

**Prognostic Importance of WBC Count in Acute Myocardial Infraction**

<sup>1</sup>Dr. Vivek Lakhawat, <sup>2</sup>Dr. Kamlesh Goyal, <sup>3</sup>Dr. Subhash Chandra

<sup>1-2</sup>Senior Resident, <sup>3</sup>Assistant Professor (Geriatric Medicine)

<sup>1-2</sup>Department of General medicine, Dr. S N Medical College, Jodhpur

<sup>3</sup>Sardar Patel Medical College, Bikaner

**Corresponding Author:** Dr. Subhash Chandra, Assistant Professor (Geriatric Medicine), Sardar Patel Medical College, Bikaner

**Citation this Article:** Dr. Kamlesh Goyal, Dr. Vivek Lakhawat, Dr. Subhash Chandra, “Prognostic Importance of WBC Count in Acute Myocardial Infraction”, IJMSIR- February - 2020, Vol – 5, Issue -1, P. No. 43 – 45.

**Type of Publication:** Original Research Paper

**Conflicts of Interest:** Nil

**Abstract**

**Background:** The objective of this study was to determine whether the WBC count is associated with in-hospital mortality & morbidity for patients with ischemic heart disease after controlling for potential confounders.

**Methods:** This study was prospective study. 100 patients presenting to Department of General medicine and cardiology, Dr. S N Medical College, Jodhpur within 24hrs with Acute MI and qualifying inclusion criteria were enrolled and written consent was taken regarding participation in the study.

**Results:** Out of 100 patients 29 had LV dysfunction, among these 29 patients 19 were having elevated WBC count and 10 had normal WBC count. Among 100 patients 15 had cardiogenic shock, among these 15 patients 9 had elevated WBC count and 6 had normal WBC count. Among 100 patients 9 patients died, among these 9 patients 7 patients had WBC count and 2 patient had normal WBC count. Total 39 patients admitted for more than 7 days, among these patients 25 had elevated WBC count and 14 had normal WBC

count.

**Conclusion:** Elevated WBC count within 24 hours was associated with high rate of complications after acute myocardial infarction.

**Keywords:** ST segment elevation myocardial infarction (STEMI), Non ST segment elevation myocardial infarction(NSTEMI), White blood cell (WBC).

**Introduction**

Introduction Coronary artery disease is the major cause of death in most modern societies across the globe. In addition, the disease leads to high morbidity, disability and loss of productivity. The clinical symptoms of coronary heart disease contain a spectrum of silent ischemia to chronic stable angina, unstable angina, acute myocardial infarction, ischemic cardiomyopathy, sudden cardiac death, arrhythmias and cardiogenic shock. Currently, 900,000 people are diagnosed with acute myocardial infarction (AMI) in the United States each year, of those about 225,000 die due to arrhythmia or heart failure.<sup>1-5</sup> Systemic inflammation is triggered by myocardial infarction which is associated with the

release of hematopoietic precursor cells from bone marrow into blood stream.<sup>6,7</sup> Understanding the cellular changes during AMI could be practically prognostic.<sup>8</sup> Likewise, it has been shown that immunologic changes following AMI have prognostic value for severity of AMI; however, are independent of risk factors and number of arteries involved.<sup>9</sup> It has been show that there is a significant correlation between the ischemic severity and the magnitude of cellular changes as a consequence of acute phase response.<sup>10</sup>

**Methods**

100 patients presenting to hospital within 24hrs with Acute MI and qualifying inclusion criteria were enrolled and written consent was taken regarding participation in the study.

**Inclusion criteria**

Patients of age of more than 18 years with ST segment elevation acute myocardial infarction (STEMI) or non-ST segment elevation acute myocardial infarction (NSTEMI) on the basis of clinical history, examination, ECG changes and biochemical markers like Troponin T, CK-MB presenting to hospital within 24 hours.

**Exclusion criteria**

Patients with prior history of infarction, known case of cardiomyopathies were excluded. Similarly, presentation with fever, recent infection with 1 week, history of trauma, malignancy, myeloproliferative disorders, and recent surgical intervention that might have altered the leukocyte count, were excluded.

**Results**

In a total of 100 patients participated in our study 70 patients were male and 30 patients were females. This distribution shows the predominance of acute myocardial infarction among males. The mean age of patients was 52.36±12.30 Yrs.

Table 1: Association Between Wbc Counts And Omplications Of Acute Myocardial Infarction

Complications	WBC count <11000/mm <sup>3</sup> (n=50)	WBC count >11000/mm <sup>3</sup> (n=50)	Total	P-value
Left ventricular failure	10	19	29	<0.05
Cardiogenic shock	6	9	15	<0.05
Long hospital stay (>7days)	14	25	39	0.001
In hospital mortality	2	7	9	<0.05

Out of 100 patients 29 had LV dysfunction, among these 29 patients 19 were having elevated WBC count and 10 had normal WBC count. Among 100 patients 15 had cardiogenic shock, among these15 patients 9 had elevated WBC count and 6 had normal WBC count. Among 100 patients 9 patients died, among these 9 patients 7 patients had WBC count and 2 patient had normal WBC count. Total 39 patients admitted for more than 7 days, among these patients 25 had elevated WBC count and 14 had normal WBC count.

**Discussion**

The leukocyte response that occurs following AMI is a central part of the inflammatory reparative response that is initiated to replace the necrotic tissue with scar tissue. This may suggest that the greater the amount of necrosis, the larger the leukocyte response, an assertion based on experimental studies that show a direct relationship between the extent of necrosis and the level of both the local and the systemic leukocyte response<sup>11,12</sup>. In our study, we found that 50% patients were having high WBC count (>11000). Association between WBC count and acute cardiogenic shock first described by Friedman et al.<sup>13</sup> in 1974

In our study we found that high WBC count was associated with more complications like left ventricular dysfunction, cardiogenic shock, long hospital stay, in hospital mortality after myocardial infarction. Same results were found by Furman et al.<sup>14</sup>.

WBC count can be a useful biochemical tool for risk stratification of acute myocardial infarction. It is readily available and rather a cheaper investigation.

### Conclusion

Elevated WBC count within 24 hours was associated with high rate of complications after acute myocardial infarction.

### References

1. Gardini E, Caravita L, Ottani F, Ferrini D, Galvani M. Coronary care units: who to admit and how long. *G Ital Cardiol (Rome)*. 2007;8(5 Suppl 1):5S-11S.
2. Jones I, Flather M, Johnson M, Barrow S, Thompson D. A description of the characteristics of patients with non-ST elevation acute coronary syndromes admitted to different settings in the 1990s. *Intensive Crit Care Nurs*. 2008;24(5):286-94.
3. Saitto C, Ancona C, Fusco D, Arcà M, Perucci CA. Outcome of patients with cardiac diseases admitted to coronary care units: a report from Lazio, Italy. *Med Care*. 2004;42(2):147-54.
4. Jairath N. Strategies for motivating CCU patients. *Dimens Crit Care Nurs*. 1994;13(6):326-33.
5. Andreoli TE, Carpenter CJ, Griggs Benjamin II. *Cecil Essentials of Medicine*. 7th edition. W.B. Saunders. 2007.
6. Biasucci LM, Liuzzo G, Angiolillo DJ, Sperti G, Maseri A. Inflammation and acute coronary syndromes. *Herz*. 2000;25(2):108-12.
7. Wojakowski W, Tendera M. Mobilization of bone marrow-derived progenitor cells in acute coronary syndromes. *Folia Histochem Cytobiol*. 2005;43(4):229-32.
8. Rho YH, Chung CP, Oeser A, Solus J, Raggi P, Gebretsadik T, et al. Novel Cardiovascular Risk Factors in Premature Coronary Atherosclerosis Associated with Systemic Lupus Erythematosus. *J Rheumatol*. 2008; 35(9): 1789-94.
9. Al-Ahmad RS, Mahafzah AM, Al-Mousa EN. Immunological changes in acute myocardial infarction. *Saudi Med J*. 2004;25(7):923-8.
10. Dimitrijevic M, Vasiljevic Z, Vuckovic-Dekic L, Spasic S. The involvement of immune reactions in cardiac damage during acute myocardial infarction: role of cell-mediated immune response. *Panminerva Med*. 1997;39(2):85-94.
11. Lucchesi, B. R. (1990). Modulation of leukocytemediated myocardial reperfusion injury. *Annual Review of Physiology*, 52(1), 561-576.
12. Chatelain, P. A. S. C. A. L., Latour, J. G., Tran, D., De Lorgeril, M., Dupras, G., & Bourassa, M. (1987). Neutrophil accumulation in experimental myocardial infarcts: relation with extent of injury and effect of reperfusion. *Circulation*, 75(5), 1083-1090.
13. Friedman, G. D., Klatsky, A. L., & Siegelau, A.B. (1974). Leukocyte count and myocardial infarction: correction. *The New England journal of medicine*, 291(25), 1361-1361.
14. Furman, M. I., Becker, R. C., Yarzebski, J., Savegeau, J., Gore, J. M., & Goldberg, R. J. (1996). Effect of elevated leukocyte count on in-hospital mortality following acute myocardial infarction. *The American journal of cardiology*, 78(8), 945-948.