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Letter to the Editor

Undetectable mannose binding lectin confers increased risk for serious infection in rheumatoid arthritis

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Dear Editor,

Wang et al.[1] recently reported in European Journal of Rheumatology on serious infections (SIs) in a cohort of patients with rheumatoid arthritis (RA), drawn from a tertiary hospital clinic in Melbourne, Australia (n = 263). We conducted a similar study in a slightly smaller RA cohort (n = 228) in Perth, Australia, which was published in 2017.[2] The Perth cohort not only was comprised of mainly private practice patients with RA (71%) but also included tertiary hospital clinic and rural patients. RA patients were followed for a mean of 5.92 years. Very similar results were obtained in the two studies. A high frequency of SIs was observed (9 per 100 patient years [PYs] among bDMARD recipients and 12 per 100 PYs in synthetic Disease Modifying Anti-Rheumatic Drug (DMARD) recipients), which closely approximates the rates that Wang et al.[1] observed (10.8 per 100 PYs). In our study, however, corticosteroids were found to confer SI risk even though prednisolone usage in the Perth cohort was low at 14.5% (incident risk ratio or IRR = 4.70, $P < .001$). In both cohorts, the top three categories of SI were lower respiratory, skin, and urological.

Importantly, in the Perth cohort, mannose binding lectin (MBL), a genetically determined component of the innate immune system, was found to confer an SI risk irrespective of treatment (for undetectable MBL or uMBL, the IRR was 4.67, $P = .001$), comparable to the use of prednisolone 10 mg or more per day. RA patients with uMBL had an SI rate of 25 per 100 PYs, which is more than three times that of RA patients with detectable MBL (8 per 100 PYs).

While the simplicity and generalizability of an SI nomogram that relies mainly on clinical characteristics plus or minus readily available laboratory data, such as low lymphocyte counts, are attractive, in our view, it is important that other biomarkers mechanistically relevant to SI susceptibility and with demonstrable predictive power should not be overlooked or excluded. When it comes to optimally informing the treating clinician about SI risk, the advantages of an accurate predictive tool, potentially incorporating all relevant biomarkers, will likely outweigh the benefits of a less accurate, albeit clinically pragmatic one.

References

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