

**Original Research Article**

Serial Measurement of High Sensitive C Reactive Protein Levels of Patients Having Acute Chest Pain –Study in a Tertiary Care Centre of Western Odisha

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ABSTRACT

Hscrp as an inflammatory marker has been used as a non invasive method which is significantly increased in patients having chest pain diagnosed to be acute myocardial infarction. Hscrp has been received great attention now a day. According to a number of studies, hscrp concentrations more than 3mg/l is more associated with disease outcome and may worsen the prognosis in patients of acute coronary syndrome. High sensitive c reactive protein levels were measured in 150 patients attended to emergency ward of cardiology department of VIMSAR, Burla for chest pain. The mean age group was 50.5 years. AMI was diagnosed in 60 patients. The final diagnosis of chest pain was based on clinical, ECG, echocardiography and two 8hrs apart Troponin I test. AMI was ruled out by negative serial Trop I test. From 60 AMI cases, only 38 cases were having STEMI, and 12 patients were having NSTEMI. 24 patients were suffering from unstable angina. Rest 66 patients were having chest pain of non cardiac origin. Hscrp level in AMI was significantly was higher than in NCCP i.e (). There was no difference between HSCRP level in STEMI and NSTEMI. In this study, patients with chestpain, high levels of HSCRP is associated with AMI, implicating the presence of inflammatory process. A negative HSCRP differential saved performing a number of time consuming processes. This negative HSCRP differential helped as a good screening test in the emergency department to rule out chest pain of cardiac origin and allowing safe discharge.

Keywords: C reactive protein and acute chest pain.

Introduction

C reactive protein is synthesized in the liver and secreted into the blood stream when there is inflammation. CRP was originally discovered by Tillet and Francis in 1930 as a substance in the serum of patients suffering from acute inflamm-

ation. Its physiological role is to bind to phosphocholine expressed on the surface of dead or dying cells in order to stimulate the complement system through C1Q complex ⁽¹⁾. CRP was previously assumed that it might be a pathogenic secretion as it is elevated in variety of

illnesses including cancer. However discovery of hepatic secretion demonstrated that it is a native protein. It is a class of acute phase reactant, as its level is dramatically increased during inflammatory processes. This increase is due to rise in the plasma concentration IL-6, which produced mainly by macrophages⁽²⁾ and also by adipocytes⁽³⁾. CRP binds to phosphocholine on the bacteria and help in complement binding to damaged cells and enhances phagocytosis by macrophages. It is a member of pentraxin family of proteins.

Different types of studies have demonstrated that inflammation plays a central role at all stages of atherosclerotic cardiovascular disease, including initiation, progression to complication⁽⁴⁻⁶⁾. This study was conducted to evaluate Hscrp levels in patients presenting with acute chest pain of cardiac and non cardiac causes and wheather AMI (STEMI and non STEMI) have higher Hscrp compared with those having non AMI. Patients having unstable angina with no evidence of myocardial infarction have higher Hscrp due to inflammation within unstable coronary plaque.⁽⁷⁾ Our aim was to determine the serum level of Hscrp in patients with acute chest pain of cardiac and non-cardiac origin, and whether AMI patients having higher Hscrp levels compared to those having non cardiac chest pain.

Material and Methods

This present study was carried out in the Department of biochemistry in collaboration with department of cardiology VSSIMSAR, Burla from Nov2014 to oct 2016.

Inclusion Criteria

The study included 150 cases who presented to the cardiology department for chest pain with age group ranges from 38yrs to 75 yrs.

Exclusion Criteria

Patients having inflammatory and infectious disease are excluded. Ethical committee approval was obtained prior to study. Written Consent of the patients was taken for active participation during the study.

Routine Investigations

- 1) FBS
- 2) CBC
- 3) Urine Microscopic Examination

Special Investigations

- 1) Serum Trop I (EIA BY AIA 360 TOSOH)
- 2) Serum Hscrp (CLIA, IMPULSE2, MONOBIND USA)

Hscrp levels measured in 150 patients evaluated for chest pain. The mean age group was 50.5yrs. Acute myocardial infarction was diagnosed in 60 patients, The final diagnosis for aetiology of chest pain was based on clinical, ECG. ECHO and two 8 hrs apart trop I test. Acute myocardial infarction was ruled out by negative serial trop I test .Blood was collected from all acute chest pain cases and serum were separated and Trop I and Hscrp level was measured. Hscrp was measured in 0 hr and 12hours.

Each case was fit into one of the three groups.

GROUP I-Chest pain patients with acute myocardial infarction including

- a) ST elevated myocardial infarction.
- b) Non ST elevated myocardial infarction.

GROUP II-Chest pain patients with unstable angina.

GROUP III-Chest pain patients of non cardiac origin.

The significance of difference of parameters in different groups were tested using student t Test analysis.

Results

Table 1 shows the clinical characteristics of all patients as well as their distribution in three clinical groups. Mean age of Gr I patients was 53 yrs, which is 7 yrs older than Noncardiac chest pain individuals. Mean age of Gr II patients was 52yrs. Diabetes is more prevalent in AMI&UA patients than the NCCP patients. But hypertension is more prevalent in Gr II patients than Gr I & Gr II patients.

Table: 1

Group I	ST elevated AMI Non-ST elevated AMI
Group II	UA
Group III	Non-Cardiac chest pain

Table 2

All patients	Nos.	Mean age	Men	Hypertension	Diabetes
	150	50.5	81%	25%	27%
Group I	60	53.3	77%	30%	31%
Group II	24	52.1	79%	37%	45%
Group III	66	46.4	86%	16%	18%

Table 3 Mean HSCRP level in mg/dl in three clinical groups:

	0 hour value	12 hour value
Group I	8.71±2.74	12.69± 4.028
Group II	5.62 ± 1.671	6.542 ± 1.48
Group III	0.79 ± 0.30	0.81 ± 0.32

Statistically Significant at p value <0.05)

Fig.-1 Bar diagram of different groups

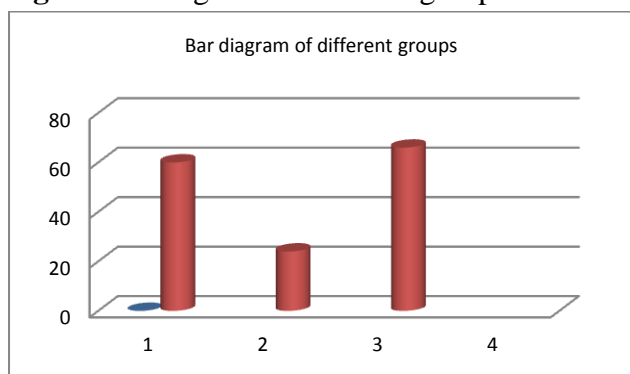


Table 2 shows mean Hscrp levels in the three groups. Mean Hscrp levels of AMI patients was significantly higher than NCCP patients, i.e 8.71±2.74 vs 0.79±0.30(p<0.0001), so as the patients of UA compared with NCCP patients (5.62±1.67vs 0.79±0.30(p<0.0001). Among AMI patients mean Hscrp levels in STEMI (9.153±2.428) was not different from Non STEMI patients (8.067± 3.09), p<0.13. Hscrp at 24 hrs showed a marked increase in patients with a final diagnosis of AMI than that of NCCP Patients.

Table 3 shows comparison of mean Hscrp levels in 0hr and 12hrs. Mean Hscrp levels at 0hr in Gr I shows 8.71±2.74 and at 12hrs it is increased to 12.69±4.028. Likewise in GrII shows 5.62±1.671 and 6.542±1.480 at 0 hr and 12 hrs respectively. In Gr III the Hscrp was 0.79±0.30 and 0.81±0.32.

Discussion

CRP is synthesized by liver in response to IL-6 during the inflammatory process (8). Atherosclerosis involves inflammatory process at all stages i.e. from fatty streaks to plaque rupture (9-11), and hence higher than normal levels of CRP increase in circulation even in the absence of myocardial necrosis. Different studies shown that CRP is a predictor of coronary disease in apparently healthy person, with step wise progression to myocardial infarction & stroke with increase CRP levels. (12-15). In our study, significantly higher percentage of AMI patients had high Hscrp compared to NCCP patients. AMI patients had higher Hscrp levels irrespective of type (ST elevated and non ST elevated). Hscrp levels vary according to genetic & environmental factors, hence its level vary in AMI patients of different geographic regions.

The study showed that mean age of AMI patients in Jordan is in the mid fifties, similar to that in other local clinical and angiographical studies (16-17). In our study mean age of AMI patients is early fifties. This is about 10 yrs younger than the mean age of AMI patients in the west. Hscrp level measurement has a lot advantage. It is a stable compound and can be measured at any time of the day and it is not changed due to biological clock. Individuals with high Hscrp can benefit from aggressive risk factor modification as well as from medication that have been found to decrease the hscrp blood levels in patients

Conclusion

In this study, we found that serum Hscrp levels is elevated not only in patients with AMI but also in patients of UA. Mean Hscrp levels of AMI Patients is significantly higher than NCCP Patients. So Hscrp estimation may be used for risk assessment, diagnostic and prognostic marker in patients with myocardial infarction.

References

1. Thompson D, PepysMB, Wood SP (Feb. 1999)” The physiological structure of human C-reactive protein & it’s complex

- with phosphocholine.”Structure 7(2):169-77,doi:10.1016150969-2126(99) 80023-9.PMID 10368284
2. Pepys MB, Hirschfield GM(June 2003). C reactive protein, a critical update “(PDF).J. Clin Invest iii(12) 1805-12 PMID 12813013.
 3. Lau, DC, DhillonB; yanH, Sznitko PE, Verma S(May 2005). “Adipokines; Molecular links between obesity & atherosclerosis”. AMJ Physical Heart circ. Physiol 228 (5):2031-41 PMID 15653761
 4. ROSSR. Atherosclerosis:an inflammatory disease; NEJM 1999;340:115-126
 5. Albert MA, GlynnRJ, RidkerPM, Plasma concentration of C Reactive protein & the calculated Framingham Coronary heart disease risk score. circulation 2003;108-116
 6. Tousoulis D, Davis G, Stefanadis C et al; inflammatory& thrombotic mechanism in coronary atherosclerosis. Heart 2003;89: 993-997
 7. Biasuccid M, GallimoreJR,Liuzzog et al, The prognostic value of C reactive protein & serum amyloid A protein in severe unstable angina,N.E.J.M1994;331:417-424
 8. Black GJ, RidkerPM,Novel clinical markers of vascular inflammation, circulation research 2001;89:763-767
 9. RifaiN, Ridker PM. High Sensitive C Reactive protein; a novel & promising marker of coronary heart disease. clin. chem 2001;47:403-411
 10. LibbyP,RidkerPM,MascricA.Inflammation &atherosclerosis.Circulation 2002,105: 113-117
 11. FarzanehA, Rudd A, Weissberg PL. Inflammatory mechanism British medical Bulletin 200;59:55-68
 12. Ridker PM, Leshman M, Stampfer MJ et al inflammation ,the risk of cardiovascular disease in apparently healthy men N.Eng.J Med 1997,336:973-979.
 13. Ridker PM, RifaiN, Rose L et al. Comparison of C reactive protein & low density lipoprotein. Cholesterol levels in the prediction of first cardiovascular events N.EnglJ.Med 2002:347:1557-1565.