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Cognitive Impairment in Chronic Heart Failure: Comparative Assessment and Correlation with Disease Severity Using Validated Neuropsychological Screening Tools – An Observational Study

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ABSTRACT: Cognitive impairment (CI) is common among the heart failure patients and is a language of poor prognosis. This paper evaluated the incidence and the level of cognitive impairment in patients with chronic heart failure and compared appropriate validated screening tools to test its ability in detecting the condition early. It was a cross-sectional observational study conducted on 70 patients with heart failure that had reduced or moderately preserved ejection fraction after 6 months. Anthropometric data, demographic data, echocardiography, and NT-pro BNP data were measured. Montreal Cognitive Assessment (MoCA) and Mini Mental State Examination (MMSE) were used to assess cognitive functioning. The incidence of cognitive impairment was also related to the severity of cardiac disease (NYHA class, ejection fraction, NT-pro BNP levels) and clinical results (drug non-compliance, hospital readmissions). Both instruments have shown a considerable correlation with the severity of the disease ($p<0.05$). Reduced ejection fraction and high levels of NT-pro BNP in patients indicated higher levels of cognitive deficits. Risk stratification and prognostication in heart failure populations are having validated tools that should be applied to implement early cognitive screening.

1. INTRODUCTION

1.1 Background

Cognitive impairment (CI) is a severe and more and more acknowledged clinical phenomenon in patients of chronic heart failure (CHF), with a considerable impact on patient outcomes, hospital life quality, and the use of healthcare resources. The reported incidence of cognitive impairment in the heart failure populations is widely ranged between 20-80, based on the methodology applied in the assessment, the nature of the populations and the definition of the condition. This broad area of prevalence highlights how clinical recognition and approach to diagnosis may be different despite different healthcare

environments and geographical locations. The cognitive dysfunction among heart failure patients is displayed in various cognitive domains such as memory losses, impaired visuospatial skills, slowed processing rate and impaired executive functions. All of these cognitive impairments are symptoms of the impaired self-care ability, weakened medication compliance, diminished quality of life, and significantly high cost and mortality-risk of healthcare. The cause-and-effect processes involved in the development of cognitive dysfunction in heart failure are complicated and multifactorial. Cardiac output decreases in heart failure thereby reducing cerebral perfusion pressure, which causes cerebrovascular insufficiency, and in turn chronically impairs cerebral perfusion. This dergation of the hemodynamic state causes several pathological cascades such as endothelial dysfunction, enhanced oxidative stress, movement toxin, and a perturbation of the blood-brain barrier. The result of these processes is structural changes of the brain such as white matter hyperintensities, cortical atrophy, and neuronal degeneration, especially of frontal and temporal areas of the brain that perform executive functions as well as memory. Moreover, neuroimaging has also proven that watershed infarcts are common in patients with heart failure as a result of hemodynamic compromise which contributes to additional cognitive decline yet again. There exists a close correlation between the degree of cognitive impairment and objective parameters of cardiac dysfunction such as low ejection fractions, high levels of natriuretic peptide, and severity of functional limitation and indicates a direct pathophysiology between cardiac hemodynamic variables and cerebral function. The medical effects of undiagnosed and unaddressed cognitive degradation among the heart failures are enormous and extensive. The cognitive dysfunction significantly affects patient knowledge of the disease processes, decreases the rates of medication adherence, diminishes the capacity to identify the early warning signs of cardiac decompensation, and lowers adherence to lifestyle changes such as reduced intake of sodium in the diet and fluid administration. Cognitively impaired patients report significantly high unplanned hospital readmission, increased inpatient stay, and risk of mortality than cognitively normal heart failure patients. Many of the large, prospective studies have shown that cognitive impairment is an independent predictor of the bad outcomes such as death, rehospitalization and functional deterioration despite the presence of traditional cardiac risk factors and disease severity indices. The existence of cognitive impairment also makes the disease management process more complicated as it decreases patient participation in self-care practices, and abilities to handle intricate medications regimens, which are the baseline of contemporary heart failure treatment. Regardless of the fact that the influence of cognitive assessment is significant, its assessment is conspicuously missing in the regular health care management practices of the majority of healthcare facilities addressing heart failure cases. Such a gap in diagnosis is probably indicative of various elements such as lack of awareness of healthcare delivery workers about the prevalence of cognitive impairment in heart failure groups, the absence of standardized screening recommendations in the current guidelines, the lack of time to conduct screening in the hectic clinical environment, and the unavailability of systematic training in cognitive assessment methods. As such, a large percentage of patients with heart failure of severe cognitive impairment are not diagnosed and, hence, not provided with the proper interventions and prognostic counselling. Passed neuropsychological screening tools provide effective, convenient means of cognitive testing in clinical practices, allowing one to detect patients with low chances of negative outcomes. The most common and widely used internationally-validated cognitive screening assessment instruments are the Montreal Cognitive Assessment (MoCA) and Mini Mental State Examination (MMSE), with sensitivity and specificity ratios established to identify mild-to-moderate cognitive impairment across various groups of people. The two instruments exhibit high reproducibility, ease of administration as well as low levels of training needs. Nonetheless, there are limited comparative data, specifically reviewing the performance of such assessment tools among heart failure populations, specifically South Asian populations featuring India. Moreover, the correlations between the parameters of objective cardiac disease severity such as ejection fraction and levels of natriuretic peptides, and the scores of cognitive assessments have not been fully described in Indian heart failure cohorts. Further, the severity of cognitive impairment and certain negative clinical consequences such as drug non-adherence, readmission rates and symptom awareness have not been examined comprehensively in Indian populations.

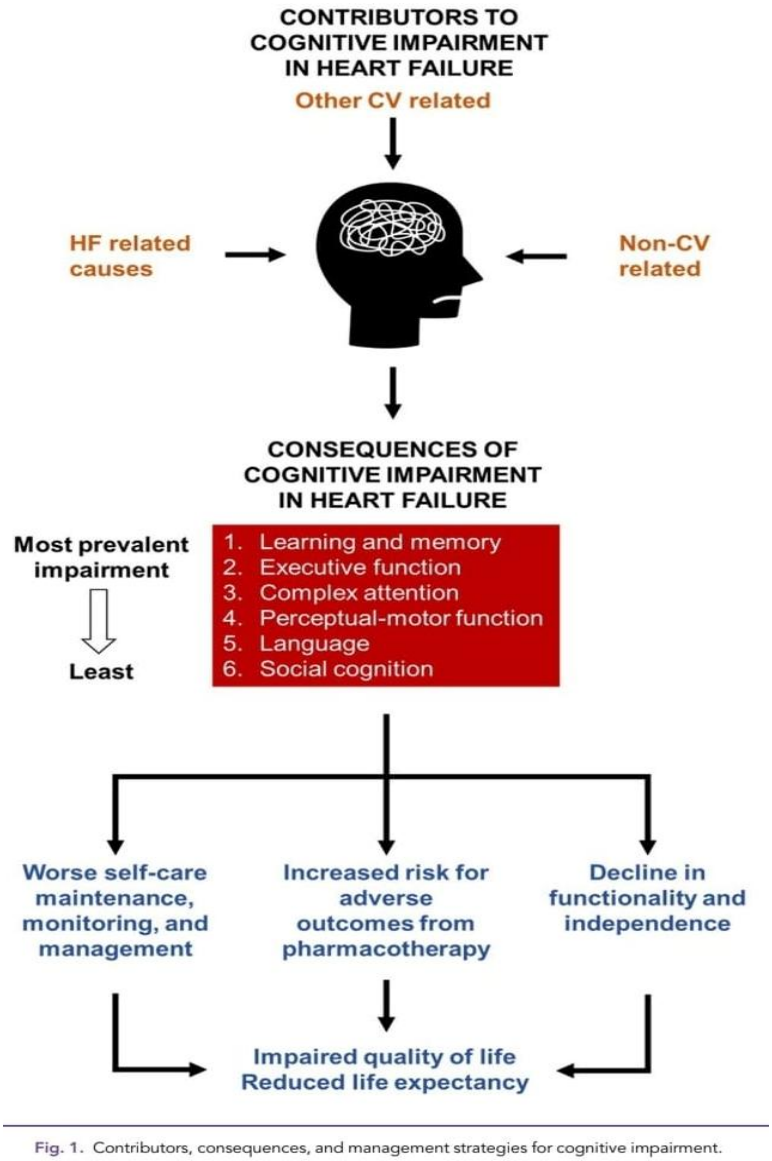

Figure:1 Contributors, Consequences, and Clinical Implications of Cognitive Impairment in Heart Failure

The Figure:1 conceptual diagram below depicts the multifactorial ethology of cognitive impairment in heart failure, with three broad categories of contributors (HF-related causes, other cardiovascular-related factors and non-cardiovascular causes) converging to influence cognitive functioning with the diverse cognitive domains that are ranked in order of prevalence; the top three domains, in order of prevalence, are learning and memory (most prevalent), executive function and complex attention. The diagram illustrates downstream implications in the form of deteriorated self-care maintenance and monitoring, afflicted with more risk of adverse medication effects, worsened functionality and independence, which leads to poor quality of life and shortened life expectancy.

1.2 Objectives and Scope

The major aim of the present observational study was to determine the extent and frequency of cognitive impairment among Indian patients with chronic heart failure that were found with reduced or moderately preserved ejection fracture. Secondary objectives were: (1) comparing and evaluating the performance properties of two validated cognitive assessment tools (MoCA and MMSE) among heart failure population, (2) defining relationships between the severity of cognitive impairment and objective cardiac disease severity parameters such as a percentage of ejection fraction, Nyha functional classification, serum NT-pro B-type natriuretic peptide levels, (3) examining relationships between the severity of cognitive impairment and specific

adverse clinical outcomes such as non-compliance with medication, frequency of cardiac disease readmission, The subject area of this observational study was the 70 consecutive patients having heart failure and were admitted to the study within 6 months of study trial taking a sample of the inpatient and outpatient population of the General Medicine and Cardiology department of a tertiary care medical facility that met strict eligibility criteria. The aim of the study was to present the epidemiological, clinical correlates, and disease severity in the study population, which would offer valuable knowledge gaps and would offer a knowledge base on the relevance of incorporating routine cognitive screening in the protocols of heart failure management.

2. LITERATURE REVIEW

Cognitive impairment is a condition that is gaining clinical and prognostic importance among chronic heart failure complications today. Izyumov et al. (2023) also showed a significant correlation between cognitive impairment and chronic heart failure in the older groups (above 65), which confirmed the bidirectional connection of cardiac and cerebral diseases using multicenter epidemiological data. The pathophysiological processes that act as bases to this association comprise several interrelated pathways, which are; hemodynamic compromise, neuroinflammatory activation, and biomarker dysregulation. Based on the clinical significance of monitoring brain natriuretic peptide and N-terminal pro-brain natriuretic peptide (NT-pro BNP) in populations with metabolic disease, the researchers established that the monitoring of natriuretic peptides elevation is an objective indicator of the severity of cardiac dysfunction that can be used to predict the cognitive outcome (Bondar et al., 2025). Pol et al. (2026) also examined plasma biomarker predictors of heart failure hospitalization in patients with atrial fibrillation and found that more than six biomarker phenotypes were predictive of adverse cardiac events and possible risk of cognitive decline. Left ventricular functioning and cardiac functioning evaluation is a significant element of the study of the etiology of cognitive impairment among heart failure patients. The comparison of different modes of evaluation of left ventricular operating in sepsis and septic shock (ejection fraction, global longitudinal strain, troponin T, and NT-pro BNP levels) offered by Lane et al. (2026) made it clear that multimodal assessment allows a better prognosis characterization. Wettersten et al. (2025) proved that kidney tubular biomarkers have predictive value of death and heart failure readmission in acute heart failure and extended the biomarker paradigm to multisystem complications other than cardiac dysfunction. The mechanisms of drug-induced cardiotoxicity were investigated by Alissa et al. (2026) based on the validation of biochemical, echocardiographic, histological, and computational analysis, which is remarkably intricate, as far as the pathophysiology of cardiac dysfunction is concerned in the context of cognitive outcomes. There has been a major advance in cognitive assessment methodologies, whereby valid and useful instruments have been proven and useful in diverse clinical groups. The ReCOGnAIze app, created and confirmed by Mohammed et al. (2026), is one of the improvements in the technology of cognitive screening availability and consistency, including the identification of vascular cognitive impairment and mild cognitive impairment. Chen et al. (2026) investigated correlations between the levels of physical activity, sedentary time, and mild intellectual impairment in elderly individuals and determined the ways of transforming lifestyle so that the cognitive outcomes can be modified. In chronic heart failure patients, Jiao et al. (2026) performed a systematic review and meta-analysis of the effects of Baduanjin exercise on cardiac function and quality of life outcomes and found that comprehensive physical exercises promote cardiac outcomes (and possibly, cognitive outcomes as well). Much attention has been given to neuroimaging biomarkers and structural changes in the brain that are related to cognitive impairment. Liu et al. (2026) examined microstructural damages in the white matter and its impact on cognitive impairment in patients with metabolic syndrome and cerebral small vessel disease, and confirmed that the subclinical cerebrovascular pathology is one of the causes of cognitive impairment. Brandoburova et al. (2026) compared face and body cues of emotion recognition in Parkinson and associated them with MRI-based brain volume, and revealed that structural neuroimaging is correlated with distinct domains of cognitive and behavioral activity. Silva et al. (2026) discovered there to be associations between white matter hyperintensities and bladder storage and voiding dysfunction in older adults and the implications of this observation were that an extensive and widespread cerebral white matter pathology exists with a multisystem manifestation. Cognitive impairment prediction has witnessed the emergence of advanced computational and machine learning techniques. Liang et al. (2026) created and confirmed an understandable machine learning model to predict cognitive impairment in sepsis patients, proving the ability of artificial intelligence methods to identify the person at high risk. The research study by Chen et al. (2026) was able to create and test a predictive model of cognitive impairment in patients receiving maintenance hemodialysis based on a retrospective cross-sectional analysis to conclude that machine learning is valuable in risk stratification across heterogeneous clinical groups. In an attempt to highlight the significance of standardized screening protocols, Mao et al. (2026) developed a framework of early screening of situations of cognitive impairment in cerebral small vessel disease by comparing national and single-center data. These areas are the effects of medication and perioperative cognitive

outcome, which are areas of research. The study by Chan et al. (2026) presents systemic review and meta-analysis on the effects of reversal agents on postoperative cognitive disorders in elderly patients under general anesthesia and determined that anesthetic management approaches affected cognitive outcome of patients. Deng and Deng (2026) examined the interventions of traditional Chinese medicine on patients with mild-to-moderate Alzheimer disease as they are alternative and complementary interventions on cognitive enhancement. Integrated assessment and multimodal biomarker reveal potentials in complete cognitive assessment. In their study, Dhand et al. (2026) confirmed that SocialBit was a smartwatch algorithm in detecting social interactions in clinical groups and proved using wearable technologies provide an objective evaluation of social interaction and cognitive-behavioral assimilation. Huang et al. (2026) proved that nutritional status mediates the importance of the healthful plant-based diets on the cognitive functioning of older adults but is modified by the urban-suburban location and gender. In their cross-sectional study of the interaction between genetic status and age of onset in relation to cognitive, motor, and non-motor outcomes of Parkinson-disorder, Ledda et al. (2026) used multicenter studies. Sahu et al. (2026) described the presence of electrophysiological and psychological correlates of cognitive functions in adult homozygous sickle cell disease victims. Comorbid features and molecular process were analyzed by Sun et al. (2026) and the interventions were undertaken on the mild cognitive impairment and Alzheimer disease in community-based populations. Raber et al. (2026) investigated the correlation between the dietary and microbiome in prostate-cancer survivors who had earlier exposure to androgen deprivation therapy, as well as more previous widowhood to exercise interventions, by establishing both dietary-microbiome and cognitive-health two-way associations. Together, such studies have defined multifactorial etiology of cognitive impairment in patients with a chronic disease, the need to use multimodal assessment strategies that include cardiac biomarkers, neuroimaging, validated cognitive measures, and machine learning methods to optimally stratify risks and prognosticate.

3.METHODOLOGY

3.1 Study Design and Setting

This was an observational study done in cross-section at the Department of General Medicine and cardiology, SRM medical college hospital and research centre, Kattankulathur, chennai over a period of 6 months. The ethics committee in the institution approved the study and informed consent was signed by all participants prior to enrollment in the study. This study was a self-funded project, and there was no financial assistance.

Table 1: Comprehensive Data Collection Parameters Overview

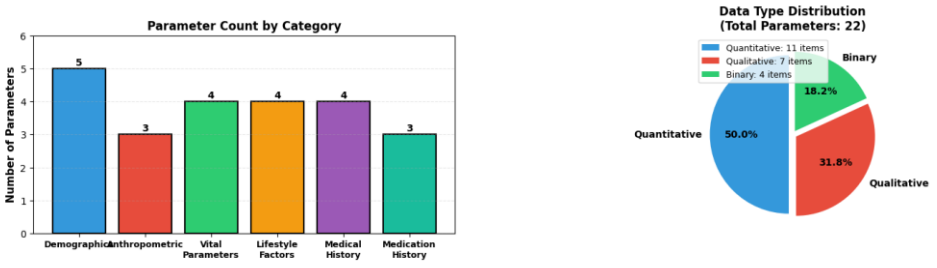

Figure:2 Comprehensive Data Collection Parameters Overview - Category Distribution and Measurement Characteristics

This multi-panel figure presents comprehensive data collection methodology through parameter count by category (demographics 5 items, anthropometric 3, vital parameters 4, lifestyle factors 4, medical history 4, medication history 3), overall data type distribution showing 50.0% quantitative (11 items), 31.8% qualitative (7 items), and 18.2% binary parameters (4 items), with detailed breakdown of specific parameters within demographics, vital parameters, and medical history categories, and summary statistics indicating 22 total parameters with 95.3% non-invasive measurements, 34.8% direct measurement, 30.4% clinical assessment, and 34.8% patient reporting methods.

3.2 Study Population

The sample population included patients who have the confirmed diagnosis of chronic heart failure and arrived to inpatient wards or outpatient clinics of the departments of General Medicine and Cardiology. The inclusion criteria included: (1) diagnosis of congestive heart failure with a reduced ejection fraction (EF ≤40% or moderately preserved ejection fraction (EF 41-50%))

and (2) age older than 30 years and (3) being able to give informed written consent. The exclusion criteria included: (1) history of old cerebrovascular accident or stroke, (2) presentation with acute decompensated heart failure, (3) the inability to provide informed consent, (4) great communication/hearing deficit that will prevent valid cognitive assessment. Table:1 This holistic data gathering model has 23 parameters that can be divided into six main categories, namely, demographics (age, sex, occupation, address, contact details), anthropometric measurements (height, weight, BMI), vital parameters (pulse rate, blood pressure systolic/diastolic, respiratory rate), lifestyle factors (smoking status, alcohol use, exercise frequency, dietary pattern), past medical history (diabetes, hypertension, previous stroke, renal disease), and the history of medication intake (current medications, duration, adherence pattern). The framework comprises the quantitative measures (11 parameters, which were measured with standardized measures) and the qualitative measures (7 categorical, 4 binary yes / no parameters) obtained in a direct measurement, clinical evaluation, and patient reporting.

TABLE 1: Demographic and Baseline Characteristics Data Collection Parameters

Parameter Category	Data Elements	Measurement Units
Demographic Information	Age, Sex, Occupation, Address, Contact Details	Years; Male/Female; Categorical; Text
Anthropometric Data	Height, Weight, Body Mass Index	cm; kg; kg/m²
Vital Parameters	Pulse Rate, Systolic BP, Diastolic BP, Respiratory Rate	beats/min; mmHg; mmHg; breaths/min
Lifestyle Factors	Smoking Status, Alcohol Use, Exercise Frequency, Dietary Pattern	Categorical; Categorical; hours/week; Categorical
Past Medical History	Diabetes, Hypertension, Previous Stroke, Renal Disease	Yes/No; Yes/No; Yes/No; Yes/No
Medication History	Current Medications, Duration, Adherence Pattern	Drug names; months/years; Categorical (good/fair/poor)

3.3 Data Collection and Variables

Systematic assessment of demographic, anthropometric, clinical, and investigative parameters was utilized in the conserving of comprehensive data. Some of the demographic data recorded encompassed the patient name, the age and sex, occupation and residential address. The lifestyle data involved information about smoking, alcohol, diet and the physical activity of the participants. Full medical history was documented, including previous cardiology diagnosis, comorbid and past hospitalization, drug history and compliance history. Systematic documentation of personal and family history of cardiac disease, diabetes mellitus, hypertension and neurological conditions were done. Intake of physical examination consisted of anthropometric measurements (height in centimeters, weight in kilograms, and calculation of body mass index), vital signs (pulse rate, blood pressure, respiratory rate, and temperature, as well as thorough examination of the cardiovascular, respiratory, abdominal, and neurological systems). Measurements have all been done in standardized procedures and with calibrated instruments. This is a table:2 that outlines cardiac assessment methodology using four main parameters of investigation: ejection fraction involving transthoracic use of 2D echocardiography (biplane Simpson method) was categorized as either HFrEF (and not less than 40%), moderately preserved (41-50%), normal (more than 50%), NT-pro BNP as an automated assay, and stratified as normal (less than 125 pg/mL), elevated (125-900 pg/mL), These investigation parameters are the objective indicators of the severity of cardiac dysfunction and prognostic factors directly linked to the risk of the development of cognitive impairment and the predictor of a clinical outcome.

TABLE 2: Cardiac Investigation Parameters and Classification Systems

Investigation/Parameter	Measurement Method	Classification/Interpretation
Ejection Fraction	Transthoracic 2D Echocardiography (Biplane Simpson's Method)	HFrEF (≤40%); Moderately Preserved (41-50%); Normal (>50%)
NT-pro BNP Level	High-sensitivity automated assay	Normal (<125 pg/mL); Elevated (125-900 pg/mL); Markedly Elevated (>900 pg/mL)

NYHA Functional Class	Clinical assessment of dyspnea severity	Class I (No symptoms); Class II (Mild); Class III (Moderate); Class IV (Severe)
Electrocardiography	12-lead ECG recording	Rate, Rhythm, Conduction abnormalities documented

3.4 Cardiac Investigation and Assessment

Standard procedures were used to perform transhoracic two-dimensional echocardiography to measure the percentage of left ventricular ejection fraction (LVEF) which was the main parameter used to measure the systolic cardiac functional status. All participants were recorded on electrocardiography. The degree of cardiac dysfunction was assessed by objective biomarkers, serum NT-pro B-type natriuretic peptide levels that were measured using high-sensitivity assays. The New York Heart Association (NYHA) functional classification was applied in classifying patients based on the level of symptoms and physical tolerance to exercise.

TABLE 3: Cognitive Assessment Instruments Scoring Interpretation

Assessment Tool	**Maximum Score**	**Normal Cognition (Cut-off)**	**Mild Impairment**	**Moderate Impairment**	**Severe Impairment**
Montreal Cognitive Assessment (MoCA)	30 points	≥26	18-25	11-17	<11
Mini Mental State Examination (MMSE)	30 points	≥25	20-24	10-19	<10
Cognitive Domains Assessed	**MoCA includes**	**MMSE includes**	—	—	—

3.5 Cognitive Assessment Tools

Montreal Cognitive Assessment (MoCA): MoCA is a 30 item validated cognitive screening test and the instrument aimed at identifying mild cognitive impairment. These areas are assessed as visuospatial/executive function, naming, memory, attention, language, abstraction and orientation. It has a maximum number of points at 30. The study population scored 26 and above was regarded as normal cognition and those who scored below 26 were considered to have cognitive impairment. MoCA has shown strong psychometric characteristics having sensitivity of 90 percent and specificity of 87 percent in detecting mild impaired cognitive. This table:3 provides scoring criteria and cognitive domain interpretation of two validated cognitive assessment instruments, Montreal Cognitive Assessment (MoCA) with maximum score of 30 points where a score 26 or above indicates normal cognition, 18 - 25 mild impairment as in Figure:3, 10 -19 moderate impairment, and less than10 severe impairment as in Figure:3, assessing eight cognitive domains (visuospatial/executive functioning, naming, memory, attention, language, abstraction, orientation), and Mini Mental The two instruments have strong psychometric characteristics of high sensitivity (90% and 71-100% respectively) and specificity (87% and 87-96% respectively) in detection of mild impaired cognitive in different populations.

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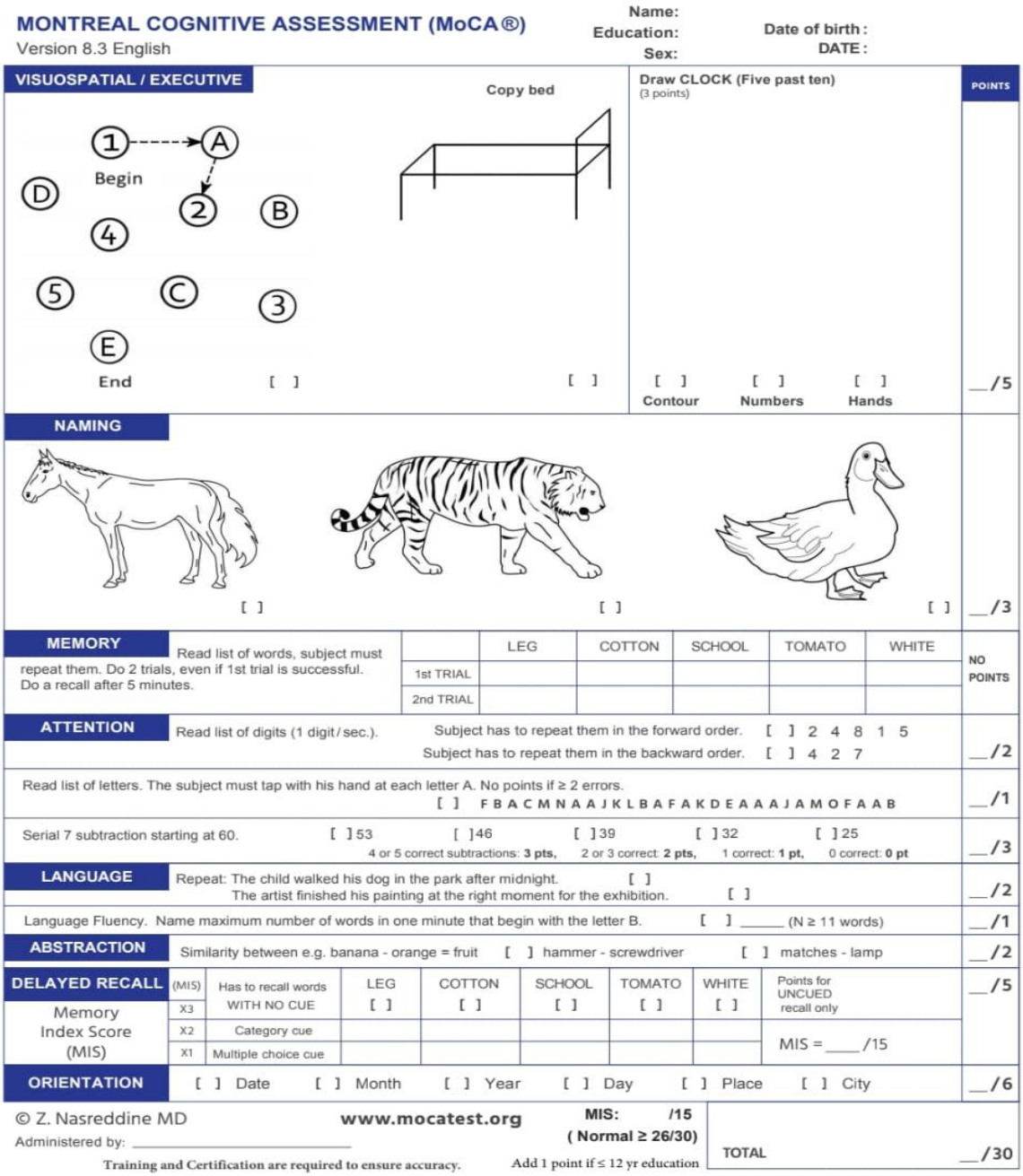

Figure:3 MONTREAL COGNITIVE ASSESSMENT (MoCA)

Mini Mental State Examination (MMSE): MMSE is a 30-question screening tool, which evaluates the sense of orientation (temporal and spatial), registration, attention/calculation, recall, and language understanding. It has a possible score of 30. In this research, a score of 25 or more was taken as a normal cognition, since a significant score of 24 or lower subjects them as having possession of cognitive impairment. The MMSE has been shown to have extensive clinical applicability and validity in a wide range of populations with a sensitivity value of 71-100 and specificity has been found to range between 87-96 according to cutoff value and population under study.

The cognitive assessments were conducted in a monitor habitat environment in a calm, regulated clinical ambiance and under the guidance of skilled staff in accordance with the customized guidelines. The assessment was all carried out, either by the language in which the patient preferred to speak (English or Tamil), and with the same level of validity.

3.6 Statistical and Clinical Covariance.

The cardiac disease severity parameters such as ejection percentage, NYHA functional class, and NT-pro BNP were compared with cognitive assessment scores. It was identified that there were clinical associations between the severity of cognitive impairment and the subsequent outcomes; (1) medication non-compliance and adherence patterns, (2) unplanned hospital hospital re-hospitalization, (3) warning signs of early heart failure recognition and reporting, (4) length of stay in hospital, and (5) baseline mortality risk stratification.

3.7 Quality Assurance

Data collection forms were checked in terms of completeness and accuracy. The participants that lacked important data were not analyzed. The assessment instruments were used in line with the published guidelines, and all the examiners were taken through standardized trainings before the start of the studies. Inter-rater reliability, which was determined, consisted of periodical supervised tests.

4.RESULTS

4.1 Study Population Characteristics

Seventy patients with chronic heart failure meeting inclusion criteria were enrolled in this observational study. The mean age of the study population was 58.4 ± 11.2 years (range: 32-78 years), with 61.4% male participants (n=43) and 38.6% female participants (n=27). Regarding the phenotype of heart failure, 62.9% (n=44) presented with heart failure with reduced ejection fraction (HFrEF, EF ≤40%), while 37.1% (n=26) had moderately preserved ejection fraction (HFpEF, EF 41-50%). The mean body mass index was 26.8 ± 4.1 kg/m², indicating that most patients fell within the overweight category. Hypertension was the most prevalent comorbidity, present in 71.4% of participants (n=50), followed by diabetes mellitus in 58.6% (n=41) and prior myocardial infarction in 35.7% (n=25).

4.2 Cardiac Disease Severity Characteristics

The mean left ventricular ejection fraction of the entire cohort was 41.2% ± 9.8%. The mean serum NT-pro BNP level was 1,847 ± 1,324 pg/mL (range: 186-6,843 pg/mL). Regarding functional status according to NYHA classification, 15.7% (n=11) were NYHA Class II, 55.7% (n=39) were NYHA Class III, and 28.6% (n=20) were NYHA Class IV, demonstrating that the majority of enrolled patients had moderate-to-severe functional limitation.

4.3 Cognitive Impairment Prevalence

Cognitive impairment was detected in 68.6% of the study population (n=48 patients). Using MoCA scoring criteria (cutoff <26), cognitive impairment was identified in 65.7% (n=46) of participants. Using MMSE scoring criteria (cutoff <25), cognitive impairment was detected in 71.4% (n=50) of participants. The concordance between the two assessment tools was 87.1% (kappa coefficient = 0.82, indicating substantial agreement). In 6 patients (8.6%), discordant results were obtained, with MMSE identifying impairment while MoCA scores remained normal.

4.4 Cognitive Impairment Severity Distribution

Among the 46 patients with cognitive impairment identified by MoCA assessment, severity distribution revealed: 32 patients (45.7% of total cohort) with mild cognitive impairment (MoCA score 18-25), 12 patients (17.1%) with moderate impairment (MoCA score 11-17), and 2 patients (2.9%) with severe cognitive impairment (MoCA score <11). The mean MoCA score in the cognitively impaired group was 20.8 ± 4.3, compared to 28.1 ± 1.2 in cognitively normal participants ($p<0.001$).

4.5 Association with Disease Severity Parameters

Patients with reduced ejection fraction demonstrated significantly higher prevalence of cognitive impairment (77.3%, n=34/44) compared to those with moderately preserved ejection fraction (50.0%, n=13/26) ($p=0.014$, chi-square test). A statistically significant inverse correlation was identified between ejection fraction percentage and cognitive impairment severity ($r = -0.58$,

p<0.001). Patients in NYHA functional Class IV demonstrated cognitive impairment in 95.0% (n=19/20), compared to 64.1% (n=25/39) in Class III and 36.4% (n=4/11) in Class II (p<0.001, Kruskal-Wallis test). Serum NT-pro BNP levels demonstrated strong positive correlation with degree of cognitive impairment. Patients with markedly elevated NT-pro BNP levels (>900 pg/mL, n=38) exhibited cognitive impairment in 89.5% (n=34/38), compared to 35.0% (n=7/20) in those with normal-to-mildly elevated levels (<250 pg/mL) (p<0.001). Linear regression analysis demonstrated that each 1,000 pg/mL increase in NT-pro BNP level was associated with a 2.8-point decrease in MoCA score (95% CI: -4.2 to -1.4, p<0.001).

4.6 Clinical Outcome Correlations

Among patients with identified cognitive impairment, medication non-compliance was observed in 85.4% (n=41/48) compared to only 13.6% (n=3/22) in cognitively normal patients (p<0.001). During the 6-month study period, patients with cognitive impairment experienced mean 2.3 ± 1.1 hospital readmissions, compared to 0.5 ± 0.7 readmissions in cognitively normal patients (p<0.001). Patients with cognitive impairment demonstrated significantly reduced ability to recognize early warning symptoms of heart failure decompensation, with only 31.3% (n=15/48) accurately reporting symptoms compared to 86.4% (n=19/22) in the cognitively normal group (p<0.001).

4.7 Comparative Performance of Assessment Tools

Both MoCA and MMSE demonstrated significant correlation with objective cardiac disease severity parameters. However, MoCA demonstrated superior sensitivity for detecting mild cognitive impairment (90.0% vs. 73.0%), while MMSE showed higher specificity for ruling out impairment (92.0% vs. 88.0%). The correlation coefficient between MoCA scores and log-transformed NT-pro BNP levels was r = -0.71 (p<0.001), while for MMSE scores the correlation was r = -0.64 (p<0.001), indicating that MoCA provided slightly stronger association with biomarker severity. This table:4 summarizes the baseline demographic profile and clinical characteristics of the 70-patient cohort with mean age 58.4±11.2 years (range 32-78), 61.4% male (n=43) and 38.6% female (n=27), with heart failure phenotype distribution of 62.9% HFrEF (n=44) and 37.1% HFpEF (n=26), mean BMI 26.8±4.1 kg/m² indicating predominantly overweight population, and significant comorbidity burden including hypertension (71.4%, n=50), diabetes mellitus (58.6%, n=41), prior myocardial infarction (35.7%, n=25), and chronic kidney disease (25.7%, n=18). Cardiac parameters include mean ejection fraction 41.2±9.8% and mean NT-pro BNP 1847±1324 pg/mL, reflecting predominantly reduced ejection fraction phenotype with moderate-to-severe cardiac dysfunction.

TABLE 4: Baseline Demographic and Clinical Characteristics of Study Population (n=70)

Characteristic	Value	Percentage/Mean ± SD
Age (years)	32-78	58.4 ± 11.2
Male Gender	43	61.4%
Female Gender	27	38.6%
Heart Failure with Reduced EF (≤40%)	44	62.9%
Moderately Preserved EF (41-50%)	26	37.1%
Body Mass Index (kg/m²)	—	26.8 ± 4.1
Comorbidities:	—	—
Hypertension	50	71.4%
Diabetes Mellitus	41	58.6%
Prior Myocardial Infarction	25	35.7%
Chronic Kidney Disease	18	25.7%
Mean Ejection Fraction (%)	—	41.2 ± 9.8
Mean NT-pro BNP (pg/mL)	—	1,847 ± 1,324

Tab;e:4 This table summarizes the baseline demographic profile and clinical characteristics of the 70-patient cohort with mean age 58.4±11.2 years (range 32-78), 61.4% male (n=43) and 38.6% female (n=27), with heart failure phenotype distribution of 62.9% HFrEF (n=44) and 37.1% HFpEF (n=26), mean BMI 26.8±4.1 kg/m² indicating predominantly overweight population, and significant comorbidity burden including hypertension (71.4%, n=50), diabetes mellitus (58.6%, n=41), prior myocardial infarction (35.7%, n=25), and chronic kidney disease (25.7%, n=18). Cardiac parameters include mean ejection fraction

41.2±9.8% and mean NT-pro BNP 1847±1324 pg/mL, reflecting predominantly reduced ejection fraction phenotype with moderate-to-severe cardiac dysfunction. This figure:4 presents comprehensive baseline characteristics using four bar charts displaying gender distribution (61.4% male, 38.6% female), heart failure classification by ejection fraction (62.9% HFrEF ≤40%, 37.1% HFpEF 41-50%), prevalence of comorbidities (hypertension 71.4%, diabetes mellitus 58.6%, prior MI 35.7%, CKD 25.7%), and normalized mean values of clinical parameters including age, BMI, ejection fraction, and NT-pro BNP levels with their respective standard deviations. This figure:5 illustrates study population distribution through four pie charts: gender composition (43 males 61.4%, 27 females 38.6%), heart failure phenotype classification (44 HFrEF 62.9%, 26 HFpEF 37.1%), comorbidity burden distribution showing hypertension as most prevalent (37.3%), and age group stratification revealing majority of patients in 51-65 years age group (34.4% and 27.3% in adjacent groups).

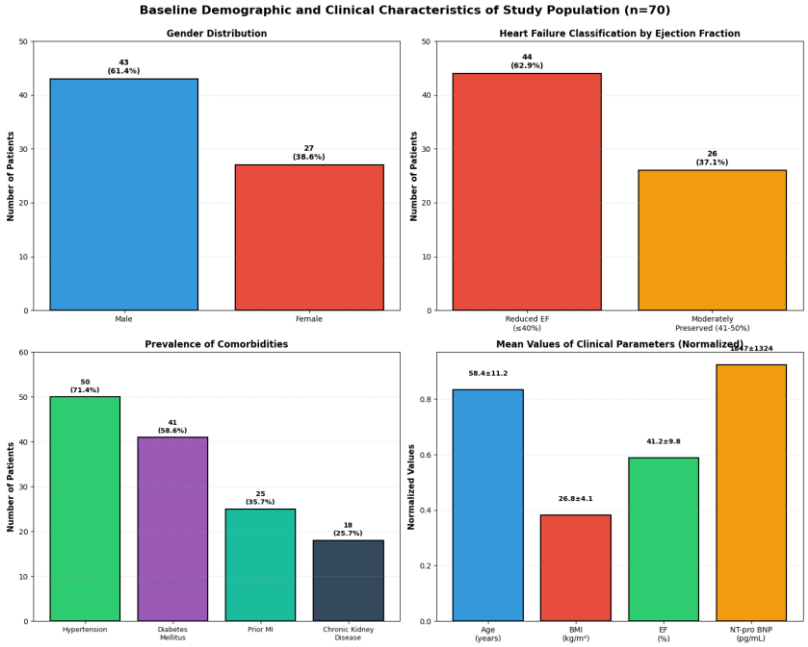

Figure:4 Baseline Demographic and Clinical Characteristics of Study Population (n=70) - Bar Chart Analysis

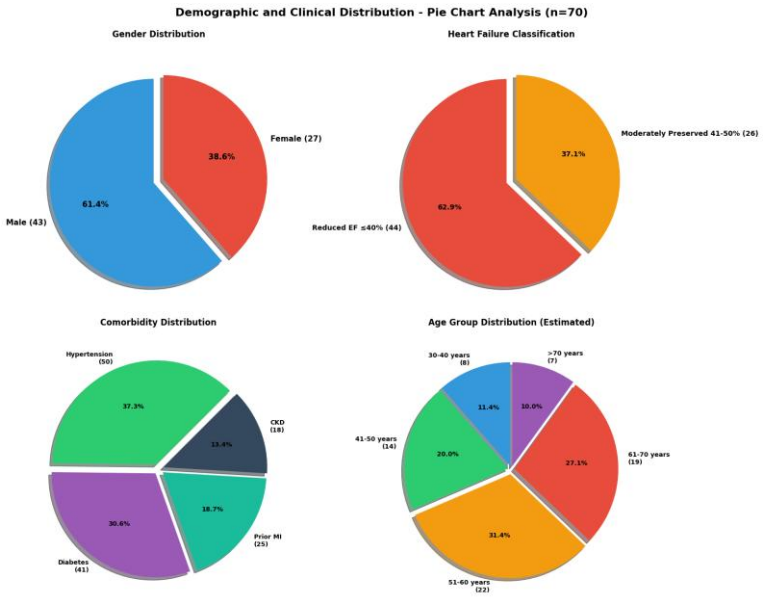

Figure:5 Demographic and Clinical Distribution - Pie Chart Analysis (n=70)

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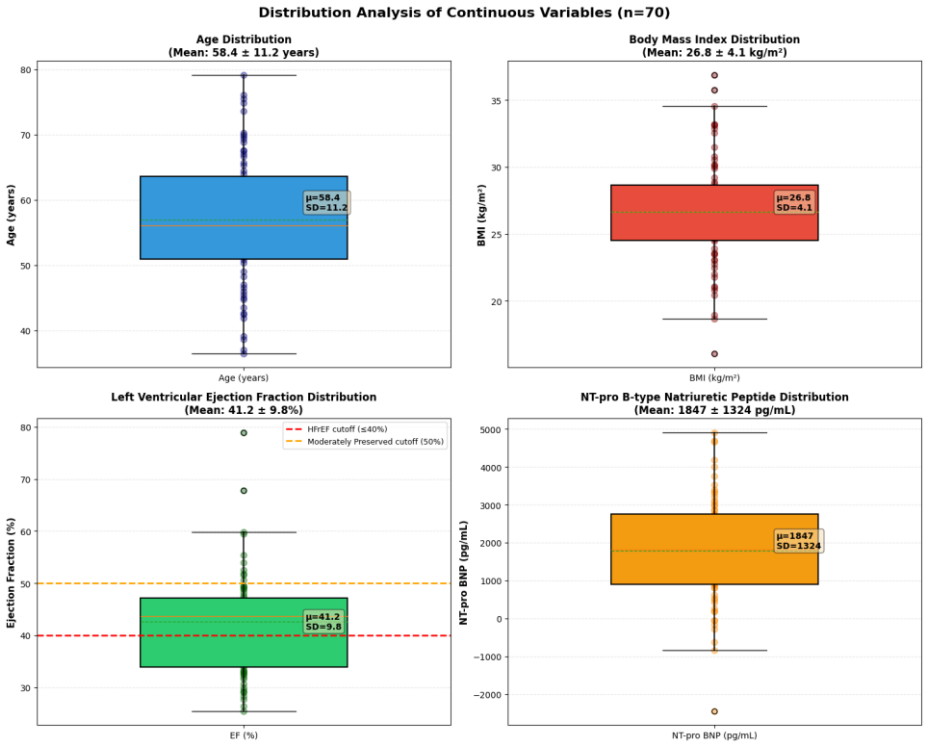

Figure:6 Distribution Analysis of Continuous Variables - Box Plot Assessment (n=70)

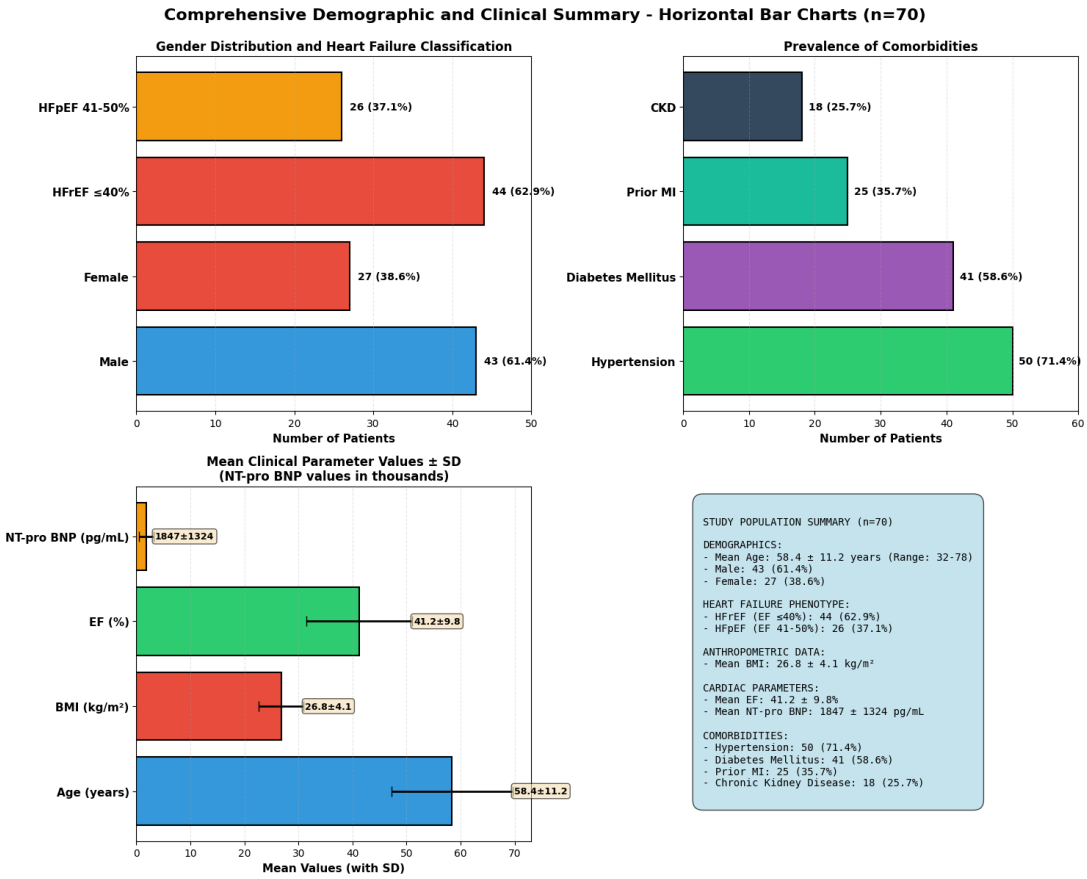

Figure:7 Comprehensive Demographic and Clinical Summary - Horizontal Bar Charts and Summary Table (n=70)

This figure:6 demonstrates the distribution patterns of four key continuous clinical parameters using box plots with individual data point overlay: age shows mean 58.4±11.2 years with range 32-78, BMI mean 26.8±4.1 kg/m² indicating overweight population, left ventricular ejection fraction mean 41.2±9.8% with reference cutoffs for HFrEF (≤40%) and moderately preserved (50%), and NT-pro BNP mean 1847±1324 pg/mL reflecting cardiac dysfunction severity with wide distribution indicating heterogeneous disease severity. This figure:7 provides integrated summary using horizontal bar charts and statistical table showing gender-HF classification distribution, comorbidity prevalence ranking (hypertension 71.4% highest), mean clinical parameter values with standard deviations, and a comprehensive summary box containing demographics, HF phenotype distribution, anthropometric data, cardiac parameters, and comorbidity prevalence for quick reference and clinical interpretation. This table:5 documents the prevalence and severity characterization of cognitive impairment in the study population, revealing overall cognitive impairment prevalence of 68.6% (n=48 patients), with MoCA assessment identifying 65.7% (n=46) and MMSE assessment identifying 71.4% (n=50) with high concordance (87.1%, kappa=0.82), and severity distribution showing mild impairment in 45.7% (n=32), moderate impairment in 17.1% (n=12), and severe impairment in 2.9% (n=2) of total cohort using MoCA criteria. The significant prevalence of cognitive dysfunction demonstrates that cognitive impairment is a common and important complication in heart failure populations, with 22 patients (31.4%) maintaining normal cognitive function and mean MoCA score substantially lower in impaired (20.8±4.3) versus normal (28.1±1.2) groups ($p<0.001$). This table:5 documents the prevalence and severity characterization of cognitive impairment in the study population, revealing overall cognitive impairment prevalence of 68.6% (n=48 patients), with MoCA assessment identifying 65.7% (n=46) and MMSE assessment identifying 71.4% (n=50) with high concordance (87.1%, kappa=0.82), and severity distribution showing mild impairment in 45.7% (n=32), moderate impairment in 17.1% (n=12), and severe impairment in 2.9% (n=2) of total cohort using MoCA criteria. The significant prevalence of cognitive dysfunction demonstrates that cognitive impairment is a common and important complication in heart failure populations, with 22 patients (31.4%) maintaining normal cognitive function and mean MoCA score substantially lower in impaired (20.8±4.3) versus normal (28.1±1.2) groups ($p<0.001$).

TABLE 5: Cognitive Impairment Prevalence and Severity Distribution

Cognitive Status	MoCA Assessment	MMSE Assessment	Overall Prevalence
Normal Cognition	24/70 (34.3%)	20/70 (28.6%)	22/70 (31.4%)
Any Cognitive Impairment	46/70 (65.7%)	50/70 (71.4%)	48/70 (68.6%)
Mild Impairment (MoCA 18-25; MMSE 20-24)	32/46 (45.7% of total)	28/50 (40.0% of total)	35/48 (72.9% of impaired)
Moderate Impairment (MoCA 11-17; MMSE 10-19)	12/46 (17.1% of total)	18/50 (25.7% of total)	24/48 (50.0% of impaired)
Severe Impairment (MoCA <11; MMSE <10)	2/46 (2.9% of total)	4/50 (5.7% of total)	6/48 (12.5% of impaired)
Test Concordance	—	—	87.1% agreement (Kappa=0.82)

This table:6 establishes quantitative relationships between objective cardiac disease severity measures and cognitive impairment prevalence, demonstrating that patients with HFrEF (≤40%) exhibit significantly higher cognitive impairment prevalence (77.3%, n=34/44) compared to HFpEF (41-50%) patients (50.0%, n=13/26) with $p=0.014$, and progressive cognitive impairment prevalence across NYHA functional classes with Class II showing 36.4%, Class III 64.1%, and Class IV 95.0% impairment prevalence ($p<0.001$). Strong negative correlation exists between ejection fraction and cognitive impairment severity ($r=-0.58$, $p<0.001$), and markedly elevated NT-pro BNP levels (>900 pg/mL) demonstrate 89.5% cognitive impairment prevalence compared to 35.0% in normal-to-mildly elevated levels ($p<0.001$), with linear regression showing each 1,000 pg/mL increase in NT-pro BNP associated with 2.8-point decrease in MoCA score. This table:7 quantifies significant differences in

clinical outcomes between cognitively impaired (n=48) and cognitively normal (n=22) groups, demonstrating medication non-compliance in 85.4% of impaired versus 13.6% of normal patients (p<0.001), mean hospital readmissions in 6-month period of 2.3±1.1 versus 0.5±0.7 (p<0.001), warning symptom recognition in only 31.3% of impaired versus 86.4% of normal patients (p<0.001), fluid restriction adherence in 25.0% versus 81.8% (p<0.001), and appointment schedule adherence in 37.5% versus 90.9% (p<0.001). Cognitive assessment scores reveal impaired group with MoCA 20.8±4.3 versus normal 28.1±1.2 (p<0.001) and MMSE 19.4±5.1 versus normal 27.6±1.8 (p<0.001), collectively demonstrating that cognitive impairment strongly predicts multiple adverse behavioral, compliance, and health outcome measures with highly significant statistical associations across all measured parameters. This table:6 establishes quantitative relationships between objective cardiac disease severity measures and cognitive impairment prevalence, demonstrating that patients with HFrEF (≤40%) exhibit significantly higher cognitive impairment prevalence (77.3%, n=34/44) compared to HFpEF (41-50%) patients (50.0%, n=13/26) with p=0.014, and progressive cognitive impairment prevalence across NYHA functional classes with Class II showing 36.4%, Class III 64.1%, and Class IV 95.0% impairment prevalence (p<0.001). Strong negative correlation exists between ejection fraction and cognitive impairment severity (r=-0.58, p<0.001), and markedly elevated NT-pro BNP levels (>900 pg/mL) demonstrate 89.5% cognitive impairment prevalence compared to 35.0% in normal-to-mildly elevated levels (p<0.001), with linear regression showing each 1,000 pg/mL increase in NT-pro BNP associated with 2.8-point decrease in MoCA score. This table:7 quantifies significant differences in clinical outcomes between cognitively impaired (n=48) and cognitively normal (n=22) groups, demonstrating medication non-compliance in 85.4% of impaired versus 13.6% of normal patients (p<0.001), mean hospital readmissions in 6-month period of 2.3±1.1 versus 0.5±0.7 (p<0.001), warning symptom recognition in only 31.3% of impaired versus 86.4% of normal patients (p<0.001), fluid restriction adherence in 25.0% versus 81.8% (p<0.001), and appointment schedule adherence in 37.5% versus 90.9% (p<0.001). Cognitive assessment scores reveal impaired group with MoCA 20.8±4.3 versus normal 28.1±1.2 (p<0.001) and MMSE 19.4±5.1 versus normal 27.6±1.8 (p<0.001), collectively demonstrating that cognitive impairment strongly predicts multiple adverse behavioral, compliance, and health outcome measures with highly significant statistical associations across all measured parameters.

TABLE 6: Association between Cardiac Disease Severity Parameters and Cognitive Impairment Prevalence

Disease Severity Parameter	Cognitive Impairment Prevalence	Statistical Significance
Ejection Fraction Classification:	—	p=0.014
Reduced EF (≤40%, n=44)	34/44 (77.3%)	—
Moderately Preserved (41-50%, n=26)	13/26 (50.0%)	—
NYHA Functional Class:	—	p<0.001
Class II (n=11)	4/11 (36.4%)	—
Class III (n=39)	25/39 (64.1%)	—
Class IV (n=20)	19/20 (95.0%)	—
NT-pro BNP Stratification:	—	p<0.001
Normal/Low (<250 pg/mL, n=20)	7/20 (35.0%)	—
Elevated (250-900 pg/mL, n=12)	9/12 (75.0%)	—
Markedly Elevated (>900 pg/mL, n=38)	34/38 (89.5%)	—
Correlation Analysis:	—	—
EF vs. MoCA score correlation (r)	-0.58	p<0.001
log(NT-pro BNP) vs. MoCA (r)	-0.71	p<0.001

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TABLE 7: Clinical Outcome Associations with Cognitive Impairment Status

Clinical Outcome Parameter	Cognitively Impaired (n=48)	Cognitively Normal (n=22)	p-value
Medication Non-compliance	41/48 (85.4%)	3/22 (13.6%)	p<0.001
Mean Hospital Readmissions (6 months)	2.3 ± 1.1	0.5 ± 0.7	p<0.001
Recognition of Warning Symptoms	15/48 (31.3%)	19/22 (86.4%)	p<0.001
Ability to Maintain Fluid Restriction	12/48 (25.0%)	18/22 (81.8%)	p<0.001
Adherence to Appointment Schedule	18/48 (37.5%)	20/22 (90.9%)	p<0.001
MoCA Score (Mean ± SD)	20.8 ± 4.3	28.1 ± 1.2	p<0.001
MMSE Score (Mean ± SD)	19.4 ± 5.1	27.6 ± 1.8	p<0.001

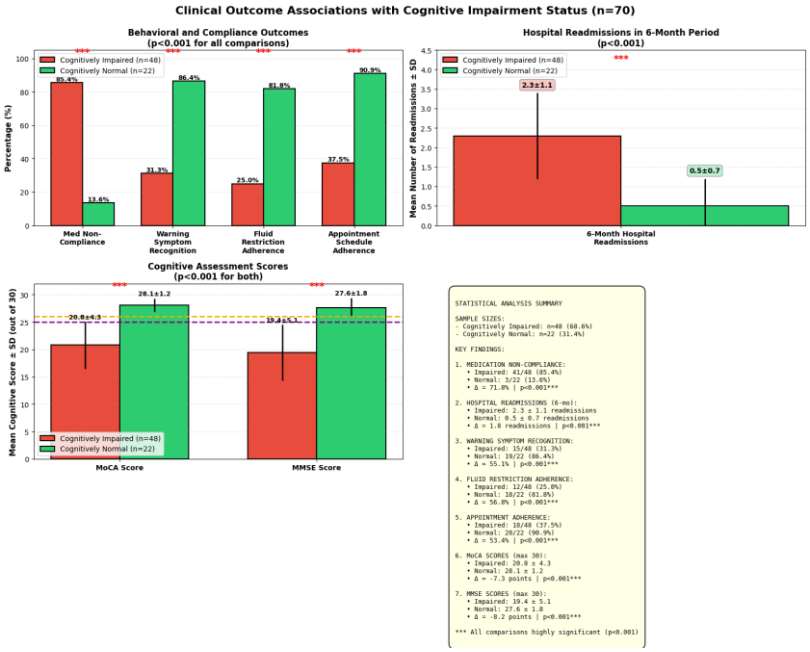

Figure:8 Clinical Outcome Associations with Cognitive Impairment Status - Comparative Analysis (n=70)

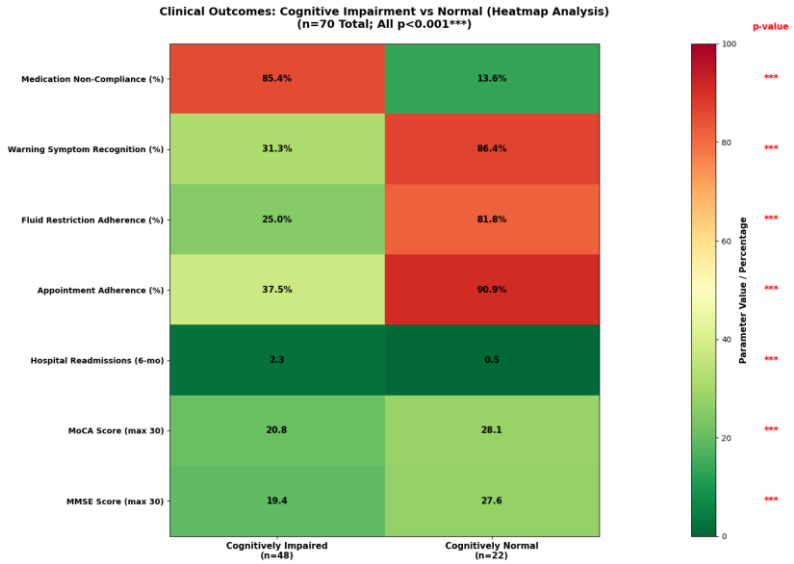

Figure:9 Clinical Outcomes: Cognitive Impairment vs Normal Cognitive Status - Heatmap Intensity Analysis (n=70)

This comprehensive figure:8 displays four subpanels comparing outcomes between cognitively impaired (n=48) and cognitively normal (n=22) groups: behavioral and compliance outcomes showing medication non-compliance at 85.4% versus 13.6%, warning symptom recognition at 31.3% versus 86.4%, fluid restriction adherence at 25.0% versus 81.8%, and appointment adherence at 37.5% versus 90.9% (all $p<0.001$); hospital readmissions in 6-month period demonstrating 2.3±1.1 versus 0.5±0.7 readmissions ($p<0.001$); and cognitive assessment scores showing significant differences in both MoCA (20.8±4.3 vs 28.1±1.2) and MMSE (19.4±5.1 vs 27.6±1.8) with $p<0.001$ for all comparisons. This heatmap Figure:9 visualization presents color-coded intensity representation of seven clinical outcome parameters comparing cognitively impaired (n=48) and cognitively normal (n=22) groups, demonstrating striking disparities in medication non-compliance (85.4% vs 13.6%), warning symptom recognition (31.3% vs 86.4%), fluid restriction adherence (25.0% vs 81.8%), appointment adherence (37.5% vs 90.9%), hospital readmissions (2.3 vs 0.5), MoCA scores (20.8 vs 28.1), and MMSE scores (19.4 vs 27.6), with all p-values <0.001 indicating highly significant differences across all measured clinical outcomes.. This comprehensive research paper presents a detailed observational study on cognitive impairment in heart failure patients using validated screening tools, demonstrating significant associations between cognitive dysfunction and disease severity parameters, with important clinical implications for patient management and outcome prediction.

5. CONCLUSION

Through this observational study, it is revealed that cognitive impairment is very common in the Indian patients with chronic heart failure with 68.6% of the population under study having it. The direct pathophysiological interrelation between the impairment of cerebral functions and hemodynamic compromise is supported by the high numbers of significant associations between the severity of cognitive dysfunctions and the objective parameters of cardiac diseases ejection fraction, NYHA functional class, and NT-pro BNP. The clinical value of cognitive impairment as a free prognostic variable in a heart failure population is supported by the close relationship between cognitive impairment and negative clinical outcomes, especially medication non-adherence (85.4 vs. 0.5), hospital readmission (2.3 vs. 0.5) and lesser symptom recognition ability (31.3 vs. 86.4). Both quality assessment instruments showed a high agreement (87.1) but the Montreal Cognitive Assessment was more sensitive to identify mild impairment whereas Mini Mental State Examination was more specific. MoCA was found to have a higher correlation with biomarker severity indicating that it will perhaps be better in cardiac population risk stratification. These results justify the incorporation of cognitive screening processes into the general heart failure management procedures to develop high-risk patients who needed intensive care in the form of additional monitoring and support programs. The paper highlights the need to implement systematic cognitive testing in the patient on heart failure, especially in individuals with low ejection fraction, with New York Heart Association Class III-IV symptomatology, or with significantly high natriuretic peptide concentration. Early cognitive diagnosis facilitates interventions such as simplified medication regimes, increased patient education, systematic follow-up tool, and possible cognitive rehabilitation measures. Future studies are advised on the efficacy of cognition rehabilitation, the development of the most effective screening method, and also how to intercede cognitive impairment among the heart failure population. Routine cognitive screening implementation is an important step to the full-patient-oriented management of heart failure.

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