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

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Abstract

Tumor necrosis factor-α (TNFα) 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 TNFα inhibitor therapy. The increased production of IFNα during TNFα inhibitor therapy is a possible pathophysiologic explanation for this paradoxical effect of the anti-TNFα.

Introduction

Tumor necrosis factor-α (TNFα) 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. TNFα 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 TNFα inhibitor.

A 32-years-old male patient with recalcitrant Crohn's disease of the ileum and descending colon (treated without improvement with prednisone, 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 sebopsoriasis (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 TNFα 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 pustolar psoriasis have all been noted, and palmoplantar pustolar disease appears to be more common than idiopathic psoriasis, accounting for up to the 50% of reported cases. Flexural psoriasis and sebopsoriasis are a rare form of presentation. In fact, to our knowledge, ther are only two articles describing cases of flexural psoriasis during infliximab treatment for Crohn's disease.[3,4]

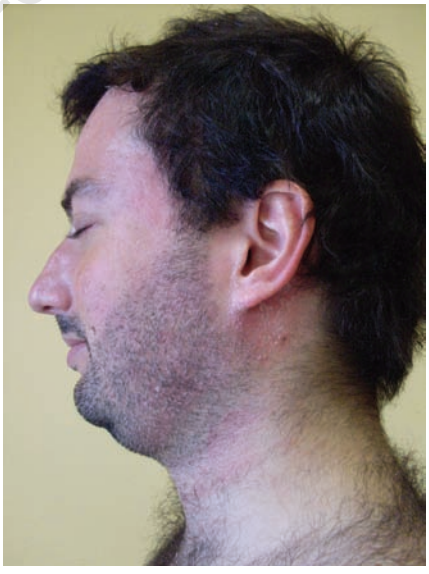

Figure 1. Erythematous patches with peripheral scaling in the axillary folds, suggestive for the diagnosis of flexural psoriasis

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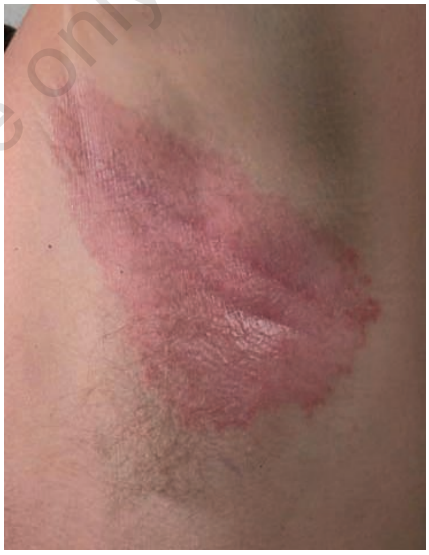

Figure 2. Typical erithemato-desquamative patches on the face and neck in sebopsoriasis.

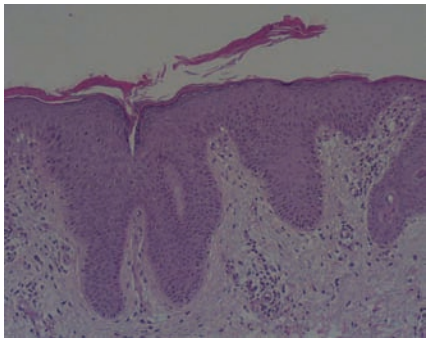

Figure 3. Histologic evaluation demonstrates psoriasiform hyperplasia, papillary dermal edema with parakeratosis and intracorneal microabscesses of neutrophils.

It is well recognized that blocking TNFα 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 TNFα is known to negatively regulate the maturation and function of plasmocytoid dendritic cells, which are the major source of IFN-α. Therapeutic inhibition of TNFα signaling would increase IFNα 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 patiens who simply continue anti TNFα 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 TNFα in treating the condition for which it was initiated and the availability of realistic therapeutic alternatives.[5]

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