the FAM111B gene c.1883 G>A resulting in a heterozygous change from a serine amino acid to an asparagine amino acid at position 628, and diagnosis of POIKTMP was established.

Genetic testing is the gold standard for diagnosis of POIKTMP, but may be unavailable or time consuming. Histopathological examination of the skin may serve as a helpful tool in diagnosing patients with POIKTMP. Scleroderma-like changes with thickened collagen fibres and disrupted elastic fibres network are distinctive, although we did not see elastic globules in the upper dermis reported by others authors.[2,3] These changes are absent in RTS and may be helpful in establishing the diagnosis. Another interesting finding in our case was abundance of eosinophils between the thickened collagen bundles, which was not previously reported. Blood eosinophilia was described in one patient in Mercier *et al.*[3] group, but the significance of this finding is unclear. Interestingly, in the early biopsies, we found lichenoid changes that have not been previously described. It is possible that skin lesions have different histopathological presentation at different stages of the disease or vary according to the morphology of the lesion.

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Various cold plasma devices for the treatment of actinic keratosis

Editor

We have read with interest the letter entitled 'Actinic keratoses treated with cold atmospheric plasma' by Wirtz *et al.*,[1] and we are glad to see that other dermatologists are corroborating our February 2017[2] report on the use of cold plasma to treat actinic keratoses. We hope this will encourage the use of plasma for other clinical indications. Undoubtedly, a number of new studies of cold plasma treatment of actinic keratoses will appear given the great advantages of this treatment modality.

The study presented by Wirtz *et al.* shows significant differences compared with our results that we believe are worth noting. While the authors utilized twice weekly treatment sessions over the course of 4 weeks, we attempted to match the less taxing treatment protocol of current destructive modalities by performing only a single treatment followed by evaluation one month later. The other main difference was that instead of a gas jet type plasma we used a nanosecond pulse dielectric barrier discharge plasma, which delivers uniform plasma directly to the tissue at a precisely controlled rate.

Wirtz *et al.* followed a much larger number of lesions than we had done in our pilot study, and they reported an overall improvement of 55% of lesions, which included the full clearance of 23% per cent of lesions, assuming that no new lesions appeared. It appears that the majority of this improvement represents Olsen II and III lesions turning into Olsen I lesions. In our study, albeit on a substantially smaller number of lesions, the total improved lesion count was 70% which included a 53% full resolution of lesions only after one single cold plasma treatment. Perhaps, this can be attributed to the different uniformity of plasma delivered by the different devices used in our study and the one by Wirtz *et al.*

The reduction of lesion thickness, scaling and haemorrhagic crust over a larger treated area reported by Wirtz *et al.* is a great benefit not only from an aesthetic viewpoint, but also because those are major factors in discomfort, irritation related to these lesions. On the other hand, from the viewpoint of preventing cancer development form actinic keratoses, we propose that further studies evaluating the efficacy of different cold plasma devices and treatment protocols evaluate the number of lesions fully cleared as a benchmark of treatment efficacy, instead of the number of lesions with decreased clinical grading for the following reasons:

The clinical grading used by Wirtz *et al.* was initially reported in the context of a clinical trial for masoprocol[3] and used since, as a way to assess the 'severity' of actinic keratoses, hypothesizing that the evolution of thin lesions through thickening eventually leads to squamous cell carcinomas. Recently, it was demonstrated that the majority of *in situ* squamous cell carcinomas

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develop in association with actinic keratoses where the cellular atypia is restricted to the basal portion of the epidermis (AK I).[4] It has also been demonstrated that there is no correlation between histological grading and the Olsen clinical grading,[5] making it impossible to assign a risk level to any actinic keratosis solely based on its clinical characteristics. Another study showed that clinical or histological thickness of actinic keratoses neither correlates with the level of histological dysplasia nor with p53 expression[6] further undermining the notion that only thick actinic keratoses represent a risk for squamous cell carcinoma development.

Because thin, Olsen I grade actinic keratoses may pose as great a risk for the development of squamous cell carcinomas as thicker lesions, treatment modalities for actinic keratoses should be evaluated by their success of fully eliminating all treated lesions, not just reducing their clinical thickness.

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Positive Jacquet's sign in traction alopecia

Editor

A 32-year-old previously healthy woman was observed with a 3-year history of hair loss. On physical examination, she had decreased hair density and smooth padded alopecic patches on the occipital and parietal scalp. The fronto/temporal hair rim was preserved. A positive Jacquet's sign was present with easy formation of several folds in the occipito/parietal scalp; the pull test was negative. The patient had been wearing a traditional religious hair cover-up for around 3 years (Fig. 1), and she was previously diagnosed and treated as lichen planus pilaris without improvement. On trichoscopy, hair casts, white pin point dots, follicular miniaturization and broken hairs were noted. A TrichoScan® (Fotofinder Derma, Teachscreen Software, Bad Birnbach, Germany) was performed, which reported 15.6% anagen, 84.4% telogen, 69.5% terminal hair, 30.5% vellus hair, 89 FU (follicular units), a hair density of 155 hairs/cm^2, vellus hair density 43.2/cm^2 and 98.5/cm^2 of terminal hair. Biopsies demonstrated broken hairs, mild perifollicular inflammation, loss of terminal hairs and trichomalacia (Fig. 2). With all these data, the diagnosis of traction alopecia (TA) was made. We suggested the interruption of the traditional religious cover-up, topical steroids and minoxidil 5% with good results.

Traction alopecia is defined as hair loss triggered by prolonged and persistent tension on the hair due to different hairstyling. It has been described mainly in Afro-American women, but has been reported in different races and both genders.[1]

The Jacquet's sign, also known as fold sign, consists of folding an area of the scalp between the hands, and if it is possible to form several folds, the sign is considered positive. This clinical assessment can be useful in the diagnosis of alopecias and



Figure 1 Clinical findings in Jacquet's sign positive. (a) Positive Jaquet's sign performed by applying pressure into the fronto-occipital region; and (b) into the bi-temporal region; (c) posterior view; and (d) superior view of patient wearing religious hair cover-up.

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