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Perspective

Perspectives of Complement System in Inflammatory Diseases

Lalit M Srivastava*

Department of Biochemistry and Lab Medicine, Sir Ganga Ram Kolmet Hospital, India

***Corresponding author:** Lalit M Srivastava, Professor, Consultant Advisor, Department of Biochemistry and Lab Medicine, Sir Ganga Ram Kolmet Hospital, Pusa Road, New Delhi-110005, India

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The Complement System provides a rapid and efficient means to protect the host from invasive microorganisms. Due to its diverse biological activities, complement is the major mediator of inflammation, a natural response to the host tissue to any injury. Evidences are available to show that complement significantly contributes to the regulation of immune response. Furthermore, the effector functions arising from complement activation carry the potential for harming the host by directly or indirectly mediating inflammatory tissue destruction. Inappropriate or excessive activation of the complement system can lead to harmful, potentially life-threatening consequences due to severe inflammatory destruction. Genetic complement deficiencies or complement depletion have been found to be beneficial in reducing tissue injury of severe complement dependent inflammation. Considerable clinical and experimental evidence along with the work done in our laboratory implicate the role of complement and complement receptors (complement receptor-1; CR-1; MCP; DAF and CD 59) in the pathogenesis of various inflammatory diseases viz. immune-complex and autoimmune disorders as also organ failure as consequence to sepsis, multiple trauma and burns. Recently considerable advances have been made towards the utility of the measurements of the complement and complement receptors for the diagnosis and assessment of disease severity and response to therapy as well as of prognostic value in early recognition of patients at risk to develop multiple organ failure after trauma or with graft rejection following renal transplantation.

The work progress during recent years on complement system and its receptors along-with complement derived break down products (C3a; C3d; C5a) or protein-protein complexes provide a comprehensive insight into the activation state of the system and have led to provide strategies towards a novel therapeutic control and approach of complement system for many inflammatory diseases. We have found in our laboratory, that the value of Complement receptor-1/Erythrocyte (CR1/E) in patients with SLE (Systematic lupus erythematosus) was significantly lower than their consanguineous relatives and healthy subjects. CR1/E was found to be stable in consecutive samples in control. Our results suggest that low levels of CR1 on erythrocytes in SLE patients are acquired during the course of the disease. In patients the numbers varied between low and high during the course of treatment. With the increasing acceptance of the importance of complement in severe inflammation, more and more interest has been shown in recent years by the investigators towards diagnostic, prognostic and follow-up studies pertaining to inflammatory disorders. It is further believed that therapeutic inhibition of complement would most likely be able to arrest the process of various inflammatory diseases. The process of efficiently inhibiting complement include utilization of soluble complement inhibitors (C1-inhibitor; recombinant soluble complement receptor-1; administration of blocking antibodies for key components of complement cascade viz. C3 and C5) or even neutralizing the effect of anaphylatoxins (C5a and C3a) generated during the pathways of complement activation.

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