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UNREPAIRED EBSTEIN ANOMALY IN ADULT WOMAN: CHALLENGES AND STRATEGIES IN LONG-TERM CONSERVATIVE MANAGEMENT – A CASE REPORT

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ABSTRACT

Ebstein anomaly is a rare congenital malformation of the tricuspid valve and right ventricle with a highly variable clinical course, and some patients survive into adulthood without surgical repair. We report a 42-year-old woman from a rural area in whom a congenital heart condition was suspected in childhood but not confirmed until her thirties. She presented with progressive exertional dyspnoea and easy fatigability. Electrocardiography showed sinus ryhtym with delta wave and short PR interval, and she later had an episode of supraventricular tachycardia that converted spontaneously. Chest radiography revealed marked cardiomegaly (cardiothoracic ratio 73%). Serial transthoracic echocardiography from 2023 to 2026 demonstrated apical displacement of the septal tricuspid leaflet by 35 mm (≈23 mm/m²) with atrialisation of the right ventricle, severe tricuspid regurgitation, right heart dilatation, and a large secundum atrial septal defect with left-to-right shunting, with preserved

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biventricular systolic function. Despite treatment with diuretics, a mineralocorticoid receptor antagonist, a beta-blocker, an angiotensin receptor blocker, and sildenafil, she was admitted twice for heart failure. Surgical repair, electrophysiological study, and right heart catheterisation were indicated but unavailable at our hospital, and the patient declined referral to a tertiary centre. This case shows that unrepaired Ebstein anomaly may be tolerated for decades despite severe anatomy. When definitive treatment is not accessible, long-term conservative management should focus on heart failure control, safe management of arrhythmias in the presence of pre-excitation, and structured clinical and echocardiographic surveillance with clear triggers for renewed referral.

Keywords: Ebstein's anomaly, adult congenital heart disease, heart failure

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INTRODUCTION

Ebstein's anomaly (EA) is a rare congenital malformation caused by tricuspid valve (TV) delamination failure. The dysplastic and tethered TV (the septal and/or posterior leaflet) is offset toward the apex of the right ventricle (RV), with subsequent atrialization of the RV inlet.[1] The resulting tricuspid regurgitation (TR) promotes dilation of right atrium (RA), annular dilatation and RV volume overload. An associated interatrial communication may allow right-to-left shunting, with cyanosis and paradoxical embolism.[2]

EA accounts for less than 1% of all congenital heart diseases (CHD), with an estimated prevalence of 1 in 200,000 live births. Associated congenital anomalies are very common, including defects in the atrial septum, patent foramen ovale in 80–90% of patients, pulmonary atresia, ventricular septal defects, and conduction anomalies, including Wolff–Parkinson–White (WPW) syndrome, placing a patient at risk of arrhythmias.[3] EA also has a wide range of clinical presentations. Neonates commonly present with cyanosis, cardiomegaly, and congestive heart failure (CHF). Older children and adults often experience additional symptoms including palpitations, arrhythmias, exercise intolerance, fatigue, and syncope.[4]

The severity of this condition varies widely, from infants who do not survive early childhood, to asymptomatic adults diagnosed incidentally in their sixties or seventies and survive without surgery.[5] Severity is determined by comparing the area of the RA and atrialized RV to the area of the remaining functional RV and left heart. Less than 50% of patients with EA will survive to five years of age, with the most common cause of death arising from tachyarrhythmias.[4]

Adults with unrepaired disease with prolonged stability may reflect an adequately sized functional RV, limited regurgitation, restricted or absent interatrial shunting, and preserved left ventricular (LV) function. It is recommended that patients with EA be managed medically with regular evaluation by a cardiologist for the assessment of arrhythmia complications or RV dysfunction. However, upon onset of symptoms, increasing exercise intolerance, cyanosis, or worsening RV dysfunction, surgical intervention should be considered, including repair or

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replacement of the tricuspid valve and right atrioplasty.[6] We report an adult woman with unrepaired EA, a large secundum atrial septal defect (ASD), and arrhythmia who has been managed medically in a rural hospital, to illustrate these challenges and the strategies available to treat them.

CASE REPORT

Clinical course

A 42-year-old woman presented to the cardiology outpatient clinic first in 2023 for evaluation of progressive dyspnea. She described episodes of dyspnoea that came and went over the past few years, accompanied by easy fatigability during routine daily activities. These complaints had gradually begun to limit her daily activities e.g walking distance and going upstairs which made her to seek medical attention.

Her early life history offered few clues to underlying heart disease. She was born at home in a rural village without the attendance of a physician, and no newborn screening for congenital heart disease was performed. According to her family, there were no perinatal complications, and no cyanosis was noted at birth or during childhood. During childhood, she visited a doctor once, at which time she was told that she might have a congenital heart condition. No further evaluation was undertaken, and because she had no remarkable symptoms, she did not seek additional medical care. She grew up without limiting cardiac symptoms.

The diagnosis was not confirmed until her thirties. In addition to her exertional symptoms, the patient reported a history of recurrent palpitations.

She was a nonsmoker, nondiabetic and nonhypertensive. At her first outpatient visit in 2023, the patient appeared well and was fully alert. Her vital signs were within normal limits, and peripheral oxygen saturation on room air was normal. No central or peripheral cyanosis was observed. Pulmonary auscultation revealed no abnormalities. Grade III holosystolic murmur was heard at the left lower sternal border. Over the following two years, the patient was observed every month in outpatient clinic with stable condition. In June 2025, she presented with palpitations and dyspnoea. Electrocardiography demonstrated paroxysmal supraventricular tachycardia (SVT). She

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remained haemodynamically stable, and the tachycardia converted spontaneously to sinus rhythm without pharmacological intervention. She had never undergone an electrophysiological study or catheter ablation. She was discharged on oral medical therapy. In August 2025 and August 2026, she was readmitted with congestive heart failure. No arrhythmia was documented during this admission, and she was treated with furosemide and other heart failure therapy.

Electrocardiography

Two electrocardiographic recordings were available. The first, obtained during an episode of palpitations, showed SVT with right axis deviation, incomplete right bundle branch block, left posterior fascicular block, and anteroinferior ischemia (Figure 1). A second recording, obtained in normal sinus rhythm with left axis deviation, poor R-wave progression across the precordial leads, and delta waves with PR interval shortening (Figure 2).

Chest radiography

The last chest radiography revealed marked cardiomegaly, with a cardiothoracic ratio of 73% and flattening of the cardiac waist. Pulmonary vascular markings were prominent, with radiographic features of pulmonary congestion (Figure 3).

Serial echocardiography

The patient underwent annual transthoracic echocardiography from 2023 to 2026. Several features were present on every examination. These included apical displacement of the septal tricuspid leaflet, dilatation of the RA and RV, diastolic flattening of the interventricular septum producing a D-shaped LV consistent with RV volume overload, and concentric LV remodelling (Figure 4).

The 2023 study demonstrated severe TR resulting from apical displacement of the septal leaflet. It also showed a secundum ASD measuring 1.1 cm with left-to-right shunting. LV systolic function was normal (ejection fraction 74%), with grade I diastolic dysfunction.

In 2024, TR was graded as massive and the LV appeared small. The ejection fraction (EF) was 63%. The echocardiographic probability of pulmonary hypertension was high.

The 2025 study described the septal leaflet as arising from the mid-interventricular septum, with moderate TR. A secundum ASD measuring 2.2 cm with left-to-right shunting was noted. The

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probability of pulmonary hypertension remained high, and LV diastolic function was reported as normal.

The most recent study, in September 2026, provided the most detailed anatomical description. The septal tricuspid leaflet was inserted on the RV aspect of the interventricular septum, 35 mm from the tricuspid annulus (≈23 mm/m²), with atrialisation of the proximal RV. TR was severe, with a vena contracta of 1.1 cm. A large secundum ASD measuring 2.0 cm with left-to-right shunting was present. A systolic pulmonary artery pressure of 26.8 mmHg was estimated from the TR jet with continuous-wave Doppler imaging. Tricuspid annular plane systolic excursion was measured as 41 mm using M-mode echocardiography. LVEF was 76%, with grade I diastolic dysfunction. In contrast to the two preceding studies, the echocardiographic probability of pulmonary hypertension was low.

Management and clinical course

The patient was managed medically and has remained under regular outpatient follow-up since 2023. Her current regimen consists of furosemide 40 mg once daily in the morning and spironolactone 25 mg once daily in the morning for symptoms of fluid overload. She also receives bisoprolol 2.5 mg twice daily, candesartan 4 mg at night, and sildenafil 10 mg twice daily. Referral to a tertiary centre was recommended for further evaluation. This would have included right heart catheterisation to confirm pulmonary haemodynamics, an electrophysiological study, and consideration of definitive surgical or transcatheter intervention. The patient declined referral and chose to continue medical therapy alone.

DISCUSSION

The clinical findings of EA spans an exceptionally wide range. At one extreme, severe tricuspid malformation may cause profound heart failure in the neonatal period and early death. At the other, patients may reach adulthood, and occasionally late adulthood, with few or no symptoms.[7] But, our patient does not fit the conventional explanation for late presentation. On the contrary, the septal leaflet was markedly displaced, the proximal RV was atrialised, RA and RV are

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dilated, and TR was severe. Despite these findings, she had no limiting cardiac symptoms until her thirties. This dissociation between anatomical severity and clinical tolerance is well recognized. In this patient, several observed features may account for it, such as preserved RV systolic function, normal LVEF, and the absence of cyanosis. The last indicates that clinically significant right-to-left shunting had not developed, an important consideration because interatrial communications in EA can permit cyanosis and paradoxical embolism in adults.[8] A notable detail in this case is that this patient did not develop jugular vein distention (JVD) or peripheral edema despite the moderate-severe RV dysfunction and TR. These symptoms may be absent due to increased compliance and dilation of the RA, allowing for greater volume accumulation before jugular backflow occurs.[6]

The electrocardiographic findings were likewise typical. Pre-excitation namely WPW syndrome, documented here as delta waves in sinus rhythm and short PR interval, is a recognised association of the lesion like this patient.[9] Supraventricular tachyarrhythmias and right bundle branch block are common presenting features in EA which also happened in this patient.[10,11] Cardiomegaly is another classic finding in EA, as was seen in this patient. The chest radiograph showed a cardiothoracic ratio of 73% with flattening of the cardiac waist, reflecting predominantly RA and RV enlargement.[4] The subsequent decompensation is consistent with the established haemodynamics of the lesion. Severe TR across an atrialised RV imposes a chronic volume load on the right heart[12], and the ASD with predominantly left-to-right flow adds a further source of right ventricular volume loading.[13] The diastolic septal flattening present on every examination is consistent with RV volume overload.

Serial transthoracic echocardiography, the only imaging modality available, illustrates further pitfalls. Reported ASD diameter ranged from 1.1 to 2.2 cm, TR was graded from moderate to massive, and the echocardiographic probability of pulmonary hypertension shifted from high in 2024 and 2025 to low in 2026. These fluctuations are more plausibly explained by differences in acoustic windows, operators, and loading conditions than by true anatomical change. Cardiac magnetic resonance imaging was not available. It provides reproducible quantification of RV volumes and function, and its measures of ventricular function have predicted adverse events in unrepaired adults.[14]

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Morbidity and mortality both greatly depend on the severity of EA measured via echocardiogram. In a study of 72 adult patients >25 years of age, Attie et al. found that the overall median survival was 16.07 years from time 0 to 25 years old. The age of diagnosis is also an important risk factor for survival, meaning that the earlier symptoms appear, the greater the probability of significant progression and mortality.[4] The diagnostic pathway, however, illustrates delay rooted in access rather than diagnostic difficulty. The patient was born at home without newborn cardiac screening. A congenital heart abnormality was suspected at a single childhood consultation but not evaluated further, and confirmation came only in her thirties. Delayed diagnosis and referral are recurring themes in CHD care in middle-income countries, where deficits in infrastructure and specialised constrain access along the entire care pathway.[15]

In this case, our patient survived into adulthood and remained asymptomatic until the age of 30, not requiring surgical intervention until age 42. The survival rate for EA patients who do not undergo surgical repair is 40% at 20 years, with 8.6% dying from sudden cardiac death within 50 years of birth. Without surgery to repair EA, <5% survive over age 50.[4] These trends make this patient's pathophysiology of disease unique and important.

By current standards, the patient met several indications for referral. The 2025 ACC/AHA/HRS/ISACHD/SCAI guideline recommends surgical intervention for adults with EA and at least moderate TR in the presence of heart failure, worsening exercise capacity, or progressive right ventricular dysfunction. It also recommends electrophysiological study in patients with symptomatic arrhythmia or ventricular pre-excitation to identify substrates for ablation.[16] Recurrent heart failure admissions with severe regurgitation, together with documented SVT and a pre-excited electrocardiogram, satisfy both criteria. Referral to a tertiary centre was recommended but declined, and none of the recommended investigations or interventions could be offered in our hospital. Management was therefore necessarily medical and symptom-directed, involves adjusting heart rate and preload and restricting exercise. This may include a low-sodium diet, oral diuretics, digoxin and a low-dose angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB).[17] Our patient was treated with diuretics, ARB, mineralocorticoid receptor antagonists (MRAs), beta blocker, and pulmonary artery vasodilator for pulmonary hypertension.

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Conservative management therefore had some specific challenges.

1. **Recurrent heart failure despite medical therapy.** In our patient, diuretics relieved congestion, but her low-normal blood pressure limited further up-titration of other heart failure drugs. She was admitted twice for decompensated heart failure within about one year, which shows that medical therapy alone did not stop the disease from progressing. *Strategy:* we use diuretics to control symptoms of congestion and other guideline-directed mediated therapy for heart failure, adjust the dose according to body weight, symptoms, and kidney function.
2. **Pre-excitation without electrophysiological study.** Because our patient never underwent an electrophysiological study, we do not know how fast her accessory pathway can conduct if atrial fibrillation develops. *Strategy:* based on guideline for SVT in adult congenital heart disease, i.v. verapamil, diltiazem, and beta-blockers should be considered, if vagal manoeuvres and adenosine fail (recommendation class: IIa). Perform electrical cardioversion if the patient becomes haemodynamically unstable (recommendation class: I).[18]
3. **Follow-up limited to transthoracic echocardiography.** Over four yearly echocardiograms, the reported size of the ASD and severity of TR ranged (Table 1). Cardiac magnetic resonance imaging, which can measure RV size and function and helps predict outcomes in unrepaired adults was not available. *Strategy:* perform echocardiography with the same protocol at every visit (Table 2), and add simple, low-cost measures: functional class, body weight, oxygen saturation, ECG, and cardiothoracic ratio. Clinical decisions should then be based on trends over time rather than on a single measurement.

CONCLUSION

Adults with unrepaired EA may stay well for many years, even when the anatomy is severe. However, once arrhythmias and repeated heart failure develop, guidelines recommend evaluation and treatment at a specialized centre. When this is not possible, as in rural hospitals, long-term

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medical care should focus on three things: treating heart failure symptoms, managing tachyarrhythmias safely in the presence of pre-excitation, and regular clinical and echocardiographic follow-up with clear signs that should prompt a new referral discussion.

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Table 1: Serial transthoracic echocardiography findings, 2023-2026

Parameter	**2023**	**2024**	**2025**	**2026**
Septal tricuspid leaflet	Apical displacement	Apical displacement	Apical displacement	Apical displacement , Inserted on RV side of IVS, 35 mm from annulus (≈23 mm/m²); atrialised RV
Tricuspid regurgitation	Severe	Massive	Moderate	Severe (VC 1.1 cm)
RA / RV	Dilated	Dilated	Dilated	Dilated
LV geometry	Concentric remodelling; D-shaped LV	Concentric remodelling; D-shaped LV; small LV	Concentric remodelling; D-shaped LV	Concentric remodelling; D-shaped LV
LVEF (by Teich)	74%	63%	76%	76%
LV diastolic function	Grade I dysfunction	Grade I dysfunction	Normal	Grade I dysfunction
LV wall motion	Global normokinesis	D-shaped septal motion	Global normokinesis	Global normokinesis
Secundum	Moderate, 1.1	Moderate, 1.1 cm,	Large, 2.2 cm,	Large, 2.0 cm,

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ASD	cm, L→R	L→R	L→R	L→R
Probability of PH	High	High	High	Low

ASD, atrial septal defect; IVS, interventricular septum; L→R, left-to-right; LV, left ventricle; LVEF, left ventricular ejection fraction; PH, pulmonary hypertension; RA, right atrium; RV, right ventricle; VC, vena contracta.

Table 2. Surveillance framework for unrepaired Ebstein anomaly in a resource-limited setting

Domain	Parameter	Frequency	Trigger for renewed referral
Clinical	NYHA class, body weight, SpO_2	Every visit	Worsening NYHA class; SpO_2 <90% or new cyanosis
Rhythm	12-lead ECG	Every visit and with palpitations	New AF/flutter; rapid pre-excited tachycardia; syncope
Imaging	TTE with fixed protocol: indexed displacement, TR vena contracta, RA/RV size, TAPSE, ASD size and shunt direction, LVEF	6–12 months, or after admission	Progressive RV dilatation or dysfunction; fall in LVEF; shunt reversal
Radiography	Cardiothoracic ratio	Annually	Progressive cardiomegaly
Events	Heart failure admissions	Ongoing	Any heart failure admission

AF, atrial fibrillation; ASD, atrial septal defect; ECG, electrocardiogram; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; RA, right atrium; RV, right ventricle; SpO_2, peripheral oxygen saturation; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation; TTE, transthoracic echocardiography.

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Figure 1: Electrocardiography when inpatient at tachycardia episode showing supraventricular tachycardia

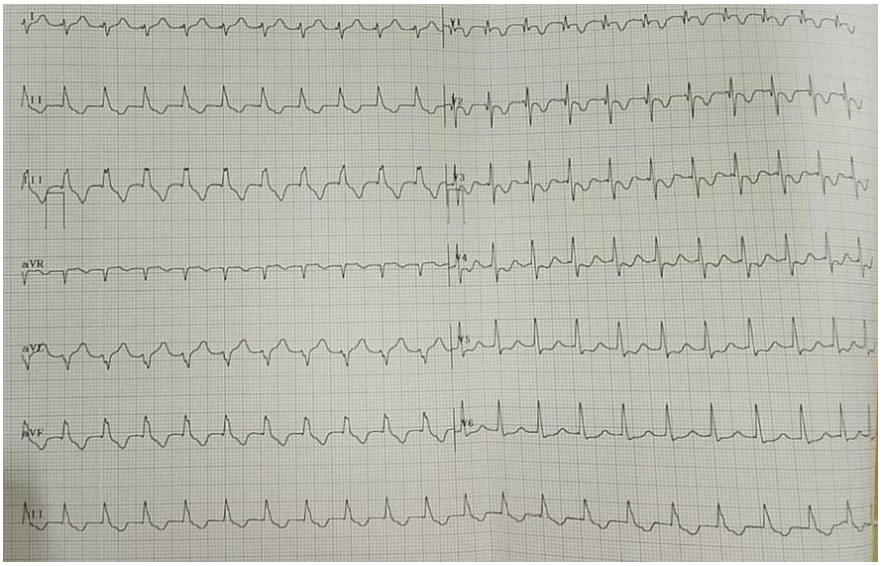
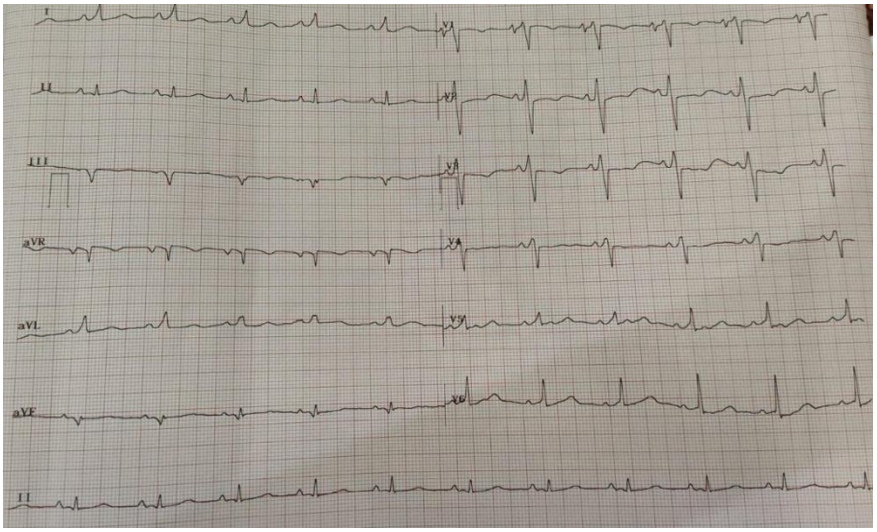
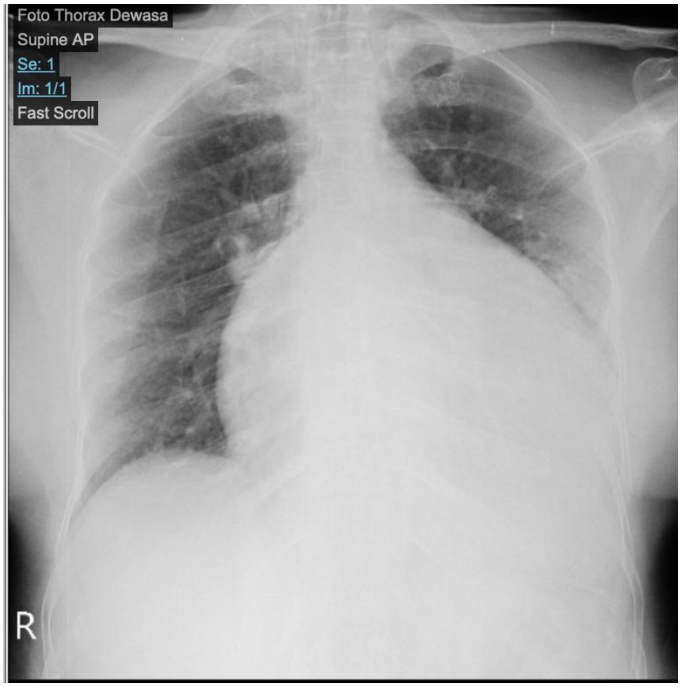


Figure 2: Electrocardiography in resting episode at outpatient clinic showing normal sinus ryhtm with delta wave and PR interval shortening suggesting for WPW pattern



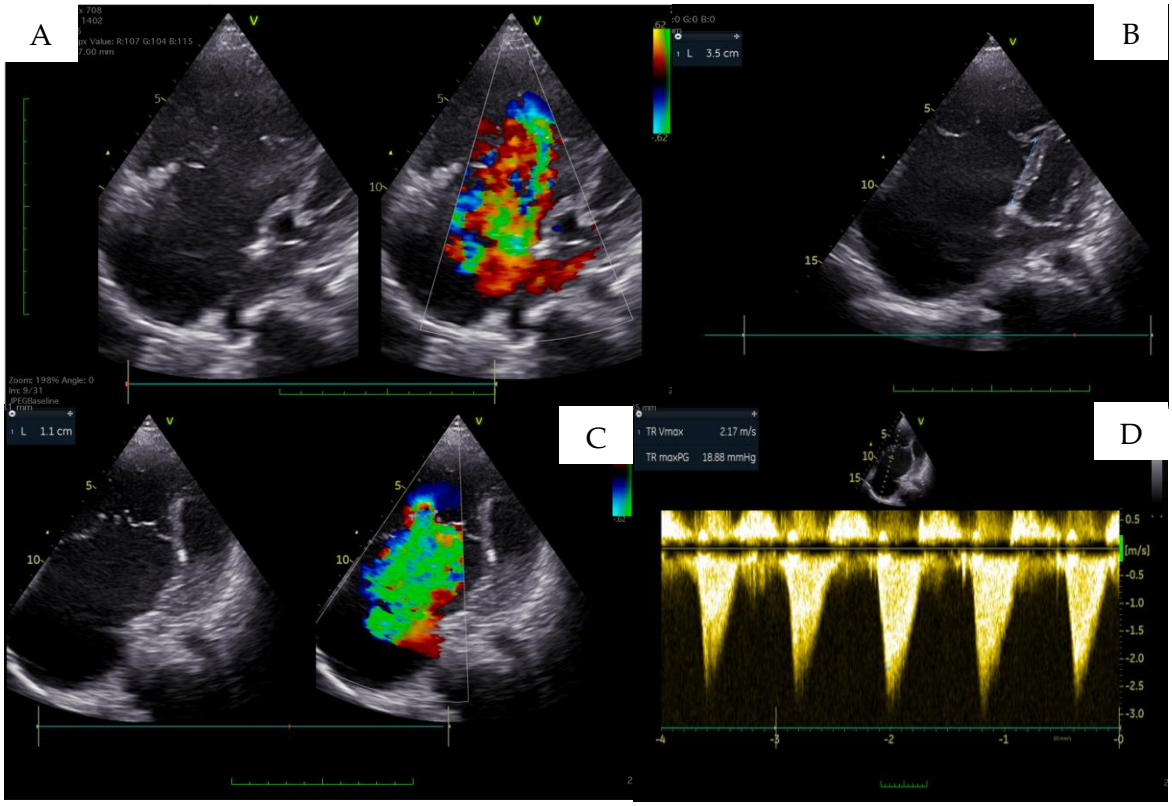
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Figure 3 : Chest xray when inpatient showing marked cardiomegaly with pulmonary vascular markings were prominent



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Figure 4 : Transthoracic colour Doppler echocardiography showing **(A)** severe tricuspid regurgitation due to apical displacement of the septal tricuspid leaflet and a large secundum atrial septal defect (2.0 cm) with left-to-right shunting, (B) apical displacement of the septal tricuspid leaflet, inserted on the interventricular septum 35 mm from the tricuspid annulus, (C) failure tricuspid leaflet coaptation with severe tricuspid regurgitation (vena contracta 1.1 cm), (D) continuous-wave Doppler of the tricuspid regurgitant jet showing a peak velocity of 2.17 m/s.



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