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Clinical and Echocardiographic profile of patients presenting with acute Heart failure; a single centered observation study

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Abstract

Background: The present study was conducted to compare the clinical and echocardiographic profile of patients presenting with acute heart failure in India. The comparison was made between types of heart failure, onset and survival ***Methods:*** This is a prospective observational study on 101 patients admitted to the emergency department in a tertiary care hospital. Detailed demographic, clinical parameters with cardiac evaluation were done and collected in the study. ***Results:*** This study compared systolic heart failure (SHF) and diastolic heart failure (DHF) patients, revealing distinct differences in demographics, clinical profiles, and outcomes. SHF patients had higher Creatine Kinase–Myocardial Band (CKMB) (5.6 ± 4.9 vs. 1.5 ± 1 ng/ml, P<0.001) and B-type Natriuretic Peptide (BNP) levels (1940 ± 3357 vs. 603 ± 420 ng/ml, P=0.009), indicating more myocardial injury and cardiac strain. In contrast, DHF patients had higher serum creatinine (1.58 ± 1.66 vs. 1.03 ± 0.33 mg/dl, P=0.018) and liver enzymes, suggesting renal and hepatic impairment. Non-survivors had higher urea levels (43.06 ± 18.85 vs. 31.12 ± 21.56 mg/dl, P=0.036), hyponatremia (P<0.001), and hyperkalemia (P<0.001), as well as elevated CKMB (7.6 ± 7.2 ng/ml, P<0.001) and BNP levels (4216 ± 5254 ng/ml, P<0.001). ***Conclusion***: This study highlights that DHF accounts for a significant proportion of acute heart failure cases. DHF and SHF exhibit similar clinical profiles, but SHF patients face significantly higher short-term mortality risks, emphasizing the need for distinct management approaches to improve patient outcomes.

Keywords: Acute heart failure (AHF), systolic heart failure (SHF), diastolic heart failure (DHF), ECHO (Echocardiography), Laboratory parameters, Onset Type and Survival.

INTRODUCTION

Cardiovascular diseases is the leading cause of mortality in India.1 In developing countries like India, heart failure (HF) has emerged as a significant contributor to death and disability, which is mainly driven by the increasing burden of vascular disease and the continued prevalence of pre-transitional conditions such as rheumatic heart disease and infectious cardiomyopathies.2

Recent study on Association of Lifestyle, Metabolic Risk, and Incident HF reported that individuals with unfavorable lifestyle habits had nearly a 3-fold higher risk of developing heart failure compared to those with favorable habits over a 10-year period.3 The growing burden of hypertension (projected to increase from 118 million in 2000 to 214 million in 2025) may contribute to 214,000–1.3 million new HF cases annually by 2025. Similarly, the increasing prevalence of obesity (5%) could result in 180,000–300,000 new HF cases per year, and diabetes could add 73,600–161,000 new HF cases annually. Taken together, the unfinished pre-transition agenda, including high rates of rheumatic heart disease and infectious cardiomyopathies, further complicates India's growing HF burden.4

HF may present as a new diagnosis or as an acute exacerbation of pre-existing disease. Acute heart failure (AHF), characterized by the rapid onset of symptoms and signs due to cardiac dysfunction, can arise de novo or from acute decompensation of chronic HF.5 AHF is a common presentation to the emergency department, which can be confused with other clinical conditions. The clinical presentation of AHF is highly variable, making timely and accurate diagnosis essential. While the diagnosis can be straightforward in classic cases, it may be challenging when symptoms and signs are atypical, as no single finding alone can define AHF.6

Thus, a comprehensive medical history, focused examination, echocardiography, and appropriate laboratory investigations form the cornerstone of diagnosis. Considering this, the aim of the present study was to evaluate the clinical and

echocardiographic profiles of patients presenting with AHF in an Indian population, identify precipitating factors and comorbidities, and differentiate between HF with reduced and preserved ejection fraction.

MATERIALS AND METHODS

This study was a hospital-based, prospective observational study conducted in the tertiary care hospital. The study was initiated after obtaining approval from the hospital's ethical committee. Study was done in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000. An informed consent form was obtained from the patient's family to use the medical reports for the study. The study was conducted over a two-year period, during which 101 patients who met the following inclusion and exclusion criteria were enrolled in the study.

Inclusion Criteria

1. Acute onset breathlessness with or without chest pain, palpitations, or syncope.
2. Patients with bilateral crepitations on auscultation and those with known congestive heart failure on optimal medical therapy who are presented with worsening symptoms

Exclusion Criteria

Patients were excluded from the study if they had a diagnosis of bronchial asthma, chronic obstructive pulmonary disease, pulmonary thromboembolism, pneumonia, sepsis, or cardiac tamponade/pericardial effusion.

Classification of Heart failure

All enrolled patient was classified sequentially by Left ventricular ejection fraction (LVEF) and etiological classification. HF was classified as SHF (LVEF ≤ 45%) or DHF (LVEF ≥ 45%) on the basis of a prior cut off value derived from a prior evaluation, which demonstrated a linear increase in mortality risk for ejection fractions (EFs) > 45%.7 De novo HF was defined as no HF diagnoses in the year before index hospitalization,8 while worsening chronic HF (WCHF) was defined by escalating signs and symptoms of HF in patients with chronic HF despite previously stable therapy.9 Outcome was reported in terms of survival and death.

Statistical Analysis:

The statistical analysis was done using SPSS (Statistical Package for Social Sciences) Version 15.0 statistical Analysis Software. Descriptive statistics, including frequencies, percentages, means, and standard deviations, were used to summarize the demographic, clinical, and laboratory characteristics of the study population. Categorical variables were expressed as frequencies, while continuous variables were presented as means with standard deviations. The chi-square test was used to analyze categorical variables. For comparisons between the two groups, the independent t-test was applied. A p-value of <0.05 was considered statistically significant for all analyses.

RESULTS

A total of 101 patients were recruited and categorized into Systolic Heart Failure (SHF, n=56; EF <45%) and Diastolic Heart Failure (DHF, n=45; EF >45%). DHF was more prevalent among patients aged <60 years (p=0.04) and in females (60%, p=0.038) (Table 1). Of these, 52 patients had De novo heart failure and 49 had worsening chronic heart failure (WCHF), with no significant demographic differences between these groups.

Among hematological parameters, neutrophil count was significantly higher in SHF (76.64 ± 4.02) than DHF (74.64 ± 5.58) (p=0.039). DHF patients had higher serum creatinine (1.58 ± 1.66 mg/dl vs. 1.03 ± 0.33 mg/dl, p=0.018), AST (127.2 ± 298.3 IU/L vs. 41.1 ± 23.2 IU/L, p=0.034), and ALT (136.7 ± 312.3 IU/L vs. 46.4 ± 29.2 IU/L, p=0.033), indicating greater renal and hepatic dysfunction. Bicarbonate levels were lower in DHF (23.9 ± 2.1) vs. SHF (24.8 ± 1.6) (p=0.014) (Table 2).

Hyperkalemia was more common in WCHF (p=0.019). Non-survivors had significantly higher urea levels (43.06 ± 18.85 mg/dl, p=0.036), more frequent hyponatremia and hyperkalemia (both p<0.001), and lower total cholesterol (157.4 ± 45.2 mg/dl, p=0.006) and LDL (103.7 ± 19.8 mg/dl, p=0.014). Dyslipidemia was more common among survivors (p=0.019) (Table 2).

Cardiac biomarkers were significantly elevated in SHF: CKMB (5.6 ± 4.9 vs. 1.5 ± 1 ng/ml, p<0.001), troponin I (4.64 ± 9.34 vs. 0.26 ± 1.03 ng/ml, p=0.002), and BNP (1940 ± 3357 vs. 603 ± 420 ng/ml, p=0.009) compared to DHF. No significant differences in biomarkers were observed between De novo and WCHF (Table 3). Non-survivors had significantly lower pH (7.22 ± 0.09), higher pCO_2 (58.5 ± 6.0 mmHg), lower pO_2 (83.6 ± 5.3 mmHg), and lower bicarbonate (23.0 ± 2.1 mEq/L) (all p<0.001). CKMB (7.6 ± 7.2 ng/ml) and BNP (4216 ± 5254 ng/ml) were also significantly elevated among non-survivors (p<0.001) (Table 3).

Atrial fibrillation was more common in DHF (40.0%) than SHF (16.1%) (p=0.007). ST elevation (21.4% vs. 2.2%, p=0.004) and QS pattern (42.9% vs. 22.2%, p=0.029) were more frequent in SHF. LVH was significantly more prevalent in DHF (44.4% vs. 17.1%, p=0.006). ST depression was more frequent in WCHF (p=0.047). Atrial flutter (p=0.019), ST elevation (p=0.026), and LVH (p=0.004) were significantly associated with survival (Table 4).

Echocardiography revealed LV dilation was more common in SHF (73.2% vs. 22.2%, p<0.001), whereas non-dilated LV and concentric hypertrophy were more common in DHF (p=0.002 and p=0.001, respectively). SHF patients had larger LV dimensions and higher rates of global hypokinesia (p<0.001 and p=0.003, respectively) (Table 5). LV dilation was more common in WCHF (p=0.036); moderate MR was more frequent in WCHF (p=0.005), while non-dilated LV was common in De novo (p=0.033). Severe MR was associated with mortality (p=0.011) (Table 5).

Valvular abnormalities were more pronounced in SHF: absence of TR was more common in DHF (62.2%, p<0.001), while higher grades of MR, AR, and MS were predominantly seen in SHF (all p<0.001, except MS p=0.013) (Table 6). Doppler findings showed restrictive filling (E>A) was dominant in SHF (76.8%), while impaired relaxation (E<A) was more frequent in DHF (51.1%, p<0.001). PAP was significantly elevated in SHF (82.1% vs. 42.2%, p<0.001), highlighting distinct structural and functional differences between the two heart failure phenotypes (Table 6).

Table 1: Age and Gender distribution according to the type of heart failure

	Total n=101	**SHF n=56**	**DHF n=45**	**p-value**	***De novo* n=52**	**WCHF n=49**	**p-value**
Age groups							
≤ 60 years	53 (52.4)	24 (42.9)	29 (64.4)	0.040	31 (59.6)	22 (44.9)	0.108
61-80 years	36 (35.6)	26 (46.4)	10 (22.2)		18 (34.6)	18 (36.7)	
≥ 80 years	12 (11.8)	6 (10.7)	6 (13.3)		3 (5.8)	9 (18.4)	
Gender							
Male	52 (51.5)	34 (60.7)	18 (40.0)	0.038	25 (48.1)	27 (55.1)	0.480
Female	49 (48.5)	22 (39.3)	27 (60.0)		27 (51.9)	22 (44.9)	
Survival	84(83.1)	42 (75)	42 (93.3)	0.014	46 (88.5)	38 (77.6)	0.143
Death	17(16.8)	14 (25)	3 (6.7)		6 (11.5)	11 (22.4)	

Data is presented as mean ± SD. p < 0.05 was considered statistically significant. CKMB: Creatine Kinase–Myocardial Band; TNI: Troponin I; MYO: Myoglobin; BNP: B-type Natriuretic Peptide.

Table 2: Comparison of Hematological and biochemical parameters Across HF Subtypes, Onset Types, and outcomes

Parameter	**SHF n=56**	**DHF n=45**	**p-value**	***De novo* n=52**	**WCHF n=49**	**p-value**	**Survival n=84**	**Death n=17**	**p-value**
Hb level (mg/dl)									
<8	1 (1.8)	6 (13.3)	0.076	3 (5.8)	4 (8.2)	0.411	6 (7.1)	1 (5.9)	0.798
8-10	28 (50)	20 (44.4)		22 (42.3)	26 (53.1)		41 (48.8)	7 (41.2)	
>10	27 (48.2)	19 (42.2)		27 (51.9)	19 (38.8)		37 (44.04)	9 (52.9)	
TLC	8354 ± 1000	8154 ± 907	0.300	8273 ± 601	8256 ± 1240	0.927	8241 ± 963	8384 ± 968	0.577
Neutrophil count	76.64 ± 4.02	74.64 ± 5.58	**0.039**	75.7 ± 5.7	75.8 ± 3.8	0.963	75.50 ± 5.24	77.00 ± 1.62	0.247
Urea (mg/dl)	30.48 ± 11.54	36.42 ± 29.42	0.169	31.6 ± 19.7	34.7 ± 23.3	0.469	31.12 ± 21.56	43.06 ± 18.85	0.036
Creatinine (mg/dl)	1.03 ± 0.33	1.58 ± 1.66	**0.018**	1.28 ± 1.15	1.27 ± 1.19	0.969	1.24 ± 1.26	1.47 ± 0.47	0.448
Hyponatremia	16 (28.6)	11 (24.4)	0.641	12 (23.1)	15 (30.6)	0.392	10 (11.9)	17 (100)	<0.001
Potassium	8 (14.3)	3 (6.7)	0.222	2 (3.8)	9 (18.4)	0.019	0 (0)	11 (64.7)	<0.001
AST (IU/L)	41.1 ± 23.2	127.2 ± 298.3	**0.034**	101.1 ± 255.4	56.4 ± 125.3	0.271	84.87 ± 222.28	52.59 ± 31.06	0.553
ALT (IU/L)	46.4 ± 29.2	136.7 ± 312.3	0.033	108.1 ± 264.2	63.8 ± 139.4	0.299	90.76 ± 232.98	66.18 ± 41.23	0.667
FBS (mg/dl)	122.1 ± 57.7	129.8 ± 83.5	0.575	129.9 ± 81.5	120.9 ± 50.0	0.505	128.70 ± 70.18	109.82 ± 54.37	0.298
PPBS (mg/dl)	199.3 ± 98.9	187.1 ± 122.2	0.580	199.3 ± 129.5	188.1 ± 91.9	0.610	195.1 ± 107.5	187.9 ± 122.3	0.809

TC (mg/dl)	177.95 ± 41.1	196.1 ± 52.6	0.055	190.9 ± 52.1	180.8 ± 40.7	0.286	191.8 ± 45.7	157.4 ± 45.2	0.006
HDL (mg/dl)	42.1 ± 18.0	38.4 ± 12	0.240	40.1 ± 16.8	40.8 ± 14.5	0.838	39.3 ± 15.5	45.9 ± 15.4	0.111
LDL (mg/dl)	128.2 ± 42.2	122.5 ± 38.8	0.488	127.2 ± 42.2	124.0 ± 39.2	0.693	130.1 ± 42.3	103.7±19.8	0.014
Dyslipidemia	26 (46.4)	24 (53.3)	0.490	27 (51.9)	23 (46.9)	0.617	46 (54.8)	4 (23.5)	0.019

Data are presented as mean ± standard deviation (SD) for continuous variables and as frequencies (percentage) for categorical variables. Statistical significance was assessed using appropriate tests, with P values <0.05 considered significant. SHF: Systolic Heart Failure; DHF: Diastolic Heart Failure; WCHF: Worsening chronic heart failure; EF: Ejection Fraction; Hb: Hemoglobin; TLC: Total Leukocyte Count; S. AST: Serum Aspartate Transaminase; S. ALT: Serum Alanine Transaminase; FBS: Fasting Blood Sugar; PP BS: Postprandial Blood Sugar; TC: Total Cholesterol; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; CKMB: Creatine Kinase MB fraction; TNI: Troponin I; MYO: Myoglobin; BNP: Brain Natriuretic Peptide.

Table 3: Comparison of Blood Gas parameters and Cardiac Biomarkers Across HF Subtypes, Onset Types, and Outcomes

Parameter	SHF n=56	DHF n=45	p-value	*De novo* n=52	WCHF n=49	p-value	Survival n=84	Death n=17	p-value
pH	7.34 ± 0.15	7.36 ± 0.06	0.264	7.35 ± 0.09	7.35 ± 0.08	0.888	7.38 ± 0.05	7.22 ± 0.09	<0.001
pCO_2	50.2 ± 5.8	52.5 ± 8	0.095	51.5 ± 6.8	58.8 ± 7.1	0.598	49.7 ± 6.1	58.5 ± 6.0	<0.001
pO_2	89.1 ± 7.04	90.8 ± 6.2	0.193	89.5 ± 6.5	90.2 ± 6.9	0.636	91.1 ± 6.3	83.6 ± 5.3	<0.001
HCO_3	24.8 ± 1.6	23.9 ± 2.1	0.014	24.3 ± 2.0	24.6 ± 1.9	0.397	24.7 ± 1.7	23.0 ± 2.1	<0.001
CKMB (ng/ml)	5.6 ± 4.9	1.5 ± 1	<0.001	4.1 ± 4.1	3.4 ± 4.4	0.422	3.0 ± 2.8	7.6 ± 7.2	<0.001
TNI (ng/ml)	4.64 ± 9.34	0.26 ± 1.03	0.002	4.0 ± 9.3	1.3 ± 3.9	0.064	2.56 ± 7.68	3.35 ± 5.07	0.686
MYO (ng/ml)	230 ± 197	172.5 ± 168	0.122	278 ± 205	179 ± 162	0.192	195.5 ± 178.4	248.9 ± 221.0	0.283
BNP (ng/ml)	1940 ± 3357	603 ± 420	0.009	1163 ± 2232	1538 ± 2939	0.470	763 ± 864	4216 ± 5254	<0.001

Data are presented as mean ± standard deviation (SD). CKMB: Creatine Kinase–Myocardial Band; TNI: Troponin I; MYO: Myoglobin and BNP: B-type Natriuretic Peptide.

Table 4: Comparison of ECG findings Across HF Subtypes, Onset Types, and Outcomes

Parameter	SHF n=56	DHF n=45	p-value	*De novo* n=52	WCHF n=49	p-value	Survival n=84	Death n=17	p-value
NSR ECG	41 (73.2)	27 (60)	0.159	37 (71.2)	31 (63.3)	0.398	57 (67.9)	11 (64.7)	0.801
Atrial Fibrillation	9 (16.1)	18 (40.0)	0.007	15 (28.8)	12 (24.5)	0.621	23 (27.4)	4 (23.5)	0.743
Atrial Flutter	3 (5.4)	0 (0)	0.115	0 (0)	3 (6.1)	0.07	1 (1.2)	2 (11.8)	0.019
Blockage No block Bifascicular LBBB RBBB	 50 (89.3) 1 (1.8) 5 (8.9)	 37 (82.2) 0 (0) 4 (8.9)	0.115	 45 (86.5) 0 4 (7.7) 3 (5.8)	 42 (85.7) 1 (2) 5 (10.2) 1 (2)	0.546	 71 (84.5) 1 (1.2) 8 (9.5) 4 (4.8)	 16 (94.1) 0 1 (5.9) 0	0.712
ST elevation	12 (21.4)	1 (2.2)	0.004	8 (15.4)	5 (10.2)	0.437	8 (9.5)	5 (29.4)	0.026
ST depression	18 (32.1)	8 (17.8)	0.101	11 (21.2)	15 (30.6)	0.047	21 (25)	5 (29.4)	0.704

QS	24 (42.9)	10 (22.2)	0.029	20 (38.5)	24 (49)	0.287	27 (32.1)	7 (41.2)	0.472
Poor r-wave	9 (16.1)	5 (11.1)	0.473	10 (19.2)	4 (8.2)	0.108	13 (15.5)	1 (5.9)	0.296
T-inversion	9 (16.1)	8 (17.8)	0.820	7 (13.5)	10 (20.4)	0.351	14 (16.7)	3 (17.6)	0.922
LVH	7 (12.5)	20 (44.4)	0.006	16 (30.8)	11 (22.5)	0.468	27 (32.2)	0	0.004

Data is presented as frequency (%). p < 0.05 was considered statistically significant. NSR: normal sinus rhythm LBBB: left bundle branch block; RBBB: Right bundle branch block; LVH: Left ventricular hypertrophy

Table 5: Comparison of structural and functional ECHO findings Across HF Subtypes, Onset Types, and Outcomes

Parameter	**SHF n=56**	**DHF n=45**	**p-value**	***De novo* n=52**	**WCHF n=49**	**p-value**	**Survival n=84**	**Death n=17**	**p-value**
LV Dilated	41 (73.2)	10 (22.2)	<0.001	21 (40.4)	30 (61.2)	0.036	40 (47.6)	11 (64.7)	0.199
LV Not enlarged	11 (19.6)	22 (48.9)	0.002	22 (42.3)	11 (22.4)	0.033	29 (34.5)	4 (23.5)	0.378
CONC LV hypertrophy	9 (16.1)	21 (46.7)	0.001	17 (32.7)	13 (26.5)	0.498	28 (33.3)	2 (11.8)	0.076
LV EDD	60.8 ± 6.6	52.7 ± 8.3	<0.001	55.7 ± 8.0	58.8 ± 8.5	0.061			
LV ESD	48.1 ± 9.8	35.6 ± 6.5	<0.001	40.8 ± 10.9	44.4 ± 9.7	0.079			
RWMA	27 (48.2)	14 (31.1)	0.082	18 (34.6)	23 (46.9)	0.208	33 (39.3)	8 (47.1)	0.552
Global hypokinesia	33 (58.9)	13 (28.9)	0.003	22 (42.3)	24 (49)	0.501	35 (41.7)	11 (64.7)	0.082
PAP	46 (82.1)	19 (42.2)	<0.001	37 (71.2)	28 (57.1)	0.059	52 (61.9)	13 (76.5)	0.253

Data is presented as frequency (%). p < 0.05 was considered statistically significant. LV: Left ventricle; LVEDD: LV end-diastolicLV ESD dimension: LV end-systolic dimension; RWMA: regional wall motion abnormality; PAP: pulmonary artery pressure

Table 6: Comparison of Valvular and Diastolic Function parameters across HF Subtypes, Onset Types, and Outcomes

Parameter	**SHF n=56**	**DHF n=45**	**p-value**	***De novo* n=52**	**WCHF n=49**	**p-value**	**Survival n=84**	**Death n=17**	**p-value**
TR Absent Mild Moderate Severe	 7 (12.5) 29 (51.8) 13 (23.2) 7 (12.5)	 28 (62.2) 8 (17.8) 3 (6.7) 6 (13.3)	<0.001	 17 (32.7) 21 (40.4) 8 (15.4) 6 (11.5)	 18 (36.7) 16 (32.7) 8 (16.3) 7 (14.3)	0.875	 31 (36.9) 32 (38.1) 10 (11.9) 11 (13.1)	 4 (23.5) 5 (29.4) 6 (35.3) 2 (11.8)	0.115
MR Absent Mild Moderate Severe	 6 (10.7) 5 (8.9) 8 (14.3) 37 (66.1)	 37 (82.2) 6 (13.3) 2 (4.4) 0 (0.0)	<0.001	 24 (46.2) 8 (15.4) 0 20 (38.5)	 19 (38.8) 3 (6.1) 10 (20.4) 17 (34.7)	0.005	 40 (47.6) 11 (13.1) 7 (8.3) 26 (31)	 3 (17.6) 0 3 (17.6) 11 (64.7)	0.013
AR Absent Mild Moderate Severe	 39 (69.6) 2 (3.6) 5 (8.9) 10 (17.9)	 34 (75.6) 11 (24.4) 0 (0.0) 0 (0.0)	<0.001	 37 (71.2) 5 (9.6) 4 (7.7) 6 (11.5)	 36 (73.5) 8 (16.3) 1 (2) 4 (8.2)	0.42	 61 (72.6) 10 (11.9) 5 (6) 8 (9.5)	 12 (70.6) 3 (17.6) 0 2 (11.8)	0.693
MS									

Absent Mild Moderate Severe	55 (98.2) 0 (0.0) 1 (1.8) 0 (0.0)	39 (86.7) 6 (13.3) 0 (0.0) 0 (0.0)	0.013	46 (88.5) 5 (9.6) 1 (1.9) 0	48 (98) 1 (2) 0 (0) 0 (0)	0.163	77 (91.7) 6 (7.1) 1 (1.2) 0	17 (100) 0 0 0	0.467
E/A E not A E > A E - A E – A	9 (16.1) 43 (76.8) 4 (7.1) 0 (0.0)	18 (40.0) 2 (4.4) 23 (51.1) 2 (4.4)	<0.001	13 (25.0) 19 (36.5) 17 (32.7) 3 (5.8)	11 (22.4) 26 (53.1) 11 (22.4) 1 (2)	0.327	20 (23.8) 32 (38.1) 28 (33.3) 4 (4.8)	4 (23.5) 13 (76.5) 0 0	0.011

Data are presented as number (percentage); p < 0.05 considered significant. TR: tricuspid regurgitation, Valvular abnormalities including MR: mitral regurgitation; AR: aortic regurgitation, MS: mitral stenosis are categorized by severity (absent, mild, moderate, severe). Doppler parameters include transmitral flow patterns: impaired relaxation (E<A), restrictive filling (E>A), normal (E≈A), and indeterminate flow

DISCUSSION

In this study it was found that diastolic heart failure was present in 44.6% of patients. Numerous epidemiologic studies and national HF registries have defined the prevalence of HFnIEF (DHF) in various HF populations and have documented a prevalence of 50% to 55%.10-12 The observed prevalence of 44.6% in our study aligns with these previously reported figures. Most cases were observed among females (60%) under the age of 60 years and had hypertension.13 Our study findings of gender distribution and associated risk factors in the DHF group are consistent with those reported by Dunlay SM et al.14

The most frequent etiological findings in the systolic and diastolic heart failure were same as found in a study done by Saoji N et al.15 Both group of patients presented with breathlessness showing no statistical difference in occurrence of PND, orthopnea, syncope. These findings were similar to a previous study done by Saoji N et al and Dhanorkar SV et al. 15,16 Though chest pain was noticed significantly higher in the SHF group and this could be attributed to higher number of patients of coronary artery disease in this group. Among the clinical findings high blood pressure and irregular heart rate was predominantly in the DHF group. This can be well explained by higher incidence of patients with hypertension and atrial fibrillation in this group. Hepatomegaly and raised JVP in SHF group indicates higher incidence of decompensation in this group.16 Among the radiological findings cardiomegaly in the SHF group was due to dilated cardiomyopathy. In this study, the left ventricle dimensions were significantly higher in the SHF group, similarly in a study of Stephanie J et al he found that larger ventricular size is associated with higher odds of HF, irrespective of age, LVEF and cardiovascular risk factors.17

Anaemia came out to be a major precipitating factor in causation of acute decompensation as found in a recent meta-analysis that reported 37.2% of heart failure patient to be anemic. Mean CKMB, BNP levels were increased in the SHF group as noticed by Siddiqui SW et al.18

De novo Vs Worsening of Preexisting Heart Failure: In this study 52% of patients presented as de novo whereas 48% presented with worsening of previously compensated symptoms. Symptomatically dyspnea was present in both the groups for shorter duration in the de novo group and longer in worsening group. Vicent, L et al. also reported similar findings in their study.19 Higher blood pressure was observed in the de novo group whereas pedal edema was found more in the worsening group. This is in line with the study conducted by Sokolska JM et al.20 Among laboratory parameters. Potassium was significantly higher in the worsening group. This could probably be due to potassium sparing drugs. There was no difference in the ECG findings except ST depression being more in the worsening group, this was consistent with the findings of Michalsen A et al.21

Factors affecting the short-term outcome of patients in acute heart failure: In the present study significantly higher proportion of patients who died had SHF (p=0.014) whereas in the study of Lang X et al. reported that HFrEF patients, SBP <130mmHg was associated with an increased risk of all-cause and cardiovascular mortality.18 Mortality was noticed more in the elderly age group (p<0.001). Similarly, patients with coronary artery disease and dilated cardiomyopathy had significant association with death. Other factors associated with death were anemia (p=0.004), those who presented with dyspnea more than 24hours, clinically had S3, hyponatremia, acidosis and increased cardiac biomarkers. The overall factors associated with high mortality were consistent with those mentioned in the European Society of Cardiology guidelines 2024.20

The strength of the study lies in its prospective design, detailed clinical, echocardiographic, and biomarker assessments, and the inclusion of patients with valvular heart disease, which reflects real-world practice, especially in regions with a high burden of rheumatic disease. Additionally, the study's comparison of de novo and worsening heart failure adds valuable insights into precipitating factors and clinical presentations.

The limitation of the study is its single-center design, which may restrict the generalizability of the findings. Larger, multicenter studies in different geographical regions are needed to validate these findings and to reduce disparities in diagnosis and management.

CONCLUSION

In this study, DHF constituted 44.6% of acute heart failure cases, with no significant symptomatic differences from SHF. Hypertension, anemia, and atrial fibrillation were common precipitating factors in both groups. Cardiac biomarkers were similarly elevated in DHF and SHF, indicating comparable disease severity, with no significant differences between de novo and worsening cases. Notably, SHF patients experienced poorer short-term mortality outcomes compared to DHF.

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