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EFFECT OF LEUCOCYTE COUNT ON LENGTH OF STAY AND IN HOSPITAL MORTALITY IN STEMI PATIENTS

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Contribution

UMB conceived the idea , designed the study and analyzed the data. JZ and NJ did data collection . RSATK and MKI did manuscript writing . MA did final review and approval. All authors contributed equally to the submitted manuscript.

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ABSTRACT

Objective: To evaluate association of total leucocyte Count (TLC) count with length of stay and in hospital mortality.

Methodology: This cross sectional study was conducted at Department of Cardiology, Jinnah Hospital Lahore from 1st October 2017 to 31st March 2018. Patients fulfilling the inclusion criteria were selected for the study. TLC count was taken at time of admission and after 48 hours. The cohort was followed up for 7 days for adverse outcome i.e. in hospital mortality. All the collected data was entered and analyzed on SPSS version 17.

Results: Total of 200 subjects were included in study. Mean age was 57.47, $\pm$ 11.942 years. About 61.0% were males. Mean hospital stay was 4.18 days $\pm$ 1.462 days. Mean TLC count at admission among patients who died was 11336.36 $\pm$ 4754.349. Mean TLC count after 48 hours was 15154.55 $\pm$2388.616. ($t = 7.226$, $p = .000$) Mean hospital stay among patients who died was 5.78+1.134. ($t = 12.920$, $p = .000$)

Conclusion: Mean TLC count and length of hospital stay is significantly associated with in patient mortality among STEMI patients.

Key Words: In hospital mortality, Hospital stay, TLC

INTRODUCTION

Cardiovascular disease (CVD) is the number cause of the death worldwide, estimated more than 17.9 million people died in 2015 and by 2030 this number is likely to grow to more than 23.6 million deaths per year.[1] Among Noncommunicable disease deaths, CVD accounts for most number of deaths.[2] In North America and Europe around 20 million patients present to emergency departments (ED) with symptoms probably suggestive of acute coronary syndrome (ACS).[3,4]

The relation between ACS and inflammatory process is multifaceted.[5] The atherosclerotic process of ACS is a chronic inflammatory disease on its own, while on the top of it an inflammatory process within the atherosclerotic plaque may lead to plaque rupture and subsequent thrombus formation with resultant myocardial ischemia.[6] The severity of this inflammatory surge determines the short- and long-term consequences of ACS.[7] The elevation of white blood cell (WBC) count in the background of an acute myocardial infarction (AMI) has been linked with adverse cardiovascular events, signifying not only a role pertaining to physiological process, but a pathologic basis.[8-10] In the recent times a special consideration had been given to WBC count as a potential risk stratification tool by the research workers because of ease to get its result, cost effectiveness, and widespread obtainability.

There has been anticipated several mechanisms for the justification of the association between WBC count and mortality in patients with AMI. The major constituent of the systemic inflammatory response to injury and the reparative mechanism is the leucocyte response after ST elevation myocardial infarction (STEMI), which then start the process of collagen replacing the infarcted tissue. The leucocyte response depends upon the extent of myocardial infarction; greater the area of the myocardial infarction, the greater the leucocyte response both locally as well as systemically.[11,12]

Even though previously an elevated WBC count after AMI, a gauge of systemic inflammation, has been acknowledged as part of the healing process, now it has often been revealed to be a prognosticator of hostile cardiovascular events. A study conducted by Nunez et al on 1118 consecutive patients admitted with the diagnosis of AMI: 569 non-STEMI and 549 STEMI. The white cells were measured after 24 hours of their admission in cardiac unit. Patients were categorized into 3 different groups depending on WBC level: WBC1 (count <10 x 103 cells/mL), WBC2 (count, 10-14.9 x 103 cells/mL), and WBC3 (count ≥15x103 cells/mL).The mortality rate was 18.5% in non-STEMI patients and 19.9% in STEMI patients.

This study was designed to establish the relation of elevated WBC-count with length of stay and in hospital mortality at 30 days and one year after STEMI in these pateints.[13]

METHODOLOGY

A cross sectional study was conducted at Department of Cardiology, Jinnah Hospital Lahore from 1st October 2017 to 31st March 2018.200 patients those fulfilling the inclusion criteria for STEMI were selected for the study. TLC count was taken at time of admission and after 48 hours. The subjects were followed up for 7 days for adverse outcome i.e. in hospital mortality. All the collected data was entered and analyzed on SPSS version 21.0. Mean and standard deviation was calculated for numerical variables and frequency and percentage were calculated for qualitative variables like history of diabetes mellitus, smoking, family history of MI. Independent t test statistics were used to compare in hospital mortality, TLC and length of hospital stay with $p < .05$ as statistical significance.

RESULTS

A total of 200 subjects included in study. Mean age was 57.47, $\pm$ 11.942, Minimum age was 23 years and maximum age was 79 years. 72.0% were fifty years and above. 61.0% were male and 39.0 % were females. 38.0% had history of diabetes mellitus and 40.0% had a positive family history of IHD. 39.0% were smokers. (Table no:1). Mean hospital stay was 4.18 days $\pm$ 1.462 days. 92.5% of subjects had hospital stay of less than 7 days. TLC at base line showed only 18.0% had a High (> 11,000 cumm) and at 48 hours 42.0% patients had High (> 11,000 cumm) TLC count. 27.5% patient died in hospital (table no:2). Mean TLC count at admission among patients who died was 11336.36 $\pm$ 4754.349. Mean TLC count after 48 hours was 15154.55 $\pm$2388.616. ($t = 7.226$, $p = .000$) Mean hospital stay among patients who died was 5.78$\pm$1.134. ($t = 12.920$, $p = .000$) (Table 3)

Table 1: Demographic and Clinical Profile of Subjects (n=200)

Variables		Frequency (n)	Percentage (%)
Age	Mean=57.47$\pm$ 11.942, Min=23, Maximum=79		
> 50 years		144	72.0
< 50 years		56	28.0
Gender			
Male		122	61.0
Female		78	39.0
Diabetes mellitus			
Yes		76	38.0
No		124	62.0
Family history IHD			
Yes		80	40.0
No		120	60.0
Smoking			
Yes		79	39.5
No		121	60.5

Table 2: Hospital Stay, TLC and Inpatient Mortality Status of Subjects (n=200)

Variables	Frequency (n)	Percentage (%)
Hospital stay days Mean=4.18±1.462, Min=3, Max=7		
< 7 days	185	92.5
> 7 days	15	7.5
TLC baseline		
High (> 11,000cumm)	36	18.0
Low (< 11,000cumm)	164	82.0
TLC 48 hours		
High (> 11,000cumm)	84	42.0
Low (< 11,000cumm)	116	58.0
In hospital mortality		
Yes	55	27.5
No	145	72.5

Table 3: In Hospital Mortality and TLC and Hospital Stay Association (n=200)

	Group Statistics				
	In hospital mortality	**n**	**Mean**	**Std. Deviation**	**T test P value**
TLC at admission	Yes	55	11336.36	4754.349	t =7.226
	No	145	7216.55	3056.823	P= .000
TL Caffer 48 hrs	Yes	55	15154.55	2388.616	t =12.480
	No	145	7990.34	3863.996	P= .000
Hospital staying days	Yes	55	5.78	1.134	t =12.920
	No	145	3.57	1.059	P= .000

DISCUSSION

Cardiovascular disease is the leading cause of death globally, accounts for nearly 836,546 deaths in the United states (US), it accounts for 1 of every 3 deaths in the US. Around 2,300 people of US die of CVD each day, an average of 1 death every 38 seconds.[1] Approximately 790,000 Americans have a heart attack every year, out of these 580,000 have first heart attack and about 210,000 people have recurrent heart attack.[14] Estimated more than 8 million patients with chest pain to emergency departments (ED) annually in US and out of these 20-25% diagnosed with ACS. It represents the second most common cause of adults visit in ED.[15] Although, 4% of patients with chest pain are of ST segment elevation myocardial infarction (STEMI), and up to 25% are of non-ST elevation acute coronary syndrome (NSTE-ACS).[16]

Inflammation have an injurious role in the context of ACS by promoting atherosclerotic plaque rupture and an evolving concept of changes in adaptive immunity is the main pathological basis of this plaque rupture.[17-19] One of the important mediators of inflammation are White Blood count Cells(WBCs). That's why WBC and its differential count have been studied to determine the outcome of CVDs. Among them Neutrophils bring atherosclerotic plaque rupture through the process of proteolytic enzymes release, arachidonic acid derivatives, and superoxide free radicals. Furthermore, in patients with ACS undergoing percutaneous coronary intervention (PCI), increased neutrophil counts due to iatrogenic injury to myocardium lead to poor functional recovery and extension of infarct size.[20-22] While on the other side of the picture, there are studies that proved that in patients with ACS the absolute and relative lymphocyte concentrations are significantly lesser and that's why such patients are at higher risk cardiac events in future.[23,24] In patients with ACS, the relative decrease in lymphocyte count is due tp stress factor. The increased (more than 2) admission neutrophil to lymphocyte ratio (NLR) is an independent predictor of increased in-hospital i.e., 7 days mortality (5.6 %).[25]

While conventionally an elevated white blood cell count (WBC), a predictor of systemic inflammation, has been acknowledged as acute myocardial infarction (AMI) healing phase response, it has been commonly revealed to be a predictor of adverse cardiovascular events after AMI. Our study was designed to assess the association between WBC and length of hospital stay and in-hospital mortality in AMI patients with ST-segment elevation (STEMI) in Pakistan. The results showed 27.5% patient died in hospital. Mean TLC count at admission among patients who died was 11336.36 ± 4754.349. Mean TLC count after 48 hours was 15154.55 +2388.616. (t =7.226, p= .000). Mean hospital stay among patients who died was 5.78±1.134. (t =12.920, p= .000). These results are consistent many studies which depicted predictive value of TLC in patients with ACS.

Nunez et al in his study found WBC on admission was an independent predictor of long-term mortality in both non-STEMI and STEMI patients Long-term mortality during follow-up was 18.5% in non-STEMI patients and 19.9% in STEMI patients.[14] Cannon CP et al in a study of 7,651 patients with ACS that a WBC of > 10,000 was related with increased 30-day and 10-month mortality (6.2% vs 3.2% to 3.6% for WBC count < 10,000; p < 0.000).[26] Barron HV et al found that WBC within 24 h of admission for an AMI is a strong and independent predictor of in-hospital and 30-day mortality as well as in-hospital clinical events.[10] Nunez J et al found N/L ratio as a useful marker to predict subsequent mortality in patients admitted for STEMI.[27]

In a study by Munir et al showed long-term mortality in patients with ACS was 6.4% in WBC1(< 7000/mm3), 18.2% in WBC2(7100-

10,000/mm3) and 40.9% in WBC3(> 10,000/mm3). categories, while short term mortality respectively was 2.6%, 3.0% and 18.2%. In comparison to patients with lower 2 WBC, patients with the highest category were 7 times more likely to die during 30 days (HR 7.83, $p = 0.017$) and more than 9 times during the total follow up period (HR 9.42, $p < 0.001$). Cox regression analysis showed WBC3 a strong independent predictor of mortality (HR 6.36, $p = 0.016$).28

CONCLUSION

Mean TLC count and length of hospital stay is significantly associated with in patient mortality STEMI patients.

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