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Challenging treatment of keloid scars: a case report

Keloids and hypertrophic scars are caused by cutaneous injury, such as trauma, insect bites, burns, surgery, vaccination, acne or viral infections. The pathophysiology of keloid formation is not well known. Keloids do not regress spontaneously [1] and are distinct from normal and hypertrophic scars [2]. Keloid formation may cause severe cosmetic outcome and dysfunction with marked itch, pain, and psychological disturbances.

Keloid management is based on a multimodal approach, however, no gold standard for their treatment exists. A recent review described treatment options available that are selected for each individual patient [1]. An updated algorithm for treatment of keloids has been presented [3].

Corticosteroids are applicable for the treatment of hypertrophic or keloid scars [4]. Among them, the most commonly used is triamcinolone acetate (TAC) injected with a 25-27G [5] or 27-30G needle [6].

A healthy 20-year-old man (182 cm, 69 kg) was treated with 0.1% tretinoin cream and oral tetracyclines followed by isotretinoin at 40 mg/d with a cumulative dose of 120 mg/kg for his acne. The acne responded well on the face and sternum area, but keloids started to form, and the remaining isotretinoin capsules were used for 10 days.

As treatment, intralesional methylprednisolone acetate was applied twice using a 30G needle (BD Microlance, 0.3x13 mm) with a two month interval, and clobetasol cream was applied daily.

After a 2.5-year follow-up, three keloids were selected for ultrapulsed CO_2-laser treatment followed by 0.1% betamethasone/2% fucidic acid cream under pressure occlusion. During follow-up, the lesions recurred with a few pustules. Keloids were topically treated with betamethasone/fucidic acid cream and pustules were broken and treated with 0.1% lapis solution. One month later, the laser-treated keloids were further treated with intralesional steroids as before. After 2-3 months, the laser-treated lesions had relapsed, and new keloids became visible (*figure 1A*).

A plastic surgeon was consulted and did not recommend surgical treatment. Therefore, intralesional steroid injections were started at 1.5 to 2-month intervals using 3+3 mg/mL betamethasone acetate-betamethasone sodium phosphate and 40 mg/mL methylprednisolone acetate, with 1.5 – 1.9 mL total injection volumes. After 24 injections, the injected amount was gradually decreased to 0.5 – 1.1 mL after the 30th treatment (*figure 1B*). Thereafter, the injection volumes were further decreased to 0.2 – 0.7 mL, with twice the amount on visits 33 (1.1 mL) and 37 (1.9 mL), until the 50th injection. Thereafter, injection volumes were 0.2 – 0.6 mL until the 54th injection.

At this point, the skin condition was stable, and the intervals between the 55th to 67th injections were prolonged to 3 to 6 months. The patient tolerated the injection pain well. At follow-up, after 10 years, the keloids were in remission (*figure 1C*). One small new

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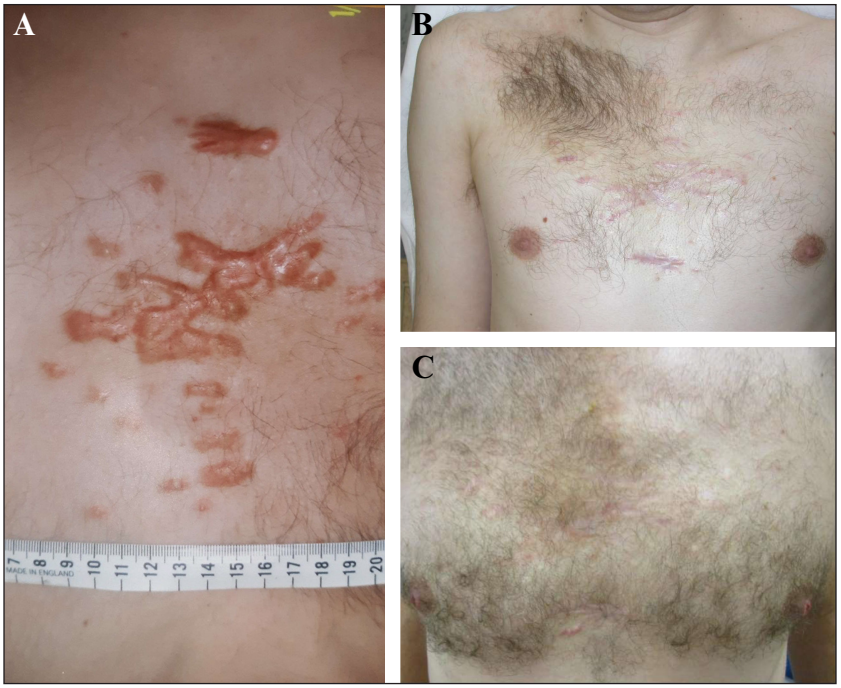

Figure 1. Clinical images of the patient at the beginning of intralesional steroid injections (December 2000) **(A)**, after 30 sessions of intralesional steroids (July 2005) **(B)**, and after 67 injections up to July 2013, followed by 10 years of follow-up (August 2023) **(C)**.

keloid appeared outside the earlier treatment area which was treated with an intralesional steroid.

The treatment of this patient's keloids was challenging. The initial progress was slow for the first 2.5 years. However, the patient was highly motivated despite the exceptionally high number of treatments. After about 4.5 years, the keloids gradually started to respond to intralesional steroids with 30 injections. Thereafter, the third phase was started with smaller injection volumes, lasting 1.5 years. The fourth treatment phase included rather small injection volumes for 5.5 years, and finally, remission was achieved after a total of 67 injections.

The best results are obtained with intralesional steroids combined with silicone dressing [1, 3, 7]. This is also our clinical experience. The number of injections reported in studies is 2-8. Follow-up times in the literature are often described as 1-3 months, occasionally 4-8 months, up to 1-2 years [8], and seldom up to 10 years [7]. TAC injections yield regression in 50-100% of cases, and the re-occurrence rate is reported at 33% after one year, and 50% after five years [9]. These results are in agreement with those of our patient who obtained remission after 13 years of steroid injections. Thus, a prolonged period of injections may be necessary for an individual patient. Interesting and effective results might be obtained when combining 5-FU (5), verapamil, hyaluronic acid [10], or intralesional cryotherapy [8] with intralesional steroid injections. ■

Ethical approval: *the patient has given his written consent for the publication of his case.*

Conflicts of interests: *none.*

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Lasthenie de Ferjol syndrome associated with skin picking disorder

We report two dramatic cases of Lasthenie de Ferjol syndrome, in which severe iron-deficiency anaemia was the result of an extreme form of skin picking disorder belonging to the group of self-induced factitious obsessive-compulsive behaviours [1].

The term "Lasthenie de Ferjol syndrome" was coined by the French haematologist, Jean Bernard, in 1967 to describe a factitious disorder characterized by severe iron-deficiency anaemia secondary to repeated, self-induced clandestine blood spoliations [2]. This name refers to the heroine of Barbey d'Aurevilly's novel '*Une histoire sans nom*', who died of anaemia after bleeding herself to death by sticking needles into her heart [3, 4]. Since then, additional cases have been reported. Most of them involved women aged 20-40 years with borderline personalities presenting with a history of recurrent iron-deficiency anaemia secondary to deliberate self-bloodletting [5].

Patient 1 was a 56-year-old unemployed single woman, with a medical history of recurrent gastritis and professional burn-out, who sought consultation for chronic facial skin ulcerations that had been evolving for at least five years. She reported a long-standing history of acne, with intense pruritus that led to compulsive scratching of the face. Clinical examination revealed four well-demarcated ulcerations on a background of scarred skin (*figure 1A*). Wound cultures were sterile. Histological examination of a skin biopsy of the ulceration margin revealed reactive epithelial hyperplasia with no evidence of malignancy, inflammatory disorder or infection. The

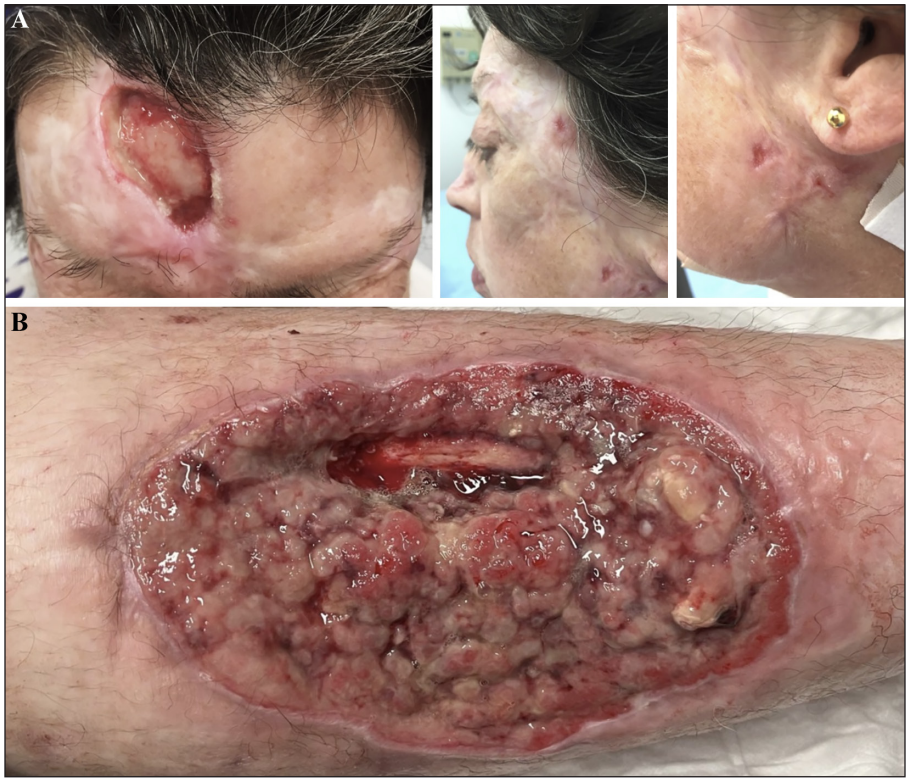

Figure 1. A) Clinical photographs of the first patient's facial lesions. These photographs show several well-demarcated angular-shaped skin ulcerations on the patient's forehead, left preauricular area, chin and nose. Initially, the ulcerated wounds largely healed with local dressings and avoidance of scratching, but relapsed quickly after initial improvement. The patient admitted to the use of tweezers to relieve irrepressible pruritus to the point of haemorrhaging. The large areas of irreversible scarring reflect the long-term chronicity of self-mutilation. The frontal ulceration led to a brain abscess two years later. **B)** Clinical photograph of a leg ulceration of the second patient. This photograph shows a large, well-defined skin ulceration of the right lower limb. On the lateromedial side, the ulceration is so deep that it exposes part of the underlying tibial bone. The patient reported that the wound sometimes bled in spurts. The evolution of this lesion was very similar to that of the lesion on her left leg, for which she had undergone amputation the previous year. When the possibility of self-mutilation was raised, the patient was lost to follow-up.