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Editorial

Medical Treatment of Ankylosing Spondylitis

Mehmet Soy[1]* and Pamir Atagündüz[2]

[1]*Department of Internal Medicine, Altinbas University, Turkey*
[2]*Department of Internal Medicine, Marmara University, Turkey*

***Corresponding author**
Mehmet Soy, Department of Internal Medicine, Altinbas University, Istanbul, Turkey, Email: mhmtsoy@gmail.com

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EDITORIAL

Spondyloarthritides (SpA) is a group of diseases, which are characterized by inflammation of the spine, asymmetrical oligoarthritis of the lower extremities and enthesitis, skin or mucous membrane lesions, uveitis and bowel inflammation. This disease group includes the classical Ankylosing spondylitis (AS) which is the prototype of SpA, juvenile ankylosing spondylitis (JAS), psoriatic arthritis (PSA), reactive arthritis, enteropatic arthritis and undifferentiated SpA [1]. Recently, a more practical classification of SpA on the basis of the type of involvement is proposed as follows: Predominantly peripheral SpA and axial SpA (axSpA). In an attempt to further classify patients without definite radiographic sacroiliitis on plane X-rays but with clinical signs and symptoms of axSpA, classification criteria for radiographic axSpA (ie. Classical AS) and non-radiographic axSpA (nr-axSpA) was developed by the Assessment of Spondyloarthritis Study Group [2].

AS frequently affects adults with a peak age of onset between 20 and 30 years. In addition to spinal disease, involvement of peripheral joints, in an asymmetric oligoarticular pattern and lower extremity dominancy, occurs in up to 50 percent of patients. Extraspinal features of AS consists of uveitis: frequently one sided, alternating anterior uveitis, intestinal mucosal lesions: frequently asymptomatic, aortic valve involvement, leading to aortic regurgitation and rarely renal amyloidosis due to chronic inflammation [1].

Autoantibodies are absent in AS but there is a strong association with the HLAB27 gene. In addition to the HLA-B27, studies have identified additional risk loci including several aminopeptidase genes (ERAP1, ERAP2, LNPEP and NPEPPS) and genes involved in the IL23 related genes. The IL-23 pathway drives the differentiation of CD4+ Th17 cells, which produce IL-17. Since its discovery of presence in the sacroiliac joints of AS patients by Braun et-al., tumor necrosis factor (TNF) is a well known therapeutic target in AS [1,3].

Diagnosis of AS is based on patient history, a trough-out physical examination, laboratory and radiologic findings. Correct identification of inflammatory back pain in a young adult and positive family history are key features for making the diagnosis early. Serum C reactive protein (CRP) and erythrocyte sedimentation rate (ESR) may be high during flares of inflammation but in a substantial proportion of AS patients these parameters may be within normal levels with high disease activity. HLA B27 may be found positive in nearly 90% of definitive cases. Demonstration of structural changes of sacroiliac joints on X-rays is essential, but this may not be possible in the early phases of disease. Use of magnetic resonance imaging (MRI) has revolutionized the imaging of AS patients [1].

TREATMENT

The goal of AS treatment is to reduce symptoms, maintain spinal flexibility and normal posture, reduce functional limitation, maintain the ability to work and decrease disease complications. A combination of drugs and physical exercises are crutial parts of therapy. Classically, Nonsteroidal Anti-inflammatory Drugs (NSAIDs) and Sulfasalazine (SZP) was the mainstay of therapy for many decades [1]. The introduction of biological drugs opened a new era in the treatment of AS. Moreover, it may causes biomechanical changes in the spine anatomy that predispose to fractures, spinal deformity and spondylodiscitis and may needs surgical interventions [4].

EXERCISE AND EDUCATION OF PATIENTS

The patient should be informed about the disease, it's possible consequences and possible treatment options. Regular exercises are mandatory for treatment of AS. Try to help patients by providing booklets or directing them to a physiotherapist and rehabilitation center.

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

NSAIDs have an important role in the treatment of AS. There are many studies that showed a rapid and excellent response to NSAIDs in inflammatory low back pain. These drugs are also effective in the improvement of the symptoms of peripheral joint involvement and entesopathy; however, they possibly have no effect on the course of the disease [5]. Interestingly, NSAID may reduce the radiologic progression. NSAIDs can be used on demand or continuously. The response to NSAIDs may vary from individual to individual. Patients who are unresponsive within 1-2 weeks should preferably be tested for another NSAID from another class [6].

SULPHASALAZINE

SZP is the most tried and tested antirheumatic drug for the

treatment of AS. Numerous double-blind controlled studies with SZP of 2-3 g / day were published. Many of them failed to show beneficial effect on spinal involvement. Some studies showed beneficial effects on peripheral arthritis [6,7]. SZP may be effective for prevention of uveitis recurrence [8].

CORTICOSTEROIDS

Systemic corticosteroids are not recommended for the treatment of AS. Local injections should be preferred instead of systemic corticosteroids in monoarticular exacerbations and localized enthesitis. In patients with active sacroiliitis despite drug therapy, sacroiliac joint fluoroscopy, CT or MRI guided injections may be performed [6].

BIOLOGICS

The discovery of these drugs has led to a new breakthrough in the treatment of AS. They were found to be significantly more effective in patients resistant to classical drugs. First drugs used for this purpose were infliximab and etanercept functioning by blocking the TNF. All of the currently available TNF inhibitors have been widely studied in AS and other SpA. All showed similar results for relieving symptoms of AS ie health-related quality of life, patient-reported outcomes and sleep quality. In addition, TNF blocking is beneficial for correction of anemia and reduction of CRP levels in patients with AS [6,9,10]. Moreover, they have beneficial effects on radiologic progression of AS. There are currently five anti-TNF blockers approved by FDA for use in AS (infliximab, etanercept, adalimumab, golimumab and certulizmumab). All anti-TNF drugs have similar efficacy in the treatment of AS. Except for increasing the risk of opportunistic infection such as tuberculosis, safety profiles are within acceptable limits [11]. In addition, before major surgical operations, they should be stopped a certain period of time [12].

Secukinumab is a FDA approved biologic agent in AS and PSA treatment, as well. It is a monoclonal IL-17A inhibitor that blocks the IL17/23 pathway. Joints, dactylitis, enthesitis, skin and nail involvements benefits from the inhibition of IL-17 [13]. Ustekinumab, is another inhibitor of Th-17 signalling pathways, approved for PSA treatment [14].

CONCLUSION

In addition to exercise and conventional drugs, newer drugs such as TNF and IL 17/23 blockers have opened up new horizons in the treatment of AS.

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