# Sustained remission of nodular inflammatory acne after treatment with infliximab

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people, there is an increased likelihood of atherosclerosis, which leads to endothelial damage and aggregation of platelet-derived growth factor (PDGF). It has also been shown that the homodimer PDGF-BB and transforming growth factor (TGF)-β1 influence osteoblastic differentiation of undifferentiated mesenchymal cells.[4] These factors also play a key role in systemic sclerosis (SSc); (TGF)-β, interleukin-1 and PDGF are upregulated in *in vitro* studies, resulting in collagen deposition.[5]

To our knowledge, there are no previously reported cases of an association of DISH with SSc. Although the occurrence of both in our patient may be a coincidence, we suggest that presence of a particular cytokine milieu and biochemical changes in DISH may encourage the fibroblast proliferation and collagen deposition that occurs in SSc, and explain how our patient developed two linked pathophysiological conditions.

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## Sustained remission of nodular inflammatory acne after treatment with infliximab

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A 22-year-old man presented with a 6-year history of severe nodular inflammatory acne.

On physical examination, acne lesions were seen on the neck, chest and posterior shoulders, without facial involvement. The nodules were fused to large, painful, oddly-shaped aggregates with or without pustules and comedones (Fig. 1). The patient had no fever. Individual lesions were present for up to 4 months. Healing of the inflammatory lesions was followed by fibrotic plaques on the side of the patient's neck and chest and by keloidal scars on his back. The inguinal folds, axillae and intergluteal cleft were free of lesions.

The patient's medical history included ulcerative colitis since the age of 14 years, which was in complete remission with azathioprine 125 mg daily. Previous treatment of the patient's acne with oral antibiotics (doxycycline), potent topical corticosteroids (clobetasol propionate 0.05%), intralesional corticosteroids and photodynamic therapy all led to only temporary and marginal benefit. Isotretinoin, up to 80 mg daily, had been tried, but discontinued after 6 months owing to lack of efficacy and development of depressive symptoms such as reduced self-esteem and confidence, loss of interest, and reduced energy.

Given the severity of the acne, we started the patient on dexamethasone pulse therapy, 300 mg per day on three consecutive days every 4 weeks. This treatment was discontinued after two courses owing to lack of efficacy and side-effects of restlessness and aggressive behaviour in the week after each pulse administration. The lesions were disabling for the patient, thus we started him on infliximab 5 mg/kg intravenously at 8-week intervals. A substantial improvement was noted after the first dose, and after three infusions, no new lesions or activation of the old lesions were noted, and the comedones had disappeared (Fig. 1). The patient's psychological condition also improved considerably. Infliximab was discontinued after eight infusions. Four months later, a very slight relapse of the acne occurred. The incidental small lesions were successfully treated with intralesional corticosteroids. The acne was very limited, and the favourable effect of infliximab was still appreciable at follow-up 1 year after discontinuation of treatment (Fig. 1).

Holland *et al.*[1] reported a pivotal role for cellular inflammatory events at all stages of acne lesion development. The response to infliximab in nodular inflammatory acne may be attributed to its anti-inflammatory effect. Tumour necrosis factor (TNF)-α, interleukin-1α and interferon-γ all play a role in hypercornification of the infundibulum. This might explain the reduction in non-inflammatory lesions seen with infliximab. It was shown *in vitro* that *Propionibacterium acnes* stimulated the production of TNF-α from keratinocytes,[2] thus a reduction in *P. acnes*-induced inflammation by TNF-α may be an additive effect of infliximab in nodular inflammatory acne.

Only two case reports have been published to date on the successful treatment of acne with anti-TNF-α drugs.[3,4] Paradoxically, the use of infliximab has been shown in some studies to provoke a new onset of acne vulgaris.[5] The pathogenesis of this infliximab-induced acne is unknown.

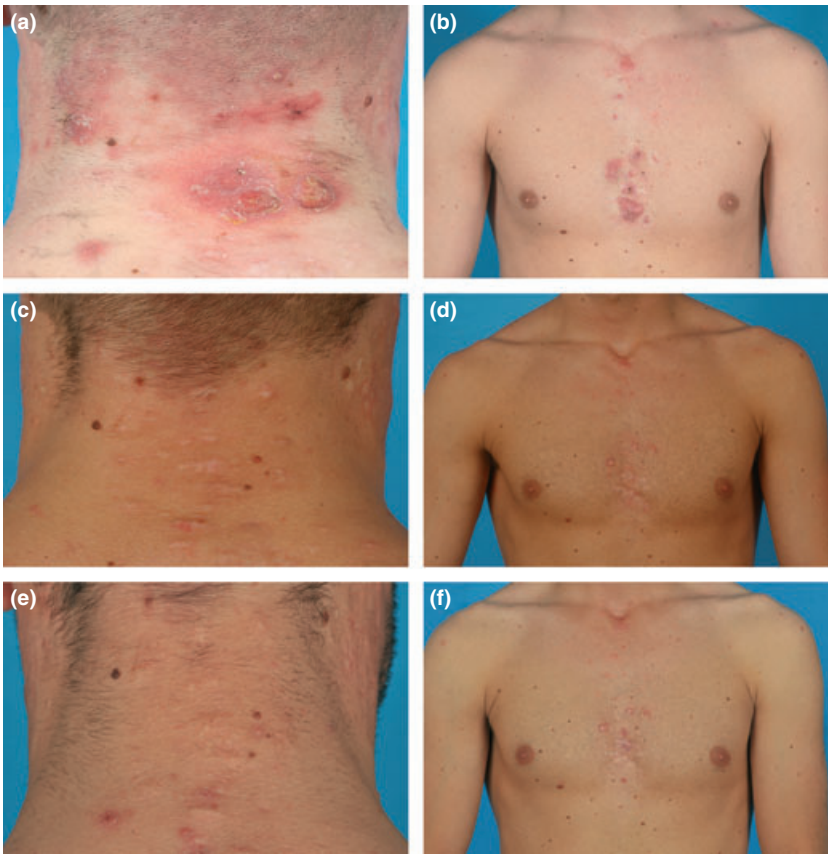


**Figure 1** Nodules, infiltrates, pustules and comedones on (a,c,e) the neck and (b,d,f) the chest after (a,b) two courses of dexamethasone pulse therapy and before treatment with infliximab; (c,d) complete remission after three infusions of infliximab at 8-week intervals; (e,f) no recurrence at follow-up 1 year after discontinuation of infliximab.

To our knowledge, this is the first case of severe nodular inflammatory acne with sustained response after discontinuation of infliximab. Therefore, we suggest that in patients with severe nodular inflammatory acne, infliximab may be a valuable treatment for inducing and maintaining sustained remission.

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## Yellow chromonychia after ascorbic acid application

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Ascorbic acid is commonly used for the treatment of melasma, and as a preservative in galenic preparations, because of its antioxidant properties.[1] We present a case of yellow chromonychia in a patient who was using an ascorbic acid galenic preparation.

A 74-year-old man presented with a 1-month history of yellowing of the nails on his hands and feet. He reported that the discoloration had been evident for a month.

On physical examination, yellowing of the nails was seen on both hands and both feet. The proximal side of each nail plate was spared (Fig. 1a). The border of the yellow area



 