CLINICAL VIGNETTE

Ankylosing Spondylitis in a Patient with Minimal Change Renal Disease

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A 41-year-old Caucasian Female with a one year history of minimal change kidney disease and hypothyroidism presented to rheumatology for evaluation of low back pain of several years. She described the back pain as inflammatory in nature, with several hours of morning stiffness that progressively improved throughout the day. Over the years, she had good response to high dose oral NSAIDs as well as IM Ketorolac; however, she stopped all NSAIDs for 1 year after diagnosis of minimal change disease. She was currently on prednisone 5 mg daily after tapering off high doses slowly over 1 year. Other medications include Lisinopril, Vitamin D weekly, and PRN Zofran and Meclizine. She denied any history of joint swelling, psoriatic rash, inflammatory eye disease or inflammatory bowel disease. She had a long history of Hashimoto's hypothyroidism on levothyroxine. Family history included a grandfather with psoriasis, as well as father with back disease of unknown etiology. She works as a registered nurse with no history of smoking or excessive alcohol use. Physical exam was unremarkable. Serological work up revealed positive HLA B27 gene, with elevated inflammatory markers. MRI of the sacrum revealed irregularity of the inferior sacroiliac joints bilaterally with peri-articular fatty marrow infiltration, which per the report was likely sequelae of prior sacroiliac inflammation. Prior MRI of cervical and thoracic spine were brought in by the patient both did not show any evidence suggestive of ankylosing spondylitis or inflammatory back disease, in particular no evidence of syndesmophytes or ossification of the anterior longitudinal ligament. After discussion with the patient, it was decided to treat the ankylosing spondylitis given inflammatory back pain, axial involvement, and high inflammatory markers. The patient was started on Etanercept after negative infectious serologies for tuberculosis and coccidiomycosis. Three months later, patient's inflammatory markers had normalized and back symptoms were improved. Her proteinuria was also stable.

Ankylosing spondylitis is an inflammatory arthritic condition that is included in a larger spectrum of inflammatory diseases called seronegative spondyloarthropathies. It can be associated with other articular features, including axial and peripheral arthritis, synovitis, enthesitis, and dactylitis. There is an association with the gene for HLA (human leukocyte antigen) B27 for the seronegative spondyloarthropathies, although research is ongoing for HLA B27 negative patients. Commonly associated non-articular features include uveitis, psoriasis and inflammatory bowel disease. A minority of patients can experience infrequent extra articular manifestations. Heart, lung, endocrine, and kidney disease in ankylosing spondylitis occur typically as a consequence of long standing inflammation. Cardiovascular manifestations include aortic regurgitation and conduction abnormalities due to sclerosing inflammation of the aortic root (aortitis), aortic valve cusps, and interventricular septum. Restrictive pulmonary disease occurs due to chest wall compromise and spinal mobility in patients with long standing or untreated disease. Autoimmune thyroid disease occurs in higher frequency in ankylosing spondylitis compared to healthy control in a cross sectional study from 2010.[1] The incidence of renal disease is rare apart from amyloidosis in patients with long time untreated inflammatory disease. A retrospective review of 212 cases of ankylosing spondylitis patients between 1978 to 2002, found renal involvement in 32 patients after an average duration of 12 years of ankylosing spondylitis.[2] Clinical findings including microscopic hematuria in 22 patients, proteinuria in 23 patients, nephrotic syndrome in 11 patients, 24 patients had decreased renal function. Renal amyloidosis and tubule-interstitial nephropathy were the most common causes of renal involvement. This retrospective study associated high inflammatory syndrome (including inflammatory bowel disease), sacroiliitis and presence of column bamboo corresponded to the diagnosis of nephropathy. Only a few case reports specifically report glomerulonephritis in ankylosing spondylitis.[3] The relationship between these two conditions is still unclear, given the limited number of clinical cases.

Treatment of ankylosing spondylitis varies based on peripheral versus axial presentation, as well as extra-articular manifestations. Patients with primarily peripheral disease, starts with traditional non-biologic DMARD therapy including methotrexate and sulfasalazine. Patients who present with radiographic axial manifestations are likely to be transitioned to biologic therapy to prevent inflammatory and erosive damage. Patients with non-radiographic changes are closely monitored but may be placed on therapy based on symptoms and change in inflammatory markers. Patients with erosive and inflammatory damage, either peripheral or axial, may still benefit from biologic therapy. Our patient was started on Etanercept, a tumor necrosis factor inhibitor that structurally is a fusion of a receptor to the Fc part of human immunoglobulin G1. Newer biologic therapy outside of the TNF inhibitors are currently in development for treatment of Ankylosing spondylitis.

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