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Original Research Article

QUANTIFICATION OF CRP, DIFFERENTIAL COUNT AND BLOOD SUGAR IN ACUTE CORONARY SYNDROME

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Abstract

Background: Acute Coronary Syndrome (ACS) refers to any condition attributed to obstruction of the coronary arteries which reduces blood flow to the heart. Predictors like blood sugar, WBC count, CRP are found to have a role in ACS disease and also with the severity of the disease. **Objectives:** To assess the extent of injury of myocardium by ejection fraction in acute coronary syndrome patients. To estimate the level of CRP, differential count, blood sugars in ACS patients. To assess the risk of morbidity & mortality in ACS patients by studying the effects of these parameters of CRP, differential count & blood sugars with ejection fraction. **Materials and Methods:** A Prospective observational study was conducted among 100 patients, between 2021 to 2022, at Govt Thiruvarur Medical College and Hospital. Based on inclusion and exclusion criteria, participants were selected and subjected to investigation. Data was collected, entered in MS Excel and analysed using SPSS. Appropriate statistics were done. **Results:** Mean age of the patients was 52 years and 79% were males. About 60% had raised JVP, 53% had lung signs, 43% had symptoms of pulmonary edema and 42% patients had symptoms of decreased urine output. ACS patients with increased random blood sugars had reduced Ejection fraction when compared to non-hyperglycemic patient. Serum level of inflammatory marker CRP is elevated in Acute coronary syndrome patients with reduced Ejection fraction. There is leucocytosis in ACS patient with reduced myocardial ejection fraction. If these parameter is elevated in ACS patients means that the ejection fraction was less than 50%. **Conclusion:** It is concluded that quantification of the three parameter namely CRP, WBC Count, Random Blood Sugar in acute coronary syndrome patients will predict the morbidity and mortality and helps roughly, to estimate the ejection fraction without echo cardiogram.

INTRODUCTION

Acute Coronary Syndrome (ACS) refers to any condition attributed to obstruction of the coronary arteries which reduces blood flow to the heart. The consequences depend on the degree and location of the obstruction and range from unstable angina to non–ST-segment elevation myocardial infarction (NSTEMI), ST-segment elevation myocardial infarction (STEMI), and sudden cardiac death. Symptoms are similar in each of these syndromes (except sudden death) and include chest discomfort with or without dyspnea, nausea, and diaphoresis. Diagnosis is by ECG and the presence or absence of serologic markers.[1] The term ACS was adopted because it was believed to more clearly reflect the disease progression associated with myocardial ischemia. Unstable angina and myocardial infarction (MI) both come under the ACS.[2]

The 2016 Heart Disease and Stroke Statistics update of the American Heart Association (AHA) has recently reported that 15.5 million persons ≥20 years of age in the USA have CHD, whilst the reported prevalence increases with age for both women and men and it has been estimated that approximately every 42 seconds, an American will suffer for an MI. According to the American Heart Association (AHA), 785,000 Americans will have an MI this

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year, and nearly 500,000 of them will experience another.[3]

India has the highest burden of ACS in the world. The CREATE registry has provided contemporary data on 20,468 patients from 89 centers from 10 regions and 50 cities in India.[2] CAD occurs in Indians 5–10 years earlier than in other populations around the world and the major effect of this peculiar phenomenon is on the productive workforce of the country aged 35–65 years.[4]

The prevalence of CAD and the incidence of ACS also are very high among Indians. India has the highest burden of ACS in the world. The rising incidence of ACS in Indians may be related to the changes in the lifestyle, the westernization of the food practices, the increasing prevalence of diabetes mellitus and probably genetic factors.[5,6]

Atherosclerosis has been increasingly recognized as a complex and multifactorial inflammatory disease rather being a simply process of lipid accumulation of the wall of the medium sized and large arteriesIt has been hypothesized that injury of the vascular wall leads to an inflammatory response that involves complex interactions between endothelial and smooth muscle cells, leucocytes and platelets1. Several epidemiological and clinical studies reported associations between the various circulating markers of inflammation, such as C- reactive protein, fibrinogen, adhesion molecules, cytokines, elevated leukocyte count and the different clinical manifestations of coronary heart disease.[2]

Elevated leukocyte count, a marker of inflammation, has long been identified as an independent predictor of an increased risk for long term mortality and myocardial infarction both in individuals without cardiovascular disease at baseline and in patients with established coronary artery disease (CAD).[3-8]

Recently, the differential leukocyte count and elevated neutrophil to lymphocyte (N/L) ratio have been the subject of interest in predicting the risk for future cardiovascular events. An elevated N/L ratio has been shown to independently indicate an increased long- term risk of mortality in patients with stable CAD and those with myocardial infarction and offer incremental prognostic value to total leukocyte count.[9-11] However, the relation between the differential leukocyte count, N/L ratio and the presence and severity of CAD has not been extensively studied. In most of the study, any one of the single parameters like total WBC count with prognosis or C reactive protein with cardiac injury or random blood sugar in acute coronary syndrome was study. But none of the study, typically assess the cardiac performance in the form of ejection fraction. In our study, we sought to investigate the relationship between the leukocyte subtypes and the presence and severity of CAD assessed in patients with suspected CAD. This study is aimed to investigate the amount of sugar in the blood, WBC count, CRP in ACS disease and to correlate with the severity of the disease. These parameter can be used to assess the cardiac performance like ejection fraction where there is scarcity of echo machine or non-availability of bed side ECHO, patients who are not able to shift to echo room, in that condition & can be utilized in Govt Taluk Hospital, Block PHC where echo is not available. The study of the effect of these factors provides a new step in the advancement of the treatment.

Objectives
1. To assess the extent of injury of myocardium by ejection fraction in acute coronary syndrome patients
2. To estimate the level of CRP, differential count, blood sugars in ACS patients
3. To assess the risk of morbidity & mortality in ACS patients by studying the effects of these parameters of CRP, differential count & blood sugars with ejection fraction.

MATERIALS AND METHODS

Place of study: Government Thiruvarur Medical College and Hospital
Study design: Prospective observational study
Study duration: 2021 to 2022
Sample size: This study is conducted in 100 patients of both sex with typical chest pain, admitted in the medicine department.

Inclusion Criteria
All Patients admitted with typical chest pain not relieved by rest, STEMI and NSTEMI with CPK-MB positive.

Exclusion Criteria
Those patients with known CAD history, severe renal or liver disease, hematologic disorders, infectious or inflammatory disease and patients on statin therapy were excluded in order to prevent the confounding anti-inflammatory effect of statin.

Method
After getting institutional ethical committee approval, Patients were taken for study who was admitted with history of typical chest pain, characterized by a pressure, tightness, squeezing, heaviness or burning and retrosternal location, after radiating to neck, jaw, shoulder or arms, sometimes epigastric regions associated with S4 gallop, mitral regurgitation, murmur, with S3 or rales. Severe ischemia and complication of myocardial infarction are evidenced by electrocardiogram and raised creatine phosphokinase-myocardium bound. (Diabetes was defined as use of insulin or oral hypoglycemics if glucose levels > 126 mg/dl. Hyperlipidemia was defined as serum total cholesterol concentration > 200 mg/dl, or serum LDL concentration $\geq$ 130 mg/dl. The patients with a history of hypertension and who were taking antihypertensive drugs were accepted as having hypertension).Then patients were categorized according to electrocardiogram, creatinine kinase-myocardium bound and duration of pain. They were classified as

Category I-ST segment elevated myocardial infarction
Category II-non STEMI
Category III-unstable angina

- For category I, treatment was started in the form of streptokinase injection 1.5 lakh IU by 1 hour who had elevated ST segment in electrocardiogram.
- Category II & III patients was treated with inj. low molecular weight heparin 5000 IU sixth hourly.
- Aspirin, clopidogrel, atorvastatin was given all three category patients.

After treatment was initiated, venous blood samples was collected & the following parameters such as random blood glucose ,cholesterol profile & renal function test were measured using standard techniques. Complete blood haemogram, total and differential leukocyte counts were measured with an automated Advia 2120 hematology analyzer (Roche). Serum CRP and other metabolic profile were measured at hospital arrival.

After patients was stabilized with proper treatment then patients was shifted to ECHO room for evaluating cardiac status from that I utilized ejection fraction to analyze the cardiac performance after myocardial injury in acute coronary syndrome. Then patients was assessed for failure symptoms in the form of pulmonary edema, reduced urine output and raised jugular venous pressure and blood pressure. These parameters were correlated with ejection fraction, differential count, raised blood sugar, C-reactive protein.

Statistical Analysis

Pearson Chi-square tests were used to compare the incidence of categorical variables among groups. Categorical variables were presented as counts and percentages. The two independent sample t tests were used to compare continuous variables between the two groups. Continuous variables were presented as mean (standard deviation) or as median (ranges). Data were entered in excel sheet and calculations by Chi square tests were done using SPSS software. Calculated p-values were considered statistically significant when they were <0.05.

RESULTS

A total of 100 ACS patients was included in the study.

Mean age of the patients was 52 years and most of them (30%) belong to age group of 46 to 55 years, followed by 36 to 45 years (27%), then by 56 to 65 years (23%) and other groups. About 79% were males. (Table 1)

Mean systolic blood pressure was 115.72 ± 17.40 & diastolic blood pressure 77.02±12.77. Mean pulse rate was 97.61 ±17.5. About 60% of ACS patients had raised JVP, 53% patients had lung signs, 43% patients had symptoms of pulmonary edema and 42% patients had symptoms of decreased urine output (Table 2)

About 73 % of the ACS patients had elevate level of CRP and remaining 27 % had normal level of CRP. About 70 % had elevated level of EF% and remaining 30 per cent had normal level. About 71% had elevated level of WBC and remaining 29 per cent had normal level. More than half (58 %) had elevated level of RBS and remaining 42 % had normal level of RBS. (Table 3, Figure 1)

The chi-square test indicates that out of 30 patients, vast majority (86.7 per cent) of the patients had elevated level of CRP when compared to EF%. Therefore, CRP will have more influence over EF%. Hence, the calculated value less than table value (.044<0.05). There is statistically significant association between CRP and their EF%. So, the research hypothesis (H1) is accepted. The chi-square test indicates that out of 30 ACS patients, vast majority (83.3 per cent) of the patients had elevated level of WBC when compared to EF%. Therefore, WBC will have more influence over EF%. Hence, the calculated value less than table value (.037<0.05). There is statistically significant association between WBC and their EF%. So, the research hypothesis (H1) is accepted. The chi-square test indicates that out of 30 patients, vast majority (83.3 per cent) of the patients had elevated RBS level when compared to EF%. Therefore, RBS will have more influence over EF%. Hence, the calculated value less than table value (.001<0.05). There is statistically significant association between RBS and their EF%. So, the research hypothesis (H1) is accepted. (Table 4)

The Karl Pearson Co-efficent Correlation test indicates that there is statistically significant relationship between CRP (-0.269**), WBC (-0.217*), RBS (-0.461**) and their EF%. Hence, the calculated value less than table value (p<0.05). So, the research hypothesis (H1) is accepted. (Table 5).

Table 1: Basic Characteristics of participants

Parameter	Category	N	%
Age	Below 35yrs	9	9.0
	36 to 45yrs	27	27.0
	46 to 55yrs	30	30.0
	56 to 65yrs	23	23.0
	66 to 75yrs	8	8.0
	76 to 85yrs	3	3.0
	Mean	52 years	SD 12 years
Gender	Male	79	79.0
	Female	21	21.0

Table 2: Vital signs and symptoms of participants

Parameter	Category	N	%
Systolic BP (mmHg)	Mean	116	SD 18
Diastolic BP (mmHg)	Mean	78	SD 13
Pulse rate (/min)	Mean	98	SD 18
JVP	Absent	40	40.0
	Present	60	60.0
Lung signs	Absent	47	47.0
	Present	53	53.0
Pulmonary edema	Absent	57	57.0
	present	43	43.0
Decreased Urinary Output	Absent	58	58.0
	Present	42	42.0

Table 3: Predictors and markers status among the participants

Parameter	Category	N	%
CRP	Normal	27	27.0
	Elevated level	73	73.0
EF%	Abnormal	30	30.0
	Normal	70	70.0
WBC	Normal	29	29.0
	Elevated level	71	71.0
RBS	Normal	42	42.0
	Abnormal	58	58.0

Table 4: Association between makers and ACS

Parameter		EF%						p value
		Elevated		Normal		Total		
		n	%	n	%	n	%	
CRP	Normal	4	13.3%	23	32.9%	27	27.0%	Chi square value 4.06, p value 0.044*
	Elevated	26	86.7%	47	67.1%	73	73.0%	
WBC	Normal	5	16.7%	24	34.3%	29	29.0%	Chi square value 4.28, p value 0.037*
	Elevated	25	83.3%	46	65.7%	71	71.0%	
RBS	Normal	5	16.7%	37	52.9%	42	42.0%	Chi square value 11.29, p value 0.001*
	Abnormal	25	83.3%	33	47.1%	58	58.0%	
*p value <0.05 – Statistically significant association is present								

Table 5: Correlation

EF%	Correlation value	Level of Significance
CRP	-0.269**	.007<0.01
WBC	-0.217*	.030<0.05
RBS	-0.461**	.000<0.01
** Correlation is significant at the 0.01 level, * Correlation is significant at the 0.05 level		
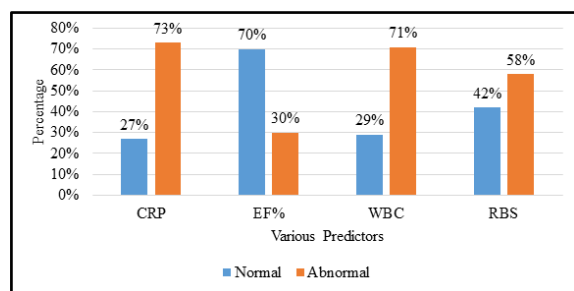
Figure 1: Status of Predictors in ACS patients

DISCUSSION

In our study, we have quantified the effects of c-reactive protein, white blood cells, random blood sugars, ejection fraction in Acute Coronary Syndrome patients. Depending on the patients load in our emergency department we have taken a total of 100 patients which include 79 males & 21 females in the mean age of 51.50 ± 11.66.

Recent studies have found that inflammation plays a important major role in atherosclerosis and Acute Coronary Syndrome. CRP belongs to the pentraxin protein family and is synthesized in hepatocytes and some extrahepatic tissues, such as vascular smooth muscle, atherosclerotic plaques, intracardial tissues. C-reactive protein is a product of inflammation whose synthesis by the liver is stimulated by cytokines in response to an inflammatory stimulus12. In our study we found that the serum level of these C-reactive protein was higher in ACS patient. This result was in consistent with other studies done by Dubey Rk etal. (2013), they gave the similar report that serum levels of CRP are higher in patients with ACS.[12]

This study was supported by the study conducted by Inder S et al 2004 they performed a retrospective analysis of the predictive value of baseline CRP level

which is measured in heart failure patient. Higher level are associated with features of more severe heart failure and are independently associated with mortality and morbidity.[13] According to him, Interleukin-6 is the primary determinant of the hepatic production of CRP and is produced in monocytes/macrophages, endothelial cells, vascular smooth muscle cells, fibroblast and cardiac myocytes under hypoxic stress.[14] left ventricular, hepatic and renal organ damage induced by low cardiac output, hypoperfusion, hypoxia and venous congestion causes increase in interleukin-6 wich in turn leads to increased CRP production.[15]

We also found that there is significant association between CRP and ejection fraction, that is patient with low ejection fraction showed significant raise in serum CRP when compared to normal ejection faction. Similar result was obtained by the study conducted by Christian stump et al in 2017 correlated the CRP level and risk of developing significant heart failure in patients with acute STEMI.[16] But this was opposed by Kennon S etal in 2003, they conclude that CRP measurement provides only little incremental prognostic information. there is no evidence that CRP is helpful for identifying groups who benefit from particular treatment in ACS. They suggest that only ECG changes and troponin measurement remain the principle tools for risk stratification and there is no evidence that CRP measurements provide additional independent information.[17]

In patients with ST-elevation MI, rise in hsCRP concentration is related to myocardial damage extent, outcomes and complication risk. According to Chan and Ng, early post infarction related rise in hsCRP is significantly and independently from other prognostic markers related to higher risk of cardiac (heart rupture, ventricular aneurysm, thrombus formation) and early mechanical complications, but does not prognosticate reinfarction . However, new coronary events after MI should be prognosticated only after hsCRP concentration returns to basal level (in 12 weeks), since primary rise in hsCRP concentration reflects acute inflammatory reaction to myocardial damage.[6]

Shah et al. investigated the prognostic value (discrimination expressed by sensitivity, specificity, AUC, calibration, calibration, reclassification) of hsCRP as the CVD risk factor in these prospective studies: NPHS-II and EAS. Later the systematic review of prospective studies (31 studies, population of 84,063, 11,252 coronary events), investigating hsCRP and CVD relations, has been done. Multifactorial analysis models prove additional clinical benefit of hsCRP to be minimal.[18] A systematic review by Schnell-Inderst et al. has analyzed the prognostic value, effectiveness and costs-benefit ratio of hsCRP together with traditional CVD risk factors and shown that there were not enough data that additional hsCRP testing would improve assessment of CVD risk and patient outcomes. Though a prognostic value of such combined models increases, it remains unclear,

whether the increment is clinically relevant. Concerning the costs-benefit ratio, hsCRP evaluation becomes significant when selecting asymptomatic patients with increased hsCRP and normal LDL cholesterol for treatment with statins.[19] Furthermore, a recent analysis by Chew et al shows that CRP predicts the risk of death or MI at 30 days among patients undergoing percutaneous coronary intervention. In this setting, the risk associated with elevated CRP was independent of, but additive to, the effect of an increased American College of Cardiology/American Heart Association lesion score.[20] The optimal cutoff point for defining high CRP levels among patients with ACS remains to be determined. The CAPTURE group found that a threshold of 10 mg/l maximized the predictive value of CRP.[21] Several other investigators have used a cutoff point of 3 mg/l for patients with ACS, while the reference ranges for primary prevention populations are lower.[22] The precise cause of these different thresholds remains unclear, but it is probably related to heightened vascular inflammation at the time of presentation with ACS.

High levels of CRP in ACS patients have been shown to be a good predictor for death but not AMI recurrence. Many studies have evaluated the association between outcomes and CRP concentrations post-AMI, or peak CRP concentrations on outcome. Acute phase levels of CRP at baseline prior to marked elevations of cTnI may prime the body to respond to any necrotic or injured tissue. This theory finds support by De Servi et al who suggest that in ACS populations there is a large variability in CRP concentrations, yet those with high CRP at baseline are perhaps more hyper-responsive to stimuli, including circulatingcTnI.[23] According to Kennon et al CRP levels were evaluated in patients with ACS and non-ST elevation on admission and after 12, 24 and 48 h; they shifted from a mean of 4.5 to one of 4.72, 7.79 and 9.99 respectively, with a statistically significant difference between patients taking and not taking aspirin prior to the onset of symptoms.[24] Furthermore, recent studies have suggested that CRP may play a direct role in promoting inflammatory atherosclerosis. CRP is not only a marker of systemic vascular inflammation but also plays an important key role in plaque disruption and subsequent thrombosis.[25] According to SK Thomas et al (2003), In ACS, the levels of troponin and CRP provide important, different, and complementary prognostic information. With increasing levels of any of the markers, there is a commensurate rise in mortality. At any detectable troponin, there is also a raised risk of a later MI. The combination of both markers allows the best prediction of mortality. The use of the combination of these markers will provide an important tool for the selection of patients for clinical trials and also for identification of patients for different treatment alternatives.[26] Another study demonstrated that a total WBC count in excess of 10 000 per/l was associated with a risk that was

approximately twice that seen when the WBC count was at or below 4000 per/l. This excess risk was independent of gender, smoking history, blood pressure, and cholesterol level.[27]

According to Hoffman et al in 2004 they suggest that Inflammation has been demonstrated to be an important risk factor for the development of cardiovascular events. Patients with elevated WBC counts have been shown to have a higher risk of developing an AMI and to be at higher risk for adverse events during the acute setting. In this review we reviewed the clinical data on the association between WBC count of AMI patients (on admission) and the prognostic outcome of these patients, we discussed possible and the possible correlation between high WBC count and the development of reperfusion injury, the no-flow phenomenon and congestive heart failure. It is possible that measuring WBC count, WBC sub-populations, cell adhesion molecule and cytokine levels should be used in order to help us to have a new and may be an improved way for risk stratification of patients admitted with AMI.[28]

Atherosclerotic plaque is characterized by infiltrates of monocytes /macrophages and lymphocytes which have transmigrated from the vascular space into the sub-endothelial layers of large and medium sized arteries.[29]In human ,myocardial necrosis begins in the sub-endocardium at 30-40 minutes after the onset of coronary occlusion. Reperfusion injury occurs due to increased fibrinolytic leading to restoration of coronary blood flow.[30] A large infract enhance cytokines synthesized & secreted by the monocytes & macrophages to induce the migration into the infracted region.[31] Next is early appearance of neutrophil in the infract zone with heavy infiltration by 1-3 days ,followed by infract healing and replacement fibrosis62.In our study, data showed that there is a strong association between leucocytosis and ejection fraction, which was made more effective by the previous study done by Tahil abmad munins et al in 2010, their study revealed that higher prevalence of total leucocytes and its sub types (i.e) neutrophils and monocyte in patients of ACS. Several experimental and clinical studies have provided compelling evidence that WBCs are important mediators of cardiac injury through release of proinflammatory cytokines. Imbalance in cytokine release has been demonstrated in patients with acute coronary syndromes in which a significant increase of interferon gamma and tumour necrosis factor-alpha production is observed accompanied by a significant decrease of interleukin- 10 production.[32] Myeloperoxidase (MPO) levels may be elevated among individuals with CAD. Myeloperoxidase is an enzyme secreted by a variety of inflammatory cells, including activated neutrophils, monocytes, and certain tissue macrophages, such as those found in atherosclerotic plaque. The enzyme is not released until leukocyte activation and degranulation.

Myeloperoxidase may convert LDL into a high-uptake form for macrophages, leading to foam cell formation, and may also deplete nitric oxide, contributing to endothelial dysfunction. In a recent case-control study, increasing levels of leukocyte-MPO and blood-MPO were significant predictors of the risk for CAD, such that after adjustment for white blood cell count and Framingham risk score, individuals in the highest quartile of blood-MPO had a 20-fold higher risk of CAD than individuals in the lowest quartile8. According to MADJID et al (2004) suggests that a high leukocyte count is associated with increased CHD related morbidity and mortality in various patient populations and clinical settings. It also appears to be an independent risk factor, regardless of atherosclerotic disease status. Thus, it may turn out to be a less expensive and more readily The increased risk of coronary thrombosis associated with leukocytosis, together with the association of UA with activation of circulating leukocytes, and the well-established risk of leukocyte plugging on reperfusion raise the question of the safety of stem cell therapies that rely on intracoronary infusion of leukocytes or systemic injections of granulocyte colony-stimulating factor or granulocyte macrophage colony-stimulating factor, which raise leukocyte counts. These approaches may increase the risk of thrombosis and may be more problematic than the small arrhythmogenic potential of sub-endocardial injections.[33]

According to Murtagh and Anderson et al(2004) , the higher level of myoperoxidase secreted by the activation and degranulation of leucocytes is an important prognostic factor in cardiac patient.[34] This was in consistent with our study.

As leucocyte assessments are cheap and routinely performed, some study suggested that it can be used for risk stratification. A study conducted by Antman Braunwald as well as Green et al suggested that leucocyte is a sensitive test for diagnosis of MI ,as it is associated with impaired perfusion in the epicardium and myocardium. This was supported by Hansen's study; they found that an elevated WBC count was accompanied by decreased epicardial & myocardial perfusion, thromboresistance and a higher rate of cardiac failure and death. According to Mueller et al hypercoagulability, endothelial dysfunction, necrosis of pro inflammatory myocytes and a no-reflow phenonmenon are all associated with leucocytosis, this is mainly due to reperfusion of the ischemic tissue causes neutrophils and platelet to form plaque in the microvasculature which causes loss of vascular reserve and enlargement of infract area leading to ventricular function and ventricular arrhythmias. Their findings showed that in the acute phase of MI, leucocytosis and neutrophilia are important predictor of heart failure-i.e patient with leucocytosis were 3.6 times more likely to develop heart failure, while those with neutrophilia had 4.29 times higher risk of developing heart failure.[35]

Our study result was in consistent with Samad Ghaffari study; they performed a single CBC analysis for the risk stratification in post STEMI complication patients. Similar result was obtained by Ratime

Eskandarian et al, they concluded that leucocytosis and neutrophilia in the acute phase of MI are important predictive factor for the development of LV systolic dysfunction. So leucocytosis can be used as risk stratification of ACS patients.[32]

Acute hyperglycemia on admission for acute coronary syndrome worsens the prognosis in patients with and without known diabetes. Postulated mechanisms of this observation include prothrombotic effects. The aim of this study was to evaluate the effect of elevated glucose levels on blood clotting in acute coronary syndrome patients.

We also got a strong association between hyperglycemia & ejection fraction .As EF decreases, the hyperglycemia increase. Our result was similar to the study conducted by Christophare et al. According to Jitender Mokta etal in 2017, Unrecognized diabetes and stress hyperglycemia at admission to coronary care unit in ACS patients increase the risk of cardiovascular events and intervention improves the outcome. This suggests that improved glucometabolic care reverse the negative effect of hyperglycemia on cardiovascular complications.

CONCLUSION

ACS patients with increased random blood sugars had reduced Ejection fraction when compared to non-hyperglycemic patient. So hyperglycemic status in ACS patient is an independent predictor of cardiovascular mortality & morbidity. Serum level of inflammatory marker CRP is elevated in Acute coronary syndrome patients with reduced Ejection fraction. There is leucocytosis in ACS patient with reduced myocardial ejection fraction. So above said three parameter are positively correlated with decreased ejection fraction .If these parameter is elevated in ACS patients means that the ejection fraction was less than 50%. So quantification of these three parameter namely CRP, WBC Count and Random Blood Sugar in acute coronary syndrome will predict the morbidity and mortality and helps roughly, to estimate the ejection fraction without echo cardiogram.

Limitation of the study
1. The sample size was small.
2. All complication of acute coronary syndrome patients was not included.
3. Correlation between the heart failure signs with these parameters was not done and justified.
4. Comparison of these parameters before & after treatment of ACS patient was done.

Conflict of Interest Nil
Funding support Nil

We also recommended the use of prophylactic betamethasone/dexamethasone injections around 34weeks of gestation to prevent the hazards of prematurity since it was the major finding in our study.

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