Tufted hair folliculitis.

Tufted hair folliculitis is an uncommon phenomenon, associated with scarring alopecia in which tufts of hair emerge from a single follicular opening. We believe this is the first case of tufted hair folliculitis to be reported from Saudi Arabia. We believe that tufted hair folliculitis is a secondary phenomenon to other causes of scarring alopecia. A 46-year-old male patient presented with an 8-year's history of recurrent bouts of burning pain and oozing lesions on his occipital scalp, at times accompanied by tender occipital adenopathy. Examination revealed an area of smooth shiny-scarred plaque devoid of hair follicles on the occipital region (Figure 1). At the periphery, grouped hairs were seen emerging in tufts from single hair openings. Still further peripherally, there was a diffuse perifollicular scaling. Pus was draining from some of the follicular openings at times of activity. The rest of skin exam, mouth and nails were normal. Lab tests including full blood count, liver function tests, and electrolytes were within normal limits. Additional investigations including antinuclear antibodies, extractable nuclear antigen, immunoglobulins, serum complement, serological tests for Syphilis were normal or negative. Fungal cultures from the affected area were negative, but bacterial culture grew staphylococcus aureus. Pathology revealed dermal granulation tissue with severe acute and chronic inflammation and fibrosis surrounding terminal hair shafts, the majority of which do not have a follicular sheath. Direct immunoflorescence for IgM, IgG, IgA, C3 and C1q all were negative. Chlorohixedine topical washes and intermittent antibiotics reasonably controlled the activity of the process orally (Cefadroxil).

Tufted hair folliculitis is a follicular and perifollicular inflammatory process of the scalp,



Figure 1 - Occipital scalp with scarring alopecia and multiple tufts of hair arising from a single opening.

characterized by recurrent courses, resulting in scarring alopecia and the appearance of multiple tufts of hair; each has 10-15 hairs emerging from a single opening. Smith and Sanderson first described this phenomenon in 1978.¹ Since that time, an additional 24 cases have been reported; most of those cases were reported from Europe and only a single case from North America.² We believe this is the first case to be reported from Saudi Arabia. All cases that have been reported where characterized by cutaneous bacterial infection with S.aureus, scarring alopecia with tufts of hair and fibrosis around the remaining follicles.³ Histological examination revealed perifollicular inflammation around the upper portion of the follicles but completely sparring the hair root region.⁴ The cellular infiltrate was mixed, composed of neutrophils as the prominent cell. Plasma cells were present in some cases. At areas of disrupted hair follicles, there was a foreign body reaction with focal collection of macrophages and foreign body giant cells. In the upper and mid dermis there was a perivascular lymphocytic infiltrate. Pathogenesis of tufted hair folliculitis is not fully understood. Whether it is a distinct disease or just a secondary phenomenon to other causes of scarring alopecia like folliculitis decalvans or dissecting folliculitis of the scalp is not known. Loewenthal in 1947 was the first to report on the normal presence of follicular groups; he named them compound follicles. His illustration, though, showed only 2-3 hairs emerging from single follicular orifices. Histology of those follicular groups in the normal scalp showed that those hairs are in anagen. Loewenthal noted that compound follicles were more common in patients with folliculitis and that pustules were confined to these follicles.⁵ Headington used the term follicular units; which is formed by 2-4 terminal hairs and 1 or 2 vellus hairs.⁶ Smith and Sanderson suggested that local inflammation caused destruction of the superficial parts of adjacent follicles which led to the formation of a common follicular duct due to local fibrosis and scarring.¹ Oakley and Scollay considered the same mechanism.7 Metz and Metz suggested an underlying nevoid abnormality which predisposes them to recurrent infection and thus scarring.8 Tong and Baden suggested the same pathogenesis;² yet this seems to be unlikely as new tufts continue to appear in previously normal areas, and we would expect it to appear at an earlier age.^{3-4,9} Oakley and Dawber postulated that there may be an underlying abnormality of the host immune response.7,10 Dalziel et al found a preponderance of telogen hairs which were confirmed histologically. Plucked hair analysis suggested that retention of telogen hair within the follicle for several hair cycles was responsible for tufting.⁴ The high this presence of number of

telogen hair is highly unusual. In the normal human scalp there is no synchronized hair cycling as occurs in animals and only 10-20% of hairs are in telogen at any time.⁴ They suggested that the local inflammatory process around the superficial parts of the follicles resulted in the retention of telogen hair. Luelmo et al noted a preponderance of anagen hair, they also noted that erythema and scaling are the first events in extension of the disease.³

Treatment. Management of tufted hair folliculitis is difficult. Topical antiseptic treatments reducing scaling with topical keratolytics and shampoos containing tar derivatives,³ and multiple courses of oral antibiotics were tried (doxycycline, tetracycline, flucloxacillin and erythromycin) cefadroxil in our case which seems to control the progression of the disease by decreasing the inflammatory process; but had no effect on the scarring or the already formed tufts. Surgical excision of the involved areas had been performed, which is possible if the involved area is small and seems to give good results.^{2,11}

In conclusion, we believe that tufted hair folliculitis is a secondary phenomenon to other causes of scarring alopecia. The association of this phenomenon with folliculitis decalvans, dissecting cellulitis of the scalp,⁹ folliculitis decalvans,¹² acne keloidalis nuchae¹³ and their clinical, pathological and evolutionary courses support our belief. We suggest also the possibility of punching out those tufts and cutting them into micrografts and transplanting them as we do when we correct a bad transplant.

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