DISSEMINATED NODULAR GRANULOMATOUS PERIFOLLICULITIS

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Abstract

Nodular granulomatous perifolliculitis is a well-recognized infection of the dermal and subcutaneous tissue caused by dermatophytes, which normally do not invade beyond the epidermis. We report here one such case that occurred in an immunosuppressed individual. The patient was a 35-year-old farmer who presented with small pruritic eruption that had initially appeared on the lower leg and then had gradually spread to hair-bearing areas of the body, finally producing nodular and pustular inflammatory lesions with exacerbations and remissions. Fungal examination by direct potassium hydroxide mount and culture revealed *Trichophyton rubrum*. Granulomatous changes were seen on histopathological examination. The patient completely responded to systemic antifungal therapy.

Key words: *Granulomatous perifolliculitis, dermatophytes, immunosuppression*

Dermatophytes generally cause infection of the stratum corneum and superficial layers of the skin and thus, are rarely found in the layers deeper than the basal cell layer. However, Domenico Majocchi has described exceptions to this—either as small perifollicular papular form or a deep subcutaneous nodular form. These are well recognized but uncommon dermal infections caused by dermatophytic fungi, occurring usually in individuals who have chronic dermatophytosis but maybe otherwise healthy. It may also occur in individuals with immunological disorders, in which case, systemic dissemination may sometimes occur. In both the conditions, trauma leads to disruption of follicle (directly or indirectly). Such a passive introduction of the organism together with keratin or necrotic material into the dermis initiates the infection. Topical agents are ineffective therapeutically due to insufficient penetration of the drug into deeper layers of the skin, and thus, oral antifungal agents are required. Here, we report a nodular granulomatous form with extensive dissemination in a patient from East Delhi, India, who recovered after a course of Terbinafine. Such cases have not very often been reported from this part of the country.

Case Report

A farmer aged 35 years, resident of East Delhi, India, presented with a six month history of pin-head sized, itchy eruption starting from the right lower leg, which later spread to involve the left leg, thigh and back, especially the hair-bearing areas (Fig. 1).

On examination, multiple lesions, varying from inflammatory nodular to pustular, were seen all over the body. Some follicular pustules with perifollicular erythema and induration were also present on the lower abdomen. There was considerable scaling in these areas. The nodules were almost painless but occasionally tender. In these areas, the lesions progressed with occasional spontaneous remission and relapses. There was also associated fissuring of soles for the past 3-4 months. The patient had received a course of oral steroids (dexamethasone, 5 mg twice daily) for almost a year. There was no other clinical abnormality or disease. A provisional diagnosis of nodular perifolliculitis was made. Skin scraping and biopsy were taken from the patient’s back, both lower legs, and the abdomen. Pus was obtained from the pustular lesions present on the back and legs. These were subjected to direct microscopic examination of 10% KOH mount followed by culture on standard Sabouraud dextrose agar (SDA) with and without chloramphenicol and cycloheximide. Growth obtained on SDA was identified on the basis of the rate of growth, texture, pigment production, and distinct morphological structures. Other physiological tests such as urea hydrolysis, growth on DTM (dermatophyte test media), and nutritional requirements were also performed. Baseline investigations, which included liver and kidney function tests and complete haemogram, were found to be within normal limits.

KOH examination showed thin septate non-pigmented fungal hyphae (Fig. 2). Growth was obtained on the culture from all the specimens after incubation at 25°C over a period of one week (Fig. 3). Lactophenol cotton blue mount of the culture isolate showed abundant hyphae and numerous teardrop shaped aleuropores present along side of thin septate hyaline hyphae. Based on these morphological features as well as other physiological tests, the isolate was identified as a typical strain of *Trichophyton rubrum*. Histopathologically, hyperkeratosis with moderate lymphocytic infiltration in the papillary dermis was seen. There was mild spongiosis with lymphocytes and neutrophils in the epidermis. However, no fungal elements were seen on histopathology (Fig. 4). A repeat

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Received: 29-11-06
Accepted: 10-02-07
biopsy with examination of several sections produced the same
observation. Dexamethasone was tapered off gradually and the
patient was started on terbinafine 250 mg twice daily. There
was a marked improvement of the lesions with reduction of
erthema and flattening of the inflammatory nodules. At the
end of two months treatment, there were no residual clinical
lesions and repeat KOH examination of specimens from earlier
involved sites could not demonstrate fungal elements.

Discussion

Disseminated nodular granulomatous perifolliculitis (a
variant of Majocchi’s granuloma) is typically described either
as small perifollicular papular form or a deep subcutaneous
nodular form. It is commonly caused by dermatophytes,
predominantly by T. rubrum. This is usually the most
frequent cause of chronic dermatophytosis; however, T.
mentagrophyte, T. violaceum, Microsporum audouinii,
M. gypseum, M. canis and T. verrucosum may also be the
causal agents. The follicular dermatophytic infection of the
dermis, in immunocompetent individuals, usually occurs
in women who shave their legs in an upward direction,
perhaps driving fungal elements into the dermis. The deep
subcutaneous nodular form, as in our case, is generally seen
in immunosuppressed individuals and is characterized by
groups of firm or fluctuant nodules occurring on scalp, hands,
legs, etc. This may even clinically resemble recurrent bacterial
pyoderma. The question is, what factors allow or contribute
to the persistence and growth of dermatophytic fungi in an
environment, which is markedly different from the superficial
epidermis and subsequently predispose certain patients
to nodular granulomatous reaction. It may either develop
following a follicular disruption or passive introduction of the
fungi into the dermis. The keratinous material present may
penetrate into the dermis and act as a substrate for the survival
of the organism. Thereafter, it sets in a chain of chronic
granulomatous reaction characterized by epitheloid cells,
small fungal cells, or occasional giant cells. In our case, the
intense itching of the widespread lesions could possibly have
led to deeper invasion by the organism. It is well documented
that production of proteinases (keratinases) by T. rubrum
leads to degradation and digestion of host tissue, and thus,
contributing to deeper penetration of the skin by the organism.
Prolonged steroid intake reduces the cell mediated immunity
and prevents an effective immune response thus predisposing
the subject to chronic dermatophytosis. Moreover, T. rubrum
produces mannan (cell wall component), which has the ability
to diminish the immune response that is far more potent than in other dermatophytes. The property of *T. rubrum* to survive as spores and remain in stratum corneum can eventually lead to deeper tissue penetration. Conditions like malnutrition, leukemias, lymphomas, or immunosuppressive drugs can affect the function of polymorphonuclear leucocytes and macrophages, which are the defenses against invasion by dermatophytes. Physical barrier of the dermis, degree of hydration and lipid composition of the stratum corneum play a major role in preventing such dermal invasions. However, disruption of such mechanism can allow the persistence and widespread dissemination of the agent. Moreover, certain researchers have hypothesized that once dermatophytosis is established it can act as a source of immunosuppression on the host and further aggravate the existing infection. It is also noted that under certain conditions, appearance of fungal cells in the dermis is prevented and may not be detected, as observed in our case. It is also well documented that serum inhibitory factors have a suppressive effect and may help to limit dermal invasion of dermatophytes. Our patient was on steroid therapy, and subsequently, when tapered off and treated with antifungal agents, the condition resolved. *T. rubrum* was demonstrable in all the deeper portions of the nodular lesions by culture methods, and histology suggestive of a granulomatous reaction was also seen. This evidently reinforced our previous diagnosis with an added element of dissemination. Hence, in such presentations, mimicking (bacterial) pyoderma, the fungal etiology should not be overlooked otherwise treatment with empirical antibiotics may subsequently result in dissemination due to the underlying fungal agent.

**Acknowledgement**

We acknowledge the help of the Pathology Department, University College of Medical Sciences and Guru Tegh Bahadur Hospital for reviewing the histopathology slides.

**References**


**Source of Support:** Nil, **Conflict of Interest:** None declared.