Tufted hair folliculitis: a case report and literature review

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KEY WORDS

tufted hair folliculitis, staphylococcal infection

ABSTRACT

A 37-year-old man presented with a one-year history of erythematous pruritic plaque on the parietal region of the scalp. Physical examination revealed multiple bundles of hair emerging from a single dilated follicular opening. Histological examination of tufted areas showed scarring in the papillary to mid-dermis with perifollicular acute and chronic inflammation. Staphylococcus aureus was found to be present in the bacterial cultures. The patient was treated with systemic antibacterial modalities that proved effective. The presented case shows a strong pathogenetic correlation between tufted hair folliculitis and staphylococcal infection, a finding that further supports infectious nature of the disease and classifies it as a subtype of folliculitis decalvans.

Introduction

Tufted hair folliculitis is a rare folliculitis of the scalp that resolves with patches of scarring alopecia within multiple hair tufts emerging from dilated follicular orifices (1). Tufting of hair is caused by clustering of adjacent follicular units due to a fibrosing process and to retention of telogen hairs within a dilated follicular orifice (2). Various pathogenetic mechanisms have been proposed including nevoid abnormalities, recurrent infections of the follicles, and retention of telogen hair in the tufts (3). We present a patient with tufted hair folliculitis who was effectively treated with antibacterial medications, verifying the infectious nature of the disease.

Case report

A 37-year-old man was referred to our department with a history of chronic, relapsing inflammatory lesions of the scalp that had been present for 1 year. He complained of intermittent itching and sticky hair in the affected area. He had no past medical history and was generally healthy. The physical examination revealed a circumscribed area of 10 cm in length and 8 cm in width on the right temporo-parietal side of the scalp that was characterized by sticky hairs and adherent yellow-white crusts. On their removal, the scalp was erythematous, infiltrated, partly erosive, and showed livid, erythematous patches of scarring with consecutive alopecia surrounded by inflamed margins. The remaining follicular openings were enlarged and contained 20 to 30 apparently normal hair shafts (Fig. 1). Results of bacteriological swabs of pus extruded from dilated follicular ostia revealed *Staphylococcus aureus*. Histopathological studies showed perifollicular inflammation of plasma cells, lymphocytes, and neutrophils around the upper portions of the follicles, sparing the hair root level. Some of the follicles were dilated and

deformed, containing an abundance of debris, keratotic material, and bacteria (Fig. 2). Routine biochemical and hematological investigations, including peripheral immunophenotyping of lymphomononuclear cells, immunoglobulins, and phagocytic capacity were normal.

According to clinical and histopathological findings a diagnosis of tufted hair folliculitis was made and treatment with intravenous ceftriaxone was started at a dosage of 1,000 mg twice per day. After two weeks of therapy, the pustular lesions regressed completely. At the follow-up visit 6 months later a residual erythematous infiltrated cicatricial area was seen, with some hair tufts emerging from a single orifice.

Discussion

Tufted hair folliculitis was first described by Smith and Sanderson in 1978 (4). Worldwide around 30 cases of tufted hair folliculitis have been reported to date. Most of the reported patients came from the United States and Europe. Four cases were found in Japan (5). The patients are 20 to 60 years old, with the greatest proportion of those affected being in their 30s (6). There is a male preponderance. The sites of predilection are the occipital and parietal areas (7). Frequently reported subjective symptoms are pruritus and pain (8). *Staphylococcus aureus* infection is most often detected in the lesions (9).

Differential diagnosis in the early stages mainly includes folliculitis decalvans, folliculitis keloidalis nuchae, kerion Celsi, perifolliculitis capitis abscedens et suffodiens (Hoffmann), trichostasis spinulosa, follicular lichen planus, and relapsing staphylococcal folliculitis (10). On the other hand, the presence of keloidal plaques in acne keloidalis nuchae, the coalescing nodules discharging purulent material in dissecting cellulitis of the scalp, the erythematous plaques covered by pustules replete with fungal elements in kerion Celsi, and the absence of follicular pustules in follicular lichen planus distinguish these diseases from tufted hair folliculitis.

Based on the clinicopathological data from a group of 44 patients with scarring alopecia, Annessi concluded that tufted hair folliculitis could be differentiated from folliculitis decalvans only by finding several hair tufts scattered within patches of scarring alopecia (1). His presumption has been confirmed by Powell et al. who performed lymphocyte staining on affected scalp biopsies and found no differences (11). These authors concluded that the two entities formed part of a spectrum of a single disease. According to Trueb et al. (3) staphylococcal infection is the initial causative factor. Underlying differences in follicular anatomy or host response may be important in determining which reaction pattern occurs in an affected individual (12). The development of atrophy with loss of hair structures (in folliculitis decalvans) or of hair tufts (in tufted folliculitis) depends upon the depth and destructive potential of the inflammatory process. Tufted hair folliculitis has been described following a scalp injury (13), pemphigus vulgaris on the scalp (14, 15), and as a consequence of tinea capitis infection (16). These observations contradict the classic infectious pathogenetic hypothesis and accentuate the crucial role of a specific host response dysfunction. However, Powell et al. did not find any evidence of local immune suppression or failure that could explain the abnormal host response to a certain presumptive superantigen (*S. aureus*, desmoglein, etc.) (11).

Figure 1. Scalp, right temporo-parietal side: erythematous, infiltrated plaque of scarring alopecia with dilated follicular openings with tufts containing 20 to 30 apparently normal hair shafts.

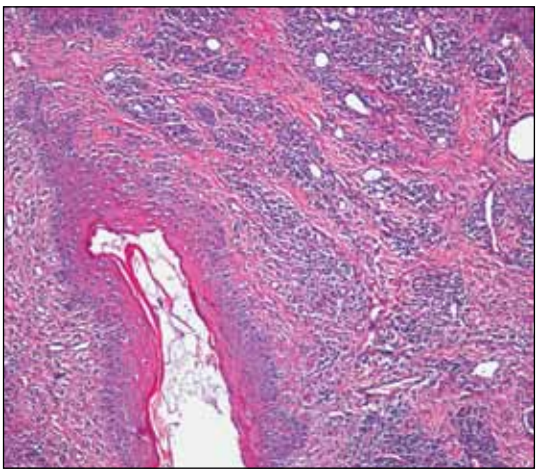

Figure 2. Dilated follicle containing keratotic material, debris, and numerous cocci. Scattered perifollicular mixed cell inflammation of plasma cells, lymphocytes, and neutrophils.

The cutaneous pathologic examination shows polymorphous inflammatory infiltrate with abundant plasma cells in the upper and mid dermis, which is mostly perifollicular, and the presence of normal and independent follicles in the deep dermis, which, while ascending, converges to a common dilated follicular channel (3). Our patient showed numerous cocci in the dilated follicles due to the inflammation. This finding corresponds to observations by Trueb, et al. on the infectious nature of the disease. The sparse inflammatory infiltrate may be in concordance with an insufficient immunologic host response evolving into this specific reaction pattern.

The therapeutic approach is problematic. All reported topical treatments have been unsuccessful. Nadifloxacin, a new quinolone antibiotic with a broad-spectrum antibacterial effect and little resistance, has been proven effective in a single patient (17). It could be used to prevent the spread of lesions or recurrence. Oral antibiotics may stabilize the disease; however,

their long-term use should be avoided to prevent possible hepatic or renal side effects. Rifampicin, as one of the best antibiotics active against *S. aureus*, has been proven as the most effective therapeutic modality to control the pustular phase of the disease, as well as to prevent possible relapses (18). More definitive results have been reported after surgical excision of the involved area (19). This therapeutic option corresponds with another pathogenetic presumption, according to which a nevoid malformation defines the area of inflammation and consecutive scar formation in tufted hair folliculitis.

We have presented an anecdotal case of tufted hair folliculitis as a consequence of a chronic bacterial infection that was effectively treated with antibiotic drugs. More clinical observations and further studies are needed to further evaluate the pathogenetic relevance of the infectious nature of this disease.

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