Review Article: Role of hCRP, Dysfunction and its Management

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ABSTRACT

CRP plays important roles in inflammatory processes and host responses to infection including the complement pathway, apoptosis, phagocytosis, nitric oxide (NO) release, and the production of cytokines, particularly interleukin-6 and tumor necrosis factorα. Early work on CRP can seem somewhat unclear and at times conflicting since it was often not specified which particular CRP isoform was measured or utilized in experiments and whether responses attributed to nCRP were in fact possibly due to dissociation into mCRP or lipopolysaccharide contamination. Each CRP isoform plays at sites of local inflammation and infection. CRP production is part of the nonspecific acute-phase response to most forms of inflammation, infection, and tissue damage.

KEYWORDS: CRP, inflammation, interleukin Scientific

INTRODUCTION

C-reactive protein (CRP) was identified in 1930 and was subsequently considered to be an "acute phase protein," an early indicator of infectious or inflammatory conditions. Since its discovery, CRP has been studied as a screening device for inflammation, a marker for disease activity, and as a diagnostic adjunct. Improved methods of quantifying CRP have led to increased application to clinical medicine. In the emergency department, CRP must be interpreted in the clinical context; no single value can be used to rule in or rule out a specific diagnosis. CRP has limited utility in the emergency department. And a normal CRP level should never delay antibiotic coverage.

C-reactive protein (CRP) is an acute-phase protein synthesized by hepatocytes in response to proinflammatory cytokines during inflammatory/infectious processes. CRP is also known to be associated with chronic inflammation.

The clinical significance of CRP in chronic inflammatory and neurodegenerative diseases, such as cardiovascular disease, type 2 diabetes mellitus, age-related macular degeneration, haemorrhagic stroke,

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Elevated blood pressure was observed in all three hCRP overexpression models, including adenoassociated virus 9 (AAV9)-transfected mice, AAV9transfected rats and hCRP transgenic (hCRPtg) rats. hCRPtg rats expressing clinically relevant high-level hCRP developed spontaneous hypertension, cardiac hypertrophy, myocardial fibrosis and impaired endothelium-dependent relaxation. Clinically relevant high-level hCRP induces hypertension and endothelial dysfunction by inhibiting AMPK-eNOS signaling, and highlight hCRP is not only an inflammatory biomarker but also a driver of hypertension. Treatment with metformin or a synthetic AMPK activator may be a potential strategy for vaso-dysfunction and hypertension in patients with high hCRP levels.

C-reactive protein (CRP) levels in diabetes predict cardiovascular events. Also, human CRP (hCRP) exacerbated the proinflammatory, pro-oxidant and procoagulant states in a spontaneous model of type 1 diabetes mellitus (T1DM), the bio breeding (BB) rat. hCRP induces endothelial dysfunction in a spontaneous model of T1DM, and this could have implications for the vascular complications in diabetics.

C-reactive protein (CRP) is an acute inflammatory protein that increases up to 1,000-fold at sites of infection or inflammation. CRP is produced as a homopentameric protein, termed native CRP (nCRP), which can irreversibly dissociate at sites of inflammation and infection into five separate monomers, termed monomeric CRP (mCRP). CRP is synthesized primarily in liver hepatocytes but also by smooth muscle cells, macrophages, endothelial cells, lymphocytes, and adipocytes. Estrogen in the form of hormone replacement therapy influences CRP levels in the elderly. Indeed, CRP values can never be diagnostic on their own and can only be interpreted at bedside. Then contribute powerfully to the management, just as universal recording of the patient's temperature, an equally nonspecific parameter, is of great clinical utility.

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