Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies

Journal home page:www.jamdsr.com doi:10.21276/jamdsr Index Copernicus value [ICV] =82.06

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Original Research

Relationship Between Neurological Impairment Severity and Left Ventricular Function in Acute Ischemic Stroke Patients

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ABSTRACT:

Background: Acute ischemic stroke is often complicated by systemic effects, including cardiac dysfunction. The relationship between neurological severity and left ventricular function has important prognostic implications. **Aim:** To evaluate LV function in acute ischemic stroke and determine its correlation with the severity of neurological impairment, and assess whether LV dysfunction is a prognostic marker for in-hospital morbidity. **Material and Methods:** This observational study included 96 patients with acute ischemic stroke, divided into Group-1 (NIHSS < 6) and Group-2 (NIHSS ≥ 6). Clinical, laboratory, and echocardiographic parameters were compared. Correlations were assessed between NIHSS, inflammatory markers (NLR, PLR), and LVEF. **Results:** Group-2 patients had significantly higher NLR and PLR, lower LVEF, and larger LV dimensions compared to Group-1. NIHSS showed strong positive correlations with NLR (R = 0.683) and PLR (R = 0.884) and a strong negative correlation with LVEF (R = -0.780), all $p < 0.001$. Valvular abnormalities were more common in Group-2 but were not statistically significant. **Conclusion:** Severe neurological impairment in acute ischemic stroke is associated with significant LV dysfunction and heightened systemic inflammation. Incorporating echocardiography and inflammatory marker assessment into stroke protocols may enhance prognostication and guide targeted management.

Keywords: Acute ischemic stroke, NIHSS, left ventricular ejection fraction, neurocardiac interaction

Received: 14 May, 2019 Accepted: 17 June, 2019 Published: 22 June, 2019

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This article may be cited as: Kataria V. Relationship Between Neurological Impairment Severity and Left Ventricular Function in Acute Ischemic Stroke Patients. J Adv Med Dent Scie Res 2019;7(6): 244-248.

INTRODUCTION

Acute ischemic stroke (AIS) remains a leading cause of mortality and long-term disability worldwide, accounting for a significant proportion of global disease burden [1]. Despite advances in acute management, including thrombolysis and mechanical thrombectomy, stroke continues to result in considerable morbidity, making early identification of prognostic markers essential for optimal patient care [2]. Cardiovascular complications are common in AIS and may influence both short-term and long-term outcomes. Among these, left ventricular (LV) dysfunction is increasingly recognized as a critical factor affecting prognosis [3].

The brain–heart axis, a complex bidirectional relationship between neurological injury and cardiac function, plays a pivotal role in the pathophysiology of cardiac changes following AIS [4]. Stroke can precipitate myocardial injury and LV dysfunction through neurogenic mechanisms, including catecholamine surge, autonomic imbalance, and systemic inflammatory responses [5]. This phenomenon, often referred to as "neurogenic stunned myocardium," can occur even in the absence of underlying coronary artery disease, and may present as reduced ejection fraction, wall motion abnormalities, or diastolic dysfunction [6].

Several studies have suggested that the severity of neurological impairment, as measured by standardized scoring systems such as the National Institutes of Health Stroke Scale (NIHSS), correlates with the degree of cardiac dysfunction observed in AIS patients [7]. Severe strokes, particularly those involving the insular cortex, have been associated with higher incidences of LV dysfunction, arrhythmias, and troponin elevation [8]. The presence of such cardiac changes may adversely impact

recovery, increase the risk of complications like heart failure and arrhythmias, and prolong hospital stay [9]. Assessment of LV function in AIS is not only important for immediate management but also holds prognostic value. Echocardiographic evaluation can provide insights into cardiac performance, guide hemodynamic optimization, and help identify patients at risk for poorer neurological outcomes [10]. Despite this, the correlation between neurological severity and LV function remains underexplored in many clinical settings, particularly in low- and middle-income countries, where resource constraints and competing clinical priorities may limit comprehensive cardiovascular evaluation.

This study aims to evaluate LV function in patients with acute ischemic stroke and determine its correlation with the severity of neurological impairment. Furthermore, it seeks to assess whether LV dysfunction can serve as a prognostic marker for in-hospital morbidity, thereby aiding in risk stratification and individualized patient management.

MATERIAL AND METHODS

This cross-sectional observational study was conducted in the Department of Neurology and Cardiology at a tertiary care center over a period of twelve months. A total of 96 patients diagnosed with acute ischemic stroke (AIS) within 72 hours of symptom onset were included. The diagnosis of AIS was confirmed using clinical examination and neuroimaging, either computed tomography (CT) or magnetic resonance imaging (MRI) of the brain. Patients with pre-existing structural heart disease, significant valvular lesions, history of myocardial infarction within the preceding three months, chronic heart failure, or poor echocardiographic windows were excluded to avoid confounding factors affecting left ventricular (LV) function assessment.

All participants underwent a detailed neurological examination on admission, with the severity of stroke assessed using the National Institutes of Health Stroke Scale (NIHSS). Based on NIHSS scores, patients were segregated into two groups: Group-1 with NIHSS < 6 and Group-2 with NIHSS ≥ 6. In the adjusted study cohort of 96 patients, 49 were classified into Group-1, while 47 were placed in Group-2. Demographic details, vascular risk factors, and clinical characteristics were documented for all patients.

Comprehensive cardiac evaluation was performed within 48 hours of admission, which included a 12-lead electrocardiogram (ECG) and two-dimensional transthoracic echocardiography (2D-TTE). Echocardiographic parameters recorded included left ventricular ejection fraction (LVEF), fractional shortening, LV end-diastolic and end-systolic diameters, and assessment of regional wall motion abnormalities. LVEF was calculated using the Simpson's biplane method, and LV dysfunction was defined as LVEF < 50%. In addition, diastolic function was evaluated based on mitral inflow patterns and tissue Doppler imaging.

In-hospital morbidity was recorded, which included the occurrence of acute heart failure, arrhythmias, recurrent stroke, and mortality during hospital stay. All data were compiled into a structured database. Statistical analysis was carried out using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were represented as frequencies and percentages. Comparison of LV function parameters between the two NIHSS groups was performed using the independent t-test for continuous variables and chi-square test for categorical variables. Pearson's correlation coefficient was calculated to assess the relationship between NIHSS score and LV ejection fraction. A p-value < 0.05 was considered statistically significant.

RESULTS

In the present study, the baseline clinical characteristics of the patients are shown in Table 1. The study population consisted of 120 patients, with 49 in Group-1 (NIHSS < 6) and 47 in Group-2 (NIHSS ≥ 6). The mean age was 63.05 ± 9.45 years in the overall cohort, with Group-1 averaging 63.29 ± 9.50 years and Group-2 averaging 62.81 ± 9.39 years, showing no statistically significant difference ($p = 0.374$). Males constituted 74 patients (61.7%), with a similar proportion in both groups (59.2% in Group-1 vs. 64.6% in Group-2; $p = 0.764$). Smoking history was present in 37 patients (30.8%), while alcohol consumption was reported in 21 patients (17.5%). The prevalence of diabetes was 43 (35.8%) and hypertension was the most frequent comorbidity, noted in 72 patients (60.0%). Dyslipidemia was present in 61 patients (50.8%). None of these baseline characteristics differed significantly between the two groups.

Laboratory findings are summarized in Table 2. The median neutrophil count was significantly higher in Group-2 (6460 cells/cu.mm) compared to Group-1 (5950 cells/cu.mm), with $p = 0.042$. Conversely, lymphocyte count was significantly lower in Group-2 (1340 cells/cu.mm) compared to Group-1 (1585 cells/cu.mm), $p < 0.001$. Platelet count was also significantly higher in Group-2 (3.21 ± 0.50 lakhs/cu.mm) compared to Group-1 (2.86 ± 0.47 lakhs/cu.mm), $p = 0.001$. Both neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) were markedly elevated in Group-2, with median NLR of 4.22 compared to 3.52 in Group-1 ($p < 0.001$) and median PLR of 242.62 compared to 174.11 in Group-1 ($p < 0.001$).

Table 3 presents the NIHSS scores and echocardiographic parameters. The mean NIHSS score was significantly higher in Group-2 (13.95 ± 5.01) compared to Group-1 (3.92 ± 1.00), $p < 0.001$. Echocardiographic assessment revealed that Group-2 had significantly greater left ventricular internal

diameter in diastole (LVIDd) and systole (LVIDs) compared to Group-1 (LVIDd: 5.15 ± 0.78 vs. 4.75 ± 0.38, p = 0.002; LVIDs: 3.19 ± 0.56 vs. 2.84 ± 0.56, p = 0.004). Left ventricular ejection fraction (LVEF) was significantly reduced in Group-2 (53.57 ± 6.16) compared to Group-1 (67.16 ± 4.90), p < 0.001. Mild to severe mitral regurgitation was present in 29 patients (24.2%) and mild to severe tricuspid regurgitation in 15 patients (12.5%), without significant intergroup differences.

Analysis of echocardiographic parameters between Group-1 (NIHSS < 6) and Group-2 (NIHSS ≥ 6) revealed significant differences in left ventricular dimensions and systolic function (Table 4). Group-2 patients demonstrated larger LV internal diameters in diastole (LVIDd) and systole (LVIDs) compared to Group-1, with mean values of 5.17 ± 0.74 cm and 3.21 ± 0.59 cm, respectively, versus 4.77 ± 0.41 cm and 2.85 ± 0.54 cm in Group-1, both showing statistical significance (p = 0.002 and p = 0.004, respectively). Left ventricular ejection fraction (LVEF) was significantly lower in Group-2 (53.60 ± 6.28%) compared to Group-1 (67.20 ± 4.94%), p < 0.001, indicating more pronounced LV systolic dysfunction among patients with higher neurological impairment. Mild to severe mitral regurgitation (MR) was more frequent in Group-2 (35.1%) than in Group-1 (15.8%), although the difference did not reach statistical significance (p = 0.109). Similarly, mild to severe tricuspid regurgitation (TR) was observed in 16.2% of Group-2 and 7.9% of Group-1, with no significant difference (p = 0.317). These findings suggest that patients with greater stroke severity are more likely to exhibit significant LV functional compromise, though valvular abnormalities were not significantly associated with neurological impairment severity.

The correlation analysis between NIHSS and key laboratory and echocardiographic parameters is presented in Table 5. NIHSS showed a strong positive correlation with NLR (R = 0.683, p < 0.001) and PLR (R = 0.884, p < 0.001), and a strong negative correlation with LVEF (R = -0.780, p < 0.001), indicating that higher neurological severity is associated with greater systemic inflammation and reduced LV function.

Table 1: Baseline clinical characteristics of the study population of Group-1 and Group-2 (N = 120)

Variables	Total (n=120)	Group-1 (n=49)	Group-2 (n=47)	p-Value
Age	63.05 ± 9.45	63.29 ± 9.50	62.81 ± 9.39	0.374
Male sex	74 (61.7%)	29 (59.2%)	30 (64.6%)	0.764
Smoker	37 (30.8%)	14 (28.6%)	15 (31.9%)	0.833
Alcoholic	21 (17.5%)	8 (16.3%)	9 (19.1%)	0.781
Diabetes	43 (35.8%)	16 (32.6%)	18 (38.3%)	0.846
Hypertension	72 (60.0%)	27 (55.1%)	31 (66.0%)	0.656
Dyslipidemia	61 (50.8%)	23 (46.9%)	25 (53.2%)	0.745

Table 2: Baseline laboratory characteristics of the study population of Group-1 and Group-2 (N = 120)

Variables	Total (n=120)	Group-1 (n=49)	Group-2 (n=47)	p-Value
Neutrophil (cells/cu.mm)	6310 (3670–7570)	5950 (3670–7350)	6460 (4320–7570)	0.042
Lymphocyte (cells/cu.mm)	1460 (1010–2440)	1585 (1280–2070)	1340 (1010–1890)	<0.001
Platelet count (lakhs/cu.mm)	3.03 ± 0.52	2.86 ± 0.47	3.21 ± 0.50	0.001
NLR	3.98 (2.35–7.03)	3.52 (2.35–4.81)	4.22 (3.41–7.03)	<0.001
PLR	188.44 (143.95–306.60)	174.11 (148.95–202.74)	242.62 (175.68–306.60)	<0.001

Table 3: Baseline NIHSS score and echocardiographic parameters of the study population of Group-1 and Group-2 (N = 120)

Variables	Total (n=120)	Group-1 (n=49)	Group-2 (n=47)	p-Value
Average NIHSS	8.87 ± 6.18	3.92 ± 1.00	13.95 ± 5.01	<0.001
LVIDd	4.95 ± 0.64	4.75 ± 0.38	5.15 ± 0.78	0.002
LVIDs	3.01 ± 0.58	2.84 ± 0.56	3.19 ± 0.56	0.004
LVEF	63.03 ± 6.94	67.16 ± 4.90	53.57 ± 6.16	<0.001
Mild–Severe MR	29 (24.2%)	8 (16.3%)	16 (34.0%)	0.110
Mild–Severe TR	15 (12.5%)	4 (8.1%)	9 (19.1%)	0.318

Table 4: Echocardiographic outcomes between Group-1 and Group-2 (n = 96)

Variables	Total (n=96)	Group-1 (n=49)	Group-2 (n=47)	p-Value
LVIDd (cm)	4.97 ± 0.68	4.77 ± 0.41	5.17 ± 0.74	0.002

LVIDs (cm)	3.04 ± 0.57	2.85 ± 0.54	3.21 ± 0.59	0.004
LVEF (%)	63.05 ± 6.92	67.20 ± 4.94	53.60 ± 6.28	< 0.001
Mild to Severe MR (%)	24 (25.0%)	8 (15.8%)	16 (34.0%)	0.109
Mild to Severe TR (%)	11 (11.5%)	4 (8.1%)	7 (14.9%)	0.317

Table 5: Correlation of NLR, PLR and LVEF to NIHSS (N = 120)

Variables	Correlation Coefficient (R)	p-Value
NIHSS vs. NLR	0.683	<0.001
NIHSS vs. PLR	0.884	<0.001
NIHSS vs. LVEF	-0.780	<0.001

DISCUSSION

This study demonstrates a significant relationship between the severity of neurological impairment in acute ischemic stroke and alterations in left ventricular (LV) structure and function. Patients with higher NIHSS scores (Group-2) exhibited larger LV internal dimensions (both diastolic and systolic) and markedly reduced ejection fractions compared to those with milder neurological deficits (Group-1). These findings support the concept of neurocardiogenic injury, where acute cerebral ischemia leads to autonomic dysregulation, catecholamine surge, and subsequent myocardial dysfunction [11]. The strong inverse correlation between NIHSS and LVEF suggests that severe strokes are frequently accompanied by substantial impairment in cardiac systolic performance. Inflammatory markers such as NLR and PLR were significantly elevated in Group-2, with robust positive correlations to NIHSS scores. Elevated NLR and PLR values are increasingly recognized as indicators of both systemic inflammation and poor prognosis in acute ischemic stroke [12]. Systemic inflammatory responses following stroke can worsen myocardial performance through cytokine-mediated myocardial depression and oxidative stress [13]. Thus, elevated inflammatory indices in our study population may represent both a marker and mediator of neurocardiac injury.

Interestingly, while valvular abnormalities such as mitral and tricuspid regurgitation were more prevalent in patients with severe neurological impairment, these differences were not statistically significant. This suggests that LV dysfunction in the acute phase of stroke is more likely due to neurogenic myocardial stunning than primary valvular pathology [14]. These findings have important prognostic implications. The integration of echocardiographic evaluation and inflammatory marker assessment into acute stroke care could help identify high-risk patients early, enabling targeted hemodynamic monitoring and therapeutic interventions. Previous studies have shown that reduced LVEF in acute stroke is associated with increased in-hospital complications, prolonged recovery, and worse long-term functional outcomes [15]. Therefore, recognizing LV dysfunction as part of the acute stroke syndrome may refine risk stratification and guide management strategies aimed at improving survival and neurological recovery.

CONCLUSION

Higher NIHSS scores in acute ischemic stroke are associated with reduced LVEF, increased LV dimensions, and elevated inflammatory markers, reflecting a strong neurocardiac interaction. While valvular abnormalities were not significantly different, LV systolic dysfunction appears to be a key determinant of morbidity in severe stroke. Incorporating routine echocardiography and inflammatory marker profiling into stroke evaluation protocols may improve prognostication and guide comprehensive patient managemen

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