

Infliximab-induced intertriginous psoriasis in patient with Crohn's disease

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Abstract

Tumor necrosis factor- α (TNF α) inhibition is an effective treatment of moderate-to-severe psoriasis and other diseases (rheumatoid arthritis, ankylosing spondylitis, psoriasis or Crohn's disease). We report a case of a 32-years-old patient affected by Crohn's disease since the age of 25 who started infliximab infusion after four years of treatment with prednisone and azathioprine per os without improvement. After the fifth infusion of infliximab, he developed a form of intertriginous psoriasis which was approached with topical steroid cream. The patient never presented psoriasis in the past. New onset of psoriasis in patients without history for skin diseases (as in our case) is a quite uncommon complication of TNF α inhibitor therapy. The increased production of IFN α during TNF α inhibitor therapy is a possible pathophysiologic explanation for this paradoxical effect of the anti-TNF α .

Introduction

Tumor necrosis factor- α (TNF α) is a proinflammatory cytokine produced by different cell types (activated T lymphocytes, keratinocytes, Langerhans cells, endothelial cells, cardiac myocytes, adipose tissue etc.) and is involved in the pathogenesis of psoriatic skin lesions. TNF α inhibitors have become established agents in the treatment of inflammatory diseases and have shown to be of great benefit in many inflammatory diseases (rheumatoid arthritis, ankylosing spondylitis, psoriasis or Crohn's disease).¹

Case Report

We report a case of unexpected induction of Psoriasis due to the use of intravenous TNF α inhibitor.

A 32-years-old male patient with recalcitrant Crohn's disease of the ileum and descending colon (treated without improvement with pred-

nisone, mesalazine and azathioprine per os) started treatment with infliximab at the dose of 5 mg/Kg at the week 0, 2, 6 and afterwards every 14 weeks. After the fifth infusion he developed erythematous patches with peripheral scaling in the axillary folds and inguinal areas, suggesting the diagnosis of flexural psoriasis (Figure 1). The face and the neck also presented a form of seborrheic dermatitis (Figure 2). The patient never had psoriasis in the past, and he did not have a familiar history of any skin disease. No signs of infection were shown.

The skin biopsy showed psoriasiform hyperplasia, papillary dermal edema with parakeratosis and intracorneal microabscesses of neutrophils (Figure 3).

The infliximab infusion was continued (seeing the good response of Crohn's disease) and a clinical skin improvement was achieved after 40 days of topical steroid treatment.

An expanding literature of experience with anti TNF α associated psoriasis is providing abundant information about this paradoxical effect. Many cases are described. The first published report of this association appeared in 2004 and concerned the development of symmetrical psoriasiform plaques in a patient treated with infliximab for Crohn's disease.² Subsequently plaque, guttate, and pustular psoriasis have all been noted, and palmoplantar pustular disease appears to be more common than idiopathic psoriasis, accounting for up to the 50% of reported cases. Flexural psoriasis and seborrheic dermatitis are a rare form of presentation. In fact, to our knowledge, there are only two articles describing cases of flexural psoriasis during infliximab treatment for Crohn's disease.^{3,4}



Figure 1. Erythematous patches with peripheral scaling in the axillary folds, suggestive for the diagnosis of flexural psoriasis

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Figure 2. Typical erythematous-desquamative patches on the face and neck in seborrheic dermatitis.

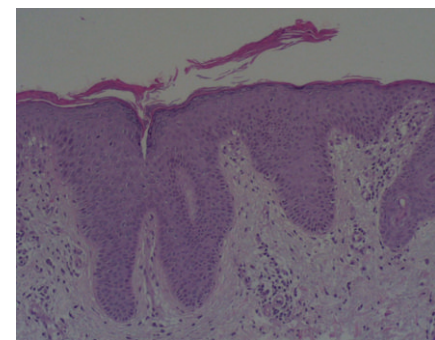


Figure 3. Histologic evaluation demonstrates psoriasiform hyperplasia, papillary dermal edema with parakeratosis and intracorneal microabscesses of neutrophils.

It is well recognized that blocking TNF α may actually favour specific autoimmune phenomena and may activate autoreactive T cells. In addition, with particular relevance to the skin, it may upregulate interferon (IFN)- α activity.⁵ Immunologically this is not unexpected because TNF α is known to negatively regulate the maturation and function of plasmacytoid dendritic cells, which are the major source of IFN- α . Therapeutic inhibition of TNF α signaling would increase IFN α activity and could trigger psoriasis in genetically susceptible individuals.⁵

On the other hand, some cases can be diagnosed as an adverse drug reaction and may contribute to stop the treatment. In literature two-thirds of patients who simply continue anti TNF α therapy improve or resolve the skin disease with steroid treatment. The decisions

need to be based on individual circumstances as the extent and severity of the disease, the efficacy of the anti TNF α in treating the condition for which it was initiated and the availability of realistic therapeutic alternatives.⁵

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