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OUTCOMES IN HEART FAILURE PATIENTS WITH COMORBIDITIES AT A TERTIARY CARE CENTRE: A PROSPECTIVE OBSERVATIONAL STUDY FROM RURAL TELANGANA

Cardiology

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ABSTRACT

Heart failure (HF) is a leading cause of morbidity and mortality in India, particularly in rural tertiary care centres facing delayed presentations and high comorbidity burden. A prospective observational study enrolled 50 consecutive HF patients at the Department of General Medicine, KIMS, Narketpally, Telangana (January 2024–December 2025). Mean age was 61.9 years; 66% were male. Hypertension and CAD were present in 56% each; smoking in 50%; DM in 44%; CKD in 34%; TB in 16%. HFrEF accounted for 54% of cases. In-hospital mortality was 18% (n=9). Smoking was the strongest mortality predictor: all 9 deaths occurred exclusively among smokers (36% vs 0%; p=0.002). Tuberculosis was significantly associated with higher mortality (OR=7.4; p=0.026). Hypertension showed a paradoxically protective effect (OR=0.16; p=0.032), likely reflecting overlap with guideline-directed medical therapy. Aggressive smoking cessation and TB screening are the highest-priority interventions in rural HF management.

KEYWORDS

Heart Failure; Comorbidities; Smoking; Tuberculosis

INTRODUCTION

Heart failure (HF) is a clinical syndrome of structural or functional cardiac abnormality leading to inadequate cardiac output, with symptoms of dyspnoea, fatigue, and fluid retention. An estimated 64 million people are affected globally[1] and 1.3–4.6 million in India, where patients present at younger ages than Western cohorts.[3] Classification is by ejection fraction: HFrEF (<40%), HFmrEF (40–49%), and HFpEF (≥50%).[2]

In rural India, comorbidities — hypertension, diabetes mellitus (DM), chronic kidney disease (CKD), smoking, and tuberculosis (TB) — interact with HF through neurohormonal, inflammatory, and haemodynamic mechanisms, compounded by delayed diagnosis and limited specialist access. , , , Outcome data from rural Indian tertiary care settings remain scarce. This study evaluated the comorbidity profile and its association with in-hospital mortality at KIMS, Narketpally.

METHODS

A prospective observational cohort study enrolled 50 consecutive HF patients admitted under a single General Medicine Unit at KIMS, Narketpally (January 2024–December 2025). HF was confirmed by Framingham criteria, ECG, chest X-ray, and 2D echocardiography. Adults (≥18 years) with confirmed HF and written consent were included; those <18 years, refusing consent, or with end-stage renal disease were excluded. Demographics, comorbidities, laboratory values, vitals, and echocardiographic data were collected. Follow-up was at 3 and 6 months. Sample size was estimated by Cochran's formula (minimum n=48; enrolled n=50). Fisher's exact test and independent t-test were used (p<0.05 significant). Ethics Committee approval was obtained (IEC, KIMS).

RESULTS

Mean age was 61.9 years; 64% were aged >60 years; 66% were male. HTN and CAD were the most prevalent comorbidities (56% each), followed by DM (44%), smoking (50%), CKD (34%), and TB (16%). HFrEF predominated (54%, mean EF 29.6%). In-hospital mortality was 18% (n=9); 28 patients improved (56%) and 13 remained morbid at discharge. A history of past HF hospitalisation was present in 23 patients (46%). Improvement declined from 56% at discharge to 42% at 6 months, with 8 additional post-discharge deaths (total 6-month mortality 34%). Baseline clinical and laboratory parameters stratified by in-hospital survival status are presented in Table 3.

TABLE – 1 : DEMOGRAPHIC AND COMORBIDITY PROFILE (n=50

Parameter	n	%
Male	33	66.0
Age >60 years	32	64.0
Hypertension	28	56.0
Coronary Artery Disease (CAD)	28	56.0
Diabetes Mellitus	22	44.0
Smoking (active/past)	25	50.0
Chronic Kidney Disease (CKD)	17	34.0
Tuberculosis	8	16.0
Past Admission (prior HF hospitalisation)	23	46.0
Anaemia (Hb <11 g/dL)	25	50.0
HFrEF (EF <40%) / HFpEF (EF ≥50%)	**27 / 23**	**54 / 46**

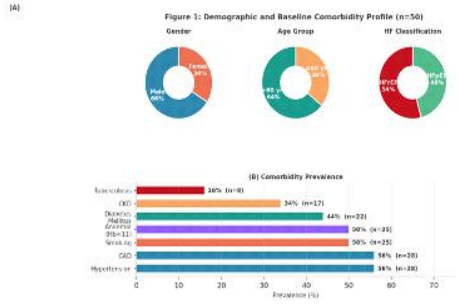

Figure 1: Demographic and baseline comorbidity profile (n=50). (A) Distribution of gender, age group (>60 vs ≤60 years), and HF classification (HFrEF 54% vs HFpEF 46%). (B) Prevalence of individual comorbidities among enrolled patients; Hypertension and CAD were most prevalent (56% each).

TABLE – 2 : ASSOCIATION OF COMORBIDITIES WITH IN-HOSPITAL MORTALITY

Variable	Deaths if Present	Deaths if Absent	OR	p value
Smoking ★	9/25 (36%)	0/25 (0%)	∞	**0.002***
Tuberculosis	4/8 (50%)	5/42 (12%)	7.40	**0.026***
Hypertension (protective)	2/28 (7%)	7/22 (32%)	0.16	**0.032***
CAD	6/28 (21%)	3/22 (14%)	1.73	0.713 NS
Diabetes Mellitus	3/22 (14%)	6/28 (21%)	0.81	0.713 NS
CKD	2/17 (12%)	7/33 (21%)	0.50	0.699 NS
Past Admission†	7/23 (30%)	2/27 (7%)	5.47	*0.062*

★ All 9 deaths occurred exclusively among smokers; the OR is undefined (∞) due to a zero-cell in the survivor arm — calculated by Fisher's exact test, not logistic regression, which would fail under complete separation. * p<0.05. NS = not significant. HTN was paradoxically protective, reflecting overlap of antihypertensives with

GDMT. † Past Admission = prior hospitalisation for heart failure; borderline significance.

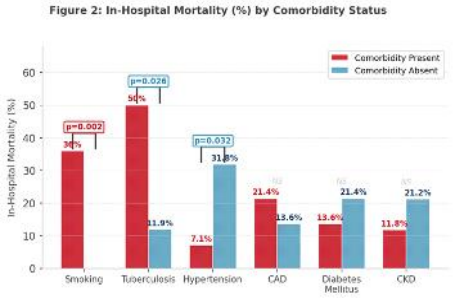

Figure 2: In-hospital mortality (%) stratified by comorbidity presence vs absence. Statistically significant associations annotated with p-values (smoking p=0.002; TB p=0.026; hypertension protective p=0.032). Non-significant associations marked NS. Smoking showed complete separation (OR=∞; 0% mortality in non-smokers).

TABLE – 3 : BASELINE CLINICAL AND LABORATORY PARAMETERS BY IN-HOSPITAL SURVIVAL STATUS (n=50)

Parameter	Survived (n=41)	Died (n=9)	p-value
Age (years)	60.9 ± 13.3	66.7 ± 12.5	0.237
Admission SBP (mmHg)	122.7 ± 26.3	130.0 ± 21.8	0.441
Admission DBP (mmHg)	73.2 ± 15.7	80.0 ± 15.8	0.244
Pulse Rate (bpm)	79.4 ± 10.6	76.7 ± 9.8	0.481
Haemoglobin (g/dL)	10.5 ± 2.6	11.1 ± 2.6	0.534
Serum Urea (mg/dL)	61.8 ± 44.4	45.6 ± 33.2	0.307
Creatinine (mg/dL)	2.1 ± 2.1	1.6 ± 2.1	0.472
Random Plasma Glucose (mg/dL)	161.1 ± 40.8	169.2 ± 47.3	0.600
Ejection Fraction (%)	42.4 ± 15.2	37.0 ± 16.8	0.344

All values are mean ± SD; p-values by independent t-test. No individual parameter showed significant difference between survival groups (all p>0.05), confirming that comorbidity burden (Table 2) was the primary in-hospital mortality determinant rather than admission vitals. SBP = systolic blood pressure; DBP = diastolic blood pressure; EF = ejection fraction.

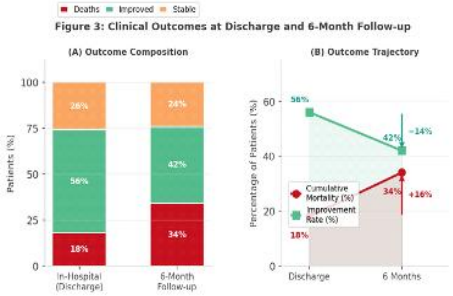

Figure 3: Clinical outcome trajectory across follow-up. (A) Stacked proportions of outcome categories (deaths, improved, stable) at in-hospital discharge and 6-month follow-up. (B) Cumulative mortality increased from 18% to 34% (absolute rise of 16 percentage points) while the improvement rate declined from 56% to 42% (absolute decrease of 14 percentage points) over the follow-up period.

DISCUSSION

The mean age (61.9 years) and male predominance (66%) are consistent with Indian HF registry data.[3] HTN and CAD were equally prevalent (56%), reflecting the cardiometabolic burden of rural India. The most clinically striking finding was the complete association between smoking and in-hospital mortality: all 9 deaths occurred exclusively among the 25 smokers (36% vs 0%; p=0.002). While this complete separation must be interpreted cautiously in a small cohort — where a single clustering effect can produce zero-cell outcomes — the mechanistic evidence for smoking as a driver of HF decompensation is robust: smoking accelerates atherosclerosis, impairs endothelial function, and promotes neurohormonal activation, synergising to worsen cardiac outcomes.[8] The high tobacco burden in this rural Telangana cohort (50%) further reflects the compounding effect of early cardiovascular disease onset and delayed care-seeking in this population. These findings are consistent with meta-analyses demonstrating that active smoking independently increases HF-related hospitalisation and all-cause mortality.[8]

Tuberculosis was associated with significantly higher in-hospital mortality odds (OR=7.4; p=0.026), corresponding to a 4.2-fold higher relative risk (50% vs 11.9%). Cardiac TB — pericardial effusion, constrictive pericarditis, and myocarditis — directly contributes to HF decompensation, compounded by malnutrition and delayed access to care.[9] This supports routine TB screening in all HF patients in high-prevalence rural settings.

Hypertension was paradoxically protective (OR=0.16; p=0.032), consistent with 'reverse epidemiology' documented in large acute decompensated HF registries.[11] In registry data, this phenomenon is partly attributed to higher admission systolic blood pressure reflecting preserved myocardial reserve; however, in our cohort, admission SBP did not differ significantly between survivors and non-survivors (122.7 vs 130.0 mmHg; p=0.441), and in fact trended higher in the deceased group, eliminating acute haemodynamics as the driver in this sample. The protective effect is therefore predominantly pharmacological: hypertensive patients are disproportionately established on ACE inhibitors, ARBs, and beta-blockers prior to admission, conferring a 'legacy' survival advantage via guideline-directed medical therapy.[10] Baseline clinical and laboratory parameters stratified by survival status are detailed in Table 3; no individual parameter showed a significant difference (all p>0.05). DM, CKD, and CAD did not reach significance, most likely due to Type II error from a small sample; these remain established predictors of acute in-hospital mortality[11] and 1-year outcomes[12] and require aggressive management regardless. HFrEF showed a 3.0-fold higher mortality than HFpEF (25.9% vs 8.7%; OR=3.67; p=0.152), consistent with Indian registry data and international registry findings demonstrating higher short-term mortality in reduced-EF cohorts, with borderline significance likely reflecting the small sample size.[3,12,13]

LIMITATIONS

This study has several important limitations. First, the small sample size (n=50) results in wide confidence intervals and reduced statistical power, making Type II errors plausible for established predictors such as diabetes mellitus and chronic kidney disease. Second, the 24-month enrollment period with 50 patients at a single department reflects the strict consecutive sampling protocol and rigorous inclusion criteria applied; this also limits generalisability. Third, because all 9 in-hospital deaths occurred exclusively among smokers, complete separation of the smoking variable precludes standard unconditional logistic regression for multivariate analysis; Firth's penalised likelihood regression was not performed and remains a priority for a larger follow-up study. Fourth, while 3- and 6-month post-discharge outcomes are reported descriptively, a formal Kaplan–Meier survival analysis was not conducted; this should be incorporated in future work. Fifth, detailed pre-admission medication histories — specifically prior adherence to beta-blockers, ACEi/ARBs, and SGLT2 inhibitors — were not systematically tabulated, limiting our ability to definitively quantify the pharmacological contribution to the observed hypertension paradox. Lastly, Framingham criteria, while widely used in resource-limited Indian settings, are considered clinically supplementary to modern echocardiographic classification.

CONCLUSION

Smoking and tuberculosis are the dominant statistically significant predictors of in-hospital HF mortality in this rural Indian cohort. The paradoxical protective effect of hypertension reflects guideline-directed therapy in hypertensive patients. Aggressive smoking cessation counselling and TB screening are the highest-priority interventions for rural HF management. Larger multicentre prospective studies with extended follow-up and standardised biomarker assessment are warranted to further characterise the comorbidity–outcome relationship.

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