The Story Behind the Acute-phase Reactants

LUIS M. AMEZCUA-GUERRA, DIANA CASTILLO-MARTINEZ and RAFAEL BOJALIL

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To the Editor:

Physicians usually try to quantify complex biological phenomena through simple laboratory assays. So several tests have been developed to assess the inflammatory response, a nonspecific intricate system of response against different aggressors that includes multiple mechanisms of innate and adaptive immunity. These tests are often used interchangeably or redundantly, on the assumption that all evaluate the same processes. In this veinCrowson and colleagues\(^1\) compared the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) to assess inflammation in patients with rheumatoid arthritis (RA). The authors conclude that it is not necessary to obtain both measures and that, where available, the CRP alone may be preferred for disease activity assessment. In the accompanying editorial\(^2\), Dr. Wolfe critically discusses these conclusions and knocks down some myths about both laboratory tests; however, we are convinced that we must go one step beyond.

The term acute-phase reaction was coined long ago, and the core concept of this response has been evolving over time. Now it is clear that the acute-phase reaction is not a global, well coordinated response; rather, the various acute-phase reactants are independently regulated\(^3\). Since each acute-phase reactant is the result of a unique immune circuit, it is ingenious to consider that it evaluates the inflammatory process as a whole. Indeed, while the ESR is a surrogate marker of the presence of various acute-phase reactants, results in inhibition of the production of proinflammatory cytokines (as tumor necrosis factor and interleukin 1β) through mechanisms involving overproduction of transforming growth factor-β (TGF-β), a prototypical antiinflammatory cytokine\(^4\).

The most dramatic expression of the importance of CRP as an anti-inflammatory molecule that prevents autoimmunity comes from systemic lupus erythematosus. The underpowered CRP response seen in patients with lupus nephritis is very likely a contributory factor leading to impaired handling of apoptotic cells and clearance of immune complexes, enabling the development of immune response to self\(^5,7\).

Briefly, we are certain that every acute-phase reaction can give us much more information than just a measure of inflammation. It must be clear that each biomarker may also have different roles in selected clinical conditions, and it is not appropriate to replace one with another. The time has come for rheumatologists to understand the story behind and the meaning of each acute-phase protein in specific clinical settings.

LUIS M. AMEZCUA-GUERRA, MD, Department of Immunology, Instituto Nacional de Cardiología Ignacio Chávez, and LaSalle University School of Medicine; DIANA CASTILLO-MARTÍNEZ, MD, Department of Dermatology, Hospital General Naval de Alta Especialidad, Mexico City, Mexico; RAFAEL BOJALIL, MD, PhD, Department of Immunology, Instituto Nacional de Cardiología Ignacio Chávez, Juan Badiano 1, Sección XVI, Tlalpan, 14080 Mexico City, Mexico, and Department of Health Care, Universidad Autónoma Metropolitana Xochimilco. Address correspondence to Dr. Bojalil; E-mail: bojraf@yahoo.com

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