Tumor Necrosis Factor-α Blocker Induced Tuberculosis

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To the Editor

Patients who are treated with anti-tumor necrosis factor-α (anti-TNF-α) blockers are at risk of reactivation of latent tuberculosis (TB) infection (LTBI). The study by Dr. Malaviya, et al describes an approach to the diagnosis of LTBI prior to anti-TNF-α therapy. Screening tests they advise are tuberculin skin test (TST), interferon-γ release assay, or Quantiferon TB Gold (QTB Gold), chest radiograph (CXR), and computed tomogram (CT) of the thorax. We describe 2 patients who developed active TB while taking infliximab, despite the recommended screening tests being negative.

Case 1: In March 2009, a 30-year-old male, with long standing ankylosing spondylitis (AS), complained of worsening of axial symptoms in spite of adequate nonsteroidal antiinflammatory drugs (NSAID). As the AS was active, infliximab therapy was offered. Baseline laboratory findings were: hemoglobin: 9.5 g/dl, leukocyte count: 10,012 cells/mm3, erythrocyte sedimentation rate (ESR) 45 mm/h, platelets: 416,000/mm3, serum glutamic pyruvic transaminase 22 IU/l, and serum creatinine 0.8 mg/dl. The LTBI screen tests including CXR, TST, and QTB Gold test were negative. CT thorax was not done. He was given 2 doses of infliximab (3 mg/kg per dose) 5 months apart. Three weeks after the second dose he developed a low grade fever and cough. There were scattered crackles on auscultation and the CXR showed consolidation in the right upper and middle zones. Although three sputum acid-fast bacilli (AFB) smears and AFB culture were negative, anti-TNF therapy (ATT) was started on clinical grounds. The patient showed clinical and radiological resolution in 6 weeks’ time.

Case 2: In March 2008, a 13-year-old boy was diagnosed to have Juvenile AS with peripheral arthritis. He improved with NSAID over 4 weeks. Six months later the AS relapsed and was refractory to NSAID and methotrexate. Laboratory reports were: hemoglobin: 10.7 g/dl, leukocyte count: 10,700 cells/cm3, platelets: 430,000 cells/cm3, ESR: 100 mm/h, and CXR: normal; TST and QTB Gold were both negative. CT thorax was not done. He was given 2 doses of infliximab (3 mg/kg per dose) 5 months apart. Three weeks after the second dose he developed low grade fever and cough. There were scattered crackles on auscultation and the CXR showed consolidation in the right upper and middle zones. Although three sputum acid-fast bacilli (AFB) smears and AFB culture were negative, anti-TNF therapy was offered. Baseline laboratory findings were: hemoglobin: 10.7 g/dl, leukocyte count: 10,012 cells/mm3, erythrocyte sedimentation rate (ESR) 45 mm/h, platelets: 416,000/mm3, serum glutamic pyruvic transaminase 22 IU/l, and serum creatinine 0.8 mg/dl. The LTBI screen tests including CXR, TST, and QTB Gold test were negative. CT thorax was not done. He was given 2 doses of infliximab (3 mg/kg per dose) 5 months apart. Three weeks after the second dose he developed a low grade fever and cough. There were scattered crackles on auscultation and the CXR showed consolidation in the right upper and middle zones. Although three sputum acid-fast bacilli (AFB) smears and AFB culture were negative, anti-TNF therapy (ATT) was started on clinical grounds. The patient showed clinical and radiological resolution in 6 weeks’ time.

In conclusion, not even a battery of tests can completely exclude LTBI. Hence, even with a negative screen, the possibility of TB flare, or development of new TB infection, must always be kept in mind while initiating anti-TNF therapy. In such patients, combination of TST and IGRA, with regular repetition of these tests, will increase the likelihood of diagnosis of LTBI. It is also recommended that LTBI prophylaxis be continued in those patients receiving anti-TNF-α therapy who have been treated for TB disease prior to initiation of the same.

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