DEVELOPMENT OF SEVERE PSORIASIS SCALP AND ALOPECIA DURING TREATMENT OF CROHN’S DISEASE WITH INFliximab: A PARADOXICAL SIDE EFFECT

INTRODUCTION
Anti-tumor necrosis factor (anti-TNF) therapy is used in a variety of autoimmune-mediated diseases and inflammatory conditions, including rheumatoid arthritis (RA), psoriasis, and inflammatory bowel disease (IBD), specifically Crohn’s disease (CD) and ulcerative colitis (UC). The complete mechanism of action of these agents is elusive; however their efficacy and safety profile has been proven in the treatment of these conditions. With an increase in the use of anti-TNF agents, including infliximab, adalimumab, and certolizumab pegol, there is also an increased recognition of paradoxical adverse effects, defined as de novo and/or exacerbation of disorders thought to be improved by anti-TNF therapy. We present a case of a patient with Crohn’s disease who developed severe psoriasis scalp and alopecia during treatment with anti-TNF-α (Infliximab) that required discontinuation and initiation of other treatments.

CASE REPORT
We report a 32-year-old woman with extensive CD, without response to conventional treatment (mesalazine and systemic corticosteroids). She had no personal or family history of psoriasis. She received Infliximab therapy at the dose of 5 mg/kg body weight for 10 years with good control of the disease. She paradoxically developed a cutaneous eruption with psoriasiform morphology and distribution and a very severe psoriasis scalp with thick, crusted plaques covering entire scalp and alopecia during treatment with Infliximab (Fig. 1-5).

RESULTS AND EVOLUTION
Skin biopsy confirmed the diagnosis of psoriasis. There was prominent hyperkeratosis and parakeratosis with intraepidermal infiltrates of neutrophils either in the stratum corneum and thickened projections of the prickle cell layer of keratinocytes. The papillary dermis between the rete ridges shows capillary loop dilatation and duplication and there is a mixed dermal inflammatory infiltrate with some neutrophils migrating into the epidermis. These lesions cleared and improved (Fig. 6-11) after topical application of corticosteroids and vitamin-D analogs and methotrexate subcutaneous 15 mg weekly. Besides cessation of the anti-TNF-α treatment was necessary. She has not yet had aggravation of intestinal manifestations but we believe it is too early to draw conclusions.

DISCUSSION
Treatment with anti-tumor necrosis factor (TNF)-α for Crohn’s disease is relatively safe, although the occurrence of new onset psoriasis in patients treated with anti-tumor necrosis factor (TNF) antibody therapy is increasingly recognized. This phenomenon is apparently paradoxical as anti TNF agents are also effective in the treatment of psoriasis. Most of current data on this apparent drug reaction come from the rhTNF antagonist induced psoriasiform lesions involves a disruption in cytokine balance by TNF blockade, allowing unopposed interferon-alpha (INFo) production by plasmacytoid dendritic cells (PCDs) in genetically predisposed individuals. Nevertheless the pathogenetic mechanism of this contradictory side effect has not yet been explained. The management options of this phenomenon include continuing with the same anti-TNF-α therapy, switching to other biological therapy or changing to an immunosuppressant therapy, although the best option is still unclear.