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C Reactive Protein as an Indicator of in Hospital Prognosis in Acute Myocardial Infarction

[1]Dr. Vidhyarahani, DNB (General Medicine), Assistant Professor, Department of General Medicine, Dhanalakshmi Srinivasan Institute of Medical Sciences and Hospital, Thuraiyur Road, Perambalur, 621212

[2]Dr. Thirumurugan Sivakumaar, DNB (General Medicine), Senior Resident Department of General Medicine, Karpagam Faculty of Medical Sciences and Research, Coimbatore, 641032

[3]Dr. Sathesh P, DNB (General Medicine), Assistant professor, Department of General Medicine, Annaporana Medical College and Hospital, Salem, 636308

[4]Prof. Dr. P. Jambulingam, M.D., (Gen. Med.), HOD, Department of General Medicine, Sri Ramakrishna Hospital, Coimbatore, 641044

Corresponding Author: Dr. Vidhyarahani, DNB (General Medicine), Department of General Medicine, Sri Ramakrishna Hospital, Coimbatore, 641044

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Abstract

The irreversible death (necrosis) of heart muscle due to prolonged lack of oxygen supply (ischemia) is Myocardial Infarction. The incidence rate of MI is 600 cases for every 1,00,000 people and is the leading cause of death throughout the globe. There are many prognostic markers for Myocardial Infarction and the easiest to detect sensitive biomarker is C-reactive Protein (CRP). CRP is a pentameric protein synthesized by the liver, whose level rises in response to inflammation. Since, Acute MI triggers an inflammatory response; CRP is inducted in the serum during MI. But, its role in prognosis and effectiveness as a marker to detect MI a rest ill in question. Hence, the present study aims at understanding the prognostic importance of CRP in acute MI patients with a sample of 214 patients admitted at Sri Ramakrishna Hospitals with acute MI. From the present study, the risk factors such as Hypertension, Diabetes, Smoking, Alcohol Consumption and Dyslipidemia are correlated with high levels of CRP. The CRP levels were also increased in patients with high BMI, lower ejection fraction and Killip Class above 2. Similarly, mortality was highly associated with high CRP levels, low Ejection fraction and Killip class above 2. Thus, the present study clearly lineated the role of CRP in

understanding the prognosis of acute MI. Hence, it is suggested to always measure CRP levels to clearly know the prognosis, since it is alsoeasy tomeasure.

Keywords: Myocardial Infarction, C - reactive protein, Hypertension, Dyslipidemia

Introduction

A heart attack or Myocardial Infarction (MI) is caused due to block in the heart that prevents flow of blood. The block is mainly because of fat or cholesterol of substances that forms a plaque in the arteries. The plaques may rupture and form a blood clot that prevents blood flow. Due to this block, heart muscle is damaged or destroyed. Depending upon whether the coronary artery is completely or partially blocked, two types of MI is reported namely STEMI and NSTEMI. During complete blockage of the artery leads to elevated ST segment which is termed as STEMI. During NSTEMI, the ST segment is not elevated because there is only partial blockage of the arteries. Another important cause of MI is spasm of coronary artery due to use of tobacco or illicit drugs such as cocaine. Myocardial infarction is usually fatal, but there is an exponential advancement in the treatment process over the years.

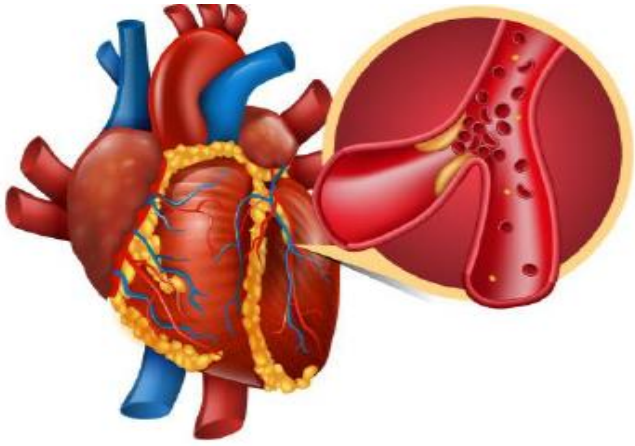


Figure 1: Blockage in the heart

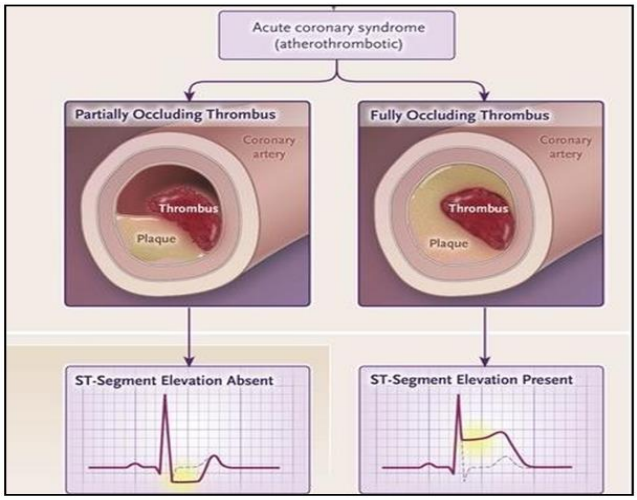


Figure 2: STEMI and NSTEMI

Risk Factors

The risk factors for MI may be inherited or acquired. The inherited risk factors for MI are Inherited High Blood pressure (hypertension), Inherited Dyslipidemia (Low levels of HDL, High levels of LDL or Triglycerides), inherited family history of heart diseases, aged men and Women, Prevalence of Type I Diabetes and Menopausal Women. The acquired risk factors for MI may be Stress, Sedentary Life style, Overweight, Individuals4who eat a diet high in saturated fat, Individuals with Type II Diabetes, Individual who smoke, Individuals who drink lot of alcohol, Individuals who have metabolic syndrome and Illicit Drug use

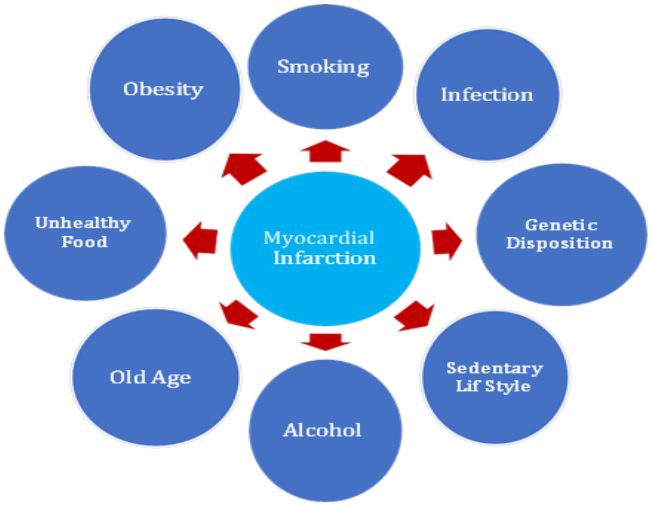


Figure 3: Risk factors for Myocardial Infarction

Objective

To find out the prognostic importance of C - reactive protein at admission in acute myocardial infarction during hospital course.

Material and Methods

Source of Data

214 consecutive patients presenting with acute coronary syndrome admitted to coronary care unit in Sri Ramakrishna hospital Coimbatore.

Study Time

December 2019 to December 2020.

Study Design

Prospective observational study

Sample Size

With prevalence rate of CHD in India[52] 13.2 % at a permissible error of 35 %, the size of sample was calculated to be 214, i.e. n=214

Using the Statistical formula,

n=4pq/L2

p = prevalence rate q = 1-p

L = Allowable error

Method of Collection of Data

The study was carried out on patients presenting with acute myocardial infarction presenting within 48 hrs.

Inclusion Criteria

All acute myocardial infarction patients having

- Chest pain lasting more than 20 minutes
- Diagnostic ECG changes with characteristic ECG alterations consisting of new pathological Q waves or ST segment and T wave changes.
- Elevated Hs trop i levels

Exclusion Criteria

- All patients with acute myocardial infarction presenting after 48 hrs.
- All patients with previous myocardial infarction
- All patients associated with any active infective or inflammatory and neoplastic condition.

Methodology

Qualifying patients were observed with detail history and clinical examination. Serum concentration of C-reactive protein was estimated at admission.

Statistical Analysis

The Collected data was analysed using SPSS Version 22. The Data was analyzed using following statistical methods

- Diagrammatic representation
- Descriptive Statistics (Mean ± standard deviation)
- Independent sample‘t’ test, One way ANOVA and Chi-Square test.
- Multivariate logistic regression tests to determine the association between C reactive protein levels with hospital mortality.

Result

The data collected were subjected to Statistical Analysis using SPSS version 22. Descriptive Statistics, Frequency analysis and Chi-Square tests were performed for appropriate variables. The probability value, p was defined as 0.05 to be ‘Significant’ and p value below 0.01 was considered ‘Highly Significant’ for all the significance tests. The results of the Statistical analysis are presented in subsequent tables.

Table 1: Age wise Distribution of the Patients

Age in years	Frequency	Percent	Cumulative Percent
<30	18	8.4	8.4
31-40	17	7.9	16.4
41-50	55	25.7	42.1
51-60	51	23.8	65.9
61-70	29	13.6	79.4
71-80	20	9.3	88.8

81-90	24	11.2	100.0
Total	214	100.0	

Graph 1: Age wise Distribution of the patients

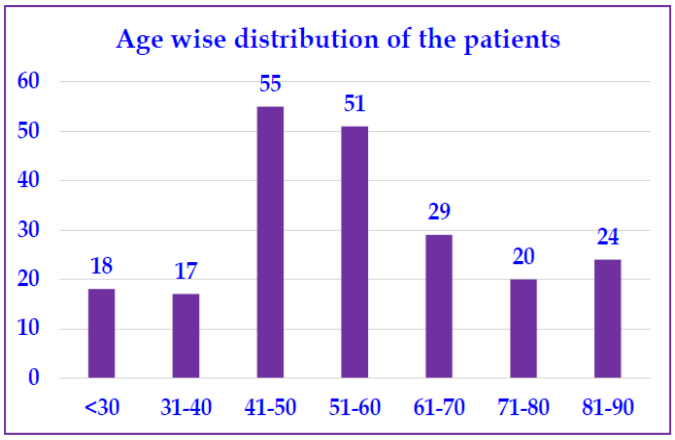


Table 2: Distribution of the Patients based on gender

Gender	Frequency	Percent	Cumulative Percent
Male	118	55.1	55.1
Female	96	44.9	100.0
Total	214	100.0	

Graph 2: Distribution of Patients based on gender

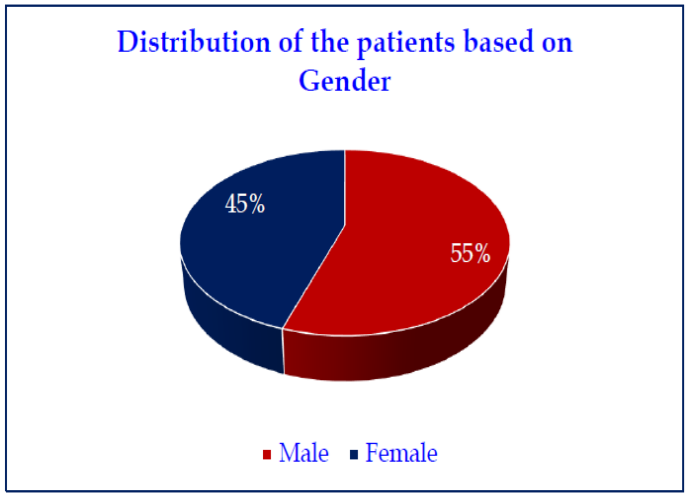


Table 3: Distribution of the patients based on presenting illness

Presenting Illness	Frequency	Percent	Cumulative Percent
Chest Pain	40	18.7	18.7
Breathlessness	37	17.3	36.0
Cough	35	16.4	52.3
Palpitation	34	15.9	68.2
Presyncope/syncope	42	19.6	87.9
Swelling of legs	10	4.7	92.5
Nausea/Vomiting	9	4.2	96.7
Other illness	7	3.3	100.0
Total	214	100.0	

Graph 3: Distribution of Patients based on Presenting Illness

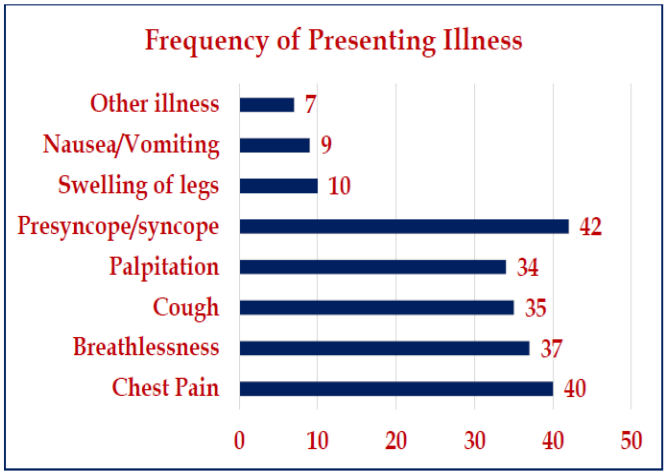


Table 4: Distribution of Patients based on past history of disease

Past Diseases	Frequency	Percent	Cumulative Percent
Hypertension	46	21.5	21.5
Diabetes	40	18.7	40.2
Rheumatic heart disease	18	8.4	48.6
Syphilis	15	7.0	55.6
Vascular Heart disease	18	8.4	64.0
TIA/Stroke	10	4.7	68.7
Others	14	6.5	75.2
None	53	24.8	100.0
Total	214	100.0	

Graph 4: Distribution of Patients based on past diseases

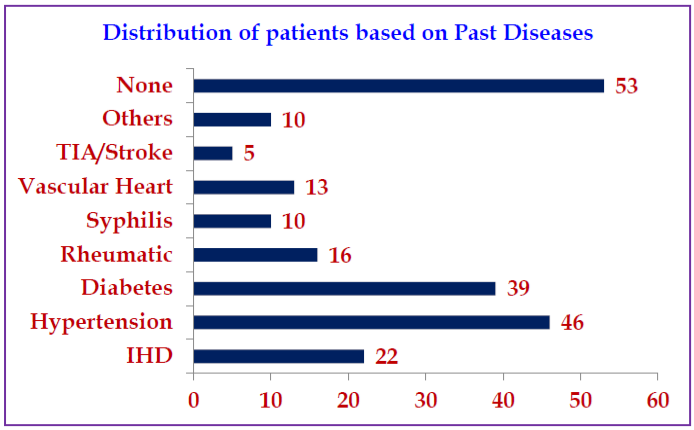


Table.5: Distribution of Patients based on prevalence of Hypertension

Hypertension	Frequency	Percent	Cumulative Percent
Yes	58	27.1	27.1
No	156	72.9	100.0
Total	214	100.0	

Graph 5: Distribution of Patients based on prevalence of Hypertension

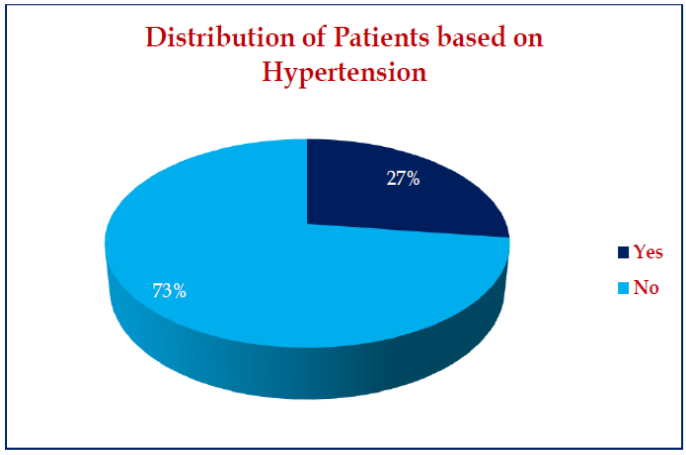


Table 6: Distribution of Patients based on Smoking Habit

Smoking Habit	Frequency	Percent	Cumulative Percent
Yes	61	28.5	28.5
No	153	71.5	100.0
Total	214	100.0	

Graph 6: Distribution of Patients based on Smoking habit

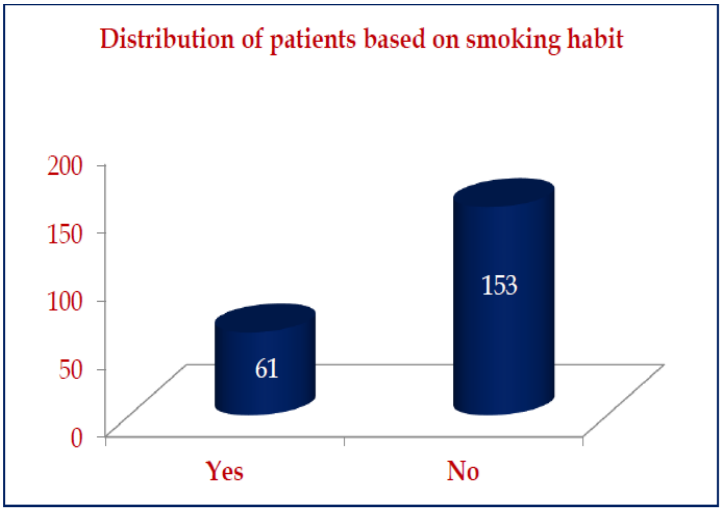


Table 7: Distribution of Patients based on their habit of alcohol consumption

Alcohol consumption	Frequency	Percent	Cumulative Percent
Yes	63	29.4	29.4
No	151	70.6	100.0
Total	214	100.0	

Graph 7: Distribution of Patients based on Drinking habit

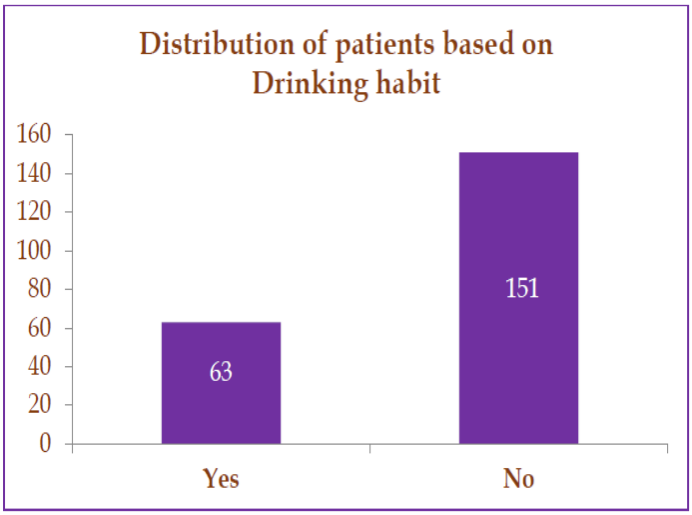


Table 8: Distribution of Patients based on prevalence of Dyslipidemia

Dyslipidemia	Frequency	Percent	Cumulative Percent
Yes	166	77.6	77.6
No	48	22.4	100.0
Total	214	100.0	

Graph 8: Distribution of Patients based on Dyslipidemia

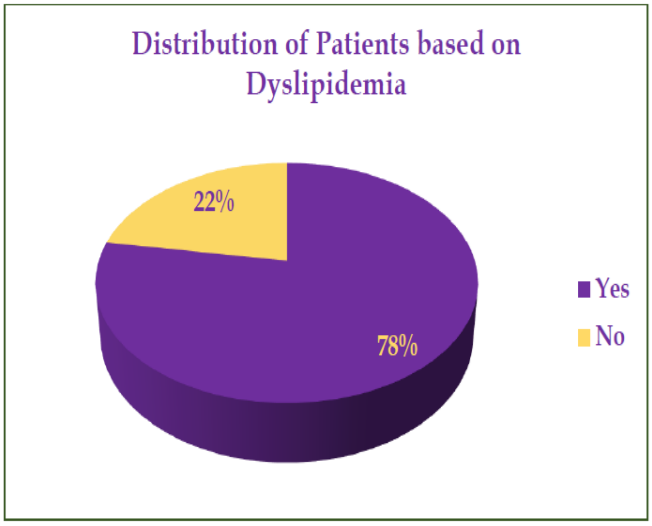


Table 9: Association between Mortality and CRP groups

Mortality	CRP Levels		Total	Chi-Square Statistic
	Group A (CRP<=3 mg/L)	Group B (CRP>3 mg/L)		
Yes	3	35	38	10.234
	7.9%	92.1%	100.0%	p<0.01
No	60	116	176	Highly Significant
	34.1%	65.9%	100.0%	
Total	63	151	214	
	29.4%	70.6%	100.0%	

Graph 9: Mortality and CRP groups

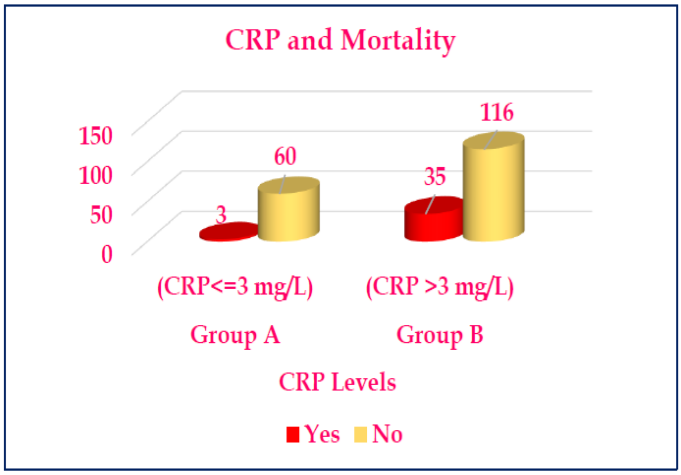
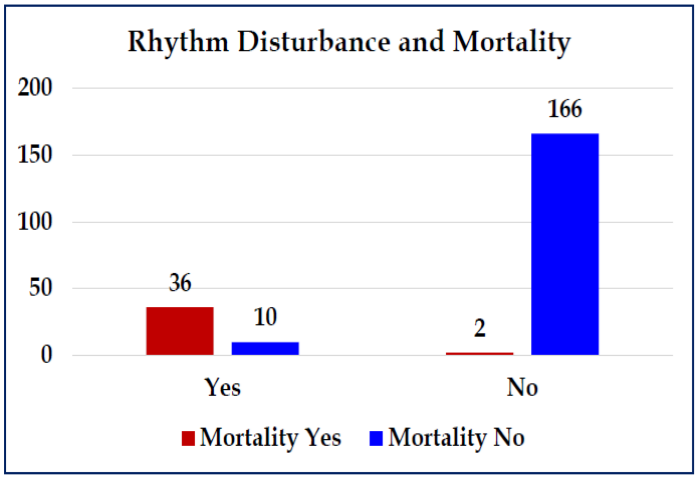


Table 10: Association between Rhythm Disturbance and mortality

Rhythm Disturbance	Mortality		Total	Chi-Square Statistic
	Yes	No		
Yes	36	10	46	146.879
	78.3%	21.7%	100.0%	p<0.01
No	2	166	168	Highly Significant
	1.2%	98.8%	100.0%	
Total	38	176	214	
	17.8%	82.2%	100.0%	

Graph 10: Rhythm Disturbance and Mortality



Discussion

Age

Majority of the patients (25.7%) in the study group are in the range of 41 to 50 years of age. This is similar to the study by Raju et al 18 where maximum patients were aged between 41 and 50 years of age.

Gender

In the study group, 55.1% of the patients are male and 44.9% of the patients are female. In contrast 78% of the patients were male and 22% were female in the study by Raju et al 18.

Presenting Illness

In the present study, 18.7% had chest pain, as against 94% in the Study by Raju et al 18. Similarly, in the study by Raju et al18 34% of the patients had breathlessness whereas 60 only 17.3% of the patients had breathlessness in the present study. 40% of the patients in the study by Raju et al18had committing and only 4.2% had nausea/vomiting in the present study.

Hypertension

27.1% of the patients in the study group had Hypertension as against 40% in the study by Raju et al [18]. Also 45% of the patients in the Maarten et al [21]

Smoking Habit

Smoking was the major risk factor in the study by Raju et al 18 as against 28.5% in the present study. Also 37% of the patients in the Maarten et al [21] study are smokers. But there is no significant association between smoking habit and CRP level.

Alcohol Consumption

70.6% of the patients do not consume alcohol and the remaining 29.4% consume alcohol. There is no significant association between drinking habit and CRP level and 69.8% of the patients who consume alcohol have CRP levels above 3 mg/L and majority of nondrinkers (70.9%) have CRP levels above 3 mg/L.

Dyslipidemia

77.6% of the patients have dyslipidemia and the remaining 22.4% do not have dyslipidemia. There is a significant association between prevalence of Dyslipidemia and CRP level and Majority of the patients with Dyslipidemia (75.9%) of the patients have CRP above 3 mg/L.

CRP levels

70.6% of the patients have CRP above 3 mg/L and the remaining 29.4% have CRP levels below 3 mg/L. CRP level in the study group is 5.957±3.287 which is much lower than the study by Toshihisa et al 19in which the mean CRP level was 14.1±11.3 mg/dL. Similarly, the CRP levels in the study by Maarten et al 21 was between 0.97 and 4.45 mg/L. The mean62CRP of Group A is 2.0719 ± 0.52572 and Mean CRP of Group B is 7.5792 ± 2.49859 andthere is a significant difference in the Mean CRP levels in the two groups.

Mortality

High C-reactive protein (CRP) levels have been associated with higher mortality ratein patients with acute myocardial infarction (AMI) as per the study by Berton et al 23. As perthe present study there is a significant association between CRP levels and Mortality 92.1%of the patients who encountered death had CRP levels above 3 mg/L which is very highwhen compared to only 4% in the study by Raju et al 18. Similarly, in the study by Lucci etal the mortality was only 2.2% in the CRP above 2mg/L group and 1.4% in the CRP below2mg/L group.

Conclusion

The present study clearly signifies the relationship between CRP levels andmortality. 92.1% of the patients who had high levels of CRP encountered death. Also, asCRP level increases there is a corresponding increase in mortality. There was a highlysignificant correlation between Mortality and CRP levels. Similarly, there was a highlysignificant correlation between Ejection fraction and CRP levels. It is suggested from thestudy it is mandatory to monitor the CRP levels systematically to prevent adverseconsequences. With advancement in techniques to measure CRP levels, it is always usefulto measure CRP level as a prognostic marker for In-Hospital patients.

References

1. Kushner I, Feldmann G. Control of the acute phase response. Demonstration of Creactive protein synthesis and secretion by hepatocytes during acute inflammation in the rabbit. J Exp Med 1978;148:466-77.
2. Oliveira EB, Gotschlich C, Liu TY. Primary structure of human C-reactive protein. J BiolChem 1979;254:489-502.
3. Oliveira EB, Gotschlich EC, Liu TY. Comparative studies on the binding properties of human and rabbit C-reactive proteins. J Immunol 1980;124:1396-402.
4. Merriman CR, Pulliam LA, Kampschmidt RF. Effect of leukocytic endogenous mediator on C-reactive protein in rabbits. ProcSocExpBiol Med 1975;149:782-4.
5. Osmand AP, Friedenson B, Gewurz H, Painter RH, Hofmann T, Shelton E. Characterization of C-reactive protein and the complement subcomponent C1t as homologous proteins displaying cyclic pentameric symmetry (pentraxins). ProcNatlAcadSci U S A 1977;74:739-43.
6. Hammerman H, Kloner RA, Hale F, Schoen FJ, Braunwald E. Dose-dependent effects of short-term methylprednisolone on myocardial infarct extent, scar formation and ventricular function. Circulation.1983; 68:446-452
7. Saffitz JE, Fredrickson RC, Roberts WC. Relation of size of transmural acute myocardial infarct to mode of death, interval between infarction and death and frequency of coronary arterial thrombus. Am J Cardiol.1986; 57:1249-1254.
8. Dellborg M, Held P, Swedeberg K, Vedin A. Rupture of the myocardium: occurrence and risk factors. Br Heart J.1985; 54:11-16.
9. Batts KP, Ackermann DM, Edwards WD. Postinfarction rupture of the left ventricular free wall: clinic pathologic correlates in 100 consecutive autopsy cases. Hum Pathol.1990; 21:530-535.
10. Mann JM, Roberts WC. Rupture of the left ventricular free wall during acutemyocardial infarction: analysis of 138 necropsy patients and comparison with 50 necropsy patients with acute myocardial infarction without rupture. Am J Cardiol.1986; 62:847-859.
11. Maarten Vanhaverbeke, Denise Veltman, NelePattyn, Nico De Crem, Hilde Gillijns, VéroniqueCornelissen, Stefan Janssens, and Peter R. Sinnaeve, C-reactive protein during and after myocardial infarction in relation to cardiac injury and left ventricular function at follow-up, ClinCardiol. 2018 Sep; 41(9): 1201–1206.
12. Nozari Y, Geraiely B. Correlation between the serum levels of uric acid and HSCRP with the occurrence of early systolic failure of left ventricle following acute myocardial infarction. Acta Med Iran. 2011;49(8):531-5. PMID: 22009810.
13. Berton G, Cordiano R, Palmieri R, Pianca S, Pagliara V, Palatini P. C-reactive protein in acute myocardial infarction: association with heart failure. Am Heart J. 2003 Jun;145(6):1094-101.
14. Tsujita K, Kaikita K, Soejima H, Sugiyama S, Ogawa H (April 2010). "[Acute coronary syndrome-initiating factors]" (in Japanese). Nippon Rinsho 68 (4): 607–14. PMID 20387549
15. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO 3rd, Criqui M, Fadl YY, Fortmann

SP, Hong Y, Myers GL, Rifai N, Smith SC Jr, Taubert K, Tracy RP, Vinicor F; Centers for Disease Control and Prevention; American Heart Association. (2003). "Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association"

16. A Fox, J Birkhead, R Wilcox, et al. Heart 2004 90: 603-609
17. Moe KT, Wong P (March 2010). "Current trends in diagnostic biomarkers of acute coronary syndrome". Ann. Acad. Med. Singap. 39 (3): 210–5.
18. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (The INTERHEART study): case-control study. Lancet 2004; 364:937-52.
19. Thygesen K, Alpert JS, White HD (October 2007). "Universal definition of myocardial infarction". Eur. Heart J. 28 (20): 2525–38. doi:10.1093/ eurheartj/ ehm355.PMID 17951287.
20. Mercado N, Poldermans D, Gardiens M, Vos J, Simoons ML. Acute myocardial infarction. Lancet 2003; 361: 847-58.
21. Thomson SG. Hemostatic factors and the risk of myocardial infarction or sudden death in patients with angina pectoris. N Engl J Med 1995; 332: 635-641.
22. Anzai T, Yoshikawa T, Shiroki H, Asakura Y, Akaishi M, Mitamura H, Ogawa S. C-reactive protein as a predictor of infarct expansion and cardiac rupture after a first Q wave acuteMI. Circulation 1997; 96:778-784.
23. Lagrand WK, Niessen JWM. C-reactive protein co localizes with complement in human hearts during acute myocardial infarction. Circulation 1997; 95:97-103.
24. Vulgari F, Cummins P. serum levels of acute phase and cardiac proteins after MI, surgery and infection. Br Heart J 1982; 48: 352-6.
25. Berk BC, Weintraub WS et al. Elevation of C-reactive protein in active coronary artery disease. Am J Cardiol 1990; 65: 168-72.
26. Petiala K, Harmoinen A. Intravenous streptokinase treatment and serum C- reactive protein in patients with acute myocardial infarction. Br Heart J 1987; 58:225-9.
27. Pietila K, Harmonien A. C-reactive protein in subendocardial and intramural myocardial infarction. clinical chemistry 1986; 32: 1596-7.
28. P Mishra Study of CRP as an indicator of prognosis in acute MI, GSVM medical college, Khanpur. JAPI vol Jan 2002; 50:36.
29. Guiseppe Berton, Rocco Cordiano, Rosa Palmieri. C-reactive protein in acute myocardial infarction: association with heart failure. American Heart Jounal 2003; 73:205 to 210.
30. S. Pandian, V Amuthan, P Sukumar. Plasma CRP level predicts left ventricular function and exercise capacity in patient with acute myocardial infarction. Indian heart journal 2005; 57:54-57.
31. Thiele JR, Habersberger J, Braig D, Schmidt Y, Goerendt K, Maurer V, et al. Dissociation of pentameric to monomeric C-reactive protein localises and aggravates inflammation: in vivo proof of a powerful proinflammatory mechanism and a new anti-inflammatory strategy. Circulation (2014) 130:35– 50.

32. Pepys MB, Hirschfield GM, Tennent GA, Gallimore JR, Kahan MC, Bellotti V, et al. Targeting C-reactive protein for the treatment of cardiovascular disease. Nature (2006) 440:1217–21.10.1038/nature04672
33. Slevin M, Matou S, Zeinolabediny Y, Corpas R, Weston R, Liu D, et al. Monomeric C-reactive protein – a key molecule driving development of Alzheimer's disease associated with brain ischaemia? Sci Rep (2015) 5:13281.10.1038/srep13281
34. Verma S, Szmitko PE, Yeh ET. C-reactive protein: structure affects function. Circulation (2004) 109:1914–7.10.1161/01.CIR.0000127085.32999.64