Erythrocyte sedimentation rate, rather than C-reactive protein, may be the preferred biomarker for hidradenitis suppurativa

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The inflammatory pathophysiology of hidradenitis suppurativa (HS) is not yet clearly defined. Despite the lack of disease specificity, inflammatory markers such as Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are used to detect acute and monitor chronic inflammatory conditions.[1] Although CRP has historically been used as an inflammatory marker for HS, ESR may be a more reliableindicator of HS disease severity.

Rouleaux formation occurs when many positively charged plasma proteins neutralize negatively charged RBCs and allow for faster erythrocyte aggregation, which further leads to ESR elevations.[1] ESR is affected by physiologic conditions that alter the size or shape or number of red blood cells, fibrinogen concentration, or acute and nonacute phase reaction protein (immunoglobulins) concentrations.[1] ESR can remain elevated for days to weeks after an acute insult.[1]

In contrast, CRP is synthesized in response to cytokines, has a short half-life, and normalizes within days after an insult; therefore, it is frequently used to determine any reinfection. Further, the elevated CRP levels have been associated with tissue damage, malignancy, trauma, autoimmune diseases, and burns.[1]

ESR and CRP both correlate to HS disease activity and predict treatment response.[2,3]

It is not surprising that the high burden of inflammation in HS causes a release of positively charged interleukins and other acute reactive proteins that promote erythrocyte aggregation, thus increasing the ESR. In addition, HS is sometimes complicated by anemia of chronic disease, which independently can increase ESR, as decreased red blood cells affect the laboratory measurements. Leukocytosis and thrombocytosis have also been associated with HS, and thrombocytosis is independently associated with elevated ESR.[4] CRP is elevated in response to only cytokines in an inflammatory state, whereas ESR can be increased in response to multiple factors related to HS including anemia, leukocytosis, and inflammation. This supports the theory that ESR would be more reflective of HS inflammatory state.

Consistent with this hypothesis, Jiménez-Gallo et al[2] compared CRP levels and ESR in patients with HS and found ESR elevation at earlier or lower inflammatory states, whereas CRP required a higher inflammatory state. ESRs was significantly elevated in both the moderate (Hidradenitis Suppurativa Physician's Global Assessment scale [HS-PGA 3]) and high (HS-PGA 4 and 5) inflammatory activity categories compared with non-HS controls. In contrast, the

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CRP levels were only significantly elevated in high inflammatory activity (HS-PGA 4 and 5) compared with non-HS controls.

Lastly, the rheumatology and gastroenterology literature has shown that baseline CRP levels can be affected by CRP autoantibodies or CRP genetic polymorphisms.[5] These factors do not affect ESR and may affect the reliability of elevated CRP as a biomarker of disease activity.

In conclusion, the frequently concomitant hematologic abnormalities that also independently elevate ESR and the demonstrated ability of ESR to detect mild HS disease suggest that ESR may be a superior marker of HS disease activity. Further research is needed to investigate and compare the inflammatory markers in HS.

Conflicts of interest

Dr Kimball is a consultant and investigator for Abbvie, Bristol Meyers Squibb, Janssen, Eli Lilly, Novartis, Pfizer, UCB, and Incyte; consultant for Regeneron and Bayer, receives fellowship funding from Janssen and Abbvie; and a member of the board of directors for Almirall. Dr Porter is a consultant and/or investigator for Abbvie, Bristol Meyers Squibb, Janssen, Eli Lilly, Novartis, Pfizer, UCB, and Incyte. Dr Gibson's fellowship was funded through the National Psoriasis Foundation. The authors have no other financial relationships or conflicts of interest relevant to this article to disclose.

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