Treatment and Prognosis of Arthritis

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Received 03 June 2021; **Accepted** 17 June 2021; **Published** 25 June 2021

Introduction

Reactive arthritis has no cure; therefore, treatment focuses on alleviating pain and inflammation, reducing long-term deficits, and limiting disease progression. Two-thirds of patients have a self-limiting course. Nonpharmacologic treatments include rest during the acute phase and local cold treatment to affected joints.Patients with enthesitis can use orthotics, insoles and heel supports to help reduce pain and increase mobility. Strengthening exercises are important during treatment to prevent muscle wasting.

Causes

Unlike in other rheumatic conditions, nonsteroidal anti-inflammatory drugs (NSAIDs), most commonly ibuprofen, indomethacin, or naproxen, are the first-line pharmacologic treatment for spondyloarthropathies and reactive arthritis. NSAIDs are beneficial for their analgesic, anti-inflammatory effects and also for slowing the development of syndesmophytes. Patients should take these agents for at least 2 to 4 weeks before trying more powerful medications. In addition to NSAIDs, consider intra-articular glucocorticoid injections for patients with reactive arthritis affecting one or more joints. Dermatologic manifestations of reactive arthritis, such as hyperkeratotic lesions, can be treated with topical corticosteroids. Low to moderate doses of systemic corticosteroids may be used in patients who have had little response to NSAIDs or glucocorticoid injections, or those who have many affected joints. To avoid adverse reactions, taper systemic corticosteroid doses to the lowest dose possible to control symptoms.

Disease-modifying antirheumatic drugs (DMARDs) such as sulfasalazine also have shown to be effective for peripheral manifestations and should be continued until patients are in remission. In patients with severe or refractory reactive arthritis, anti-tumor necrosis factor (TNF) agents such as etanercept and infliximab may be effective. Anti-TNF therapy is associated with symptom improvement and has led to remission in one-third of patient cases. Antibiotic therapy for reactive arthritis is controversial, as evidence shows that it does not alter the disease course.

Diagnosis

To reduce the chance of developing reactive arthritis, patients should be given appropriate antibiotics at the time of initial infection or if the antecedent infection remains active. In patients with chronic reactive arthritis, routine follow-up and screening are crucial to prevent further manifestations. For example, ECG, echocardiogram, and ophthalmologic referral are recommended. Furthermore, patients diagnosed with reactive arthritis should continue to follow up with a rheumatologist as indicated for maintenance and prevention of long-term complications. Reactive arthritis can develop into a serious and chronic disease if not diagnosed and treated promptly. The diagnosis is easy to miss because of the similarity between clinical manifestations of other diseases and variability in the disease course itself. Clinicians should be aware of the ACR general guidelines and consider the possibility of reactive arthritis in patients presenting with urethritis, cervicitis, conjunctivitis, uveitis, and arthritis following a recent infection.

This is due to factors such as delays in culturing infectious fluids, a failure to isolate infectious organisms during arthrocentesis, and a lack of specific serologic tests for some of the causative agents. Laboratory testing of the inflammatory markers erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) also can be quite helpful in the diagnosis of reactive arthritis, especially in the acute phase. However, the absence of elevated inflammatory markers does not rule out reactive arthritisSeptic arthritis can present similarly to reactive arthritis. Septic arthritis is monoarticular and can be distinguished from reactive arthritis with synovial fluid and Gram stain analysis. Synovial fluid analysis also can help differentiate gout from reactive arthritis. In gout, crystals will be present on synovial fluid analysis and patients may have uric acid tophi.

Cite this article: Oleg N, Treatment and Prognosis of Arthritis. J Arthritis, 2021, 10(6), 001.