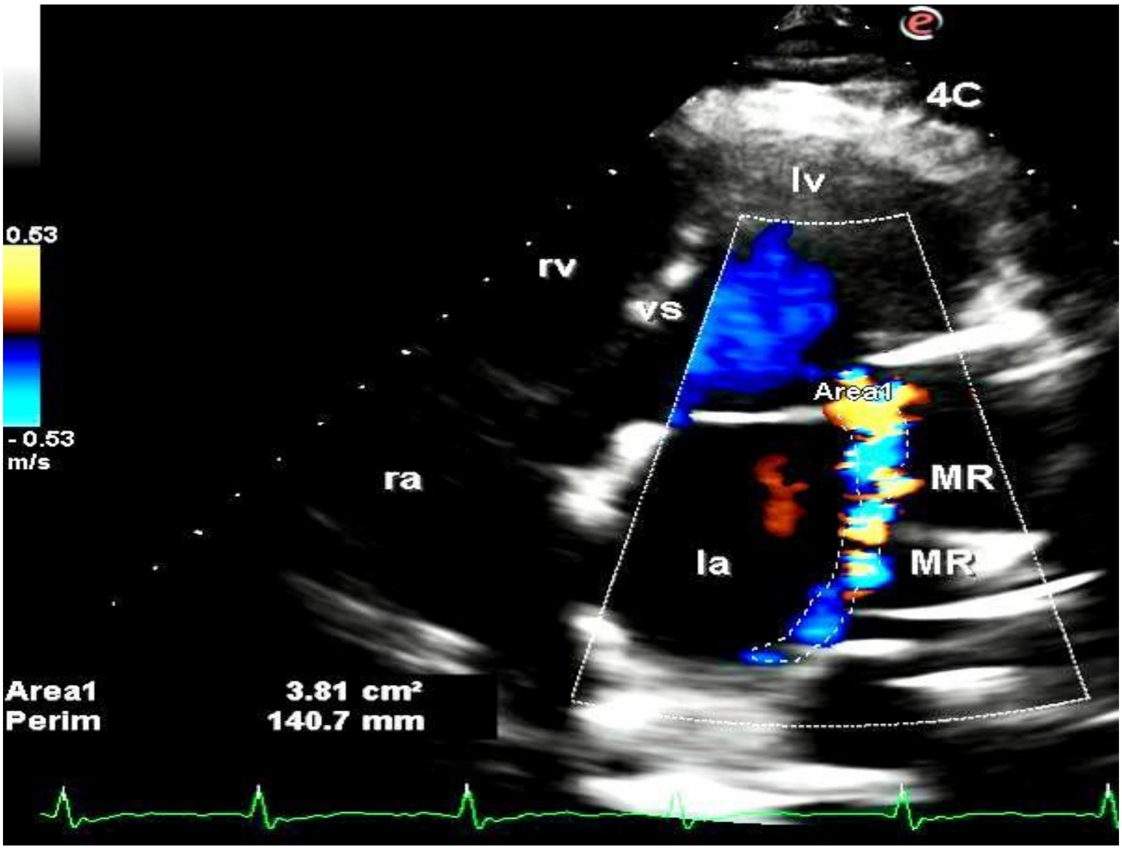

Figure 15: Moderate sized mitral regulation central jet with an area of 3.81 sqcm was revealed in the 4C view.

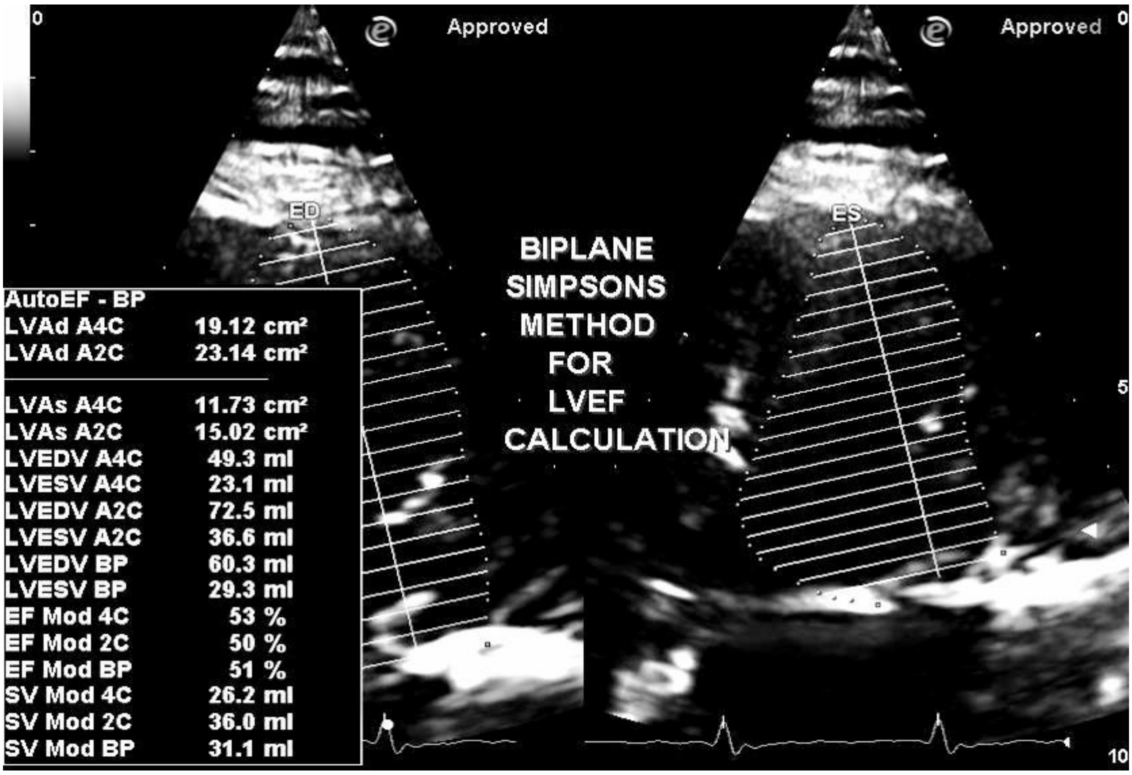

Figure 16: LVEF estimation by biplane Simpson's method.

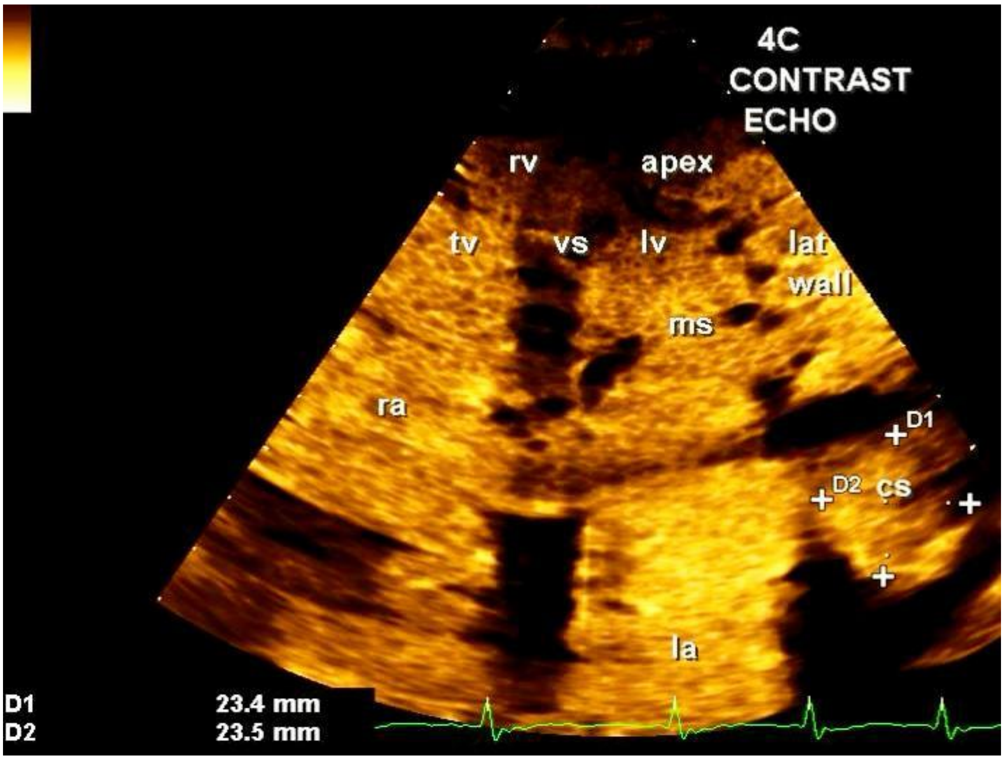

Figure 17

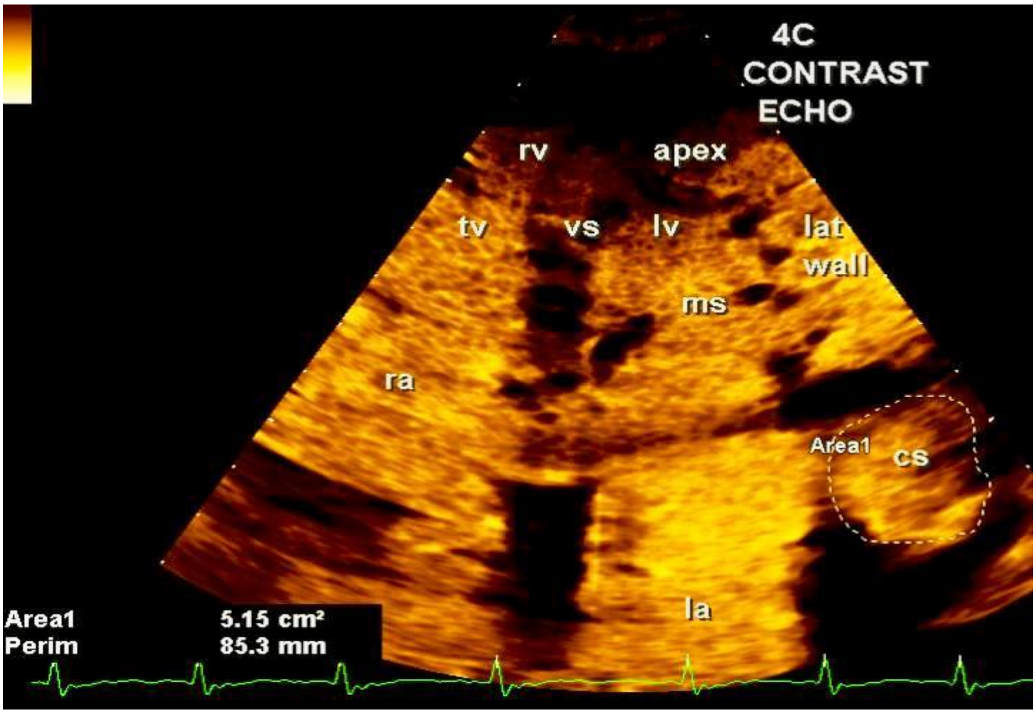

Figure 17: Contrast echocardiography in 4C view clearly demonstrates mitral stenosis, dilated right, left atrium and coronary sinus (cs) with dimensions.

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, Significantly 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

Coronary sinus (CS) is the largest vein draining the blood from heart. It is a muscular tube of about 2 cm to 3 cm length and 1 cm in caliber.[8]

Coronary sinus is the blood conduit in the continuation of the Great Cardiac Vein (GCV) situated between the valve of GCV of Vieussens or the point of entrance of the Oblique Vein of Left Atrium (OVLA) and the Thebesian Valve (ThbV) of the ostium of the CS. It is situated in the posterior part of the coronary sulcus of the heart, usually covered by muscular fibers from the Left Atrium (LA). It continues to run on the posteroinferior region of Atrioventricular groove (AV groove) until it ends at its orifice located between the Inferior Vena Cava (IVC) and the tricuspid valve on the inferior region of the atrial septum. The CS and its tributaries return blood to the Right Atrium (RA) from the whole heart including its septa except the anterior region of the Right Ventricle (RV) and small, variable parts of both atria and Left Ventricle (LV).[8]

The CS has become a clinically important structure through its role in providing access for different cardiac procedures.

The coronary venous system has become important in many electrophysiological procedures, viz. biventricular pacing, arrhythmia ablation and for deployment of an array of cardiac devices. The advent of advanced invasive and interventional cardiac treatment and management tools for common disorders like heart failure has made understanding of CS anatomy necessary. There is also an increased interest in the CS as an access point for interventionists for ablation procedures as an arrhythmia source and for mapping.[8]

Currently one of the most common uses of the cardiac venous system is the placement of left sided pacing leads in cardiac resynchronization therapy. In this, the leads are permanently placed within the cardiac venous system to minimize delayed conduction in the LV. The anatomy of these veins provides a unique access to myocardium of LV.[8]

Detailed anatomical knowledge of CS is clinically relevant, but not much data is available on its morphology and morphometry.

Dilated Coronary Sinus – Causes

A dilated coronary sinus is typically caused by elevated right-sided heart pressure or abnormal blood flow draining directly into the sinus. The condition is rarely seen in healthy individuals and is usually categorized into two primary mechanisms: structural heart issues and congenital venous anomalies.[9]

1. Elevated Right Atrial Pressure

- **Severe Tricuspid Regurgitation:** Leakiness of the tricuspid valve allows blood to flow backward into the right atrium, transmitting high pressure into the coronary sinus.[10]
- **Pulmonary Hypertension:** High blood pressure in the arteries that serve the lungs forces the right side of the heart to work harder, leading to increased right atrial pressure.[9]
- **Right Ventricular Dysfunction or Heart Failure:** Poor pumping function in the right ventricle causes blood to back up into the right atrium and coronary sinus.[11]

2. Congenital Venous Anomalies

- **Persistent Left Superior Vena Cava (PLSVC):** This is the most common congenital anomaly causing a dilated coronary sinus. Instead of draining properly into the right atrium via the usual pathways, the left- sided veins drain into the coronary sinus.[11]
- **Total Anomalous Pulmonary Venous Return (TAPVC):** A condition where oxygenated blood from the lungs drains improperly into the right atrium or its veins (such as the coronary sinus) rather than the left atrium.[11]
- **Coronary Artery Fistula:** An abnormal connection allowing high-pressure oxygenated blood from a coronary artery to shunt directly into the low-pressure coronary sinus.[11]

In our index patient CS dilatation was due to multiple factors:

a) Long standing large ASD
b) Accompanying rheumatic mitral stenosis and mitral regurgitation.
c) Concurrent co-existence of Pulmonary hypertension.

CONCLUSION

Following a thorough physical assessment and 2D Color Doppler TTE, our patient who had undergone ASD surgery and presented with rheumatic mitral stenosis was found to exhibit an enlarged coronary sinus. While TTE can suggest dilation indicative of CS, advanced imaging methods such as CT or MRI are critical for precise evaluation, particularly to detect an associated left superior vena cava or a bridging innominate vein. Transesophageal echocardiography may aid in diagnosis but carries a risk of misinterpretation, making it preferable for use during surgical intervention rather than as a primary diagnostic tool.

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