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Relationship between cognitive impairment and decompensated chronic heart failure in hospitalized older adults

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Abstract

Chronic heart failure (HF) is frequently associated with cognitive impairment in older adults, affecting up to 50% of these patients. This relationship has multiple causes including low cardiac output, comorbidities, and aging itself, negatively impacting prognosis and increasing mortality and hospitalizations.

A prospective observational study was conducted at General Hospital Zone 20 "La Margarita" in Puebla, evaluating 41 geriatric patients with acutely decompensated chronic HF over six months. The sample included 51.2% men and 48.8% women, with an average age of 79.6 years and 6.89 years of education. Arterial hypertension was the most frequent comorbidity (82.9%).

Using the Mini-Mental State Examination and Framingham criteria, cognitive status was evaluated along with other functional variables. Results showed that 34.1% of patients had normal cognitive function, while 36.6% presented some degree of cognitive impairment. Depression affected 19.5% of participants, anxiety 27.5%, and dementia 7.3%, predominantly vascular type. Delirium occurred in 17.1% of cases.

Statistical analysis confirmed a moderate relationship between cognitive impairment and heart failure (p=0.05, r=0.5), with contributing factors being the number of hospitalizations and infectious processes. This study demonstrates the importance of comprehensively evaluating cognitive status in patients with chronic heart failure.

Keywords: Heart failure, older adults, cognitive impairment, cardiac output, dementia

Introduction

Cognitive impairment is a frequent manifestation in patients with chronic heart failure (HF), whose multifactorial etiology includes factors related to age, low cardiac output, the cardiac pathology itself, and associated comorbidities.

The PRICE and EPISERVE epidemiological studies report an HF prevalence of 6.8% and 4.7%, respectively, in the general population. This prevalence shows a progressive increase with age: 1.3% between 45-54 years, 5.5% between 55-64 years, 8% between 65-75 years, and 16.1% in those over 75 years.

Approximately half of geriatric patients with HF develop cognitive impairment, establishing a direct association between both pathologies and a higher risk of dementia. This condition significantly worsens the clinical prognosis, increases mortality and rehospitalization rates, interferes with self-care and therapeutic adherence, and results in adverse health outcomes.

In Mexico, HF represents a relevant health problem, responsible for 3-6% of mortality and 3-5% of hospital admissions, constituting the main cause of hospitalization in the population over 65 years of age ^[1].

Cognition represents the intellectual functioning that allows interaction with the environment ^[1]. Mild cognitive impairment (MCI), according to NIA-AA criteria, characterizes patients who do not meet dementia criteria and who maintain functional independence ^[2], presenting impairment in cognitive domains greater than expected for their age and educational level, without reaching the diagnostic threshold for dementia ^[3].

MCI constitutes a major public health and social problem, considered a transitional state between cognitive normality and dementia, where patients preserve functionality in basic activities but present a pathological condition not attributable to normal aging.

Corresponding Author: Dorcas María Sosa Mendóza, Geriatrician, Universidad Autónoma de Yucatán, Yucatán, Mexico Its population prevalenace varies between 1-29%. In Mexico, Cárdenas *et al.* identified MCI in 24% of 142 subjects over 60 years of age, especially in the older population with lower educational levels ^[4].

The PARAGON-HF study demonstrated that MCI in HF with preserved ejection fraction is associated with worse clinical outcomes, including a higher risk of hospitalization and cardiovascular mortality, suggesting cognitive impairment as a potential prognostic marker. This is related to a deficit of brain-derived neurotrophic factor in HF, leading to synaptic damage, neurotransmitter reduction, and alterations in precuneal neuroconnectivity, increasing the risk of executive and social cognitive impairment. Additionally, HF generates a reduction in cerebral blood flow, producing neurodegeneration and an environment conducive to the development of cognitive impairment.

Physiological brain changes associated with aging include loss of volume and frontal cortical thinning, non-uniform neuronal volume decrease, synaptic and dendritic changes, neurotransmitter and receptor reduction, and decreased cerebral blood flow and oxygen consumption. Around age 60, impairment in memory, verbal fluency, mathematical logic, and analytical efficiency becomes manifest ^[5].

Risk factors for MCI include advanced age, female sex, low educational level, genetic factors, and depressive disorders. Associated psychological and behavioral symptoms include depression, irritability, anxiety, apathy, and agitation. Memory impairment constitutes the main dysfunctional indicator, affecting complex instrumental activities such as cooking, financial management, or medication management [6]

Evaluation uses the Peterson criteria and the Mini-Mental State Examination (MMSE), which has been extensively validated with 79.8% sensitivity, 81.3% specificity, 86.3% positive predictive value, and 73.0% negative predictive value, with scores lower than 24 considered indicative of dementia [7].

Current research incorporates biological and neural markers, including electroencephalogram (EEG), which in the early stages of Alzheimer's shows an increase in theta waves, a decrease in beta and subsequently alpha activity, with an increase in delta waves in advanced stages ^[6]. However, there is empirical divergence in differentiating between healthy subjects and those with MCI ^[8].

Biomarkers include TAU protein and beta-amyloid 42 peptide. The hyperphosphorylation of P-TAU promotes the formation of neurofibrillary tangles affecting the medial temporal lobes and associative cortical structures. The beta-amyloid peptide, an essential component of neuritic plaques and the main neurodegenerative marker, generates localized inflammatory processes and neuronal changes through TAU phosphorylation, potentially causing dysfunction and neuronal death [9].

Sociodemographic variables and life experiences influence geriatric cognitive functioning. Circumstances in early and middle age modify cognition in older adulthood through lifestyle patterns correlated with socioeconomic status, educational level, and ethnicity [10].

Cardiovascular disease increases the risk of MCI by 50-60% in subjects with a vascular history or subclinical vascular disease. HF shows a variable prevalence of MCI between

30-80% depending on the study series and evaluation instruments, suggesting generalized screening [11].

The HF syndrome has a high prevalence and is the most common cause of geriatric hospitalization, with an unfavorable prognosis and an annual hospital mortality of 30-40% during exacerbations, responsible for prolonged hospitalizations and a high readmission rate [12].

Clinically, it is characterized by typical symptoms (dyspnea, malleolar edema, fatigue) accompanied by signs such as elevated jugular venous pressure, pulmonary rales, and peripheral edema, caused by structural or functional cardiac abnormalities resulting in reduced cardiac output and/or elevated intracardiac filling pressures [13].

It can present with preserved or reduced left ventricular ejection fraction, frequently coexisting with coronary artery disease, arterial hypertension, valvular heart disease, atrial arrhythmias, or multiple organ dysfunction, requiring immediate medical attention and urgent hospitalization [14].

The Forrester classification distinguishes four groups: warm-wet (well-perfused-congested), cold-wet (hypoperfused-congested), cold-dry (hypoperfused without congestion), and warm-dry (compensated without congestion), providing therapeutic guidance and prognostic information [14].

Clinical manifestations include asthenia, fatigability, and congestive symptoms such as progressive dyspnea, orthopnea, nocturnal cough, paroxysmal nocturnal dyspnea, abdominal distension, and edema due to hydrosaline retention. In the geriatric population, the presentation can be atypical, manifesting as a confusional syndrome or anxious episodes [15].

Physical examination reveals tachypnea, Cheyne-Stokes respiration, tachycardia, a third-heart-sound gallop, heart murmurs, and congestive signs such as jugular engorgement, hepatojugular reflux, crackles, wheezing, pleural effusion, hepatomegaly, edema, and ascites, constituting the Framingham criteria for clinical diagnosis [16]

Guidelines recommend follow-up echocardiograms only in cases of clinical worsening, therapeutic changes, or exposure to cardiotoxic drugs. The Canadian guidelines specify evaluation for de novo HF, after 3 months of therapeutic optimization, prior to resynchronization devices, and following hospital decompensation, suggesting reevaluation every 1-3 years in stable patients [17].

The true prevalence of cognitive impairment in HF requires determination using adapted and validated tests to justify the need for screening, given that the loss of cognitive performance significantly interferes with self-care and therapeutic adherence, resulting in worse health outcomes.

Materials and Methods

An observational, cross-sectional, and prospective study was conducted at the General Zone Hospital number 20 (HGZ20) in Puebla. The sample consisted of 41 patients aged 60 or older (average age of 79.6 years), who were hospitalized with a diagnosis of decompensated chronic heart failure. The Mini-Mental State Examination (MMSE) was used for cognitive assessment, and the HF diagnosis was supported by the Framingham criteria. The statistical analysis sought a correlation (Pearson) between the variables, considering a p-value ≤ 0.05 as significant.

Results

Statistical Analysis and Sample

Statistical analysis was performed using SPSS version 25. For quantitative variables, measures of central tendency and dispersion were reported, while qualitative variables were expressed in frequencies and percentages. The Shapiro-Wilk test was used to determine data distribution, and Pearson's correlation was used to assess the relationship between variables. A statistical significance level of $p \le 0.05$ was established.

The study included 41 patients. The sample showed a nearly equal sex distribution, with 51.2% men (n=21) and 48.8% women (n=20). The average age of the participants was 79.6 \pm 8.4 years, with a range of 66 to 99 years.

Regarding other characteristics (Table 1):

- The average body mass index (BMI) was 27.0 ± 7.3 .
- 61.0% (n=25) of the patients were married.
- The average schooling was 6.89 ± 3.7 years.

Table 1. Sociodemographic and clinical characteristics of the patients who participated in the study.

	N (%) or mean ± SD	
Number of patients	41	
Sociodemographic characteristics		
Age	79.6 ± 8.4	
Sex		
Female	20 (48.8)	
Male	21 (51.2)	
Weight	69.2 ± 20.1	
Size	9.16 ± 33.0	
Body mass index (BMI)	27.0 ± 7.3	
Marital status		
Married	25 (61.0)	
Bachelor	14 (34.1)	
Schooling (years)	6.89 ± 3.7	
Comorbi	idities	
Diabetes	17 (41.5)	
Chronic obstructive pulmonary disease (COPD)	7 (17.1)	
Chronic kidney disease (CKD)	13 (31.7)	
High blood pressure	34 (82.9)	
Ischemic heart disease	16 (39)	
Cerebral Vascular Disease (CVD)		
Ischemic	3 (7.3)	
Hemorrhagic	0 (0)	
Liver disease	3 (7.3)	
Degenerative joint disease	2 (4.9)	
Hypothyroidism	6 (14.6)	
Prostatic hyperplasia	6 (14.6)	
Cardiac arrhythmia	11 (26.8)	
Cancer	2 (4.9)	

Comorbidities and Functional Status

Patient comorbidities were investigated. Arterial hypertension was the most frequent, affecting 82.9% (n=34) of the sample. The second most common comorbidity was diabetes, present in 41.5% (n=17). The least frequent were degenerative joint disease and cancer, both with a prevalence of 4.9% (n=2).

Various functional tests were performed. Key results include:

- Charlson Comorbidity Index (CCI): Mean score of 5.46 ± 2.24.
- Mini Nutritional Assessment (MNA): Mean of 17.88 ± 6.75.
- Mini-Mental State Examination (MMSE): Average score of 20.7 ± 8.28 .
- **Katz Index:** Mean of 4.38 ± 1.98 .

Neuropsychiatric Findings

The presence of various neuropsychiatric comorbidities was assessed (Table 2).

- **Depression:** Found in 19.5% (n=8) of patients. Of these, 12.2% (n=5) met the diagnostic criteria of the DSM-V.
- **Anxiety:** Was present in 27.5% (n=11) of the sample.
- **Benzodiazepine Use:** Reported by 87.7% (n=36) of patients, with consumption being mostly acute (87.7%).
- **Sleep Disorders:** 34.1% (n=14) reported sleep maintenance problems, and 24.4% (n=10) reported sleep onset/initiation problems.
- Cognitive Impairment and Dementia: According to the MMSE, 34.1% (n=14) had a normal score, but 36.6% (n=15) presented some degree of impairment (mild, moderate, or severe). Dementia was diagnosed in 7.3% (n=3), with vascular dementia being the most prevalent (4.9%).

Table 2: Frequencies and characteristics of neuropsychiatric comorbidities of patients who participated in the study.

	N (0/) CD
N. I. C. C.	N (%) or mean ± SD
Number of patients	41
	ric comorbidities
	ression
No	33 (80.5)
Yes	8 (19.5)
	Depression Criteria
Yes	5 (12.2)
No	36 (87.7)
GDS-15 Geriatric Depression Scale Score	4.35 ± 3.6
Anxiety	diagnosis
Yes	11 (27.5)
No	29 (72.5)
Generalized Anxiety Scale Score (GAD-7)	4.39 ± 5.1
Use of Ben	zodiazepines
Yes	36 (87.7)
No	5 (12.2)
Time of use of	benzodiazepines
Acute	36 (87.7)
Chronic	5 (12.2)
Sleep	disorder
None	17 (41.5)
Home/Conciliation	10 (24.4)
Maintenance	14 (34.1)
	impairment
Mini-Mental Test Score (MMSE)	20.76 ± 8.2
Normal	14 (34.1)
Mild impairment	6 (14.6)
Moderate impairment	5 (12.2)
Severe impairment	4 (9.8)
	nentia
No	35 (85.4)
Yes	3 (7.3)
	dementia
No dementia	35 (85.4)
Vascular	2 (4.9)
Mixed	1 (2.4)
	\· ·/

Delirium

Characteristics of delirium episodes were evaluated (Table 3).

- 17.1% (n=7) of patients presented with delirium.
- The most common types were mixed and hyperactive (7.3% each).
- The most identified precipitating factor was infectious (9.8%).
- The majority of cases (95.1%) did not require pharmacological treatment.

Table 3: Characteristics and treatment of the delirium episode presented by the patients in the study.

	N (%) or mean ± SD
Number of patients	41
Deli	rium
Presence	of delirium
Yes	7 (17.1)
No	34 (82.9)
Type of	delirium
None	34 (82.9)
Mixed	3 (7.3)
Hypoactive	1 (2.4)
Hyperactive	3 (7.3)
Place where d	lelirium began
Not applicable	34 (82.9)
Emergency	1 (2.4)
Hospitalization	5 (12.2)
Institutionalization	1 (2.4)
Precipita	ting factor
Not applicable/None	35 (85.4)
Metabolic	1 (2.4)

Infectious	4 (9.8)	
Drug	1 (2.4)	
Duration of delirium	0.20 ± 0.56	
Pharmacological treatment		
No	39 (95.1)	
Yes	2 (4.9)	
Score Confusion Assessment Method (CAM)	1.07 ± 3.9	

Hospitalization and Life Expectancy

Variables related to hospitalization and life expectancy were analyzed (Table 4).

- 78.0% (n=32) of patients reported no hospitalizations in the last year.
- The majority (82.9%, n=34) did not have an advance directive.
- The estimated mean life expectancy for the sample was 6.07 ± 3.96 years.

Table 4: Deterioration variables and life expectancy of the	e patients in the study	١.
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	N (%) or mean ± SD
Number of hospitalization	s over the past year
No	32 (78.0)
1	4 (9.8)
2	1 (2.4)
3	2 (4.9)
More than 3	1 (2.4)
NECPAL Score	0.66 ± 1.3
Advance dire	ective
No	34 (82.93)
Yes	7 (17.0)
Graduation des	tination
Domicile	7 (17.1)
Death	2 (4.9)
Reference to another unit	1 (2.4)
Life expecta	ancy
10-year Charlson index score (ICC)	5.46 ± 2.24
Lee's Life Expectancy Score	12.12 ± 3.96
5-year survival percentage (Lee)	48.42 ± 19.21
Schönberg Life Expectancy Score	13.94 ± 3.62
5-year survival percentage (Schönberg)	48.20 ± 18.78
Life expectancy (years)	6.07 ± 3.96

Correlation Analysis

Finally, the relationship between cognitive impairment and other variables was examined.

- A statistically significant positive correlation was found between cognitive impairment and the Katz index (r=.392, p=.039), the Barthel index (r=.572, p=.001), and the Lawton and Brody scale (r=.420, p=.023).
- A significant negative correlation was observed with the presence of depression (r=-.418, p=.024), with the Schönberg life expectancy score (r=-.397, p=.033), and with the Schönberg 5-year life expectancy percentage (r=-.405, p=.029).
- No other significant correlations were identified.

Discussion

This study implemented a comprehensive geriatric assessment in elderly adults hospitalized for acute heart failure at HGZ20, using the Mini-Mental State Examination to evaluate five cognitive domains: orientation, immediate memory, attention-calculation, delayed recall, and visuospatial-language skills. The results allowed for the classification of patients according to their cognitive functioning.

The findings revealed that 34% of patients maintained normal cognitive function during exacerbations, while 35% presented some degree of cognitive impairment, distributed as: 14% mild impairment, 12% moderate, and 9.8% severe.

These results are consistent with the Health ABC Study, which demonstrated a 35% higher probability of moderate-to-severe cognitive impairment in patients with heart failure compared to controls without this pathology [18]. The American Heart Association (2024) reports that up to 81% of patients with heart failure develop cognitive impairment affecting memory, language, and executive function [19].

A recent Chinese cohort study by Hanyu Li *et al.*, which followed 29,614 adults for 6.6 years, observed that global cognition, executive function, and memory were the areas primarily affected post-incident of heart failure, with greater impairment in the elderly, women, and white individuals ^[20]. Although this current study showed a male predominance among those affected, it aligns with the pattern of initial global and executive cognitive impairment.

The results are consistent with the Framingham Offspring Study, which estimates cognitive impairment in 25-75% of patients with acute-on-chronic heart failure, increasing with age and recurrent hospitalizations. In the present investigation, 7.3% developed dementia, primarily vascular (4.9%) followed by mixed (2.4%), correlating with observations from the Framingham Offspring Study since 1974 regarding a higher risk of dementia and Alzheimer's in the context of heart failure with low cardiac output [21, 22].

The meta-analysis by Hailing Zhang *et al.* in China, including 12 studies with 9,556 patients, found that cognitive impairment increases the risk of all-cause

mortality by 88% and the risk of rehospitalization by 48% compared to patients without impairment ^[23]. Although no in-hospital deaths were recorded in patients with cognitive impairment or dementia in this study, a history of multiple hospital readmissions was documented.

Comprehensive geriatric assessment should incorporate cognitive screening (MMSE) in patients with heart failure, given that the relationship between both pathologies was confirmed. Exacerbations can destabilize baseline cognitive status due to hypoxia, delirium, adverse drug effects (diuretics, digitalis), electrolyte disturbances, or associated infections [24].

Cognitive impairment and dementia are predictors of mortality, hospital readmission, and poor prognosis in heart failure. Timely diagnosis allows for the implementation of management strategies: involving caregivers, simplifying treatments, and evaluating cognitive-functional support interventions that improve the patient's evolution and prognosis.

Conclusion

A significant relationship is established between cognitive impairment and acutely decompensated heart failure. Patients with cognitive impairment show a proportional increase in the probability of hospitalizations and cardiovascular mortality as the severity of the cognitive compromise advances.

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Author's Contribution

Not available

Conflict of Interest

Not available

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Not available

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