CASE REPORT

Infliximab-Induced Psoriasis in a Patient with Crohn’s Disease

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ABSTRACT

Infliximab is a well-known treatment for inflammatory bowel diseases (IBDs) and psoriasis. Paradoxically, there have been numerous reports of new-onset psoriasis following tumor necrosis factor-α antagonist therapy in patients with IBD. Here, we report a case with Crohn’s disease who developed Infliximab-induced plaque-type psoriasis 4 months after initiation of treatment with Infliximab.

Key words: Biological therapy, Crohn’s disease, Infliximab, psoriasis, tumor necrosis factor-alpha inhibitors

INTRODUCTION

Biologic therapy has a well-known established role in the management of many inflammatory conditions including inflammatory bowel disease (IBD), rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis and psoriasis. Unfortunately, in recent years a paradoxical effect has been reported with anti-tumor necrosis factor-α (TNF-α) therapy in patients with Crohn’s disease (CD), which is the development of psoriatic skin lesions. Numerous case reports, case series, and reviews have been published, mainly describing new-onset or worsening of existing psoriasis and psoriasiform exanthemata in adult patients following TNF-α antagonist therapy. However, researchers have recognized an association between IBD and psoriasis, with an increased prevalence of psoriasis in patients with IBD and vice versa. These data suggest that genetic variants common to both conditions may coexist and research for genetic polymorphisms common to these conditions is being carried-out.

CASE REPORT

A 19-year-old Arab female who had been diagnosed with CD the year before was given an infusion of Infliximab which was initiated (5 mg/kg body weight) at weeks 0, 2, 6 with maintenance infusions given every 8 weeks. There was a significant improvement of the bowel disease. However, after the 4th infusion of Infliximab (4th months), the patient gradually developed erythematous and scaly plaques all over her body, including the upper and lower limbs [Figure 1]. The rash initially improved with topical steroids then reappeared after each infusion of Infliximab. There were no associated symptoms. Both parents had psoriasis. On physical examination, there were erythematous papules and plaques with silvery scales on the trunk, buttocks, arms, legs and scalp. Skin punch biopsy was consistent with early lesions of psoriasis. A diagnosis of plaque-type psoriasis was made and treatment with calcipotriol/betamethasone ointment was initiated. The lesions improved after a few weeks but recurred upon every Infliximab infusion, with a total of four infusion cycles. Since the psoriatic lesions improved
Psoriasis is a chronic skin disorder affecting almost 2% of the population. Biologic therapy is one of the treatment options for resistant cases of psoriasis. Unfortunately, it can be seen as a paradoxical side effect with biologics. In the literature, there are 209 cases of anti-TNF-α induced or exacerbated psoriasis. IBD accounts for 20% of all the reported cases, a vast majority of which have CD. In a cohort of >700 Infliximab treated patients with CD, a variety of skin eruptions occurred, almost 10% of which were severe enough to warrant dermatology consultation, and for which psoriasiform exanthema was confirmed. It has been suggested that these skin eruptions are different from classic psoriasis when there is no anti-TNF-α exposure, and the preferred term here is “psoriasiform”. The onset of psoriasis following TNF-α antagonists does not appear to be drug specific, and there is a risk of recurrence following the use of an alternate anti-TNF-α agent. It may occur at any age (mean: 30.1 years), and in most patients, there is no personal or family history of psoriasis. The development of the psoriatic lesions may occur any time after the introduction of anti-TNF-α. Clinically, palmoplantar pustular psoriasis is the predominant form (52-56%), followed by plaque psoriasis (49-50%), and guttate lesions (12-15%). In IBD patients, the majority of cases described are plaque-type (61%), mainly affecting the palmo-planter region (82%).

The study by Seneschal et al. of skin biopsy specimens from 11 patients who developed psoriasiform lesions following TNF-α antagonist therapy showed the following variable histologic features: Spongiosus, increased type 1 Interferon expression and increased expression of the chemokine receptor (CXCR). Different reports indicate that patients continued Infliximab therapy because their skin eruptions were successfully treated with topical therapy. However, Rahier et al. and Cullen et al., reported that up to 40% of patients with IBD with a new-onset or worsening existing psoriasiform skin lesions discontinued anti-TNF-α therapy. The pathogenesis of this paradoxical process is not yet fully understood, but there is evidence to support the role of increased interferon-alpha expression as a result of TNF-α blockade. It is possible that variants in the IL-23 axis play a role in the development of this paradoxical adverse event.

In a study done by Sherlock et al., a significant association was found between polymorphisms in the IL-23R gene and the subsequent development of psoriasis/psoriasiform lesions, indicating that variants in the IL-23 axis play a role in the development of this side-effect. An explanation for the occurrence of psoriasis in IBD patients could simply be the simultaneous concurrence of the two diseases, as the incidence of psoriasis in patients with IBD ranges from 6-11%, compared to 2-3% in the general population. However, the majority of reported cases had no history of psoriasis before the start of biological therapy and upon withdrawal of the drug there was a complete regress of the clinical lesions. This adverse event seems to indicate a true increase in the prevalence in anti-TNF-α treated patients and the Food and Drug Administration now recommends that manufacturers of TNF-α antagonist products record this in the prescribing information of the product.

REFERENCES

Results from the infliximab multinational psoriatic arthritis controlled trial (IMPACT). Arthritis Rheum 2005;52:1227-36.


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