

Severe Psoriasis With Positive Tuberculosis Test and Treatment With Biologic Therapy: A Case Report

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Psoriasis and tuberculosis (TB) are 2 conditions with high prevalence in the general population, often present simultaneously in the same patient. The crucial cytokine involved in the pathogenesis of psoriasis, tumor necrosis factor α (TNF- α), also is important in defending against mycobacteria. Therefore, it is important to screen patients for a latent TB infection (LTBI) while they are undergoing treatment with TNF- α antagonists. We present a case of a patient with psoriasis and LTBI who underwent treatment with etanercept.

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Psoriasis is a chronic skin condition with important social relevance because of its comorbidities and high prevalence in the general population.¹ Tuberculosis (TB) is an infectious disease caused by bacteria belonging to the *Mycobacterium tuberculosis* complex. More than 3.8 million new cases of TB are reported to the World Health Organization each year. In 90% of the reported cases, TB presents as a latent infection; the other 10% of cases present in the active form. The high prevalence of both psoriasis and TB explains how they can be present simultaneously in the same patient.¹

Tumor necrosis factor α (TNF- α), the cytokine involved in the pathogenesis of psoriasis, is essential in the defense of mycobacteria.² Therefore, when

considering a therapy with TNF- α antagonists for psoriasis, it is crucial to screen patients for TB to avoid the possible reactivation of a latent TB infection (LTBI) by choosing an alternative treatment or performing appropriate prophylaxis. We present a case of a patient with psoriasis and LTBI who was treated with etanercept.

Case Report

A 63-year-old man with a history of hypercholesterolemia and hypertension presented to our dermatology department for a severe psoriasis eruption on his legs (psoriasis area and severity index [PASI] score of 18) (Figure, A). He underwent a session of narrowband UVB phototherapy with nearly no clinical response (PASI 17). Tuberculosis screening was performed with a plan to initiate treatment with TNF- α antagonists. Both the Mantoux intradermoreaction test and QuantiFERON[®]-TB Gold (QFT-G) test were positive for TB, but a chest radiograph was negative. Based on these results, a diagnosis of LTBI was determined.

Before initiating therapy with TNF- α antagonists he was administered oral isoniazid 300 mg daily and vitamin B₆ 25 mg daily to reduce the risk for peripheral neuropathy; 15 days later therapy was disrupted due to the onset of fever, sweating, and a palmoplantar psoriatic eruption (Figure, B and C). Laboratory testing revealed a white blood cell count of 4000/ μ L (reference range, 4500–11,000/ μ L), platelet count of 117,000/ μ L (reference range, 150,000–350,000/ μ L), C-reactive protein level of 14.17 nmol/L (reference range, 0.76–28.5 nmol/L), and erythrocyte sedimentation rate of 33 mm/h (reference range, 0–20 mm/h). A chest radiograph revealed hyperdensity of striae in the right apical region, which was fibrosclerotic, likely because of a specific type of TB; therefore, the prophylactic treatment was suspended. An alternative antitubercular therapy with oral rifampin

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Clinical features of psoriasis on the legs, hands, and feet before (A, B, and C, respectively) and after treatment with etanercept (D, E, and F, respectively).

600 mg daily and oral pyrazinamide 0.5 mg 3 times daily was administered; 1 month later prophylactic treatment was suspended due to a 10-fold increase in aminotransferase.

The QFT-G test was still positive, but echography revealed aspecific nonhomogeneous changes of hepatic parenchyma and wall thickening of the descending colon. Three months after prophylactic treatment was suspended, repeat blood tests were

performed and were within reference range; however, because his cutaneous involvement was severe and negatively affected his quality of life (dermatology life quality index, 27), he was started on subcutaneous etanercept 50 mg weekly. After only 4 weeks of treatment he achieved great clinical improvement (PASI 4)(Figure, D–F). The QFT-G test was repeated and was negative for TB. After 12 weeks of treatment the test was repeated again as well as at the end of the

treatment period (24 weeks) and after 16 weeks from suspension. The result was still negative, indicating that there was no reactivation of LTBI.

Comment

The cytokine TNF- α is deeply involved in the pathogenesis of chronic inflammatory diseases such as psoriasis and plays a key role in immune response to mycobacteria infections. As a result, TNF- α antagonists, which are effective in treating psoriasis, may be responsible for inducing new TB infections or LTBI reactivations and for reducing macrophage activity and the ability to create granuloma.³ Therefore, it is clear that patients should be accurately screened for TB and undergo specific prophylaxis starting at least 6 months prior to the administration of TNF- α antagonists. In patients with severe psoriasis who are adequately compliant, it is possible to start biologic treatment after only 1 month of TB prophylaxis under strict medical monitoring. The risk for new infection or reactivation of TB is higher in patients treated with monoclonal antibodies than with the human-soluble receptor of TNF- α , etanercept.⁴ A study by Saliu et al⁵ demonstrated that the inhibition of the activation of T lymphocytes and production of IFN- α caused by infliximab and adalimumab is one of the main mechanisms involved in determining a different risk for TB infection observed in the 3 different biologic therapies.

In our patient, the Mantoux test and QFT-G test were both positive for TB. Because of the severity of

our patient's psoriasis clinical examination, concern for quality of life, and blood tests that were within reference range, we treated him with subcutaneous injections of etanercept 50 mg weekly with strict follow-up. During the 24 weeks of treatment and after 16 weeks from suspension, the QFT-G test was negative.

Conclusion

We report a patient with LTBI and psoriasis with specific incomplete prophylaxis who was treated with etanercept.

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