# ABOUT NODULAR ERUPTION: Case Report

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## OBSERVATION:

A 52-year-old woman was referred to us for evaluation of an intensely pruritic eruption, most marked on the extremities. The eruption consisted of numerous 0.5-1.5 cm nodules (Figure 1).

Many lesions were hyperpigmented and had a crateriform morphology (Figure 2). The lesions had been appearing over a two years period, and treatment with antihistamines and corticosteroid ointments had been unsuccessful.

Otherwise the patient was healthy and a laboratory examination, including serum bilirubin, urea nitrogen, glucose and hemoglobin was normal. The family history was unrevealing, and was negative for an atopic diathesis.

A biopsy specimen did not show a specific histologic features but just hyperkeratosis, hyperacanthosis and infiltration of the dermis by a mononuclear cells.

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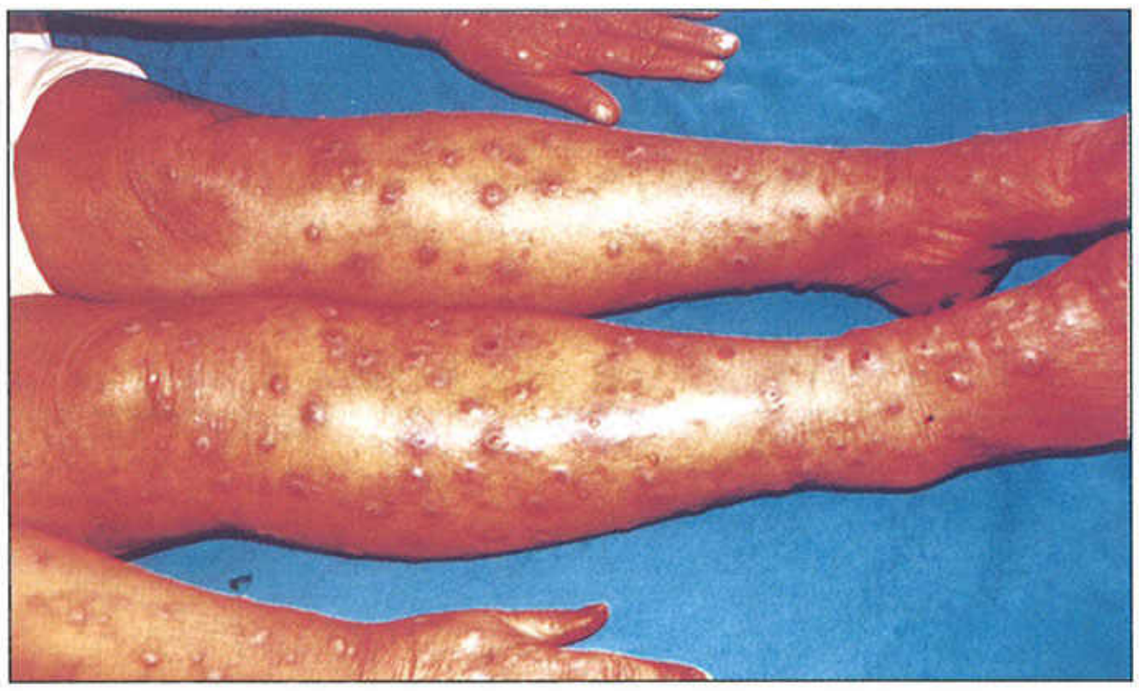

*Fig. 1: Nodular eruption on the legs and the forearms*

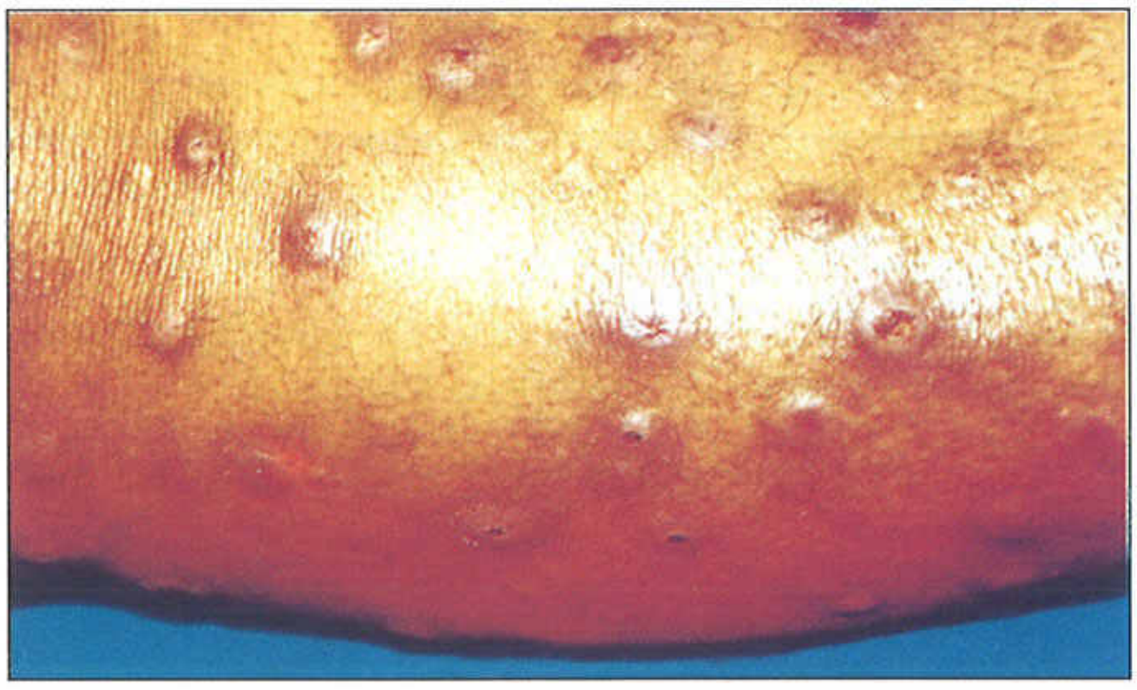

*Fig. 2: The crateriform aspect of some lesions.*

## COMMENT:

Prurigo nodularis (PN) is an uncommon disease, first described by Hyde [1] in 1909. The clinical diagnosis of PN is by an eruption of numerous and intensely pruritic, pink papules and nodules, often with a verrucous or crateriform morphology.

Lesions range from 0.5 to 3.0 cm in diameter and are often distributed over the extensor surfaces of the extremities.

Histologic features are non-specific and include: hyperkeratosis of the edpidermis; irregular acanthosis; infiltration of the dermis by histiocytes, lymphocytes, mast cells and eosinophils[2]. In 1934, Pautrier described "massive hyperplasia of nerves",

but neural hyperplasia is not the most prominent feature of PN[3]. The number of Merkel cells is increased in biopsy specimens of PN and may be a component of the neurocutaneous abnormalities associated with this disorder[4].

Various illnesses have been reported in association with PN including anemia, uremia, hepatic dysfunction[3], hepatitis[5], gluten intolerance(6), and Hodgkin's disease[7]. PN may resemble hypertrophic lichen planus, chronic eczema and drug eruptions, and may be misdiagnosed as such.

The etiology of PN is unknown but many factors may contribute to its pathogenesis. Atopic dermatitis has been associated with many cases of PN and some authors distinguish between an "atopic" form of PN and non-atopic form. Patients with the former have an earlier age of onset and cutaneous hypersensitivity to various environmental allergens. The non-atopic form is characterized by a later age of onset and absence of cutaneous hypersensitivity[8]. In some cases of PN, patients are found to have allergic contact dermatitis, and the eruption has successfully cleared after avoiding the offending contactant[9]. Some authors believe that there is a continueum between PN and contact dermatitis(3).

Many patients claim that the eruption began with an insect bite reaction(3,6). In some cases, psychosocial factors are felt to precipitate episodes of pruritis(3,6).

Treatment of PN is difficult, and various remedies have been tried with variable success. Recommended therapies include(6): intralesional corticosteroids; systemic antihistamines; sedatives; PUVA; and oral antibiotics such as erythromycin.

Topical doxepin would also be a reasonable therapy. Thalidomide has been used successfully in a photosensitive and HIV positive patient with PN(10). Patch testing may also prove useful if an allergen can be identified(9).

In conclusion, we feel that PN is an uncommon disease, usually of unknown etiology. However, it is a diagnosis of exclusion, and other diseases associated with pruritus(cutaneous or systemic) must be considered before the patient is labeled as having PN.

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