C-REACTIVE PROTEIN (CRP) AS A DIAGNOSTIC MARKER FOR HIGH GRADE FEVER

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ABSTRACT:
C-reactive protein (CRP) is an annular (ring-shaped), pentameric protein found in blood plasma, whose circulating concentrations rise in response to inflammation. It is an acute-phase protein of hepatic origin that increases following interleukin-6 secretion by macrophages and T cells. This cross-sectional study was conducted among the patients presenting in the medical outdoor department of different hospitals. Name, age, gender and history of fever and levels of C-reactive protein were noted on a predefined proforma. All the data was entered and analyzed with SPSS Ver. 23.0. A total of 80 patients were included in this study i.e., 40 males (50%) and 40 females (50%). The mean age of the patients was 29.34±3.44 years. Out of 80 patients, fifteen patients had raised CRP levels with high grade fever. Thirty patients had high grade fever but CRP levels were not raised.

KEYWORD: C-REACTIVE PROTEIN (CRP)
INTRODUCTION:
C-reactive protein (CRP) is an annular (ring-shaped), pentameric protein found in blood plasma, whose circulating concentrations rise in response to inflammation. It is an acute-phase protein of hepatic origin that increases following interleukin-6 secretion by macrophages and T cells. Its physiological role is to bind to lysophosphatidylcholine expressed on the surface of dead or dying cells (and some types of bacteria) in order to activate the complement system via C1q. CRP is synthesized by the liver in response to factors released by macrophages and fat cells (adipocytes). It is a member of the pentraxin family of proteins. It is not related to C-peptide (insulin) or protein C (blood coagulation).

C-reactive protein was the first pattern recognition receptor (PRR) to be identified. Traditional CRP measurement only detected CRP in the range of 10 to 1,000 mg/L, whereas high sensitivity CRP (hs-CRP) detects CRP in the range of 0.5 to 10 mg/L. hs-CRP can detect cardiovascular disease risk when in excess of 3 mg/L, whereas below 1 mg/L would be low risk. Traditional CRP measurement is faster and less costly than hs-CRP, and can be adequate for some applications, such as monitoring hemodialysis patients. In healthy adults, the normal concentrations of CRP varies between 0.8 mg/L and 3.0 mg/L. However, some healthy adults show elevated CRP at 10 mg/L. CRP concentrations also increase with age, possibly due to subclinical conditions. There is also no seasonal variations of CRP concentrations. Gene polymorphism of interleukin-1 family, interleukin 6, and polymorphic GT repeat of the CRP gene do affect the usual CRP concentrations when a person does not have any medical illnesses. The plasma half-life of CRP is 19 hours, and is constant in all medical conditions (1-3).

MATERIAL AND METHODS:
This cross-sectional study was conducted among the patients presenting in the medical outdoor department of different hospitals. Name, age, gender and
history of fever and levels of C-reactive protein were noted on a predefined proforma. All the data was entered and analyzed with SPSS Ver. 23.0. The quantitative variables were presented as mean and standard deviation. The qualitative variables were presented as frequency and percentages.

RESULTS:
A total of 80 patients were included in this study i.e., 40 males (50%) and 40 females (50%). The mean age of the patients was 29.34±3.44 years. Out of 80 patients, fifteen patients had raised CRP levels with high grade fever. Thirty patients had high grade fever but CRP levels were not raised.

DISCUSSION:
When there is a stimulus, the CRP level can increase 10,000-fold from less than 50 µg/L to more than 500 mg/L. Its concentration can increase to 5 mg/L by 6 hours and peak at 48 hours. Therefore, the only factor that affects the blood CRP concentration is its production rate, which increases with inflammation, infection, trauma, necrosis, malignancy, and allergic reactions. Other inflammatory mediators that can increase CRP are TGF beta 1, and tumor necrosis factor alpha. In acute inflammation, CRP can increase as much as 50 to 100 mg/L within 4 to 6 hours in mild to moderate inflammation or an insult such as skin infection, cystitis, or bronchitis. It can double every 8 hours and reaches its peak at 36 to 50 hours following injury or inflammation. CRP between 100 and 500 mg/L is considered highly predictive of inflammation due to bacterial infection. Once inflammation subsides, CRP level falls quickly because of its relatively short half-life. CRP concentrations between 2 and 10 mg/L are considered as metabolic inflammation: metabolic pathways that cause arteriosclerosis and type II diabetes mellitus. Recent research suggests that patients with elevated basal levels of CRP are at an increased risk of diabetes, hypertension and cardiovascular disease. A study of over 700 nurses showed that those in the highest quartile of trans fat consumption had blood levels of CRP that were
73% higher than those in the lowest quartile. Although one group of researchers indicated that CRP may be only a moderate risk factor for cardiovascular disease, this study (known as the Reykjavik Study) was found to have some problems for this type of analysis related to the characteristics of the population studied, and there was an extremely long follow-up time, which may have attenuated the association between CRP and future outcomes. Others have shown that CRP can exacerbate ischemic necrosis in a complement-dependent fashion and that CRP inhibition can be a safe and effective therapy for myocardial and cerebral infarcts; so far, this has been demonstrated in animal models only. It has been hypothesized that patients with high CRP levels might benefit from use of statins. This is based on the JUPITER trial that found that elevated CRP levels without hyperlipidemia benefited. Statins were selected because they have been proven to reduce levels of CRP. Studies comparing effect of various statins in hs-CRP revealed similar effects of different statins. A subsequent trial however failed to find that CRP was useful for determining statin benefit.

In a meta-analysis of 20 studies involving 1,466 patients with coronary artery disease, CRP levels were found to be reduced after exercise interventions. Among those studies, higher CRP concentrations or poorer lipid profiles before beginning exercise were associated with greater reductions in CRP. To clarify whether CRP is a bystander or active participant in atherogenesis, a 2008 study compared people with various genetic CRP variants. Those with a high CRP due to genetic variation had no increased risk of cardiovascular disease compared to those with a normal or low CRP. A study published in 2011 shows that CRP is associated with lipid responses to low-fat and high-polyunsaturated fat diets (4-6).

REFERENCES: