Commentary

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Supplementation on Autoimmune Diseases with Metabolic Syndrome

Timo Kielmann*

Department of Haematology, Leeds Teaching Hospitals, Leeds, United Kingdom

***Corresponding author:** Timo Kielmann, Department of Haematology, Leeds Teaching Hospitals, Leeds, United Kingdom, Email: timkman@yahoo.com

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Description

ReA occurs worldwide and predominantly occurs in adults aged 20–40 years. It begins with an enteric, urogenital, or possibly an upper respiratory tract infection by a triggering microorganism The male:female ratio is 3:1. Women often have less severe disease than men. Bas et al postulates that the male predominance of urogenital ReA may be due to a less potent antibody production in response to chlamydial infection in men compared with women, which may lead to systemic dissemination of the organisms to the joints. ReA occurs in 1–4% of patients a few days to 6 weeks after infection of the urogenital or enteric tract, and 30– 70% of these patients are positive for HLA-B27. It is important to note that the arthritic symptoms may be present before the full resolution of the initial infection. The prevalence in adolescents and young adults varies widely by country and is difficult to accurately estimate because of differences in classification between adults and children and lack of national registries. Ravelli and Martini estimate that ERA accounts for 3–11% of those diagnosed with JIA. Among adult populations, the prevalence is estimated to be 30–40 per 100,000 persons. The incidence of ReA is related to the population prevalence of HLA-B27 and the incidence of urethritis, cervicitis, and infectious diarrhea caused by a predisposing organism. In Europe, the incidence of ReA is estimated to be 10–30 per 100,000 persons. Some studies have shown the incidence of the enteric type to be as high as 7% after shigella and 29% after Salmonella enteritidis. Examined the incidence of urogenital ReA in an urban STD clinic in Alabama using a survey and confirmatory physical examination. They found that 4.1% of patients with urethral inflammation or infection had features of ReA. It is likely that the true incidence of ReA is higher than what is shown by the population-based studies because mild cases can go unrecognized.

No diagnostic or classification criteria have been established for ReA, but the American College of Rheumatology has issued general guidelines, and in 1999, the 4th International Workshop on ReA discussed the term ‘reactive arthritis’, proposing its use only for a clinical picture and microbes associated with HLA-B27 and spondyloarthritis.3 Consequently, the definition of ReA is now based on a diagnostic criterion that largely focuses on enteral or urethral infections. However, we previously reported a case of clinical ReA after HIV infection4 ; a syndrome consistent with ReA has also been reported with dengue and chikungunya virus, parvovirus B19, rubella and measles vaccines. In our case, the arthritis occurred precisely 3 weeks after the infectious episode; without a competing source of identifiable extra-articular infection, and synovial fluid cultures negative for bacteria, we are strongly led to a diagnosis of clinical ReA.

Autoimmune hemolytic anemia

ReA tends to occur most often in men between ages 20 and 50. A 30–50% of patients with ReA carry HLA-B27. Although patients without HLA-B27 can develop ReA, as is in our case, some degree of genetic susceptibility is considered necessary, since ReA occurs in only 7–15% of infected population-level subjects. The association of been linked to more severe disease, higher frequencies of sacroiliitis and extra-articular manifestations, and an increased likelihood of persistent arthropathy. A limitation of our study is that viral arthritis is also a known entity and we are unable to completely exclude an acute viral arthritis, though this typically occurs during the acute fever episode and the pattern described here was notably different. Additionally, bacterial coinfections are reported in severe COVID-19 patients and ReA due to an occult bacterial respiratory coinfection is possible. However, sputum and blood cultures prior to initiation of empirical antibiotics failed to identify a competing bacterial cause, and the clinical course was not consistent with occult coinfection. Finally, our patient developed mild hyperuricemia, a known side effect of favipiravir and raising the possibility of a crystal arthropathy as a cause of symptoms. Careful synovial fluid analysis did not identify MSU crystals, which ruled out gouty arthritis. To conclude, we report the first case of ‘ReA’ after SARS-CoV-2 infection. Our findings offer an opportunity to improve both early diagnosis and treatment of ‘ReA’ during the COVID-19 pandemic. A ‘definite’ diagnosis of ReA is based on the fulfilment of both major criteria and a relevant minor criterion, while a ‘probable’ diagnosis is characterised by both major criteria but no relevant minor criterion or one major criterion and one or more of the minor criteria. The identification of the trigger infection is also required.

Definitions of disease, disease severity and disease phase

Although there is an established link between pathogens and development of ReA, there is strong evidence against the efficacy of broad-spectrum antibiotic therapy. Different than other rheumatic conditions, it is well established that NSAIDs are first-line drugs for the management of spondyloarthropathies and reactive arthritis, not only for the analgesic anti-inflammatory effects, but also because they retard the development of syndesmophytes. DMARDs such as Sulfazalasine are effective for the peripheral manifestations, but not with the axial involvement, most experts consider glucocorticoids use in ReA contraindicated, except for an occasional intra-articular injection. Since IL-17 was discovered and found to be importantly involved in the pathogenesis of spondyloarthropathies, despite this anti-IL17A monoclonal antibodies have not been tried in ReA. At this moment, there are only three anti-interleukin-17 drugs commercially available: secukinumab, ixekizumab, and brodalumab. Good results have been reported in a small cohort of ten patients in ReA with anti-TNF, but there is no data on anti-IL-17A in ReA. Novel treatments include microbial ecosystem therapeutics and fecal microbiota transplant (FMT), potentially useful especially in SpA.

Bacterial antigens, whether produced by a viable intrasynovial bacterium or brought into the articular cavity by monocytes, have an immunostimulatory action. These antigens can most likely persist in the joint either “stuck” to the extracellular matrix or within antigen presenting cells and by the various mechanisms described below can trigger a lymphocyte reaction potentially responsible for arthritis.