Section: Cardiology

Original Research Article

CORRELATION BETWEEN ADMISSION WHITE BLOOD CELL COUNT AND LEFT VENTRICULAR EJECTION FRACTION IN STEMI

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ABSTRACT

Background: ST-elevation myocardial infarction (STEMI) is associated with significant morbidity, mortality, and ventricular dysfunction despite advances in reperfusion therapy. Inflammation is key to myocardial injury and remodelling. This study evaluated the association between admission white blood cell count and left ventricular ejection fraction in patients with STEMI. **Materials and Methods:** This cross-sectional study included 60 patients with STEMI at a tertiary care centre, categorised by admission WBC count. Demographic and cardiovascular risk factors were recorded. Admission blood samples were analysed for WBC using an automated haematology analyser. LVEF was assessed using two-dimensional echocardiography and categorised as normal (>50%), mild dysfunction (40-50%), and moderate-to-severe dysfunction (<40%). **Result:** Most patients were >60 years old (40%) and male (80%). Anterior wall myocardial infarction was the most common (46.7%), with 43.3% presenting within <6 hours. Primary PCI was performed in 56.7% of the patients. A WBC count of 8000-11000 cells/mm³ was found in 50% of patients, while 53.3% had moderate-to-severe LVEF dysfunction (<40%). In patients with WBC counts <8000 cells/mm³, 70% had preserved EF (≥40%), whereas 65% of those with WBC counts ≥8000 cells/mm³ had reduced EF (<40%). A significant association was found between the WBC count and LVEF (p=0.011). Spearman correlation showed a weak but significant negative correlation between WBC count and EF% (r=-0.21, p=0.042). **Conclusion:** A higher admission WBC count was significantly associated with reduced LVEF in patients with STEMI. The admission WBC count may be a simple and useful biomarker for early risk stratification and ventricular dysfunction assessment.

INTRODUCTION

Cardiovascular disease is the leading cause of global mortality, with STEMI being a severe acute form. ST-Elevation Myocardial Infarction (STEMI) results from complete coronary artery occlusion after atherosclerotic plaque rupture, causing myocardial ischaemia and necrosis.[1,2] Despite advances in reperfusion therapy, especially primary PCI, STEMI is associated with significant morbidity, mortality, and long-term ventricular dysfunction.[3] Early coronary blood flow restoration is crucial to reduce myocardial damage and preserve cardiac function; however, many patients experience adverse ventricular remodelling, heart failure, and recurrent events post-reperfusion.[2] Early risk stratification is vital in STEMI management to identify high-risk patients. Inflammation plays a central role in the pathophysiology of STEMI, affecting myocardial injury and remodelling. Atherosclerotic plaque rupture involves inflammatory cell infiltration, releasing enzymes and mediators that destabilize the fibrous cap and promote thrombosis.[4]

After coronary occlusion, inflammation is associated with leukocyte activation, cytokine release, oxidative stress, endothelial dysfunction and microvascular injury. Neutrophils infiltrate the ischaemic myocardium, releasing cytokines such as tumour necrosis factor-α and transforming growth factor-β, worsening injury, and promoting fibrotic remodelling.[4] Excessive inflammation is linked to infarct expansion and adverse remodelling, increasing the risk of heart failure and mortality after post-STEMI.[5] Systemic inflammation reflects ongoing myocardial activity; therefore, haematological inflammatory markers are gaining attention for STEMI prognosis. Among these, the WBC count is a simple, inexpensive, and routinely available parameter that can be rapidly assessed on admission. Elevated WBC count correlates with

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larger infarct size, impaired reperfusion, adverse remodelling, and increased mortality in acute coronary syndromes.[6] Leukocytes reflect the inflammatory response intensity and myocardial injury severity. Activated leukocytes contribute to microvascular obstruction through endothelial damage, oxidative stress, and prothrombotic mediator release, worsening ischaemic injury, and impairing recovery.

Studies have highlighted the prognostic significance of an elevated admission WBC count in patients with STEMI undergoing PCI. A study by Reindl et al. found that increased WBC levels were significantly associated with larger infarct size and reduced LVEF post-reperfusion therapy.[6] Higher WBC counts correlate with increased heart failure, arrhythmias, and mortality following STEMI.[7] Inflammatory indices, such as the neutrophil-to-lymphocyte ratio, predict adverse cardiovascular outcomes.[8] Changes in WBC count during hospitalisation are linked to long-term mortality, underscoring the impact of inflammation on post-infarction outcomes.[9] LVEF is crucial for cardiac systolic function and prognosis after STEMI. Reduced LVEF indicates myocardial damage, which is linked to heart failure, cardiogenic shock, recurrent ischaemic events, arrhythmias, and increased mortality. Despite therapy improvements, reduced LVEF remains associated with significant morbidity and poor survival.[10]

Inflammation may contribute to ventricular dysfunction after STEMI via myocyte necrosis, matrix degradation, oxidative injury, and fibrotic remodelling. Leukocytes release mediators and reactive oxygen species that exacerbate myocardial injury. Inflammatory biomarkers like WBC count may indicate post-infarction ventricular dysfunction. Identifying the early predictors of reduced LVEF can guide STEMI therapy. While studies have evaluated leukocytes as a prognostic marker, the link between WBC count and left ventricular function remains unclear. Previous studies have shown variable correlations due to population, timing, and treatment differences.[6,11] Most data from well-resourced systems limit applicability to developing settings lacking advanced imaging. Inexpensive markers, such as WBC count, may aid in early risk assessment. Given the role of inflammation, further evaluation of the association between WBC count and LVEF is needed to improve prognostic assessment and identify high-risk STEMI patients early. This study evaluated the association between these factors in patients with STEMI.

Aim and Objectives

This study aimed to evaluate the association between early WBC count and LVEF in patients with STEMI, determine the correlation between admission WBC count and LVEF, and compare LVEF across different WBC levels.

MATERIALS AND METHODS

This hospital-based cross-sectional study included 60 patients with ST-elevation myocardial infarction (STEMI) who were admitted to a tertiary care centre. Ethical approval and written informed consent were obtained from the participants before the study initiation.

Inclusion and Exclusion criteria

Patients aged ≥18 years with ischaemic symptoms, ST-segment elevation on electrocardiogram, and elevated cardiac biomarkers within 48 h of symptom onset were included. Patients with chronic inflammatory diseases, active infections, haematological disorders, malignancies, chronic kidney disease, and other severe systemic illnesses were excluded.

Materials: The materials used in the study included standard monitoring equipment, electrocardiography, an automated haematology analyser, laboratory reagents for blood investigations, a two-dimensional echocardiography machine, an appropriately sized intravenous cannula, and all necessary emergency drugs.

Methods: Sixty patients were categorised into Groups I (<8000 cells/mm³), II (8000-11000 cells/mm³), and III (>11000 cells/mm³) based on their admission white blood cell count. Demographic details, including age, sex, and cardiovascular risk factors such as hypertension, diabetes mellitus, and dyslipidaemia, were recorded. Blood samples were collected at admission, and the total white blood cell count was measured using an automated haematology analyser. Left ventricular ejection fraction (LVEF) was assessed using two-dimensional echocardiography during hospitalisation and categorised as normal (>50%), mild dysfunction (40-50%), and moderate to severe dysfunction (<40%).

Statistical analysis: Data are presented as mean, standard deviation, frequency, and percentage. Variables were compared using Fisher's exact test for categorical data and Spearman's correlation analysis. Statistical significance was set at $P<0.05$, and the analysis was performed using IBM-SPSS version 21 (IBM-SPSS Science Inc., Chicago, IL, USA).

RESULTS

Among patients, most were >60 years (24, 40%), followed by 51-60 years (22, 36.7%), and ≤50 years (14, 23.3%). Males predominated with 48 (80%) while females were 12 (20%). Hypertension was absent in 36 (60%) patients and present in 24 (40%) patients. Diabetes was absent in 40 (66.7%) patients and present in 20 (33.3%) patients. Dyslipidaemia was absent in 32 (53.3%) and present in 28 (46.7%) patients [Table 1].

The anterior wall myocardial infarction was the most common, occurring in 28 (46.7%) patients, followed by inferior wall in 22 (36.7%) and lateral wall in 10 (16.7%). Time to presentation: 26 (43.3%) within <6

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hours, 20 (33.3%) between 6-12 hours, and 14 (23.3%) after >12 hours. Killip classification: Class I in 38 (63.3%), Class II in 14 (23.3%), and Class III-IV in 8 (13.3%). Primary PCI was performed in 34 (56.7%), thrombolysis in 18 (30%), and medical management alone in 8 (13.3%) patients. Heart failure was the most common complication 12 (20%), followed by arrhythmias 10 (16.7%) and cardiogenic shock 6 (10%) [Table 2].

Table 1: Distribution of demographic characteristics and comorbidities

			N (%)
Age (years)		≤50 years	14 (23.3%)
		51-60 years	22 (36.7%)
		>60 years	24 (40%)
Sex		Male	48 (80%)
		Female	12 (20%)
Comorbidities	Hypertension	Present	24 (40%)
		Absent	36 (60%)
	Diabetes Mellitus	Present	20 (33.3%)
		Absent	40 (66.7%)
	Dyslipidaemia	Present	28 (46.7%)
		Absent	32 (53.3%)

Table 2: Distribution of infarct characteristics, clinical presentation, treatment modalities, and complications

		N (%)
Infarct Location	Anterior wall MI	28 (46.7%)
	Inferior wall MI	22 (36.7%)
	Lateral wall MI	10 (16.7%)
Time to Presentation	<6 hours	26 (43.3%)
	6-12 hours	20 (33.3%)
	>12 hours	14 (23.3%)
Killip Class	Class I	38 (63.3%)
	Class II	14 (23.3%)
	Class III-IV	8 (13.3%)
Mode of Treatment	Primary PCI	34 (56.7%)
	Thrombolysis	18 (30%)
	Medical management	8 (13.3%)
Complications	Heart failure	12 (20%)
	Arrhythmias	10 (16.7%)
	Cardiogenic shock	6 (10%)

Among the study patients, 30 (50%) had a WBC count of 8000-11000 cells/mm³, 20 (33.3%) had <8000 cells/mm³, and 10 (16.7%) had >11000 cells/mm³. Moderate to severe LVEF dysfunction (<40%) was most common in 32 (53.3%) patients, followed by mild dysfunction (40-50%) in 16 (26.7%) patients, and normal LVEF (>50%) in 12 (20%) patients [Table 3].

Table 3: Distribution of white blood cell count and left ventricular ejection fraction categories

		N (%)
WBC Count (cells/mm³)	<8000	20 (33.3%)
	8000-11000	30 (50%)
	>11000	10 (16.7%)
LVEF Category	>50% (Normal)	12 (20%)
	40-50% (Mild dysfunction)	16 (26.7%)
	<40% (Moderate-severe dysfunction)	32 (53.3%)

Among patients with WBC counts <8000 cells/mm³, 14 (70%) had preserved left ventricular ejection fraction (≥40%), while 6 (30%) had reduced ejection fraction (<40%). Conversely, among patients with WBC ≥8000 cells/mm³, 26 (65%) had a reduced ejection fraction (<40%), and 14 (35%) had a preserved ejection fraction (≥40%). The association between WBC count and left ventricular ejection fraction was significant (p=0.011) [Table 4].

Table 4: Association between white blood cell count and left ventricular ejection fraction

		Left Ventricular Ejection Fraction		p-value
		Reduced EF (<40%) n (%)	Preserved EF (≥40%) n (%)	
WBC Category	<8000	6 (30%)	14 (70%)	0.011
	≥8000	26 (65%)	14 (35%)	

It shows a weak but statistically significant negative correlation between admission WBC count and LVEF (Spearman r = −0.21, p = 0.042). Higher WBC counts were associated with lower LVEF, suggesting that increased inflammatory response at admission may be linked to poorer left ventricular systolic

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function. However, the weak correlation ($r^2 \approx 4.4\%$) indicates that WBC count explains only a small proportion of LVEF variability, and the finding should be interpreted cautiously given the sample size of 60 patients [Figure 1].

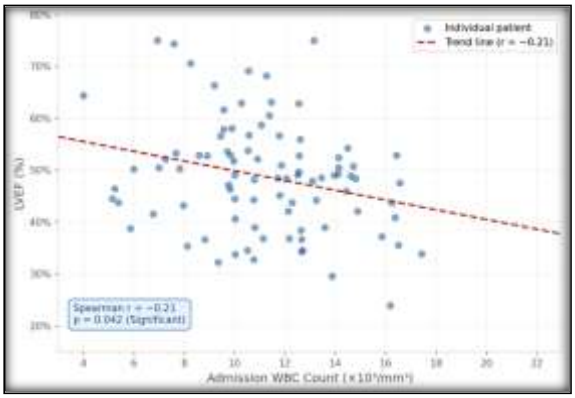

Figure 1: Correlation between admission white blood cell count and left ventricular ejection fraction.

DISCUSSION

Our study found a significant link between admission WBC count and LVEF in patients with STEMI. Elevated WBC counts correlated with reduced LVEF, with a significant negative correlation ($r = -0.21$, $P = 0.042$), indicating that increased inflammation was associated with poorer ventricular function. Although statistically significant, the observed correlation was weak, indicating that WBC count alone may not be a strong predictor of ventricular dysfunction. Most patients were over 60 years of age (40%), and 80% were male, similar to Arcari et al., who reported a mean age of 59 ± 12 years and 89% male among reperfused STEMI patients. Hypertension was present in 56%, dyslipidaemia in 46%, and diabetes in 15% of the population.[12]

Reindl et al. reported a mean age of 57 ± 10 years, with 64%, 62%, and 9% of patients with STEMI undergoing PCI having hypertension, hyperlipidaemia, and diabetes, respectively.[6] Hussain et al. found 71.4% were male, mostly aged 41-60.[11] The demographic profile of our study was comparable to that of previous studies on STEMI patients. The most common type of myocardial infarction was anterior wall myocardial infarction (46.7%), followed by inferior wall myocardial infarction (36.7%). Hussain et al. reported anterior wall infarction in 53.1% and inferior wall in 46.9% of STEMI patients.[11] The predominance of anterior infarction in both studies may explain the higher incidence of ventricular dysfunction due to the larger infarct size and impaired systolic function.

In our study, moderate-to-severe left ventricular dysfunction (LVEF <40%) was observed in 53.3% of patients, which was higher than Hussain et al.'s 32.7%.[11] Arcari et al. reported a median LVEF of 45% (40-53) among reperfused STEMI patients.[12] The higher proportion of reduced LVEF may be related to delayed presentation and increased inflammation. Patients with WBC count ≥8000 cells/mm³ had a significantly higher prevalence of reduced EF (<40%) than those with WBC count <8000 cells/mm³ (65% vs. 30%; p=0.011). This aligns with Hussain et al.'s finding of a significant negative correlation between WBC count and ejection fraction (Pearson correlation = -0.462, p=0.001).[11]

Reindl et al. showed that peak WBC count correlated with infarct size (r=0.31 to 0.41; P<0.01) and inversely with LVEF (r=-0.29 to -0.39; P<0.01) in the acute and chronic stages post-STEMI, supporting that increased inflammation contributes to myocardial injury and dysfunction.[6] Ferrari et al. showed that the admission leukocyte count correlated with creatine kinase (r=0.256, p=0.021) and troponin (r=0.247, p=0.026) levels, indicating that an elevated WBC count reflects a larger infarct size and necrosis.[13] Purayil et al. reported 78.5% of patients with moderate/severe LV dysfunction had raised WBC counts at presentation, while 76.6% with poor LV function during follow-up also had elevated leukocyte counts.[14]

The prognostic significance of elevated WBC counts has been highlighted in clinical outcome and mortality studies. Starčević et al. reported elevated admission WBC count (>11000/mm³) in 57.14% of STEMI patients undergoing PCI, observing lower ejection fraction, higher creatine phosphokinase, and greater heart failure incidence.[15] Patients with elevated WBC counts had a three-fold higher 30-day mortality risk and a two-fold higher one-year mortality risk. Tashchuk et al. showed that STEMI patients with reduced LVEF had significantly higher leukocyte counts (p<0.05), neutrophil counts (p<0.01), neutrophil-to-lymphocyte ratio (p<0.01), and systemic immune inflammation index levels (p<0.01), explaining the link between inflammatory markers and ventricular dysfunction.[8]

Our study supports the evidence that the admission WBC count reflects the inflammatory response intensity and myocardial injury severity in STEMI. Elevated leukocyte counts may be associated with inflammatory and microvascular mechanisms contributing to impaired left ventricular systolic function. As WBC count is inexpensive, available, and routinely measured at admission, it may serve as a practical biomarker for early risk stratification and identification of STEMI patients at risk of ventricular dysfunction and adverse cardiovascular outcomes.

CONCLUSION

This study showed a significant association between admission white blood cell count and left ventricular ejection fraction in patients with STEMI. Higher WBC counts were associated with lower LVEF, suggesting a relationship between inflammatory response and impaired ventricular function. Spearman correlation analysis showed a weak but significant negative correlation between WBC count

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and LVEF. These findings support the potential role of inflammatory processes in myocardial injury and ventricular dysfunction following STEMI. As an inexpensive and routinely available laboratory parameter, admission WBC count may provide useful prognostic information and assist in early risk stratification, particularly in resource-limited settings. However, given the weak correlation and relatively small sample size, larger prospective studies are required to validate these findings and further determine the prognostic utility of WBC count in predicting post-STEMI outcomes.

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