photosensitizer into the abnormal target cells.[3] Clinically, the lesions of KWE are erythematous. Pretreatment lesional erythema significantly correlates with the cure rate for PDT in actinic keratoses. Erythema is likely to reflect the amount of oxygen available locally.[4]

The KWE gene locus has been mapped to 8p22–p23. To date, specific gene mutations in this region have not been identified.[5] The abnormality within the gene locus is preferentially expressed on the palms and soles, but can also be expressed, albeit transiently, in some cases on the limbs or even the face, as has been seen in this family.[2] PDT seems to have the ability to modify this expression.

PDT may be a successful treatment for KWE. The underlying pathogenesis of KWE is thought to be caused by a focal triggering event that leads to a precipitous halt to normal differentiation of keratinocytes, with subsequent apoptosis high in the epidermis and rapid recovery with proliferation of the basal cells. The initiating trigger is conducted to the neighbouring keratinocytes, and the lesion expands laterally. We believe that PDT destroys the keratinocytes in the target area, therefore changing the susceptibility to the trigger. Further studies are required to compare gene expression between PDT-treated sites and the neighbouring skin that still expresses the disease.

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Conflict of interest: none declared.

Accepted for publication 11 June 2010

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Systemic sclerosis in a patient with diffuse idiopathic skeletal hyperostosis

doi: 10.1111/j.1365-2230.2010.03992.x

A 62-year-old Guyanian woman presented with a 5-month history of patches on her arms. She worked as a nurse and had a 5-year history of diffuse idiopathic skeletal hyperostosis (DISH) involving her thoracic spine.

On physical examination, erythematous and hyperpigmented patches were seen on the patient's hands and forearms. There were no other skin changes and no systemic symptoms.

On histological examination of a biopsy taken from a lesion, thickening of the dermal collagen and aggregates of lymphocytes around mid and deeper dermal vessels were seen.

Laboratory investigations showed that the patient was positive for anti-nuclear antibodies (1 : 640) and anti-centromere antibodies, and negative for double-stranded DNA antibodies. Routine biochemistry tests and a chest X-ray gave normal results.

The patient was diagnosed with limited scleroderma. Over the next few months she began developing progressive difficulty in swallowing, shortness of breath, further tightening and thickening of the skin on her forearms, dactylitis, and calcinosis. There was involvement of the face with microstomia and beaking of the nose. There were no symptoms of Raynaud disease or dyspepsia reported. The patient was started on oral prednisolone 60 mg/day, and methotrexate was introduced. Several months later, with the patient on 20 mg/week methotrexate, the patches had flattened and the patient's quality of life had much improved.

DISH or Forestier disease was first described over 50 years ago[1] as an ossifying noninflammatory, nonerosive enthesopathic skeletal disease. It involves calcification and ossification of soft tissue, in particular of ligaments and entheses. Another classification[2] describes involvement of at least four contiguous vertebral bodies (predominantly vertebrae 7–10) as an important diagnostic criterion. DISH affects 3–6% of the population over 40 years of age[1] and 11% of those aged over 70 years. It also predisposes to vascular calcification.[2] Patients who have DISH are also found to have high growth hormone (GH) and insulin-like growth factor (IGF) levels. GH can induce the local production of insulin, and IGF-1 and IGF binding proteins in chondrocytes and osteoblasts,[3] leading to osteoblast cell growth and proliferation. These circulating hormones may also have a role in scleroderma. Case reports suggest that GH suppressants such as octreotide improve gastric motility in patients with scleroderma, and may therefore play a role in long-term prognosis.[3]

The cause of DISH is unknown, but particular risk factors are implicated on the basis of its frequent association with obesity, hypertension, diabetes mellitus, hyperinsulinaemia and dyslipidaemia.[3] In predisposed

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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Conflict of interest: none declared.
Accepted for publication 26 September 2010

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Sustained remission of nodular inflammatory acne after treatment with infliximab

doi: 10.1111/j.1365-2230.2010.03998.x

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.