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Case Report

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SEVERE ANEMIA PRESENTING AS HEART FAILURE WITH PRESERVED EJECTION FRACTION

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ABSTRACT

In chronic anemia, the reduced oxygen-carrying capacity of hemoglobin is typically offset by an elevated cardiac output. Patients with chronic severe anemia but no other cardiovascular disease rarely develop symptoms of congestive heart failure, a condition known as non-cardiac circulatory congestion. Severe anemia can induce a hyperdynamic circulation and chronic volume overload, potentially resulting in significant alterations to echocardiographic parameters, especially global left ventricular strain. This case describes a 16-year-old male patient with severe anemia who developed congestive heart failure, particularly pulmonary congestion. A comprehensive clinical examination, along with pathological, radiological, ECG, and two-dimensional echocardiographic investigations, was performed, and their distinctive features are presented herein. This study focuses on severe anemia, high output heart failure, and the assessment of left ventricular dysfunction using 2D strain imaging and global longitudinal strain via echocardiography.

KEYWORDS: *Severe anemia, High output heart failure, LV strain imaging, Global Longitudinal Strain, Left ventricular dysfunction, 2D Strain imaging, Echocardiography.*

INTRODUCTION

It is well-established that chronic anemia is associated with both cardiac dilatation and hypertrophy. Anemia is the most prevalent condition that can elevate cardiac output at rest. In men, chronic anemia typically increases cardiac output when hemoglobin levels fall below 7 g/dL, yet significant cardiac enlargement occurs only with extreme reductions in hemoglobin (below 4 g/dL). Although the clinical signs of a hyperkinetic state in anemia are often striking, they can be rapidly reversed in nearly all cases by partially correcting the anemia. Circulatory congestion resembling that observed in congestive heart failure is a rare yet serious complication of severe anemia[2] [Fig. 1, 2].

Anemia is a widespread health issue globally, with the highest prevalence observed among the Indian population. Anemia is defined by a decrease in both the number of red blood cells and the oxygen-carrying capacity of hemoglobin.[3,4] In chronic anemia, the heart experiences structural alterations and functional decline as a response to decreased hemoglobin levels. Furthermore, anemia serves as a risk factor for cardiovascular disease outcomes in the general population.[5] Furthermore, there is limited prior data on the impact of low hemoglobin levels on left ventricular (LV) diastolic function in patients without overt heart disease.[6-9]

Strain imaging is the preferred method for accurately quantifying ventricular function. This echocardiographic technique assesses regional left ventricular strains in the longitudinal, radial, and circumferential directions. Chronic anemia is typically associated with an increase in cardiac mass.[10] Furthermore, left ventricular global longitudinal strain (GLS) has been demonstrated to be a

superior indicator of left ventricular dysfunction.[11] However, there is a scarcity of literature exploring the relationship between low hemoglobin levels and left ventricular (LV) function, as well as the utility of two-dimensional strain patterns, specifically global longitudinal strain (GLS), for the early detection of LV dysfunction in patients with severe anemia.

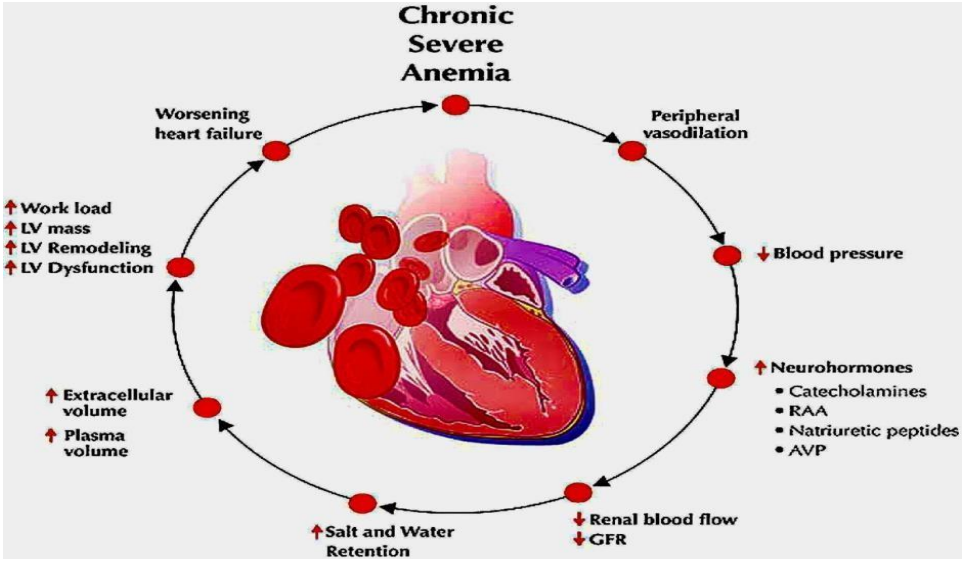


Figure 1: Possible sequence of Events Involved in the the Pathogenesis of Heart Failure in Chronic Severe Anemia AVP = arginine vasopressin; GFR = glomerular filtration rate; LV = left ventricle; RAA = renin – angiotensis-aldosterone.

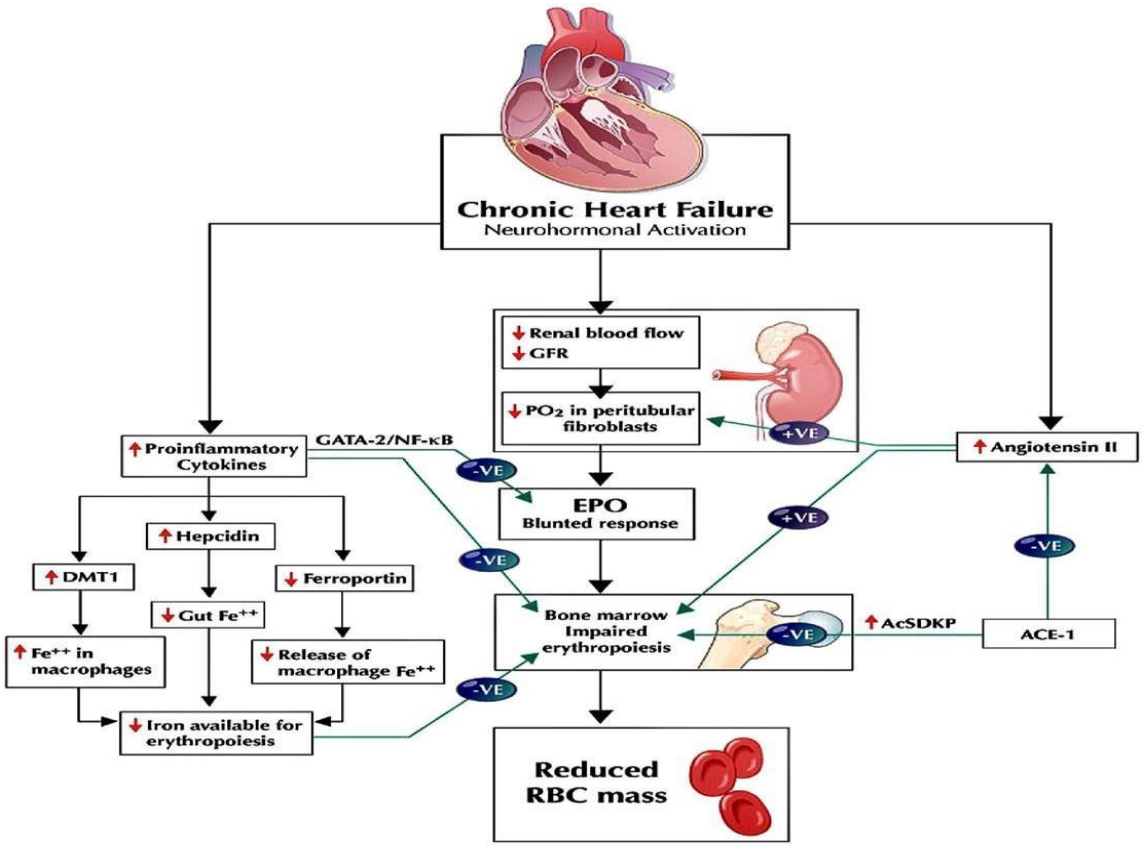


Figure 2: Possible Mechanisms Involved in the Genesis of Anemia in Heart Failure.

Shown is diagram of the possible mechanisms of anemia in patients with heart failrw. ACE-I = angiotensin-converting enzyme inhibitor; AcSDKP = N-acetly-seryl-aspartyle-lysyl-proline; ARB = angiotensin receptor blocker; DMT1 = divalent metal transporter; EPO = epoetin; GFR = glomerular filtration rate; HF = heart failue; NF-kB = nuclear facor-kappa B; RBS = red blood cell mass.

CASE REPORT

A 16 year old adolescent presented to our cardiac OPD with a history of 2-3 months of gradually progressive breathlessness on exertion, weakness, fatigue and general anasarca. From last one week he was experiencing recurrent spells of orthopnea while sleeping.

On clinical examination he had a striking pale appearance (Figure 3) consistent with severe anemia. There was conspicuous swelling all over the body, particularly over hands (Figure 4) and abdomen. Moreover, elevated Jugular Venous Pressure (JVP) (Figure 6), was demonstrated.

Fig. 3: Facies of our index patient. Striking pallor is obvious.

Fig. 4.

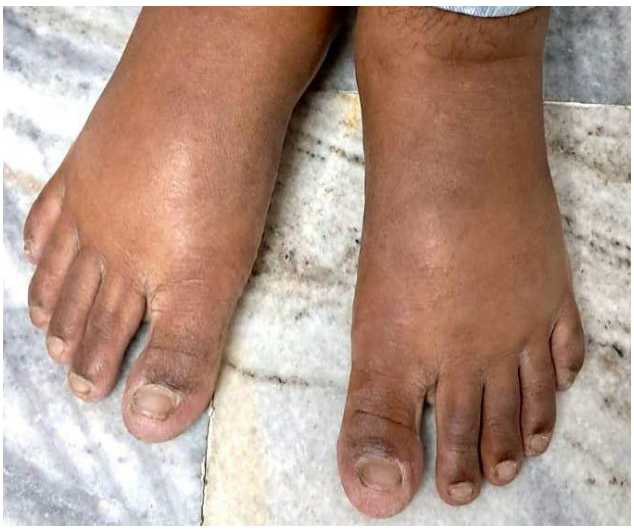


Fig. 5

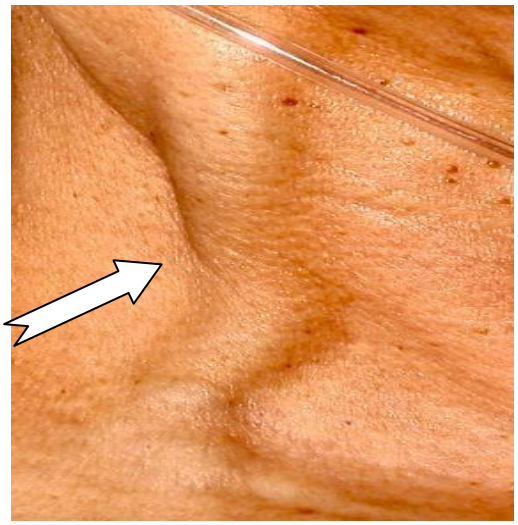


Fig. 6

Fig. 4) Swelling of both hand and all fingers; Fig. 5) Swelling of feet; Fig. 6) A distinctive elevated JVP was demonstrated.

His height was 145 cm, weight was 54 kg, respiration rate was 25, pulse rate was 105/min, BP was 90/60 mmHg and spo2 was 99%. He was afebrile. On cardiovascular examination, the heart sounds were normal with presence of soft systolic murmur in the pulmonary area and the apex. No gallops or clicks were audible. Besides generalized edema and elevated JVP rest of the systemic examination was normal.

X-Ray chest (PA) of the patient displayed normal cardiac size with increased pulmonary vascular markings, suggestive of pulmonary plethora (Figure 7).

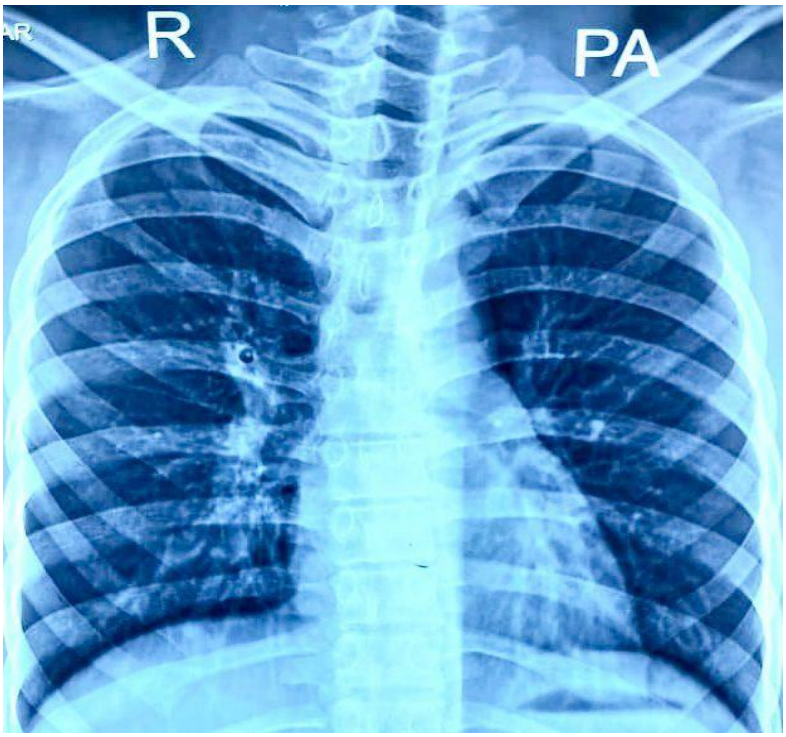


Fig. 7: There characteristic pulmonary plethora accompanied with normal cardiac size.

Resting ECG exhibited sinus tachycardia with a ventricular rate of 120 beats/min and a normal QRS axis along with non-specific T wave inversions in L_2, L_3, AVF, V_{1-4}. There was no evidence of sinus node or AV nodal disease or arrhythmia. (Figure 8).

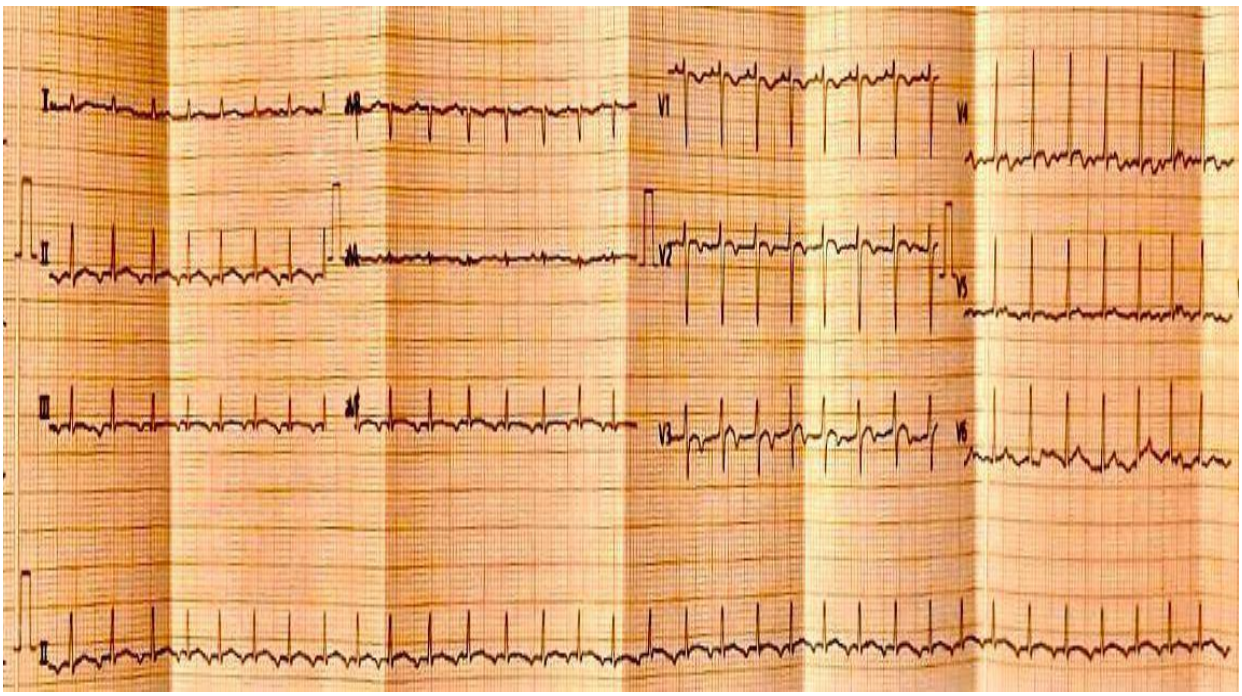


Fig. 8: Resting ECG exhibited sinus tachycardia with a ventricular rate of 120/min with a normal QRS axis.

PATHOLOGICAL INVESTIGATION: 26/04/2026

HAEMOTOLOGY TEST REPORT

COMPLETE BLOOD COUNTS (CBC)			**Reference Range**	**Units**
Haemoglobin (Hb)	**6.0**	**L**	13.0-16.0	g/Dl
Total Leukocyte Count (TLC)	**3500**	**L**	4000-11000	/cumm
Differential Leukocyte count (DLC)				
Neutrophil	64		40.0-70.0	%
Lymphocyte	30		20.0-40.0	%
Eosinophils	4		1.0-6.0	%
Monocytes	2		00.0-10.0	%
Basophils	0		00.0-02.0	%
Platelet Count (Machine)	1.53		1.5-4.0	Lac/cumm
RBC Count	**2.60**	**L**	4.50-6.0	m/cumm
NRBC Count	**0.32**	**H**		%
MCV	**74**	**L**	80.0-100.0	Fl
MCH	**23**	**L**	27.0-32.0	Pg
MCHC	**31**	**L**	32.0-37.0	gm/Dl
PACKED Cell Volume	**19**	**L**	35.0-45.0	%
ESR (Westergen)	**19**	**H**	0.0-9.0	mm/1st hr
Immature Platelet Fraction	**9.40**	**H**	0-7.5	%
RBCs – These are normocytic, normochromic type. No normoblasts seen. WBCs – TLC shows normal counts. DLC shows normal distribution of components. No immature cells of WBC series are seen. IMPRESSION: MICROCYTIC HYPOCHROMIC ANAEMIA				

SEROLOGY TEST REPORT

CRP (C-REACTIVE PROTEIN)-HIGH SENSITIVE			**Reference Range**	**Units**
CRP (C-Reactive Protein)-Quantitative	**6.78**	**H**	0/00-6.00	mg/L
Unsaturated Iron binding Capacity	693.00			
Total Iron Binding Capacity	**726**	**H**	225.0-535.0	ug/Dl
% Transferrin Saturation	**4.55**		13.00-45.00	%
Ferritin	**542.4**	**H**	25.0-350.0	ng/ml
TYPHIDOT IGG/IGM				
Typhidot IgG Antibody	**NEGATIVE**			
Typhidot IgM Antibody	**NEGATIVE**			

BIOCHEMISTRY TEST REPORT

CREATININE			**Reference Range**	**Units**
S.Creatinine	0.66		0.40-1.50	mg/Dl
URIC ACID				
Serum Uric Acid	**3.4**	**L**	3.4-7.0	mg/Dl
S. TOTAL PROTEIN				
Protein, Total	**4.65**	**L**	6.0-8.3	gm/Dl
Albumin	**2.31**	**L**	3.5-5.1	gm/Dl
Globulin	2.34		2.00-3.50	gm/Dl
A:G (Albumin:Globulin) Ratio	**0.99**		1.20-2.0	gm/Dl
S. ALANINE AMINO-TRANSFERASE				
SGPT (ALT)	45		5.0-50.0	IU/L
ALKALINE PHOSPHATASE (ALP)				
ALKALINE PHOSPHATASE (ALP)	**137.0**		60.0-446.0	IU/L

IRON STUDIES-I (IRON,TIBC, Ferritin)			**Reference Range**	**Units**
Iron, Serum	**33.00**	**L**	59.0-158.0	ug/Dl
Unsaturated Iron binding	693.00			

Capacity				
Total Iron Binding Capacity	**726**	**H**	225.0-535.0	ug/Dl
% Transferrin Saturation	**4.55**		13.00-45.00	%
Ferritin	**542.4**	**H**	25.0-350.0	ng/ml

IMMUNOASSAY TEST REPORT

			Reference Range	Units
NT-ProBNP (N-TERMINAL PRO B TYPE NATRIURETIC PEPTIDE)	3650		<75 Years: 0-125 >75 Years: 0-450	**pg/ml**
Thyroid Stimulating Hormone (TSH)	**5.620**		**0.3-6.0**	**uIU/ml**

CLINICAL PATHOLOGY TEST REPORT
URINE ROUTINE EXAMINATION

PHYSICAL EXAMINATION		**Reference Range**	**Units**
Colour	**STRAW**		
Appearance	**SLIGHTLY TURBID**		
pH	6.00	ACIDIC : <6 NEUTRAL : 6-7 ALKALINE : >7	
Specific gravity	1.015	1.003-1.030	
CHEMICAL EXAMINATION			
Urine Protein	NIL		mg/dL
Urine Sugar	NIL		mg/dL
Bilirubin	NIL		
MICROSCROPIC EXAMINATION			
Pus cells/Leukocytes	NIL		
Epithelial Cells	OCCASIONAL		
Blood	NIL		
Casts	NIL		/HPF
Crystals	NIL		/HPF

SUMMARY OF PATHOLOGICAL INVESTIGATIONS

There was severe microcytic hypochromic anemia with hypoproteinemia. A considerable reduction of Hb %, serum iron and serum albumin levels were illustrated. NT-proBNP, a diagnostic biomarker for heart failure (HF) and cardiac dysfunction was severely elevated confirming the diagnosis of congestive heart failure (CHF) in our patient.

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.

Furthermore left ventricular a speckle tracking echocardiography was implemented to demonstrate any early features of subclinical LV dysfunction. The important findings of our comprehensive color doppler transthoracic echocardiography (TTE) are outlined.

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	8.3 mm
LVID d	46.2 mm
LVPW d	7.3 mm
IVS s	12.2 mm
LVID s	26.9 mm
LVPW s	12.9 mm
EF	73 %
% LVFS	42 %
LVEDV	98.4 ml
LVESV	26.9 ml
SV	71.6 ml
CO	6.94 l/min
%IVS	48 %
% PW	75%
LV Mass	115 g

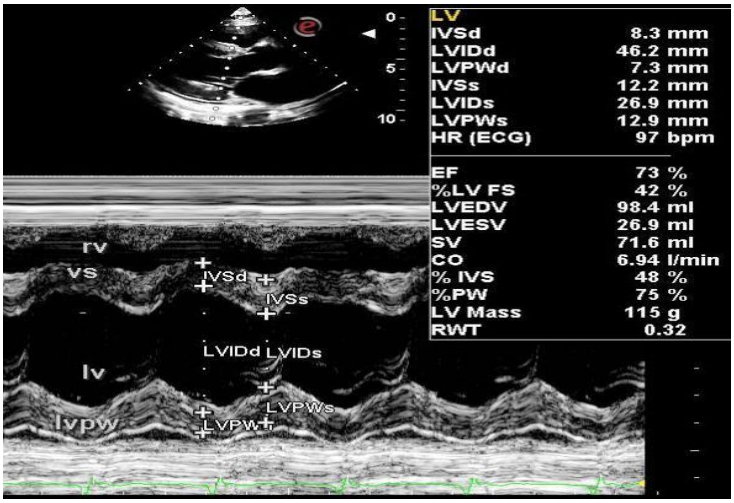


Fig. 9: M-mode echocardiography of LV.

Summary of M-mode echocardiography

The LV end-diastolic and LV end-systolic volumes, LV systolic function & dimensions were normal. The LVEF was 73% and LV mass was 115 gm.

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	27.69 cm^2
LVAd A2C	28.59 cm^2
LVEDV (MOD A4C)	85.8 ml
LVEDV (MOD A2C)	82.4 ml
EF (MOD A4C)	63 %
SV (MOD A2C)	55.7 ml
LVAs A4C	14.98 cm^2
LVAs A2C	14.81 cm^2
LVESV (MOD A4C)	31.6 ml
LVESV (MOD A2C)	26.7 ml
EF (MOD A2C)	68 %
SV (MOD A2C)	54.2 ml

Other distinctive features were

a) E/A ratio of mitral and tricuspid inflow velocities showed a ration of 2:5:1 and 2:1 respectively, suggestive of restrictive physiology **(Figures 15-16).**

b) Dilated right and left atrium.

c) Dilated inferior vena cava (IVC) with reduced respiratory excusrsion **(Figure 10).**

- IVC (Expiration) D = 23.5 mm
- IVC (Inspiration) D = 17.7 mm

d) There was normal LV dimensions, volumes and systolic function LVEF was normal, 63 %. *(Figures 17-19).*

e) Mild circumferential pericardial effusion wasdetected with an estimated fluid of approximately 100ml **(Figures 17-18).**

f) There was no evidence of pericardial constriction.

g) No PAH was demonstrated.

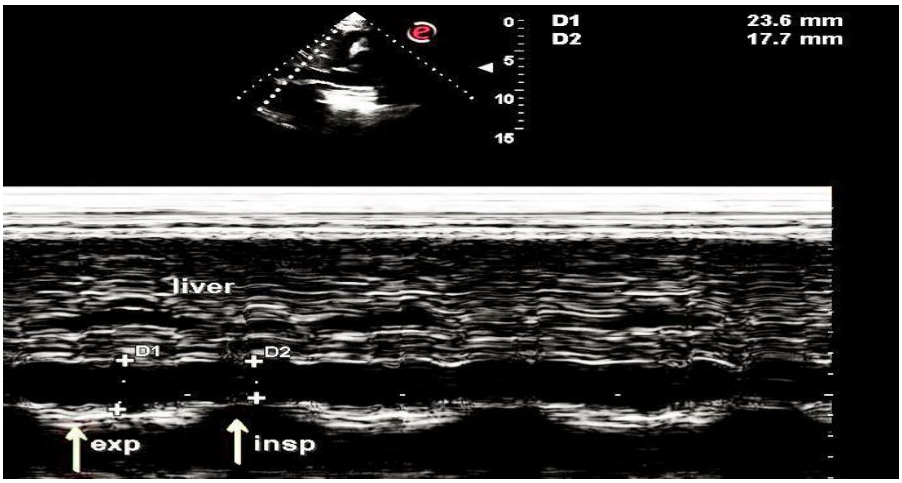


Fig. 10.

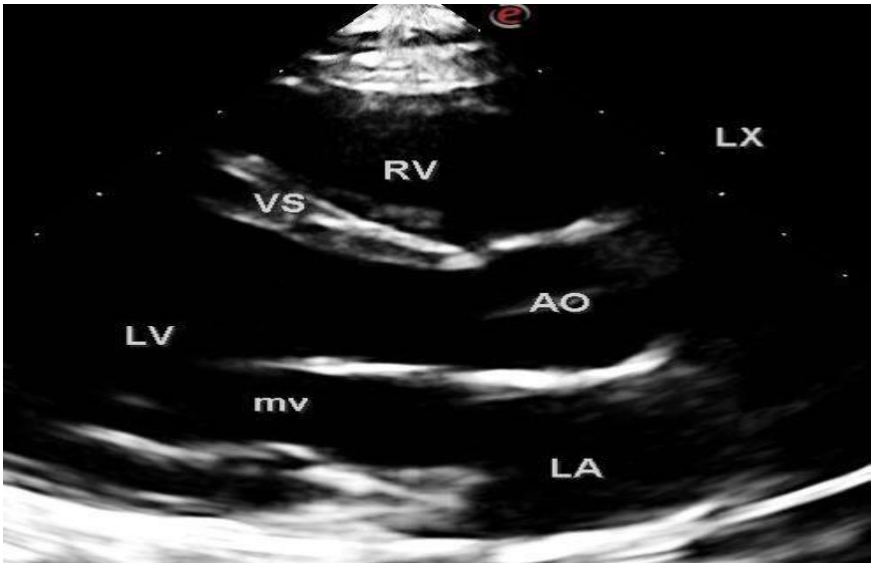


Fig.11.

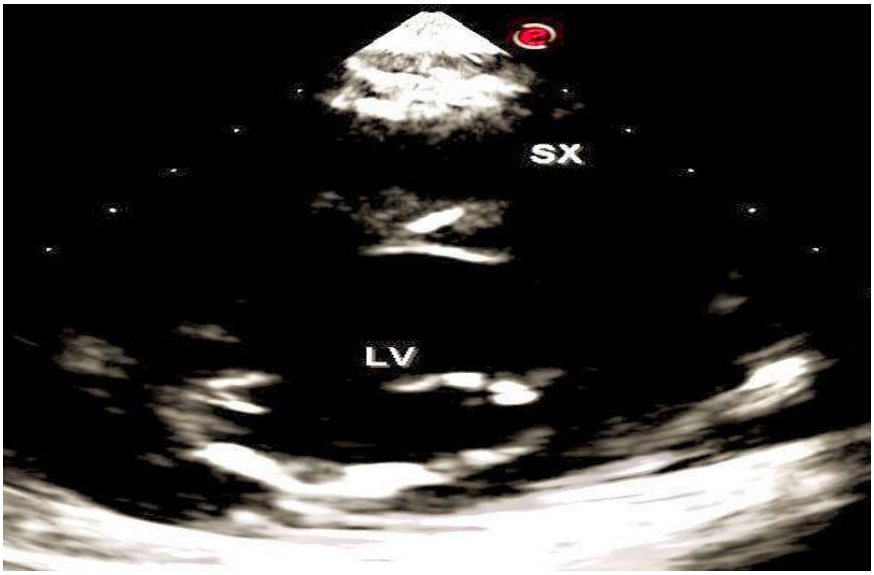


Fig. 12.

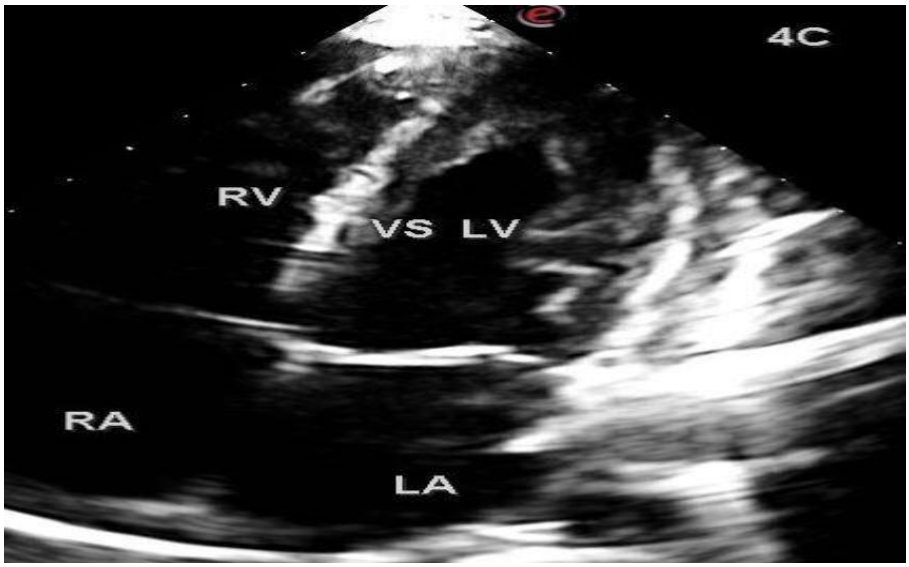
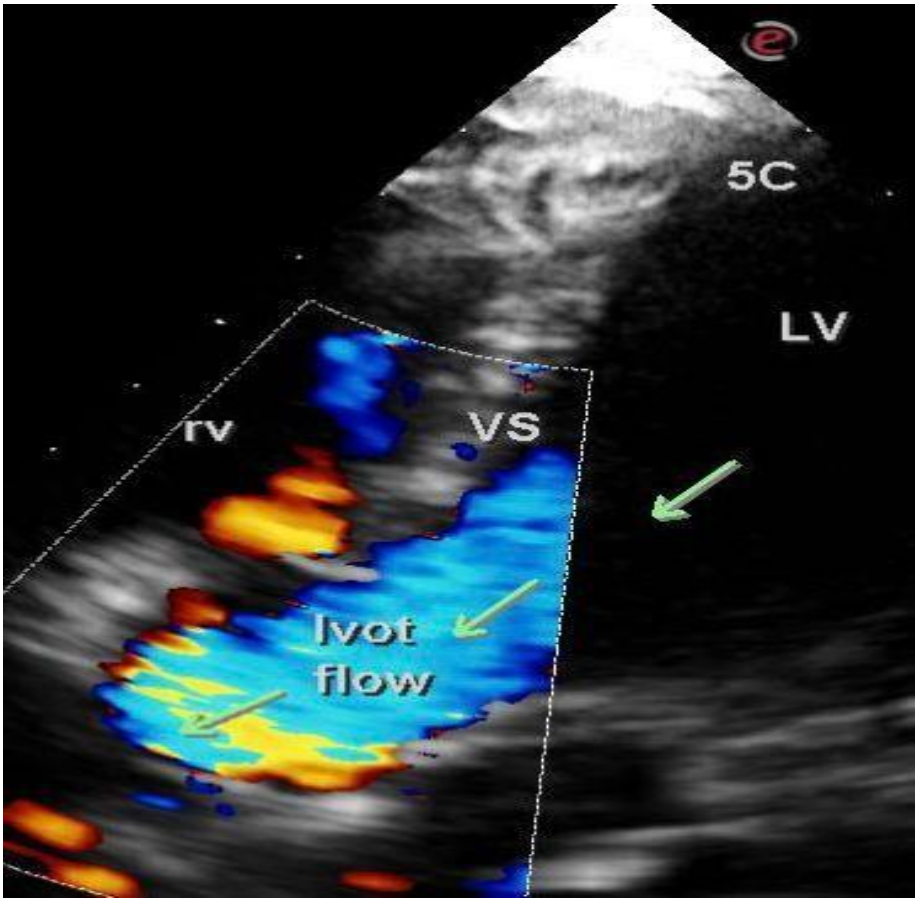


Fig. 13


5C
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VS
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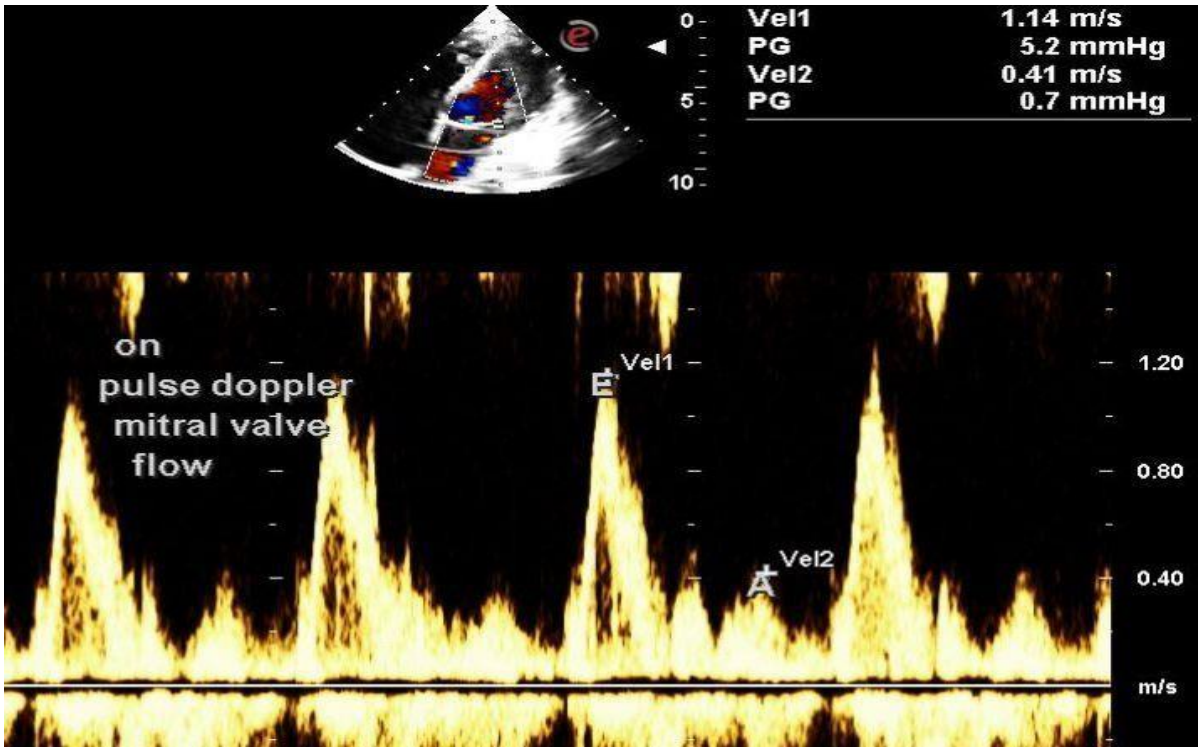
Fig. 14.


Vel1 1.14 m/s
PG 5.2 mmHg
Vel2 0.41 m/s
PG 0.7 mmHg
0
5
10
on
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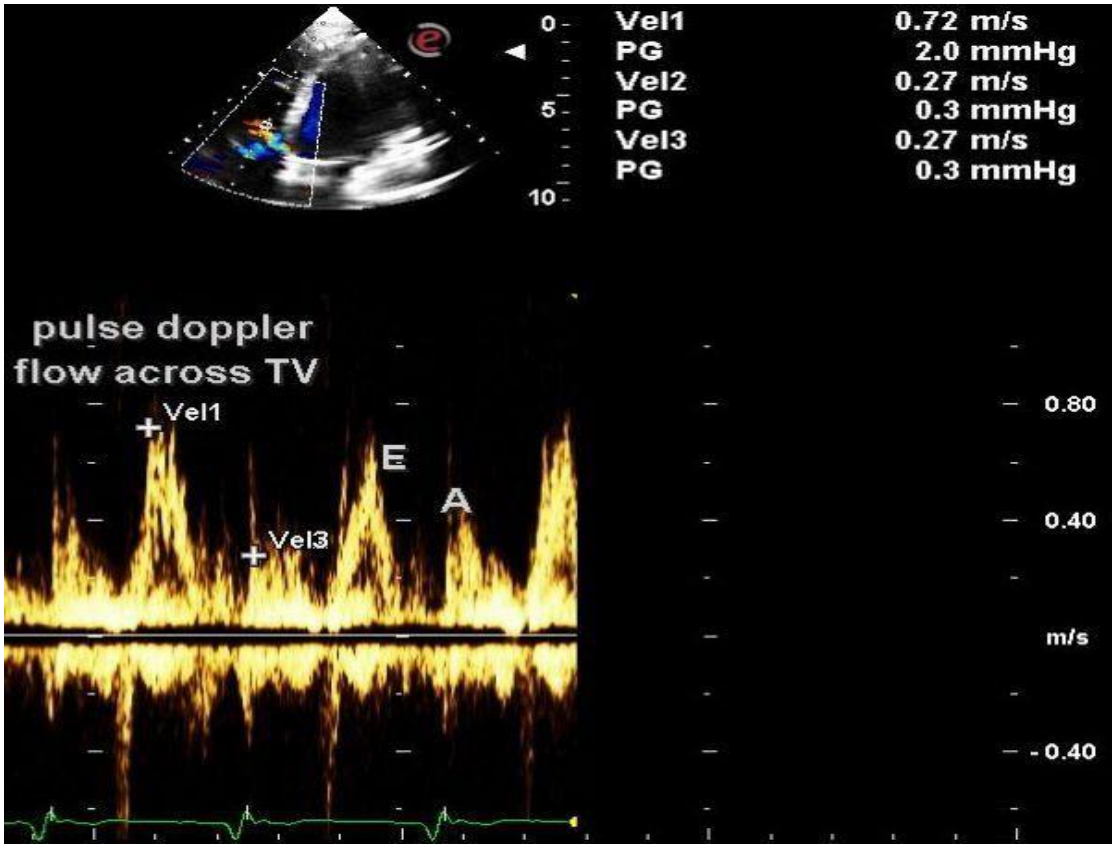
Vel2
1.20
0.80
0.40
m/s

Fig. 15.


Vel1 0.72 m/s
PG 2.0 mmHg
Vel2 0.27 m/s
PG 0.3 mmHg
Vel3 0.27 m/s
PG 0.3 mmHg
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Vel1
E
A
Vel3
0.80
0.40
m/s
-0.40

Fig. 16.

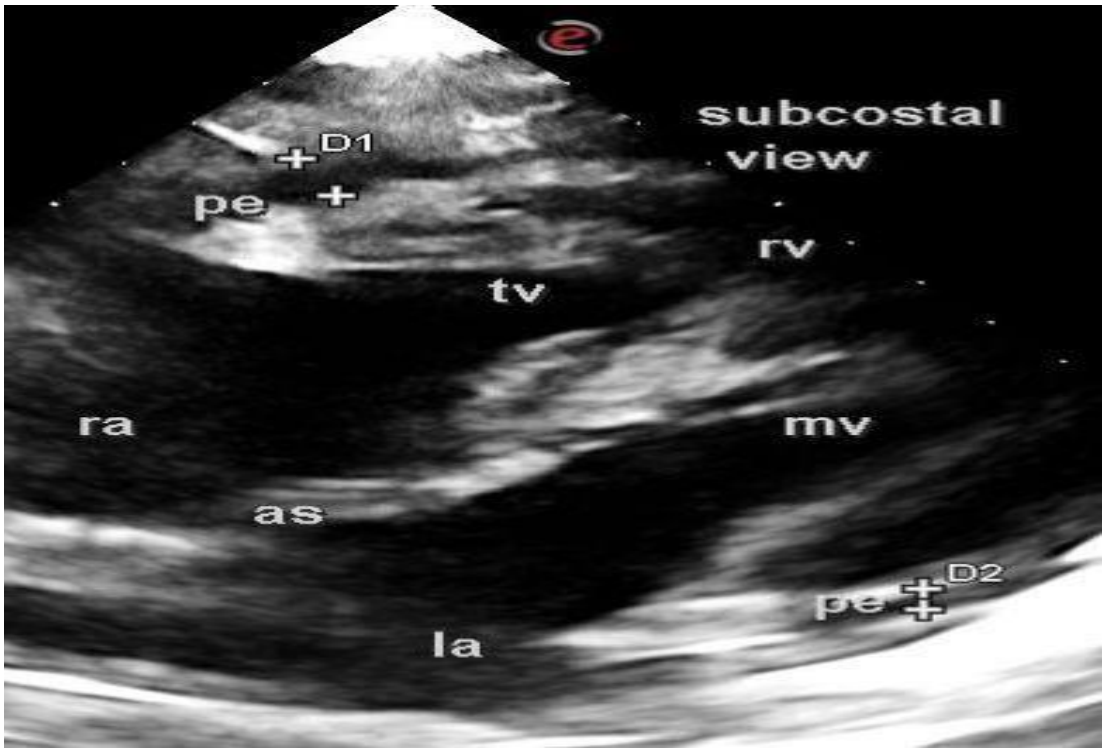
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Fig. 17.

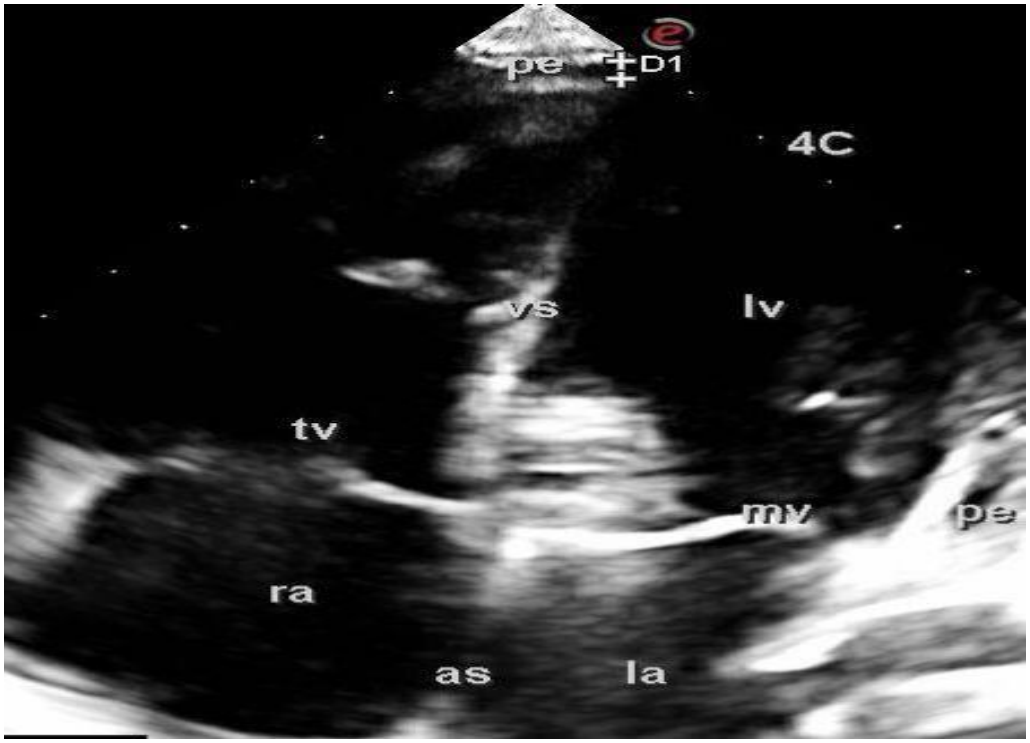
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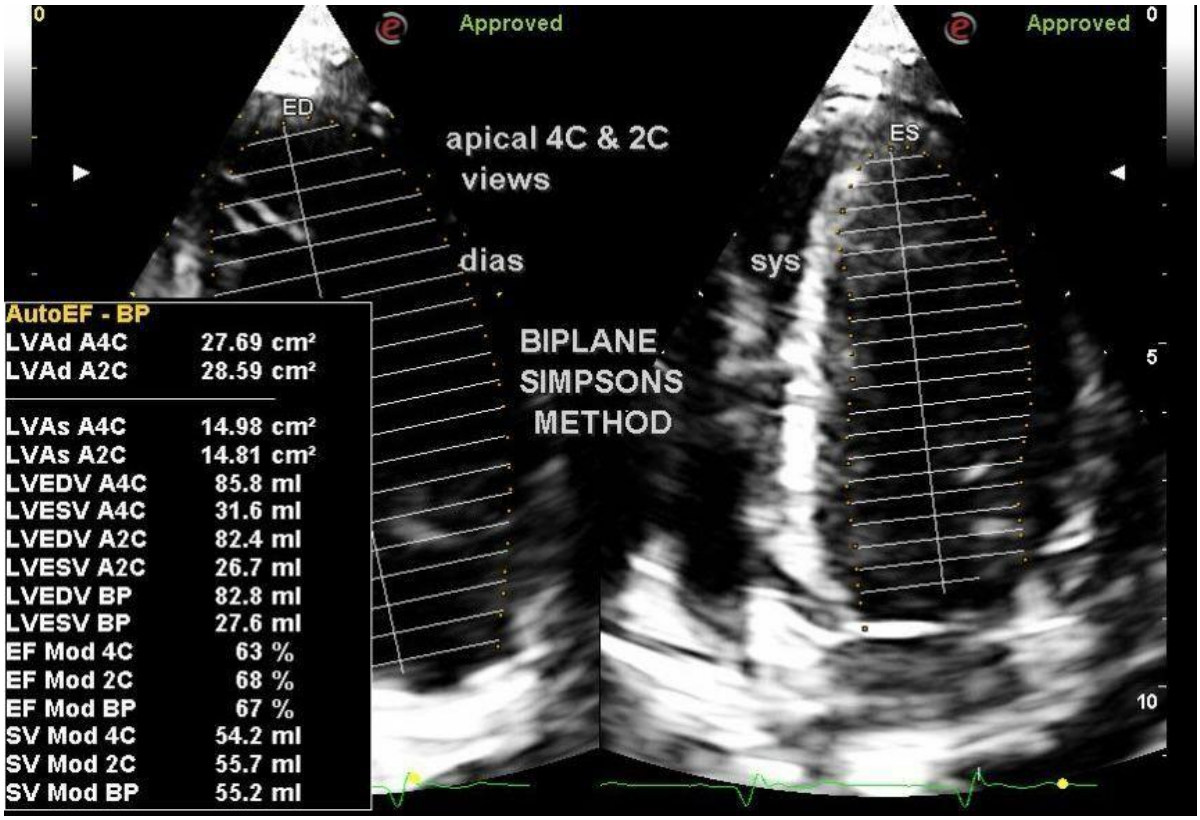
Fig. 18.


Approved
Approved
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dias
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BIPLANE
SIMPSONS
METHOD
AutoEF - BP
LVAd A4C 27.69 cm²
LVAd A2C 28.59 cm²
LVAs A4C 14.98 cm²
LVAs A2C 14.81 cm²
LVEDV A4C 85.8 ml
LVESV A4C 31.6 ml
LVEDV A2C 82.4 ml
LVESV A2C 26.7 ml
LVEDV BP 82.8 ml
LVESV BP 27.6 ml
EF Mod 4C 63 %
EF Mod 2C 68 %
EF Mod BP 67 %
SV Mod 4C 54.2 ml
SV Mod 2C 55.7 ml
SV Mod BP 55.2 ml

Fig. 19.

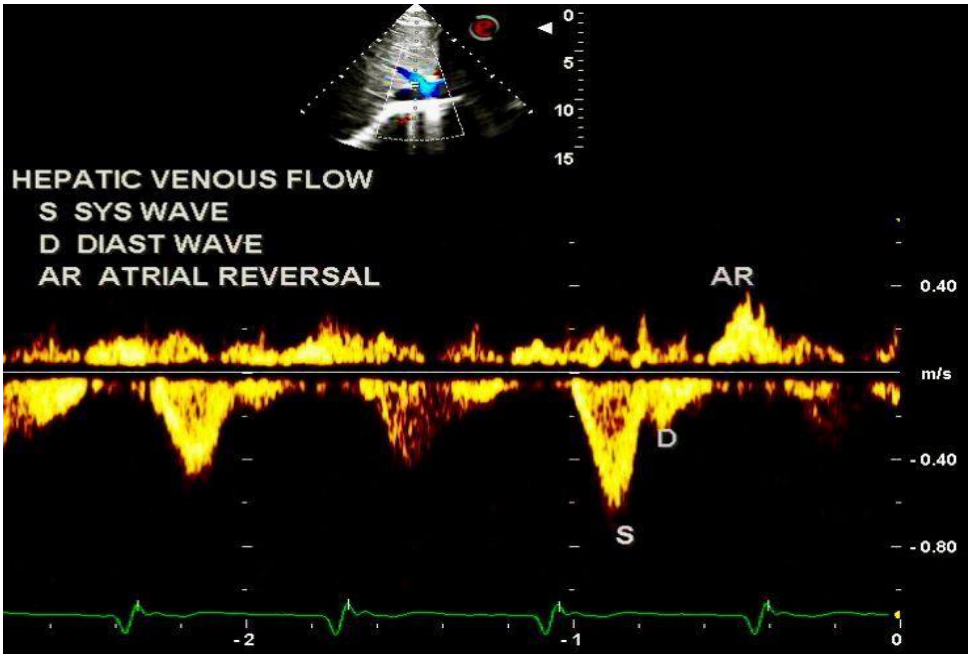

Fig. 20.

Figures 10-20: Fig. 10: On subcostal view a dilated IVC is seen with reduced respiratory excursion during inspiration indicating presence of congestive heart failure; **Fig.11**: LV anatomy and dimensions were normal of LX view; **Fig. 12**: In the SX view the LV was normal; **Fig.13**: 4C view was normal; **Fig.14**: In the 5C view there was not LV outflow obstruction; **Fig. 15:** On pulse wave doppler analysis the mitral inflow velocity showed a E/A ratio of 2.5 : 1 , indicating a restrictive physiology; **Fig. 16:** Tricuspid inflow velocity similarly demonstrated a restrictive physiology with an E/A ratio of 2.1:1; **Fig. 17:** Mild circumferential pericardial effusion was observed in the subcostal view , pericardial effusion; **Fig. 18**: Similarly 4C view exhibited a mild pericardial effusion; **Fig. 19**: EF was 67% on Biplane-Simpson's method; **Fig. 20**: In the subcostal view hepatic venous flow displayed predominant systolic wave and contracted diastolic wave .

Summary of 2D Color Echocardiography

The LV cavity dimensions and systolic functions were normal. Nonetheless, mitral & tricuspid inflow velocities were consistent with restrictive physiology with dilatation of LA and RA and dilated IVC with reduced respiratory excursion.

Hepatic venous flow displayed prominent systolic wave and contracted diastolic wave, suggestive of hyperdynamic circulation. Moreover a mild circumferential pericardial effusion was detected.

LV Strain Echocardiography by speckle tracking.

Speckle tracking echocardiography (STE) was executed to demonstrate any subclinical evidence of LV dysfunction. This maybe reflected by reduction of global longitudanal strain (GLS) and moreover decerement in values of few or multiple segments of 17-segment module of LV segmental strain imaging (Fig 21-23, Table 3).

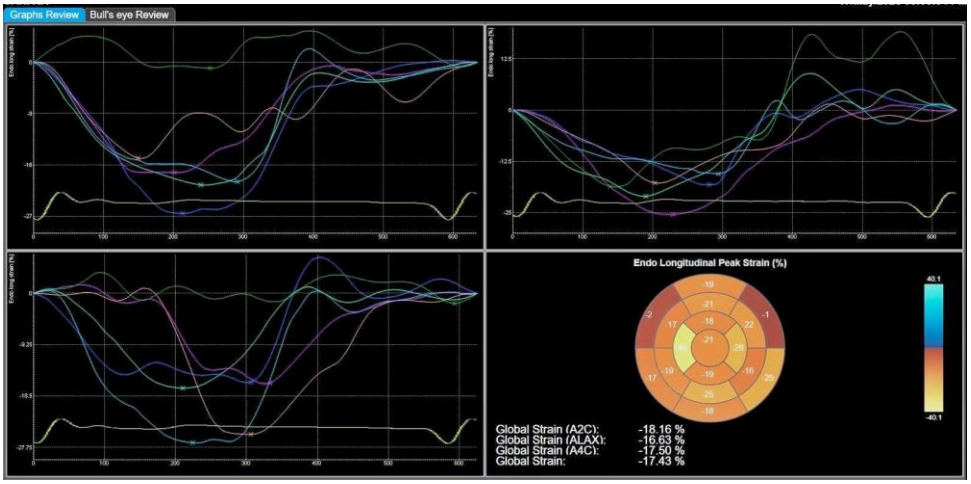

Fig. 21.

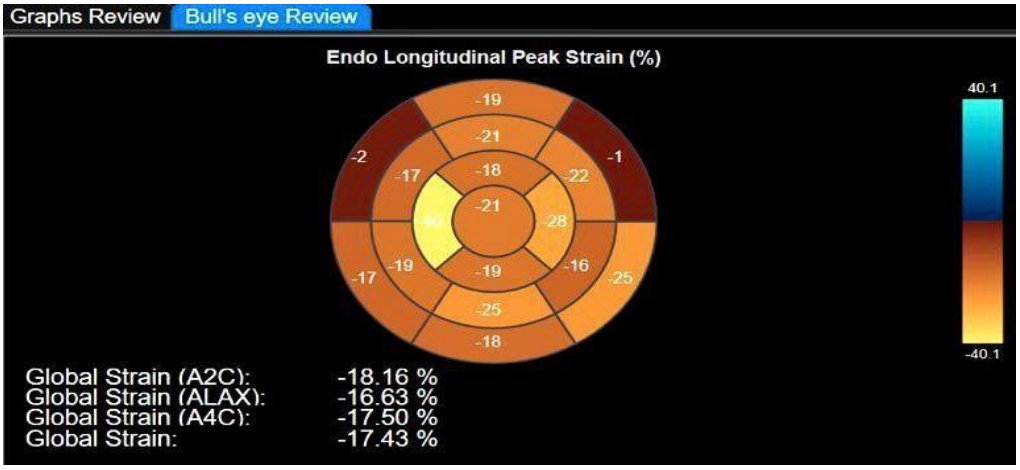

Fig. 22.

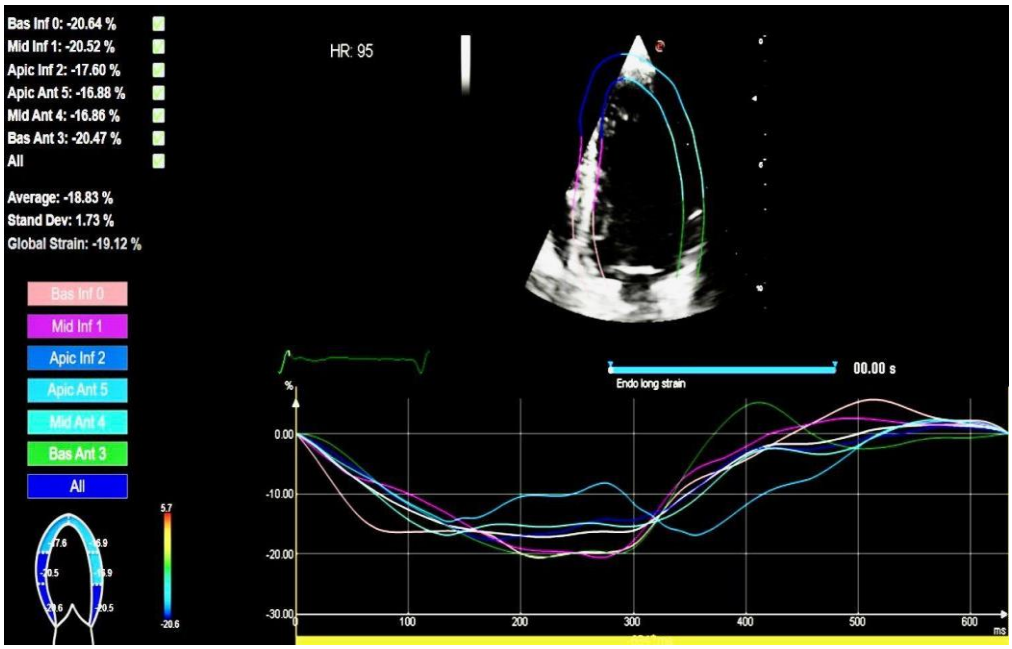

Fig. 23 a).

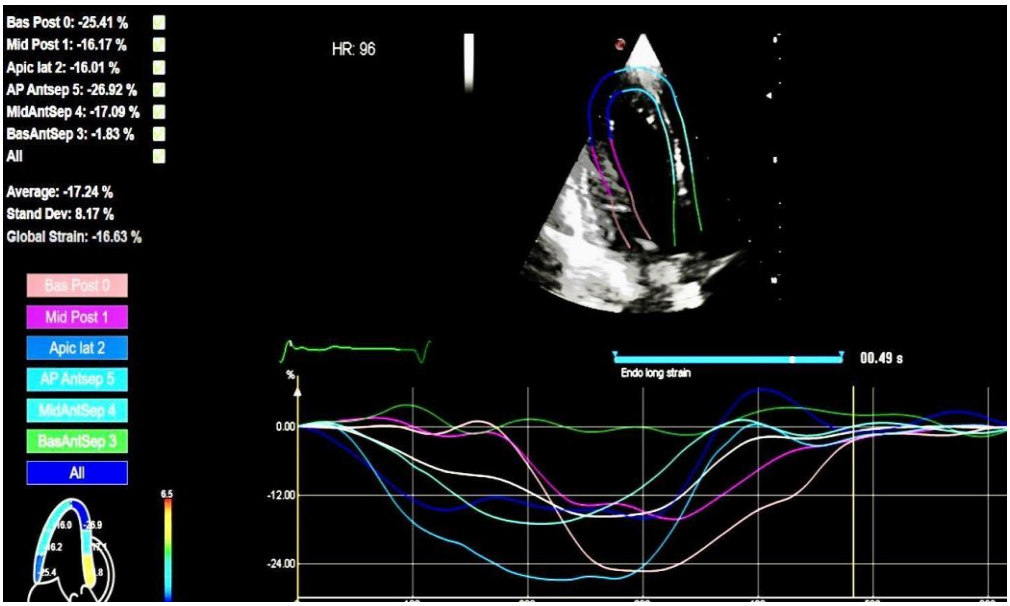

Fig. 23 b).

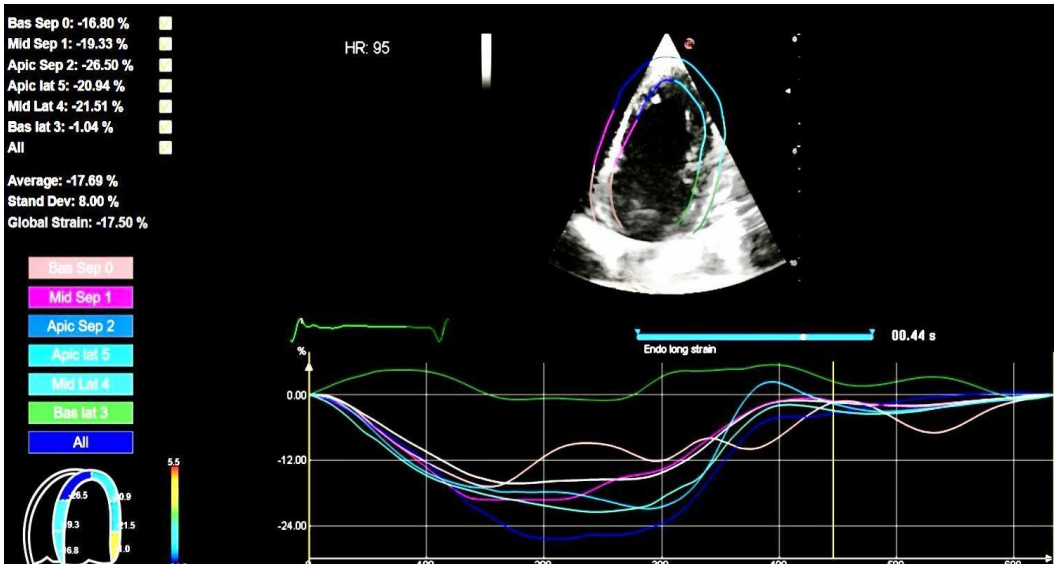

Fig. 23 c).

Figure 21-23: LV strain imaging by speckle tracking echocardiography.

Fig. 21) a) Graphs and Bulls Eye plot of apical 2C, apical LAX & apical 4C views. b)LV Global strain was - 17.43 % ; **Fig. 22).** Bulls Eye Plot of apical LV strain echocardiography. Global Strain was - 17.43 %; **Fig. 23a)** Apical 2C Longitudinal Strain was - 19.12 %; **Fig. 23b)** Apical LAX longitudinal strain was - 16.63 %; **Fig. 23c)** Apical 4C longitudinal strain was - 17.50 %

Table 3: Segmental Longitudinal strain values obtained from Bull's eye plot.

Bas Ant	-18.58 %	★
BasAntSep	-1.83 %	★
Bas Sep	- 16.80 %	★
Bas Inf	-17.71 %	★
Bas Post	-25.41 %	
Bas lat	-1.04 %	★
Mid Ant	-20.98 %	
MidAntSep	-17.09 %	★
Mid Sep	-19.33 %	
Mid Inf	-25.39 %	
Mid Post	-16.17 %	★
Mid Lat	-21.51 %	
Apic Ant	-18.31 %	★
Apic Sep	-40.07 %	
Apic Inf	-19.17 %	
Apic lat	-27.71 %	
Apex	-20.67 %	
Global Strain (A2C)	-18.16 %	★
Global Strain (A4C)	-17.50 %	★
Global Strain (ALAX)	-16.63 %	★
Global Strain	-17.43 %	★

★**Subclinical dysfunction**

Summary of LV strain imaging

Our index patient demonstrated reduction in values of GLS in A2C, A4C, ALAX views (-18.16 %, - 17.50% and -16.63% respectively), accompanied by adversely affected GLS with a value of -17.43 %.

Additionally in 17-segment straining, atleast 8 segments exhibited subclinical dysfunction as indicated by their strain values being lower than 19%.

Future course of action

We immediately initiated anticongestive treatment for the emergent management of CHF in the form of intravenous diuretics, cardioselective beta blockers and ivabradine for heart rate control. Furthermore, we urgently requested for two units of fresh whole blood. After administration of anti-congestive therapy and two units of fresh blood, patient dramatically improved in the next 48 hours. Moreover oral supplementation of Iron, proteins and Vitamin B12 were strongly recommended. We discharged the patient in a totally asymptomatic stage, without any clinical evidence of CHF. His Hb% and serum Albumin levels at discharge were 9.98 gm% and 3.52 g/dL, respectively.

DISCUSSION

Approximately 50% of heart failure patients exhibit preserved ejection fraction. The prevalence of anemia in heart failure with preserved ejection fraction (HFpEF) ranges from 19% to 68%. While anemia is associated with poor outcomes in heart failure with reduced ejection fraction (HFrEF), its prognostic significance in heart failure with preserved ejection fraction (HFpEF) remains debated. At present, no specific therapy has been shown to reduce mortality in HFpEF, and there are very few evidence-based treatment strategies that improve morbidity. Comorbidities, including anemia, significantly contribute to the morbidity and mortality associated with HFpEF. However, the impact of anemia on outcomes in patients with HFpEF remains unclear. A post-hoc analysis of the Candesartan Cilexetil in Heart Failure Assessment of Reduction in Mortality and Morbidity (CHARM)-Preserved trial, along with a patient-level meta-analysis by the MAGGIC group, demonstrated that anemia is associated with increased mortality.[12,13] However, other studies have shown inconsistent findings.[14,15] The WHO defines anemia as hemoglobin levels below 13 g/dL in adult males and below 12 g/dL in adult females. HFpEF was defined by clinical features indicative of heart failure with an ejection fraction exceeding 50%. Although the exact pathogenesis of anemia in heart failure patients remains unclear, previous studies suggest that hemodilution, reduced erythropoietin production (particularly in those with chronic kidney disease), and impaired iron supply are likely contributing factors. Multiple mechanisms may account for the increased mortality and hospitalization rates observed in patients with anemia. In patients with HFpEF, myocardial energy efficiency is already compromised and is further impaired by anemia, which reduces oxygen delivery to the myocytes. Furthermore, research indicates that anemia is linked to a 25% increase in cardiac mass, alterations in left ventricular geometry, and reduced global longitudinal strain.[16] The aforementioned factors can act synergistically to induce remodeling and decompensation, resulting in increased mortality and hospitalization rates among patients with anemia. Anemia also impairs quality of life and exercise tolerance in patients with HFpEF.[15]

CONCLUSION

Cardiac dysfunction, particularly affecting left ventricular diastolic function, is not uncommon in patients with severe anemia. Patients with severe anemia are found to have an increased left ventricular mass. Patients with significant anemia may develop left ventricular hypertrophy, leading to decreased compliance and diastolic dysfunction that can manifest as features of congestive heart failure. Conventional echocardiography is a straightforward method for detecting left ventricular dysfunction in patients with severe anemia, indicating that correcting the anemia is necessary to reverse and prevent this condition.

It is universally accepted that in patients with HFpEF, anemia is associated with a higher risk of mortality and overall hospitalization compared to those without anemia. Treating anemia may potentially improve clinical outcomes in these patients.

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