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EVALUATION OF GLOBAL LONGITUDINAL STRAIN IN HEART FAILURE WITH PRESERVED EJECTION FRACTION CAUSED BY SEVERE ANEMIA

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ABSTRACT

Iron deficiency anemia (IDA), a prevalent blood disorder, arises from insufficient iron reserves that hinder adequate red blood cell production, leading to reduced hemoglobin levels and compromised oxygen delivery. This condition is more common in women and is associated with a stronger impact on left ventricular mass. Severe cases often result in elevated left ventricular mass with eccentric hypertrophy being more frequent. Echocardiography reveals hemodynamic alterations linked to IDA, which can enhance oxygen delivery. Severe IDA may also cause an elevated heart rate and increased stroke volume, thereby raising cardiac output. Assessment of left ventricular (LV) global longitudinal strain (GLS) via speckle tracking echocardiography (STE) offers a non-invasive, cost-effective method to quantitatively evaluate early declines in LV systolic function among IDA patients. STE, being non-invasive, affordable, and widely used in clinical settings, could serve as a practical tool for assessing GLS in severe anemia cases, as impaired GLS may signal silent myocardial dysfunction. Speckle tracking technology can detect cardiac involvement at very early stages. A delicate boundary exists between overt myocardial dysfunction and asymptomatic LV dysfunction in severely anemic individuals. Thus, advanced imaging techniques are essential to identify subtle cardiac changes in the initial phases. We present a case of a teenage male with severe anemia complicated by heart failure with preserved ejection fraction (HFpEF), where detailed 2D echocardiography, including GLS analysis via STE, was conducted.

KEYWORDS: *Iron Deficiency Anemia, Global Longitudinal Strain, Left Ventricular Function, 2D Strain Imaging, Echocardiography, Cardiac Dysfunction, Heart Failure.*

INTRODUCTION

Iron deficiency anemia, a widespread blood disorder, is defined by low iron levels that disrupt red blood cell formation, reducing hemoglobin and impairing oxygen transport. It is a global health concern affecting all age groups, though it is most prevalent among children, adolescents, fertile women, and the elderly. India accounts for over half of the world's malnourished population.[1] A WHO study between 1993 and 2005 found that 25% of the global population was anemic.[2] The National Family Health Survey-3 (NFHS-3) reported that 56% of individuals may be anemic, while the National Nutrition Monitoring Bureau Survey (NNMBS) from 2006 indicated 69.7% of women and 68.6% of men suffered from anemia.[3,4]

Grading of severity of anemia (Table 1, Table 2).

Table 1: WHO Grading of Anemia Severity.[5]

Population	Value	Severity
Adult Male (>15 years)	11.0 – 12.9 g/dL	Mild
	8.0 – 10.9 g/dL	Moderate
	<8.0 g/dL	Severe

Adult Females (Non-pregnant, > 15 years)	11.0 – 11.9 g/dL	Mild
	8.0 – 10.9 g/dL	Moderate
	< 8.0 g/dL	Severe
Pregnant Females	10.0 – 10.9 g/dL	Mild
	7.0 – 9.9 g/dL	Moderate
	< 7.0 g/dL	Severe

Table 2: ICMR Grading of Anemia Severity.[6]

Value	Severity
<4 g/dL	Very Severe
4 – 6.9 g/dL	Severe
7 – 9.9 g/dL	Moderate
10 – 10.9 /dL	Mild

While the primary clinical effects of IDA stem from its blood-related impacts, emerging research suggests it may also influence left ventricular (LV) function. The LV, a critical part of the circulatory system, pumps oxygenated blood throughout the body. Alterations in LV function can significantly affect cardiovascular health and overall well-being.

Studies have explored potential links between IDA and LV changes to better understand the cardiovascular consequences of iron deficiency.[7,8]

Iron is vital for processes like muscle contraction, oxygen utilization, and heart energy metabolism. In IDA, reduced iron availability may impair cardiac function, leading to LV hypertrophy, weakened contractions, and oxygen supply-demand imbalances[9] (Figures 1,2).

Additionally, IDA has been associated with endothelial dysfunction, oxidative stress, and systemic inflammation, which can worsen cardiovascular diseases, including LV failure. Chronic iron deficiency might also contribute to structural and functional heart remodeling.

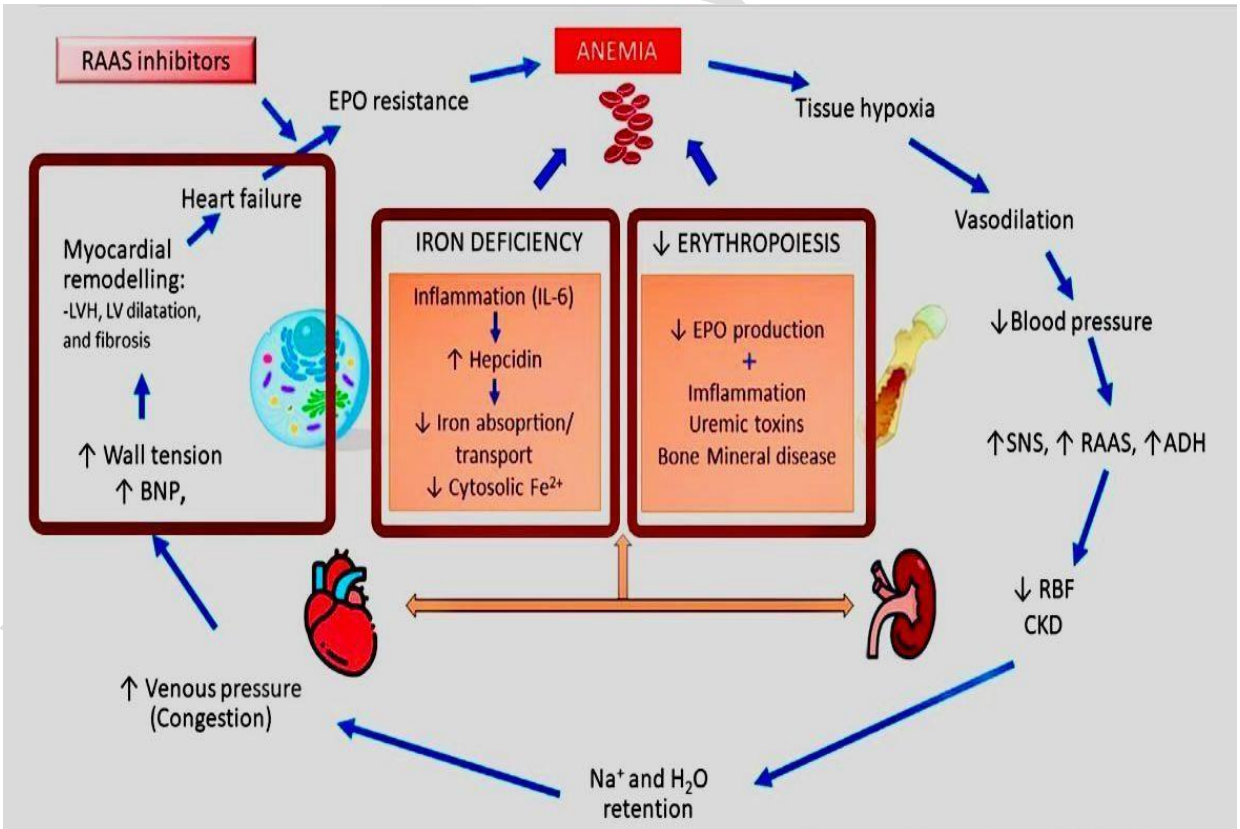

Figure 1: Pathophysiology of Severe Anemia causing HfpEF.

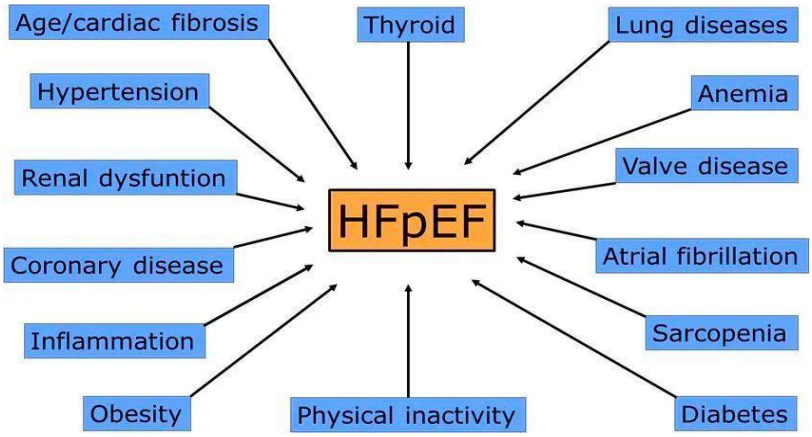

Figure 2: Principle mechanisms/causes of heart failure with preserved ejection fraction (HFpEF). Each cause could determine acute cardiac decompensation.

Despite growing research on IDA's relationship with LV function, findings remain inconsistent. Some studies report reduced LV systolic and diastolic function along with impaired STE values in IDA patients, while others find no clear correlation. These discrepancies may arise from differences in study populations, sample sizes, methodologies, and LV function assessment techniques[9] (Table 3).

Table 3: Normal baseline values of Global Longitudinal Strain.

Age Group	Global Longitudinal Strain		Reference
	Mean (%)	Range (%)	
Healthy Adolescent	-19	-19 to -22	[10]
Healthy Pediatric & adolescent	- 20.2	- 16.75 to - 23.6	[11]

Given the potential cardiovascular impacts of IDA, further research on LV function in these patients is critical. Advanced imaging methods like cardiac MRI and STE can comprehensively evaluate LV function, offering insights into how IDA affects heart structure and performance[12,13] (Figures 3,4).

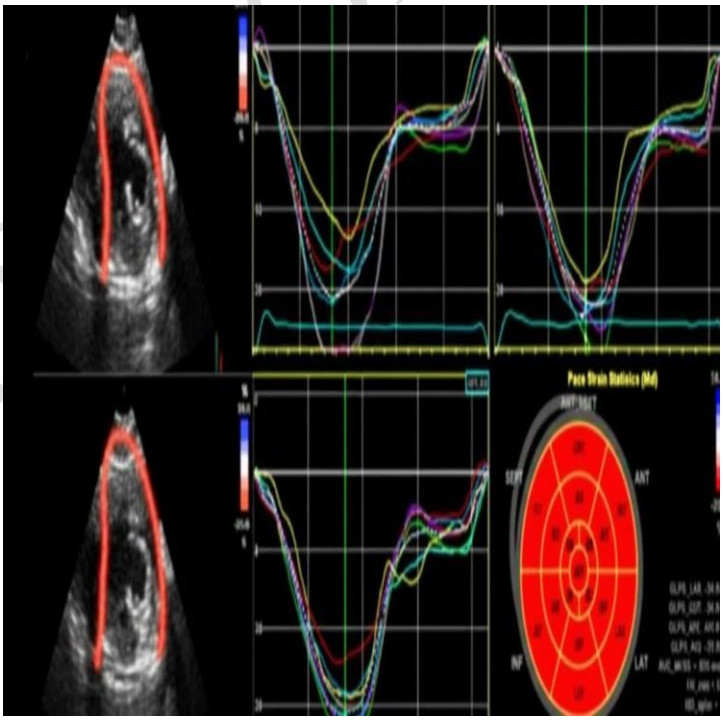

Figure 3: Two-dimensional speckle tracking: from recorded multiple heart beats during breath-hold, an apical four-chamber view, an apical two-chamber view, and an apical three-chamber view have been stored. Once the specific views have been named, it is required to trace the endocardial border in a still frame, setting the ROI. Finally, the evaluation of the deformation graphs and values may be calculated. The segmental peak systolic strains are represented in bullseye. ROI indicates region of interest.

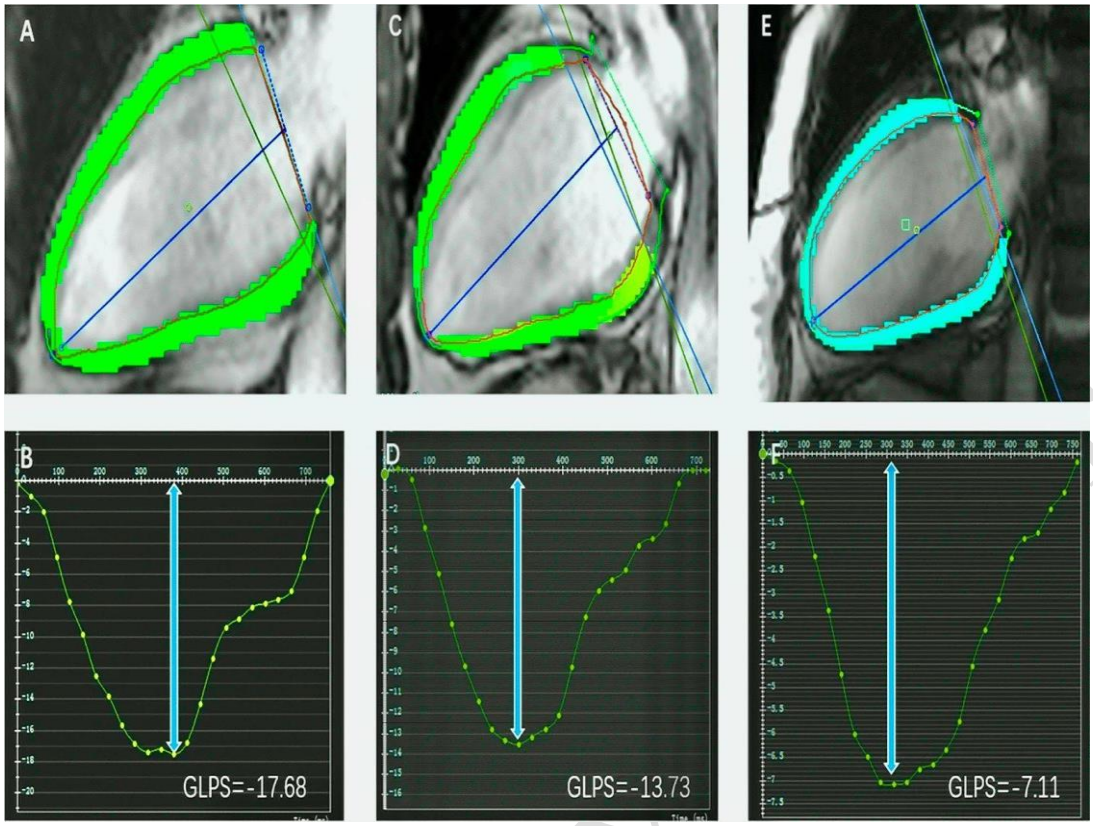

Figure 4 : The worsening effect of anemia on left ventricular function and global strain: Representative CMR pseudocolor images at the end-diastole and CMR-derived peak strain curves, A,C,E: LV pseudocolor images in the vertical 2-chamber long axis; B,D,F,: LV global peak strain curve in the longitudinal direction; A, B: a patient of control group; E,F : a T2DM patient with anemia. GLPS, global longitudinal peak strain.

In order to guide therapeutic interventions for affected persons, identify cardiac issues early, and stratify risk, it is important to understand the link between IDA and LV function.

Examining left ventricular function in this patient population has substantial therapeutic importance and may lead to better patient care and outcomes, especially given the rising prevalence of iron deficiency anaemia and its possible cardiovascular consequences.

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 5) consistent with severe anemia. There was conspicuous swelling all over the body, particularly over hands (Figure 6) and abdomen. Moreover, elevated Jugular Venous Pressure (JVP) (Figure 8), was demonstrated.

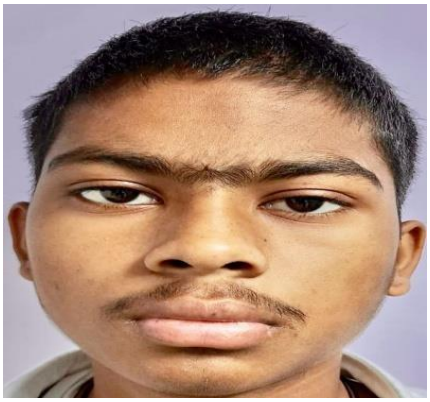

Fig. 5) Facies of our index patient. Striking pallor is obvious.

Fig. 6

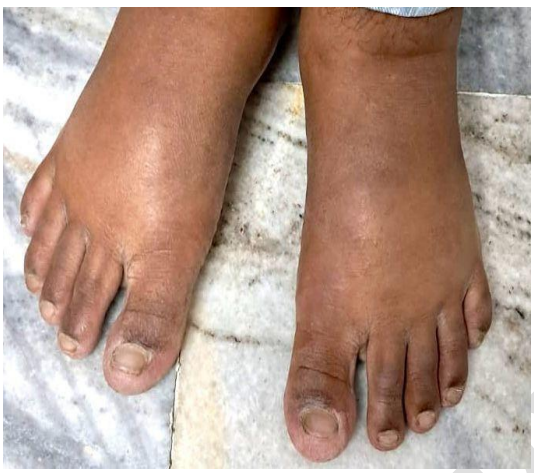

Fig. 7

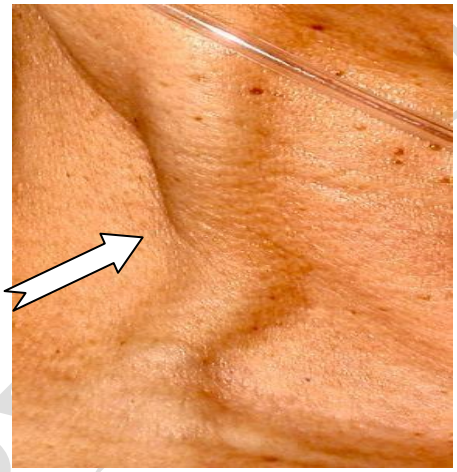

Fig. 8

Fig. 6) Swelling of both hand and all fingers; Fig. 7) Swelling of feet; Fig. 8) 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 9)

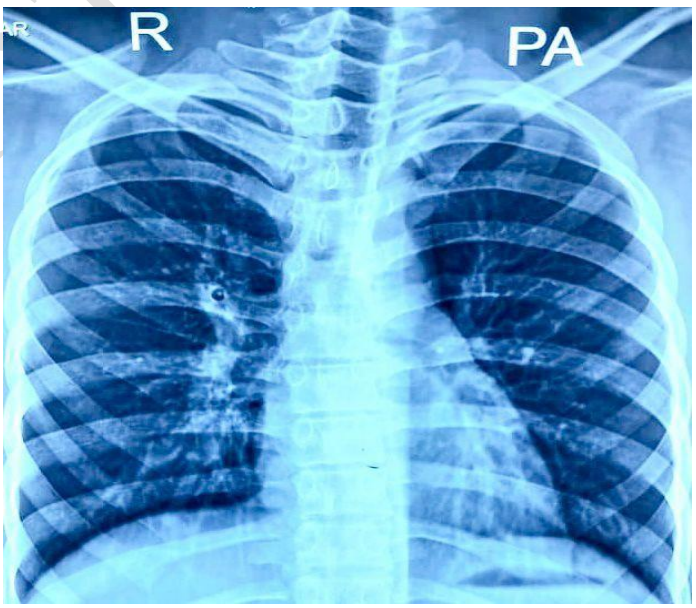

Fig. 9) There characteristic pulmonary plethora accompanied with normal cardiac size.

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 L2, L3, AVF, V1-4. There was no evidence of sinus node or AV nodal disease or arrhythmia. (Figure 10).

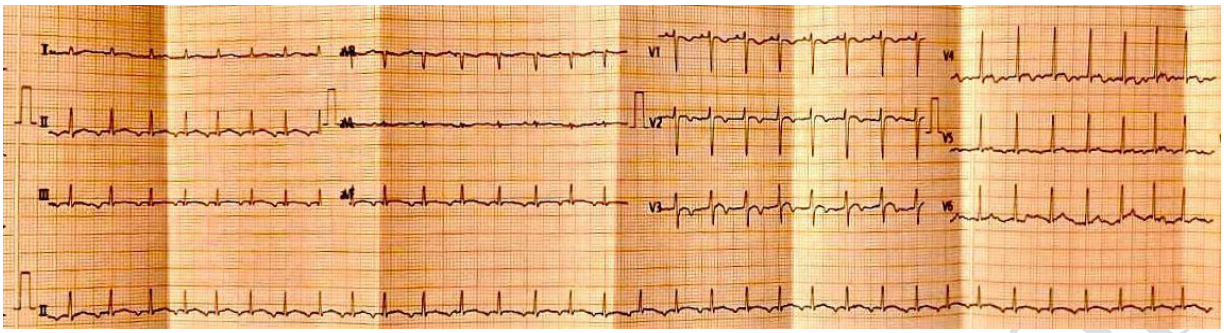

Fig. 10) Resting ECG exhibited sinus tachycardia with a ventricular rate of 120/min with a normal QRS axis.

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 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

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

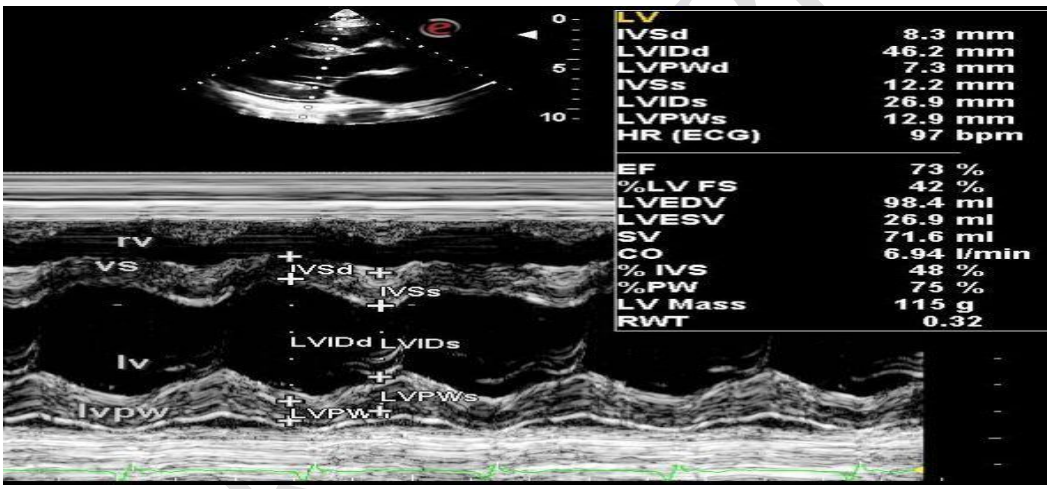

Fig.11: M-mode echocardiography of LV.

1. 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 17-19).**
b) Dilated right and left atrium.
c) Dilated inferior vena cava (IVC) with reduced respiratory excursion **(Figure 12).**

- 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 19-20).
e) Mild circumferential pericardial effusion was detected with an estimated fluid of approximately 100ml **(Figures 19-21).**
f) There was no evidence of pericardial constriction.
g) No PAH was demonstrated.

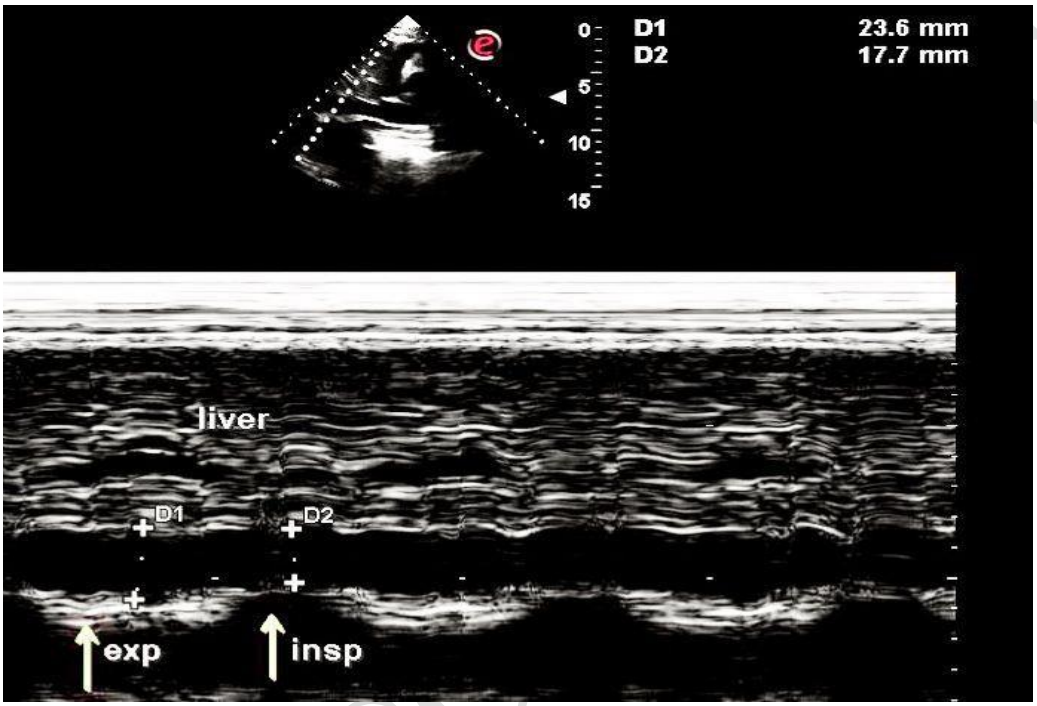

Fig. 12

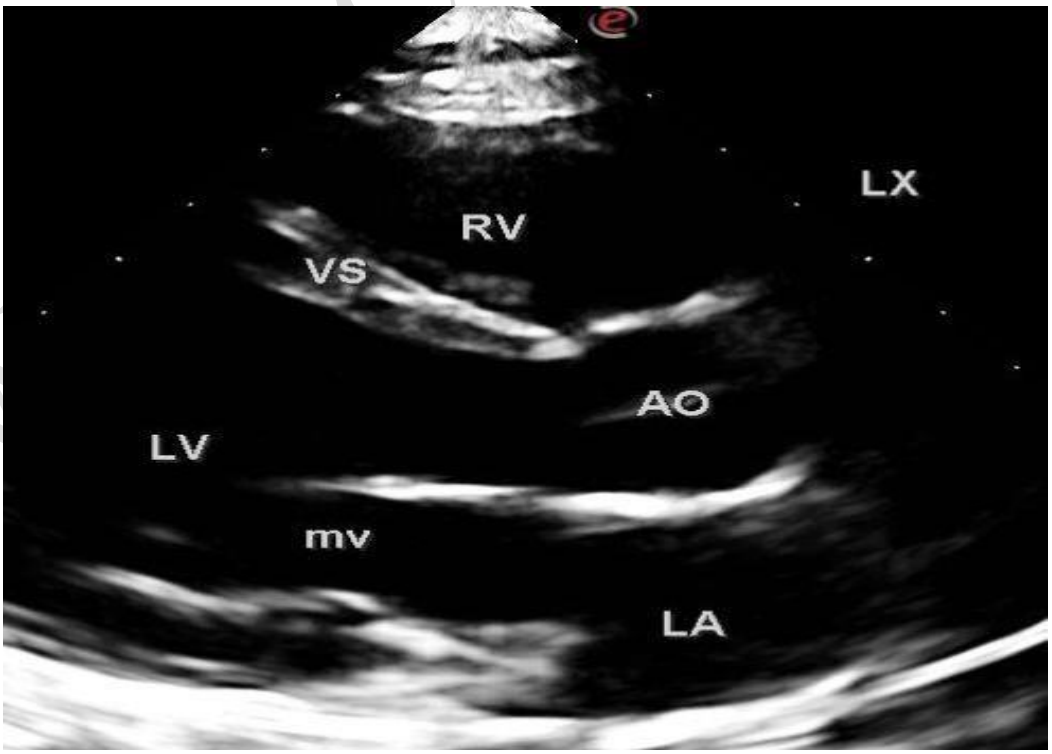

Fig.13

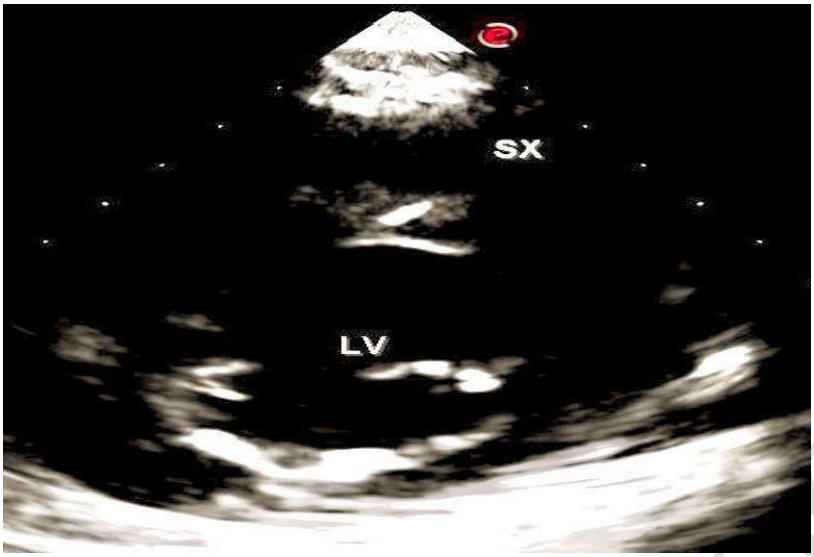

Fig. 14

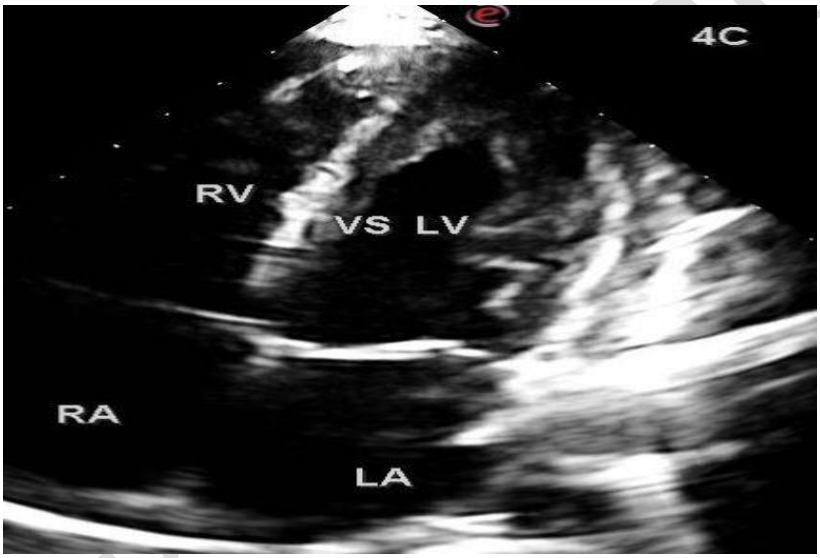

Fig. 15

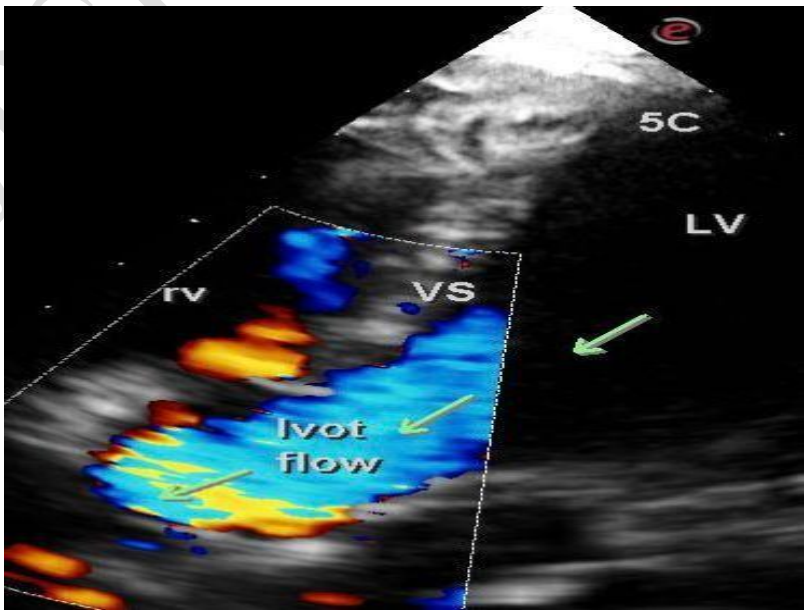

Fig. 16

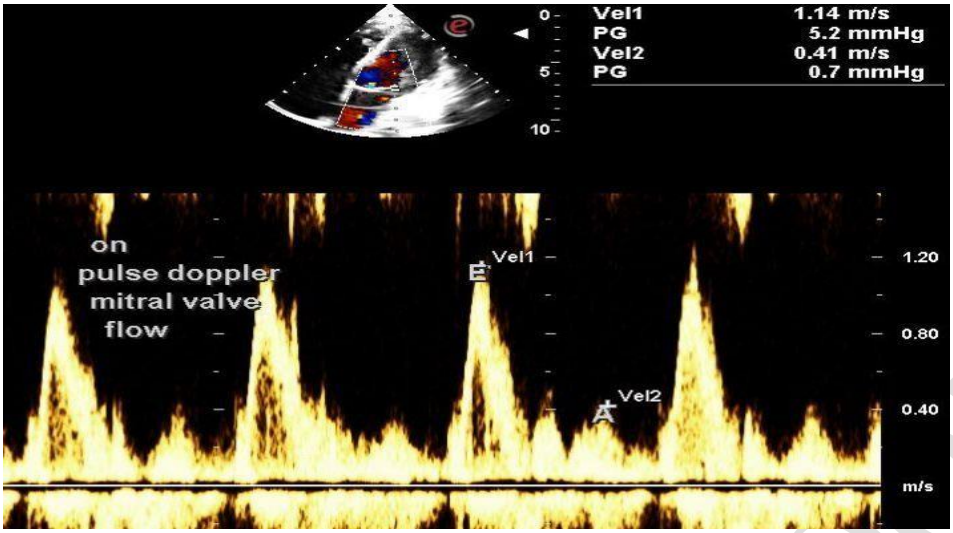

Fig. 17

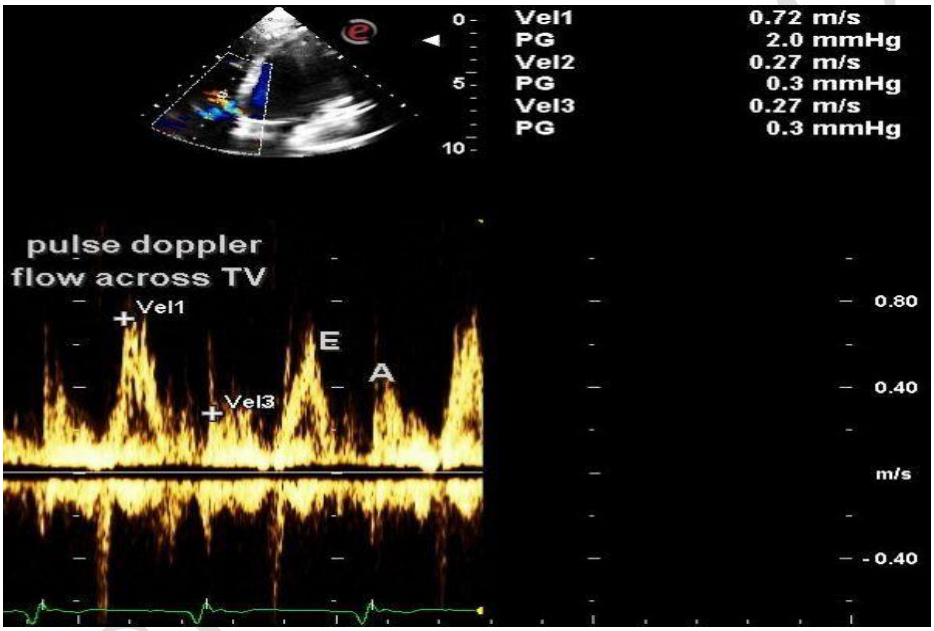

Fig. 18

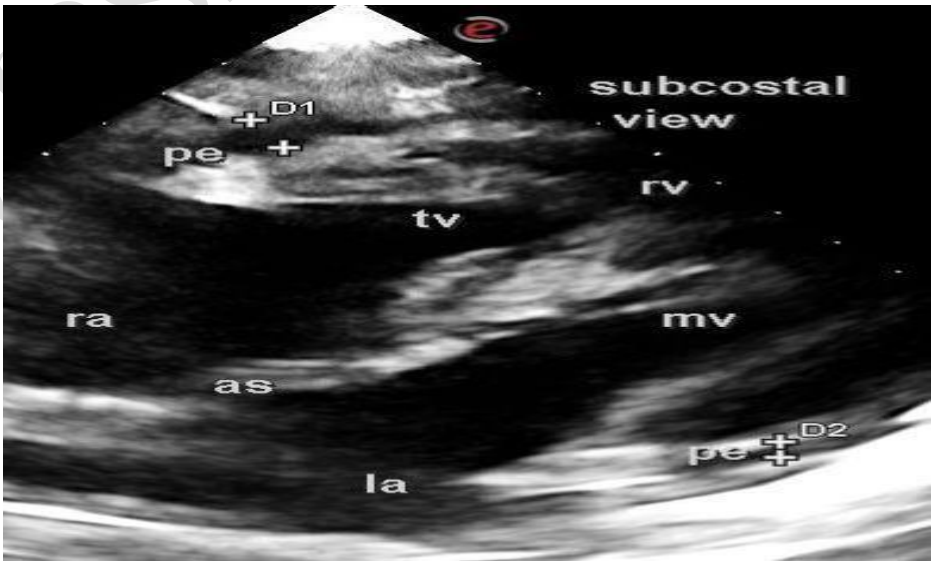

Fig. 19

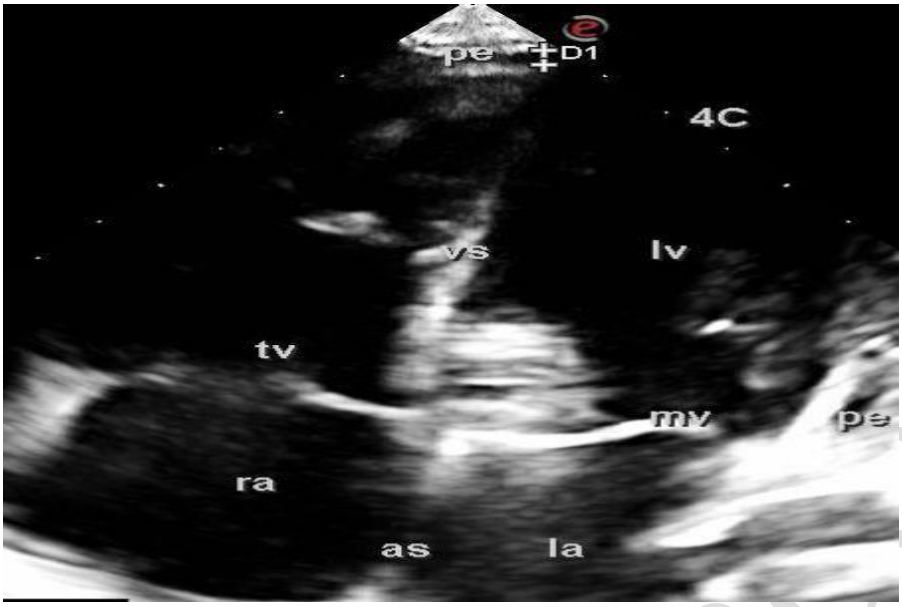

Fig. 20

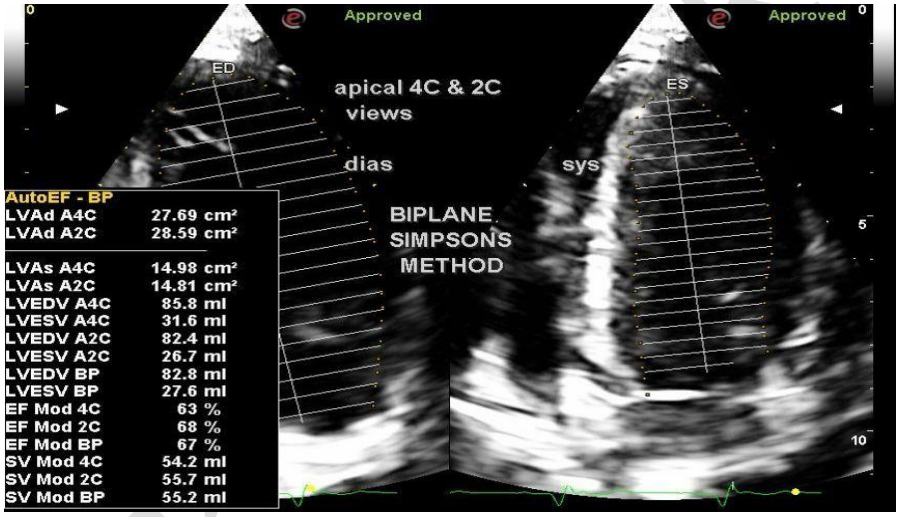

Fig. 21

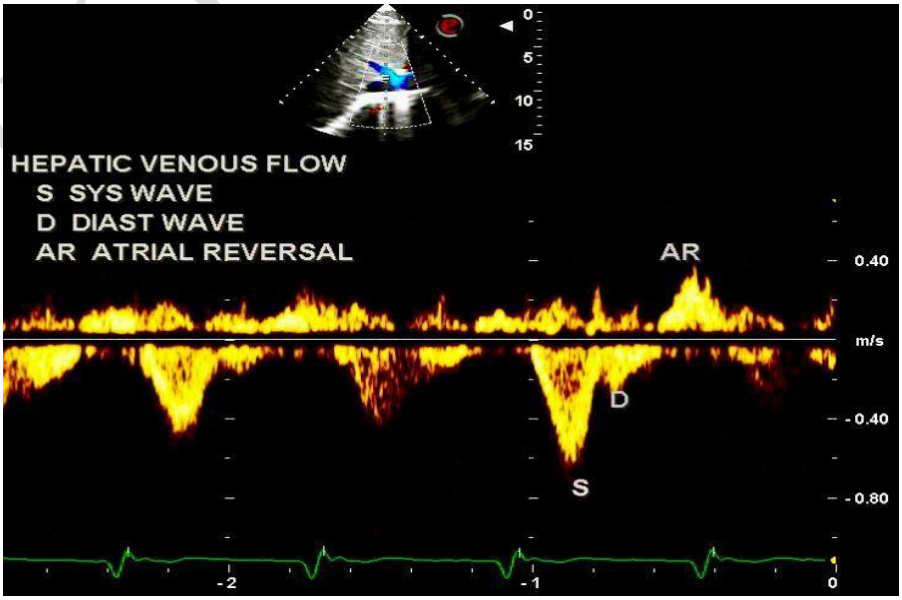

Fig. 22

Figures 12-22: Fig. 12: On subcostal view a dilated IVC is seen with reduced respiratory excursion during inspiration indicating presence of congestive heart failure; **Fig.13**: LV anatomy and dimensions were normal of LX view; **Fig. 14**: In the SX view the LV was normal; **Fig.15**: 4C view was normal; **Fig.16**: In the 5C view there was not LV outflow obstruction; **Fig. 17:** On pulse wave doppler analysis the mitral inflow velocity showed a E/A ratio of 2.5 : 1, indicating a restrictive physiology; **Fig. 18:** Tricuspid inflow velocity similarly demonstrated a restrictive physiology with an E/A ratio of :1; **Fig. 19:** Mild circumferential pericardial effusion was observed in the subcostal view, pericardial effusion; **Fig. 20**: Similarly 4C view exhibited a mild pericardial effusion; **Fig. 21**: EF was 67% on Biplane-Simpson's method; **Fig. 22**: 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 longitudinal strain (GLS) and moreover decrement in values of few or multiple segments of 17-segment module of LV segmental strain imaging (Fig 21-23, Table 3).

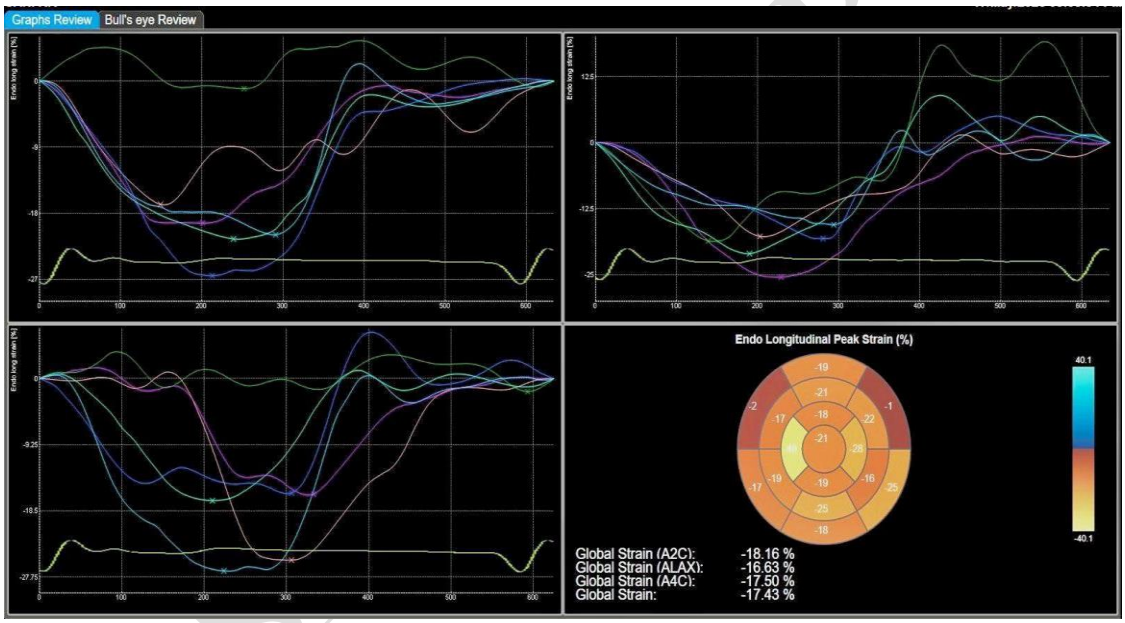

Fig. 23

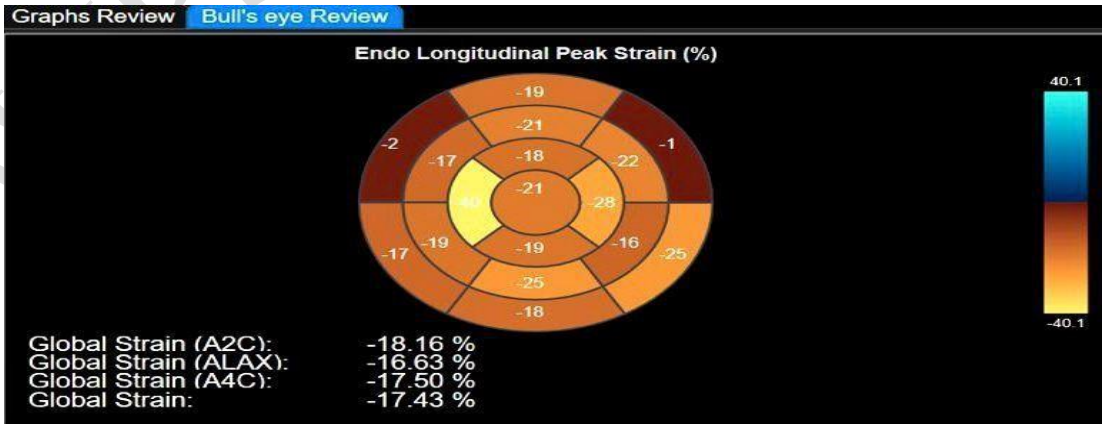

Fig. 24

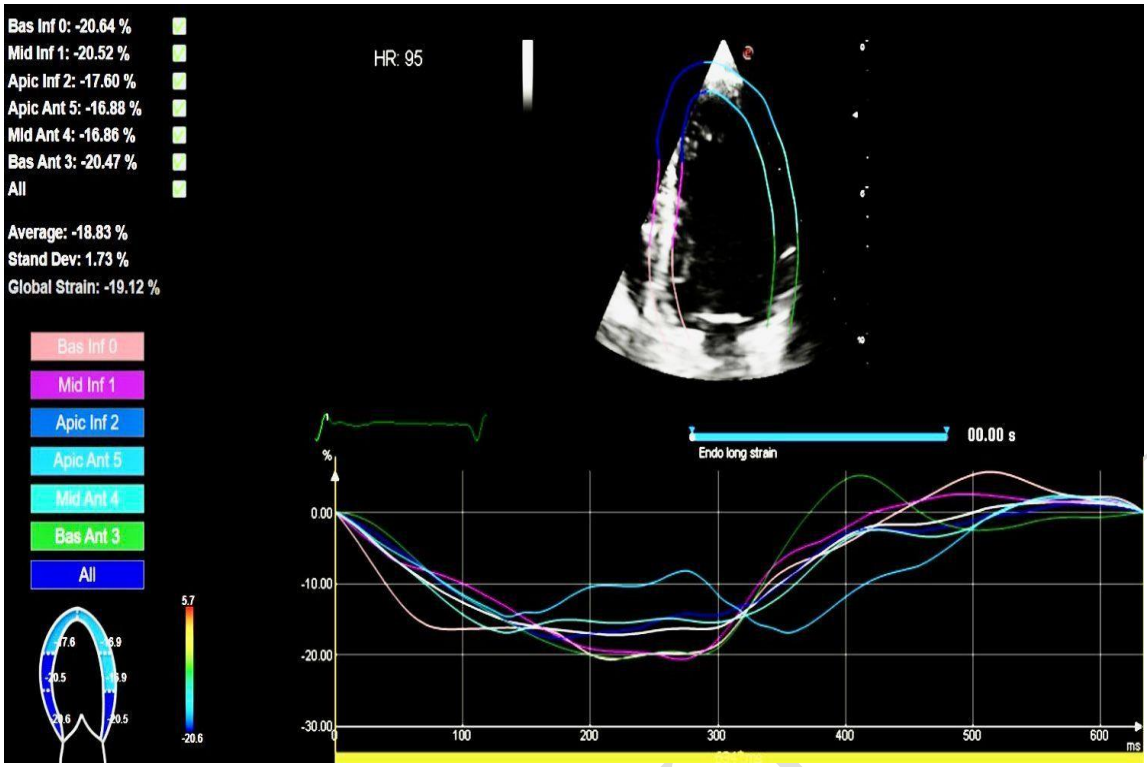

Fig. 25 a)

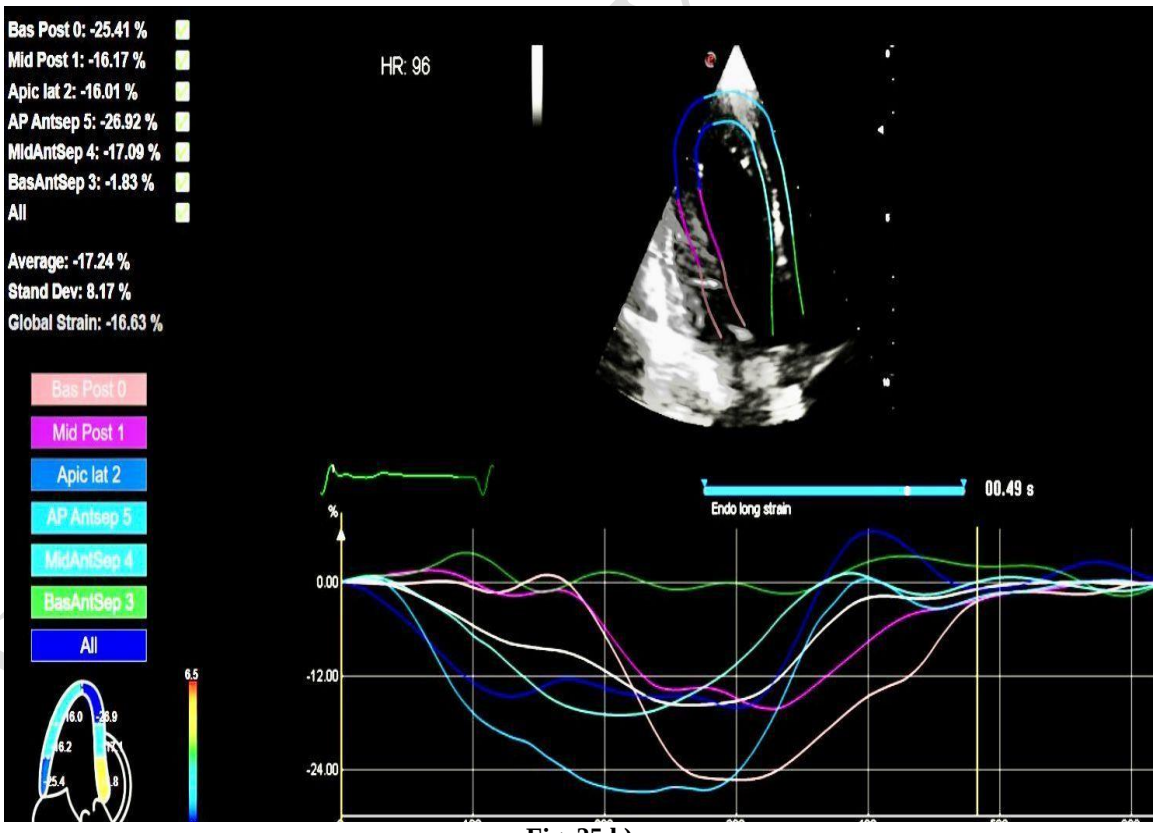

Fig. 25 b)

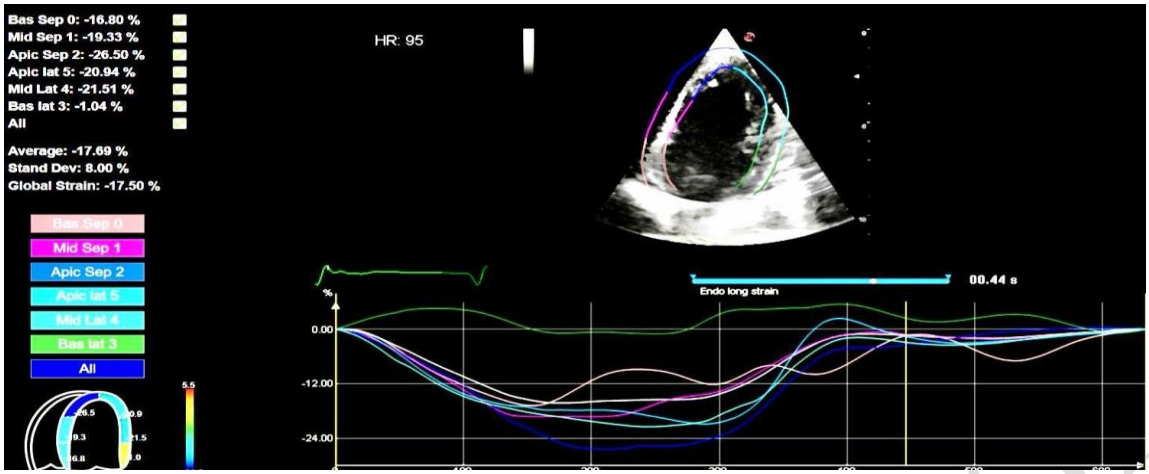

Fig. 25 c)

Figure 22-25: LV strain imaging by speckle tracking echocardiography.

Fig. 23) Graphs and Bulls Eye plot of apical 2C, apical LAX & apical 4C views. LV Global strain was - 17.43 %; **Fig. 24).** Bulls Eye Plot of apical LV strain echocardiography. Global Strain was - 17.43 %; **Fig. 25a)** Apical 2C Longitudinal Strain was - 19.12 %; **Fig. 25b)** Apical LAX longitudinal strain was - 16.63 %; **Fig. 25c)** 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 appropriately implemented. 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

Pathophysiology of Anemia in Heart Failure[14]

Anemia is frequently observed in individuals with both acute and chronic systolic heart failure (HF), as well as in HF with preserved ejection fraction (HFpEF), affecting up to half of patients depending on diagnostic criteria and study populations. Low or declining hemoglobin levels, alongside anemia, are recognized as strong independent indicators of poor prognosis in HF.

Addressing anemia could potentially enhance HF outcomes and may represent a new avenue for treatment. Initial research and pooled analyses have indicated that anemia correction in HF patients might alleviate symptoms, improve heart function, and enhance quality of life. However, due to safety concerns regarding elevated haemoglobin levels associated with erythropoietin-stimulating agents (ESAs) in chronic kidney disease (CKD) patients, medical professionals may hesitate to pursue anemia treatment until further evidence emerges from trials like the darbepoetin alfa study in HF, which assesses event reduction and mortality.

Factors Linked to Anemia in HF Patients[14]

Individuals with HF and anemia tend to be older, more likely to have diabetes or chronic kidney disease (CKD), and often exhibit more severe HF. This includes higher New York Heart Association (NYHA) class scores, reduced exercise tolerance, lower quality-of-life ratings, increased swelling, lower blood pressure, greater reliance on diuretics, and more pronounced neurohormonal or inflammatory markers. Research from the Valsartan Heart Failure Trial (ValHeFT) database identified renal impairment, diabetes, edema, elevated brain natriuretic peptide (BNP) or C-reactive protein (CRP), low albumin, low body weight, and reduced diastolic pressure as independent risk factors for anemia. Notably, anemia does not appear to correlate with diminished left ventricular (LV) function in studies where such measurements were available. Hemoglobin levels inversely relate to ejection fraction (EF): lower haemoglobin levels coincide with higher EF.

Additionally, spontaneous haemoglobin increases over time may signal worsening LV function, and in CKD patients, haemoglobin elevation has been linked to reduced LV function in a dose-dependent manner.

Anemia as a Risk Factor for Cardiovascular Outcomes[22]

Anemia is recognized as an independent contributor to cardiovascular disease (CVD) risks[15,16] in the general population and is associated with higher illness and death rates in HF patients. Severe anemia can trigger hemodynamic shifts, such as increased blood volume, reduced vascular resistance, and elevated cardiac output. HFpEF, accounting for 40–50% of HF cases, carries significant health burdens and mortality.[17] Despite numerous randomized trials targeting therapies for HFpEF, no effective treatments have emerged, leaving mortality rates stagnant over the past two decades.[18] Advanced imaging methods like speckle-tracking echocardiography and cardiac MRI may improve the identification of HFpEF subgroups.[19,20] (Figure 26).

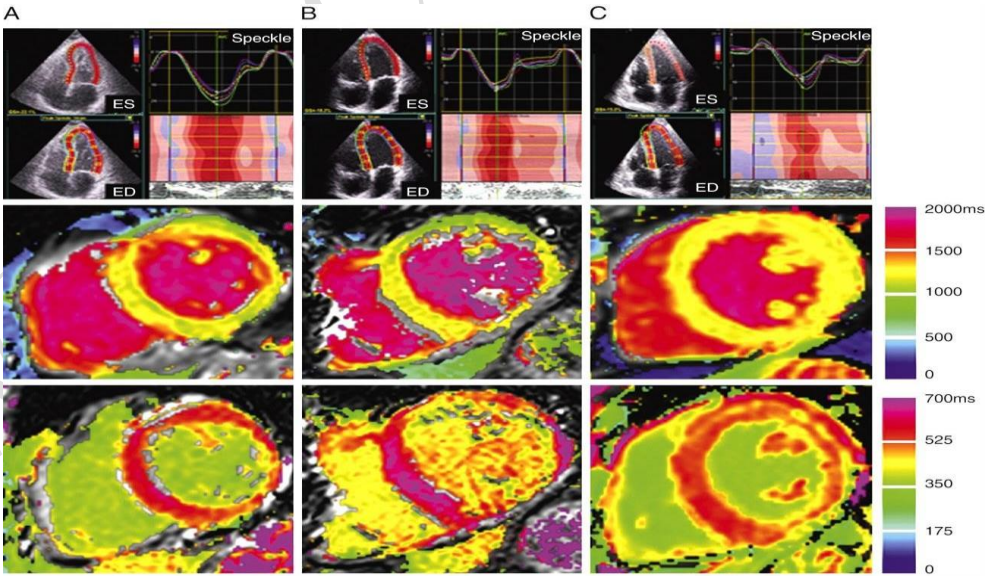

Figure 26: Tagged CMR Global Longitudinal Strain (eGLS), Top Row: end-diastole [ED] and end- systole [ES] and pre- and post-contrast T1 mapping (**middle** and **bottom** rows, respectively). **(A)** A male healthy volunteer with an eGLS of -22.1% is shown. Mean native T1 was 1212.7 ms., post contrast T1 was 586.1 ms. Extracellular volume (ECV) was calculated at 24.9%. **(B)** A hypertensive male with an eGLS of -18.2% is shown. Mean native T1 was 1169.0 ms, post-contrast T1 was 593.5 ms. ECV was calculated, at 31.2%. **(C)** A male with heart failure with preserved ejection fraction (left ventricular ejection fraction with an eGLS of -15.0%) is shown. Mean native T1 was 1265.0 ms, post-contrast T1 was 484.8 ms. ECV was calculated at 36.3%.

Diagnosing heart failure with preserved ejection fraction (HFpEF) remains complex. While echocardiography-based guidelines[21] provide clear parameters, similar findings are frequently observed in hypertensive individuals without HFpEF. Moreover, left ventricular global longitudinal strain (GLS) is a critical tool for identifying exercise limitations, which are prevalent in HFpEF patients.[23]

Recent advancements in speckle-tracking echocardiography have revealed subclinical left ventricular (LV) dysfunction in HFpEF patients, often associated with reduced GLS.[21] Furthermore, diminished GLS has emerged as an independent predictor of poor outcomes in this population.[22] A study by Kosmala et al.[23] emphasized that subclinical systolic dysfunction, detected via lowered GLS, is the most effective indicator distinguishing HFpEF patients with exercise restrictions from those without.

To assess LV function in IDA patients, various echocardiography techniques have been explored.

Notably, 2D and 3D speckle-tracking LV-GLS has become a standard method, capable of identifying early myocardial changes before LVEF alterations. ZHOU et al.,[24] concluded that iron therapy can reduce heart failure hospitalisation, increase cardiac function, improve quality of life and decrease in NT-pro-BNP level in patients with HFpEF. Abnormal GLS correlates with heart failure progression, mortality, and LV remodeling.[25] This may stem from GLS's reliance on endocardial longitudinal fibers supplied by distal coronary vessels, which are prone to ischemia and hypoxia, leading to reduced systolic and diastolic function. Additionally, chronic IDA-induced high-output states increase LV pre- and afterload, gradually diminishing myocardial performance and GLS.

Goyal et al.[27] confirmed a strong association between severe anemia and LV dysfunction, advocating for 2D strain echocardiography to identify LV issues before structural changes occur. Their study showed that severely anemic patients had significantly lower GLS scores. A GLS score below -16% is abnormal, above -18% is normal, and -16% to -18% is borderline. In Goyal et al.'s cohort, the case group averaged - 16.59% + 1.303%, compared to -19.23 + 0.633% in controls. Similarly, Eroglu et al.[26] reported HF patients had a mean GLS of -9%, versus -20% in controls, with worsening GLS indicating advanced systolic dysfunction.

Our index patient (Hb 6 g/dL) displayed a GLS of -17.43%, which was significantly lower than the normal range for adolescents (-19% to -22%) and pediatric/teenagers (-16.75% to -23.6%).[12,13]

CONCLUSION

GLS derived by 2-Dimensional STE is of tremendous value in patients with severe anemia with HFpEF for the effective management of such cases due to it's high temporal resolution, accessibility and robust prognostic validation.

It is highly recommended for routine clinical utility due to its considerable accuracy in detecting early myocardial dysfunction in individual afflicted with severe anemia.

Early IDA treatment can improve the prognosis of HFpEF, repair the myocardium, promote effective myocardial work and restore the patient's activity endurance. Assessment of cardiac trends in IDA patients can help guide medical therapy and prevent HFpEF.

Tagged CMR is perhaps the gold standard for determining the GLS However its availability is exceedingly low in India due to high cost and affordability issues.

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