Sir,
Superficial granulomatous pyoderma (SGP) is a rare, chronic variant of pyoderma gangrenosum (PG), first described by Winkelmann et al. (1). We present here a case of SGP, which demonstrates its ability to invade regional important structures.

CASE REPORT

A 65-year-old man presented with a 10-year history of a slowly, progressive, erythematous, crusted plaque on both sides of his face. This caused bilateral lower eyelid ectropion and scleromalacia of his right eye (Fig. 1a).

Skin biopsies were taken (Fig. 2). Gram and Ziehl-Neelsen stains, immunofluorescence studies, and fungal cultures were all negative. No foreign material was identified. Auto-antibody tests were also negative and blood dyscrasia was not detected. There was no clinical evidence of malignancy, such as lymphadenopathy, or an associated disease, such as inflammatory bowel disease. The clinicopathological features were consistent with SGP.

Topical clobetasol propionate ointment 0.05% and oral prednisolone failed to suppress his disease. Cyclosporine (100 mg twice a day, body weight 91 kg) halted the progression of the condition. His facial plaque resolved after 5 months and there has been no relapse on continued cyclosporine for more than one year (Fig. 1b).

DISCUSSION

SGP shares several features with PG, such as a similar clinical appearance, pyodermatous histology and pathergy. However, there are differences between them: in SGP there is a much slower progression, less tendency for the edges to be undermined, and a lack of regular association with other diseases. Although there are no pathognomonic histological features of SGP; foreign-type granulomas, sinus tract formation and pseudoepitheliomatous hyperplasia are regularly present in SGP but far less in PG (2, 3).

Suppurative granulomatous infections should be considered as the main histological differential diagnosis of SGP, such as those caused by mycobacteria or deep-seated fungi. Halogenoderma is another histological consideration, however, the lack of contact with iodine or bromide, excludes this diagnosis in our patient.

The site of predilection of this condition is on the trunk, but unusually our case presented on the face. Successful treatment of SGP has included the use of antibiotics, dapsone and oral corticosteroids. Recalcitrant cases have resolved with cyclosporine, mycophenolate mofetil and intravenous immunoglobulins (1–5). Lachapelle et al. have suggested that cyclosporine should be a first-line treatment for this condition (5). However, in our opinion, as this condition progresses slowly, treatments with less significant complications should be tried first.
The juxtaocular location of the lesion in our patient was sight-threatening. The more common truncal SGP are unlikely to cause damage to vital structures as they enlarge. Although pathergy is more likely in SGP than PG, the benefit of a reparative operation of his bilateral ectropion is worth considering. However, our patient has repeatedly opted not to undergo such a procedure.

This case illustrates the difficulties of treating SGP and confirms that oral cyclosporine is a useful treatment for SGP. It also highlights that it is a diagnosis of exclusion. There should be particular effort to find an infective aetiology as the treatment may involve potent immunosuppressive therapy. The location of the lesion and the presence of damaged neighbouring structures are important factors that determine how aggressively it should be treated.

ACKNOWLEDGMENTS

We are grateful for Dr Eduardo Calonje, St John’s Institute of Dermatology, London, UK, for reviewing the histology.

REFERENCES