Introduction

Reactive arthritis, also known as Reiter syndrome, is a spondyloarthropathy that typically follows a urogenital or gastrointestinal infection, and is characterized by conjunctivitis, urethritis, and arthritis. The frequency of reactive arthritis in the United States is estimated at 3.5 to 5 patients per 100,000. Physician assistants (PAs) can manage the condition; therefore, they should be familiar with the disease's signs and symptoms, diagnostic criteria, and treatment regimens. Without proper management, reactive arthritis can progress to a chronic destructive arthritis. Prompt recognition of the condition is key to early intervention and a better patient outcome with fewer complications.

Causes

Reactive arthritis is a rare condition that is easily missed in clinical practice because of the variability in its symptomatology and presentation. Prompt recognition and treatment can reduce progression to chronic arthritis and other poor outcomes. Given the shortage of rheumatologists in the United States and the large number of physician assistants (PAs) in the field, PAs should be able to diagnose and treat complex rheumatologic conditions such as reactive arthritis.

Diagnosis

Reactive arthritis most commonly occurs up to several weeks after a gastrointestinal or urogenital infection. It also can occur following certain upper respiratory infections and after Bacillus Calmette-Guerin (BCG) treatment for bladder cancer. Organisms commonly associated with reactive arthritis include Chlamydia trachomatis, Shigella flexneri, Salmonella enteritidis, Campylobacter jejuni, Clostridium difficile, and Ureaplasma urealyticum. Less commonly associated infections include Escherichia coli, Borrelia burgdorferi, Neisseria gonorrhoeae, and Chlamydia pneumoniae. One common similarity between each of these pathogens is that they are intracellular organisms and can reach the joints as a complete form or as fragments causing a sterile synovitis. Additionally, inheritance of the HLA-B27 allele is strongly associated with the development of reactive arthritis. Patients with this gene have a 50% increased risk of developing reactive arthritis, although the exact association between the two is unclear. Current research includes theories of molecular mimicry between the gene and pathogen, which incites the immune response; the gene being a receptor for various pathogens; and the gene driving cross-reactivity between antigen and host. The presentation of reactive arthritis varies significantly in severity, number of clinical features, and symptom onset. Patients can be asymptomatic or symptomatic with urogenital, rheumatologic, ophthalmologic, dermatologic, and visceral manifestations. Studies have shown that symptom severity and frequency is increased in patients who are HLA-B27 positive.

Cite this article: Pinar Borman, Reactive Arthritis Overview. J Arthritis, 2021, 10(6), 001.