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## Role of c-reactive protein in cardiovascular diseases - a review

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**Abstract:** Circulating level of C - reactive protein depicts the cardiovascular condition. By reviewing available studies, the current study determines that CRP level ( c- reactive protein level ) within the upper quantile of the normal range constitutes an increased risk for cardiovascular events or diseases. CRP levels within the upper quartile/quintile of the traditional range indicates increased risk for cardiovascular events. High CRP levels in acute myocardial infarct after treatment shows an alarming sign. This is due to the response of the body to the inflamed atherosclerotic (coronary) vessels and adjacent myocardium. Localized CRP in infarcted myocardium activates the complement system, resulting in inflammation and thrombosis. After reviewing many literatures with correlation between CRP and cardiovascular disease, the current study explains the pathogenesis and its role in diagnosing myocardial infarction. The background of this current study may provide directions for prevention of cardiovascular events.

**Keywords:** C- reactive protein ; cardiovascular disease risk factors ; genetics and structures of CRP; Function of CRP ; atherosclerosis ; ischemia ; necrosis ; coronary heart disease ; CRPhs ; inflammation innovative technique.

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### INTRODUCTION

C- reactive protein is a blood test marker seen in the inflammatory condition produced from the liver (Ansar and Ghosh, 2016b)(Ansar and Ghosh, 2016b)(Ansar and Ghosh, 2016b)(Ansar and Ghosh, 2016b).It is an acute phase reactant protein which is an exquisitely sensitive systemic marker of disease with broad clinical utility for monitoring and differential diagnosis (Ansar and Ghosh, 2016a)(Ansar and Ghosh, 2016a)(Ansar and Ghosh, 2016a)(Ansar and Ghosh, 2016a).CRP has a capacity to precipitate the somatic c-polysaccharide of streptococcus pneumoniae ('C-Reactive Protein', no date, *Website*, no date a)('C-Reactive Protein', no date, *Website*, no date a)('C-Reactive Protein', no date, *Website*, no date a)('C-Reactive Protein', no date, *Website*, no date a)

CRP may be a sensitive systemic marker of inflammation and tissue damage. It's a member of the pentraxin family of plasma proteins, which are a part of the lectin fold super family , of calcium dependent ligand binding and lectin ( carbohydrate - binding ) proteins. The sole molecule that displays an equivalent remarkable sensitivity, speed and dynamic range of responses as CRP is Serum Amyloid A-protein (SAA). Of the bug selection of inflammatory biomarkers that are studied, high-sensitivity C-reactive protein (hsCRP) has received the foremost attention for its use in screening and risk classification and as a predictor of clinical response to statin therapy. CRP involved in the immunologic process increases the deposition of plaque leading to increased CV disease (CVD) risk. But, definitive randomized evidence for its role as a causative thought about atherothrombosis is lacking. Measuring the hsCRP levels in assessing vascular nature in atherosclerosis and other cardiovascular disease is still under debate.

CRP is the forerunner within the search for inflammatory markers and is subject to intensive research in numerous studies worldwide. Unlike other markers of inflammation, CRP levels are stable over long periods, haven't any variation, are often measured inexpensively with available high-sensitivity assays, and have shown specificity in terms of predicting the danger of CHD. CRP may have a task within the genesis of atherosclerotic lesion, since it reduces the expression of gas (NO) synthase and prostacyclin synthase, and binds LDL-C and promotes its uptake by macrophages, a key step in atherogenesis (*Website*, no date b)(*Website*, no date b)(*Website*, no date b)(*Website*, no date b).

CRY plays a major role in expressing the adhesion molecules in endothelial cells (EC). These factors regulate atherogenesis. Multiple prospective cohort studies have established that increased CRP levels are associated with increased CHD risk in both genders, across a wide age range (*Website*, no date c)(*Website*, no date c)(*Website*, no date c)(*Website*, no date c)These findings are consistent in different populations with different settings. They help

in prediction of risk factors related to cardiovascular disease, myocardial infarction, stroke stroke, sudden cardiac death, peripheral artery disease and incident diabetes and new onset hypertension. CRP levels have also been shown to predict risk of both recurrent ischemia and death among those with stable and unstable angina, those undergoing percutaneous angioplasty, and people presenting to emergency rooms with acute coronary syndrome (ACS).(Website, no date d)(Website, no date d)(Website, no date d)(Website, no date d)

Our department is passionate about research we have published numerous high quality articles in this domain over the past years (Abraham *et al.*, 2005; Devaki, Sathivel and BalajiRaghavendran, 2009; Neelakantan *et al.*, 2010, 2015; Arja *et al.*, 2013; Ramshankar *et al.*, 2014; Sumathi *et al.*, 2014; Surapaneni and Jainu, 2014; Surapaneni, Priya and Mallika, 2014; Ramamoorthi, Nivedhitha and Divyanand, 2015; Manivannan *et al.*, 2017; Ezhilarasan, 2018; Ezhilarasan, Sokal and Najimi, 2018; J *et al.*, 2018; Ravindiran and Praveenkumar, 2018; Malli Sureshbabu *et al.*, 2019; Mehta *et al.*, 2019; Krishnaswamy *et al.*, 2020; Samuel, Acharya and Rao, 2020; Sathish and Karthick, 2020)(Abraham *et al.*, 2005; Devaki, Sathivel and BalajiRaghavendran, 2009; Neelakantan *et al.*, 2010, 2015; Arja *et al.*, 2013; Ramshankar *et al.*, 2014; Sumathi *et al.*, 2014; Surapaneni and Jainu, 2014; Surapaneni, Priya and Mallika, 2014; Ramamoorthi, Nivedhitha and Divyanand, 2015; Manivannan *et al.*, 2017; Ezhilarasan, 2018; Ezhilarasan, Sokal and Najimi, 2018; J *et al.*, 2018; Ravindiran and Praveenkumar, 2018; Malli Sureshbabu *et al.*, 2019; Mehta *et al.*, 2019; Krishnaswamy *et al.*, 2020; Samuel, Acharya and Rao, 2020; Sathish and Karthick, 2020)(Abraham *et al.*, 2005; Devaki, Sathivel and BalajiRaghavendran, 2009; Neelakantan *et al.*, 2010, 2015; Arja *et al.*, 2013; Ramshankar *et al.*, 2014; Sumathi *et al.*, 2014; Surapaneni and Jainu, 2014; Surapaneni, Priya and Mallika, 2014; Ramamoorthi, Nivedhitha and Divyanand, 2015; Manivannan *et al.*, 2017; Ezhilarasan, 2018; Ezhilarasan, Sokal and Najimi, 2018; J *et al.*, 2018; Ravindiran and Praveenkumar, 2018; Malli Sureshbabu *et al.*, 2019; Mehta *et al.*, 2019; Krishnaswamy *et al.*, 2020; Samuel, Acharya and Rao, 2020; Sathish and Karthick, 2020)(Abraham *et al.*, 2005; Devaki, Sathivel and BalajiRaghavendran, 2009; Neelakantan *et al.*, 2010, 2015; Arja *et al.*, 2013; Ramshankar *et al.*, 2014; Sumathi *et al.*, 2014; Surapaneni and Jainu, 2014; Surapaneni, Priya and Mallika, 2014; Ramamoorthi, Nivedhitha and Divyanand, 2015; Manivannan *et al.*, 2017; Ezhilarasan, 2018; Ezhilarasan, Sokal and Najimi, 2018; J *et al.*, 2018; Ravindiran and Praveenkumar, 2018; Malli Sureshbabu *et al.*, 2019; Mehta *et al.*, 2019; Krishnaswamy *et al.*, 2020; Samuel, Acharya and Rao, 2020; Sathish and Karthick, 2020)

The aim of this review article is to analyse the role of CRP in pathogenesis of cardiovascular diseases.

## MATERIALS AND METHODS

By reviewing various available studies about c-reactive protein involved in cardiovascular disease and using the keywords such as, inflammation, atherosclerosis, Myocardial infarction, stroke, Coronary heart disease in search engines like PUBMED, SCOPUS, GOOGLE SCHOLAR, the information have been gathered.

Inclusion criteria :

- Patients with high CRP level
- case study CRP at sites of inflammation and infection.

Exclusion criteria :

- Disease not related to elevation of CRP..

## DISCUSSION

**Genetics and structure of CRP:** The CRP gene is located on chromosomes (Mukherjee, 1978)(Mukherjee, 1978)(Mukherjee, 1978) belongs to a small pentraxin family. The monomer has 224 amino acids and a molecular mass of 25,106Da. In serum, it assembles into stable pentameric structure with a discoid shape (Brundha, Pathmashri and Sundari, 2019; Topdagi Yilmaz *et al.*, 2020)(Brundha, Pathmashri and Sundari, 2019; Topdagi Yilmaz *et al.*, 2020)(Brundha, Pathmashri and Sundari, 2019; Topdagi Yilmaz *et al.*, 2020)(Brundha, Pathmashri and Sundari, 2019; Topdagi Yilmaz *et al.*, 2020).

CRP Reflects Inflammation of (Coronary) Vessels by Pathogenic Agents

CRP Reflects Inflammation of (Coronary) Vessels by Pathogenic Agents CRP may increase in disorder in response to infectious (viral, bacterial) agents inducing inflammatory reactions within the (coronary) vessels. Although this possibility can't be excluded definitely, infectious agents in coronary vessels or myocardium haven't been demonstrated convincingly so far. Notably, chronic infections elsewhere within the body also are related to an increased risk for disorder. Furthermore, the sequence of infection by pathogens and therefore the initiation and progression of disorder still remains to be elucidated. These infections likely are related to raised CRP levels. Hence, chronic infections could also be related to disorder by a coinciding rise of plasma CRP thanks to elicitation of an acute phase response.

**Function of CRP:** CRP binds to the phosphocholine expressed on the surface of dead or dying cells and a few of bacteria (Website, no date e; He *et al.*, 2020)(Website, no date e; He *et al.*, 2020)(Website, no date e; He *et al.*, 2020)(Website, no date e; He *et al.*, 2020). This activates the complement system, promoting phagocytosis by

macrophages, which are necrotic and apoptotic cells and bacteria . This so-called acute phase response occurs as a result of accelerating concentrations of IL - 6 which is produced by macrophages also as adipocytes in response to a wide range of acute and chronic inflammation conditions. Like bacterial viral or fungal infections, rheumatic and other inflammatory diseases, malignancy and tissue injury and necrosis (P Jannathulferdioz, no date)(P Jannathulferdioz, no date)(P Jannathulferdioz, no date)(P Jannathulferdioz, no date).These conditions cause release of interleukin - 6 and other cytokines that trigger the synthesis of CRP and fibrinogen by the liver(Li *et al.*, 2020)(Li *et al.*, 2020)(Li *et al.*, 2020)(Li *et al.*, 2020).

### **C – Reactive protein and Cardiovascular Disease:**

In association between circulating CRP and cardiovascular diseases, CRP may increase in response to infections (viral, bacterial) agents including inflammatory reactions within the (coronary) vessels (‘International Journal of Pharmaceutical Sciences And Research’, 2016; Kumar, Ashok Kumar and Brundha, 2016)(‘International Journal of Pharmaceutical Sciences And Research’, 2016; Kumar, Ashok Kumar and Brundha, 2016)(‘International Journal of Pharmaceutical Sciences And Research’, 2016; Kumar, Ashok Kumar and Brundha, 2016)(‘International Journal of Pharmaceutical Sciences And Research’, 2016; Kumar, Ashok Kumar and Brundha, 2016)(‘International Journal of Pharmaceutical Sciences And Research’, 2016; Kumar, Ashok Kumar and Brundha, 2016). Although this possibility cannot be excluded definitely, infection agents in coronary vessels or myocardium have not been demonstrated convincingly so far (‘C-reactive protein and insulin-resistance in obese children and adolescents’, 2003)(‘C-reactive protein and insulin-resistance in obese children and adolescents’, 2003)(‘C-reactive protein and insulin-resistance in obese children and adolescents’, 2003)(‘C-reactive protein and insulin-resistance in obese children and adolescents’, 2003). Notably, chronic infections elsewhere within the body also are related to increased risk factor for cardiovascular disease (Tousoulis and Stefanadis, 2013; Hannah *et al.*, 2019)(Tousoulis and Stefanadis, 2013; Hannah *et al.*, 2019)(Tousoulis and Stefanadis, 2013; Hannah *et al.*, 2019)(Tousoulis and Stefanadis, 2013; Hannah *et al.*, 2019). These infections could also be related to cardiovascular diseases by a coinciding rise of plasma CRP due to elicitation of an acute phase response.(Martínez and Ramón González-Juanatey, 2009)(Martínez and Ramón González-Juanatey, 2009)(Martínez and Ramón González-Juanatey, 2009)(Martínez and Ramón González-Juanatey, 2009)

The most important aspect is that the biomarker has an independent predictive potential, being called high-sensitivity CRP (hs-CRP). Thus, cardiovascular risk is categorized counting on the extent of hs-CRP:

- low risk-hs-PCR<1mg/L
- moderate risk-hs-PCR between 1-3mg/L
- high risk-hs-PCR>3mg/L. (Bonaca and Morrow, 2008)(Bonaca and Morrow, 2008)(Bonaca and Morrow, 2008)(Bonaca and Morrow, 2008)

CRP Reflects inflammation related to the atherosclerotic process :

CRP levels are considered to pick the extent of inflammatory reactions within the atherosclerotic vessels(Hanna, 1995)(Hanna, 1995)(Hanna, 1995)(Hanna, 1995). Thus virtue of its acute phase behaviour CRP may be a marker for severity and progression of atherosclerotic processes within the vessels(*Website*, no date f)(*Website*, no date f)(*Website*, no date f)(*Website*, no date f).Thus there's an opportunity that CRP is linked to disorder because it increases in response to clot formation superimposed on atherosclerotic lesions within the vessels is unlikely (Shreya and Brundha, 2017; Yari *et al.*, 2020)(Shreya and Brundha, 2017; Yari *et al.*, 2020)(Shreya and Brundha, 2017; Yari *et al.*, 2020)(Shreya and Brundha, 2017; Yari *et al.*, 2020).

CRP reflects extent of (Myocardial) ischemia :

CRP level does not rise in association with Myocardial ischemia without necrosis, as demonstrated in patients with Variant angina pectoris with documented episodes of myocardial ischemia (Ashokachakkaravarthy *et al.*, 2020)(Ashokachakkaravarthy *et al.*, 2020)(Ashokachakkaravarthy *et al.*, 2020)(Ashokachakkaravarthy *et al.*, 2020). Thus, CRP is not tenable in association with myocardial ischemia (Gallone *et al.*, 2020)(Gallone *et al.*, 2020)(Gallone *et al.*, 2020)(Gallone *et al.*, 2020).

CRP reflects extent of (Myocardial) necrosis :

The extent of necrosis in part determines the CRP response, that is, CRP in association with Myocardial necrosis rises the CRP level (Wu, 2003; Khimion and Boiko, 2020)(Wu, 2003; Khimion and Boiko, 2020)(Wu, 2003; Khimion and Boiko, 2020)(Wu, 2003; Khimion and Boiko, 2020). Recanalization of an infarct related coronary artery improves prognosis after AMI as it decreases infarct size (Crea, Lanza and Camici, 2013; Pogosova *et al.*, 2020)(Crea, Lanza and Camici, 2013; Pogosova *et al.*, 2020)(Crea, Lanza and Camici, 2013; Pogosova *et al.*, 2020)(Crea, Lanza and Camici, 2013; Pogosova *et al.*, 2020). It also attenuates the CRP response suggesting that successful reperfusion may limit inflammation in infarcted myocardium , though it also may induce reperfusion injury(Kalaiselvi and Brundha, 2016)(Kalaiselvi and Brundha, 2016)(Kalaiselvi and Brundha, 2016)(Kalaiselvi and Brundha, 2016) . Thus CRP responses after AMI predict clinical outcome such as 6 – months mortality

irrespective of infarct size (Gawda and Czubak, 2017)(Gawda and Czubak, 2017)(Gawda and Czubak, 2017)(Gawda and Czubak, 2017).

CRP – radiated inflammation in cardiovascular disease :

After AMI, CRP contributes to inflammation in ischemic myocardium by activating complement (34). Fragments were found to possess increasing plasma levels of CRP and activated complement have also been found in human atherosclerotic vessels (*Website*, no date g)(*Website*, no date g)(*Website*, no date g)(*Website*, no date g). Thus CRP may constitute a cardiovascular risk factor because it localizes in ischemic myocardium and atherosclerotic lesions, thereby promoting local complement activation (Sami *et al.*, 2019)(Sami *et al.*, 2019)(Sami *et al.*, 2019)(Sami *et al.*, 2019).

Ligands for CRP in Cardiovascular disease :

Postulated ligands for CRP in atherosclerosis include lipoproteins. CRP can bind to phosphatidylcholine vesicles containing lysophosphatidylcholine (Heinecke, 2006)(Heinecke, 2006)(Heinecke, 2006)(Heinecke, 2006). It is generated from phospholipids by phospholipase A2 (PLA2) enzymes and are demonstrated in infarcted myocardium (Timothy, Samyuktha and Brundha, 2019)(Timothy, Samyuktha and Brundha, 2019)(Timothy, Samyuktha and Brundha, 2019)(Timothy, Samyuktha and Brundha, 2019). Therefore postulate that lysophospholipids constitute the ligand for CRP in ischemic myocardium. The inner and outer leaflet of the cell wall of normal cells differ in phospholipid composition; sphingomyelin and phosphatidylethanolamine mainly within the inner ischemic cells may generate micro vesicles, which, on interaction with PLA2 enzymes also may constitute binding sites for CRP (Slevin and Molins, 2019)(Slevin and Molins, 2019)(Slevin and Molins, 2019)(Slevin and Molins, 2019). Ligand bound CRP activates the classical pathway complement, and this activation subsequently enhances inflammation and contributes to myocardial tissue damage of dysfunction.

Measuring CRP levels :

Learning your CRP involves a simple biopsy and is comparatively inexpensive. CRP levels gradually rise with age but remain generally stable over a period of months or a few of years. it's not a test you'd wish to repeat on a day to day. And you'd wish to avoid taking the test while you've any quite infection or inflammation, quite cold or the flu. Because CRP levels reflect inflammation, the blood levels are getting to be very high in these situations. High-sensitivity CRP levels rise alongside various risk factors indicating aging, smoking and obesity. So if you smoke or are overweight and have high CRP levels, the CRP are often related to these risk factors rather than indicate a further risk for disorder. In atherosclerosis, the hs-CRP blood level signals what proportion low-grade inflammation is present within the vessel wall. Currently, there is no evidence that the CRP molecule itself could even be a drive of atherosclerosis.

In a study of D.L. Cozlea et al , disorder (CVD) is that the foremost explanation for premature death worldwide. many risk factors are associated with disorder. Recent extensive evidence supports inflammation as a key pathogenetic mechanism within the event and progression of atherosclerosis and in triggering clinical atherothrombotic CVD events. C-reactive protein (CRP) is one possible marker of vascular inflammation and plays a moment role in promoting vascular inflammation, vessel damage and clinical CVD events. Material and method. They evaluated 100 patients with cardiovascular risk factors, using the systematic coronary risk evaluation (SCORE) charts for top risk regions of Europe which we determined the CRP level, using the nephelometric method. By their SCORE chart, 44% of the patients are within the moderate risk category, and almost 40% within the high risk category, the rest of them (16%) are within the low and really high risk category. A statistically significant p value ( $p < 0.05$ ) was observed between patients with  $CRP < 10\text{mg/L}$ , who had a lower systolic blood pressure than patients with  $CRP \geq 10\text{mg/L}$ . The CRP level over  $10\text{mg/L}$  is correlated with an over 4% risk of developing a fatal CVD in 10 years. The acute phase reactant, CRP, a simple downstream marker of inflammation, has now emerged as a major cardiovascular risk factor.

**Inference:** Recent research and studies have shown that CRP test can reveal risks of underlying cardiovascular diseases; like attack and strokes. Standard CRP test can measure CRP content ranging between 3 – 5 mg/L. It's helpful in determining quite high levels of CRP content in blood. It helps in confirming the presence of certain inflammatory diseases within the body (*Website*, no date h)(*Website*, no date h)(*Website*, no date h)(*Website*, no date h). The prevention of cardiovascular events in persons with high-normal or elevated plasma CRP levels could even be achieved by anti-inflammatory agents like aspirin, reducing the synthesis of CRP (cytokine-antagonists), preventing the binding of CRP to membranes (phosphorylcholine-like drugs) or inhibiting CRP-induced

activation of the classical complement pathway (C1-esterase-inhibitor). Future studies should reveal whether these approaches are indeed efficacious(Website, no date i)(Website, no date i)(Website, no date i)(Website, no date i) .

## CONCLUSION

CRP production, alongside the rise of other pro-inflammatory Cytokines, starts during the primary stage of the inflammatory process. This shows the role of the risk marker during this process. Many studies have been demonstrated that increased CRP Concentrations are related to an increased risk of MI (Myocardial infarction), stroke, peripheral arterial disease, and sudden cardiac death. Certain limitations in with inflammatory screening remains and available studies suggest HS CRP has potential adjunct global risk assessment.

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