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PRURIGO NODULARIS

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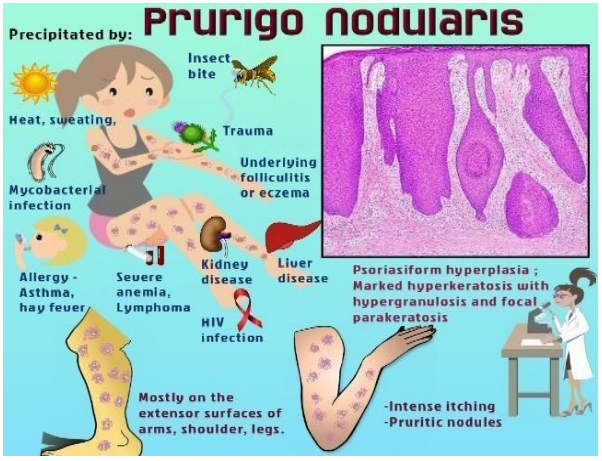
ABSTRACT
Prurigo nodularis (PN), or chronic prurigo, is a distinct disease characterized by the presence of chronic pruritus and multiple localized or generalized pruriginous lesions resulting from persistent scratching. We report a case of a 22-year-old female presented to the female dermatology ward with chief complaints of generalized itching persisting from one year and half years, with relative sporing of the trunk and face, and itchy lesions over the bilateral limbs and trunk for one year. On examination, multiple hyperpigmented papules and nodules with excoriations over the bilateral limbs and trunk, a few erosions, crusting, oozing, and tenderness were also present. Skin biopsy showed hyperkeratosis, parakeratosis, acanthosis, focal spongiosis and hypergranulosis, elongated and blunt rete ridges. Also seen perivascular and periadnexal inflammatory infiltrate composed of lymphocytes and plasma cells and fibrocollagenous thickening suggestive of PN. Serum vitamin D was low (18.2nmol/l). The patient was managed with antihistamines, topical corticosteroids, vitamin D supplementation and methotrexate. Significant improvement was noted. This case highlights the importance of recognizing nutritional factors like vitamin D deficiency and the role of immunomodulators in resistant prurigo nodularis.

KEYWORDS: Prurigo Nodularis (PN), Chronic Itch, Dermatology Case Report, Inflammatory skin disease, Interleukin, Interleukin-31, Immunomodulators.

INTRODUCTION
Prurigo nodularis (PN) is a long-lasting inflammatory skin disorder. It typically manifests as one or more intensely itchy nodules that are symmetrically located on the trunk or limbs. These nodules are firm, range in color from flesh to red, and have a hyperkeratotic appearance.[1] The condition can manifest in individuals of any age. It is frequently linked to another skin hypersensitivity disorder, such as atopic dermatitis or chronic itching from various causes. Diagnosis is primarily based on clinical evaluation, although some conditions may mimic it, necessitating careful differentiation. This condition is associated with considerable physical and psychological distress and often proves resistant to treatment.[2]



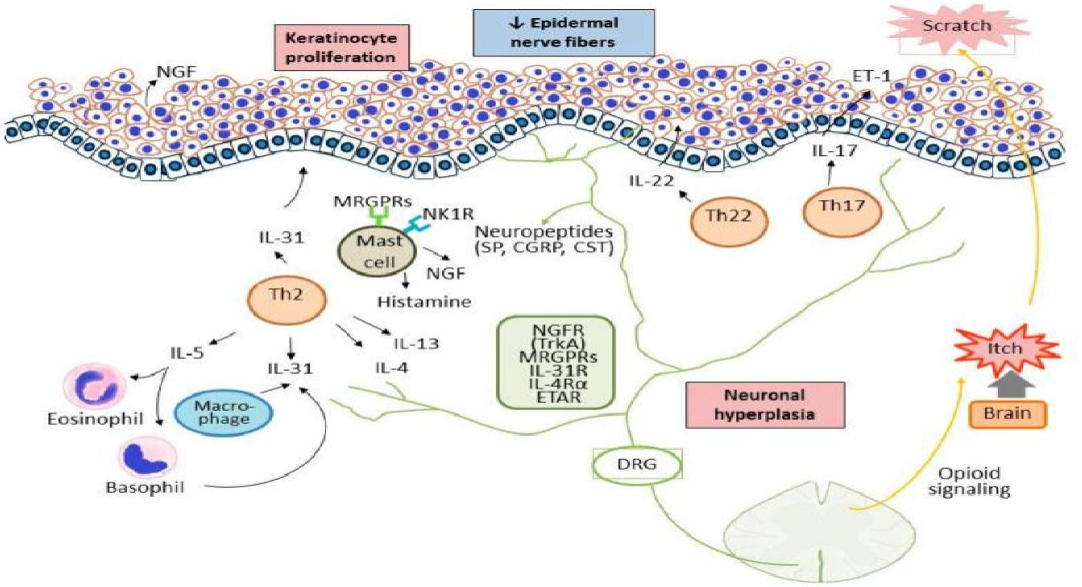
EPIDEMIOLOGY
An estimated 125,322 ambulatory care visits and 87,634 PN cases occur annually. According to a different estimate, the prevalence of PN is between 36.7 and 148.53 per 100,000 people.[1] Additionally, because comorbidities are more common and specialty care is used more frequently, PN presents a substantial health-care burden.[3] Ethnicity and genetic predisposition seem to play a role since African-Americans are 3 to 4 times

more likely to have PN than white patients.[4] Other conditions that have been reported to induce PN include internal malignancy, renal failure, and psychiatric conditions. In HIV-positive patients, PN has been reported to be predictive of advanced immunosuppression.[5]

ETIOLOGY

The precise cause of prurigo nodularis is still unclear. It is believed to be linked to nerve dysregulation, as skin biopsies have revealed an increased number of nerve cells in the dermis but fewer in the epidermis. Additionally, there is a higher presence of Merkel cells in the epidermis and the papillary layer of the dermis, along with more neutrophils and mast cells, leading to increased cytokine release, which might account for the intense itching. This itchiness can be triggered by factors such as insect bites, stress, dermatitis, or other underlying skin or systemic conditions. There is also a connection with brachioradial pruritus, which is caused by traction on spinal nerves in the neck. Scratching can lead to skin thickening and inflammation, exacerbating the condition.[6] Individuals with PN also show elevated levels of interleukin 31 (IL-31), a T-cell–derived highly prurigogenic cytokine.[10]

PATHOGENESIS



The interaction between the skin, immune, and nervous systems in individuals with PN is complex. Keratinocytes are a major source of growth factors and inflammatory cytokines, which trigger immune activation. The increased presence of Th2, Th17/IL-17, Th22/IL-22, eosinophils, and mast cells initiates inflammation and encourages the excessive proliferation of keratinocytes. At the same time, neuronal hyperplasia in the dermis releases neuropeptides like substance P, which stimulate the immune response by interacting with immune cells and keratinocytes. The cycle of itching and scratching worsens the inflammation, and scratching causes mechanical damage to peripheral nerve fibers in the epidermis. SP stands for substance P; NK1R is neurokinin 1 receptor; CST refers to cortistatin; NGF is nerve growth factor; TrkA is tyrosine kinase receptor A; MRGPRs are Mas-related G-protein-coupled receptors; DRG is dorsal root ganglion.[9]

CLINICAL MANIFESTATIONS

elevated lumps on your skin that typically have a thick, dry crust on top. The bumps may match the color of your skin. They may also be black, brown, dark red, or light pink. The depth and size of the bumps may differ. They can occasionally impact both your dermis, or middle layer of skin, and epidermis, or top layer of skin, severe itching, Burning or stinging at times.

PN bumps could be classified as
Nodules that penetrate your epidermis and have a diameter greater than one centimeter. Papules are only found on the skin's surface and have a diameter of less than one centimeter.

Plaques are scaly, shallow lesions that are larger than one centimeter in diameter but do not penetrate below the skin's surface.[7]

DIAGNOSIS AND CLINICAL ASSESSMENT

A comprehensive clinical evaluation is usually the first step in the diagnosis of prurigo nodularis. A thorough patient history, including any prior skin conditions, family history, and lifestyle factors, will be taken by medical professionals. The appearance and distribution of the nodules will be the main focus of a physical examination.

Tests for Diagnosis

Additional tests may be carried out to rule out other conditions, even though a clinical evaluation is frequently adequate for diagnosis. These could consist of.

Skin biopsy: To confirm the diagnosis, a tiny sample of skin may be obtained for analysis in a lab. Laboratory tests: To look for infections or underlying conditions, blood tests may be carried out.

Allergy testing: Tests to identify particular allergens may be carried out if an allergic reaction is suspected.[8]

CASE PRESENTATION

A 22-year-old female presented to the female dermatology ward with complaints of generalized itching persisting from one year and half years, with relative sporing of the trunk and face, and multiple itchy lesions over the bilateral limbs and trunk for one year. The lessions were gradually increasing in size and number.

PHYSICAL AND SYSTEMIC EXAMINATION

General physical examination revealed a moderately built female, afebrile, with stable vitals. No pallor, icterus, or lymphadenopathy noted.

Skin Inspecion

Multiple hyperpigmented papules and nodules which excoriations over bilateral lower limbs and trunk Surface: Rough, scaly, and excoriated; few lesions with crusting and lichenification.

PARAMETERS	DAY-1	DAY-2	DAY-3	DAY-4	DAY-5	DAY-6	DAY-7
TEMPERATURE(°F)	98.2	98	97	97	97	98	98.2
BLOOD PRESSURE(mm/hg)	120/80	120/80	120/70	110/70	110/70	110/80	120/70
PULSE RATE (bpm)	82	80	80	80	80	78	80
RESPIRATORY RATE(cpm)	18	20	20	20	17	18	16
CVS	S1S2	S1S2	S1S2	S1S2	S1S2	S1S2	S1S2
RS	BAE+	BAE+	BAE+	BAE+	BAE+	BAE+	BAE+
P/A	Soft,Non tender	Soft,Non tender	Soft,Non tender	Soft,Non tender	Soft,Non tender	Soft,Non tender	Soft,Non tender

Vitals remained stable throughout the hospital stay

COMPLETE BLOOD PICTURE

Detailed laboratory investigations on case report

PARAMETERS	RESULTS	BIOLOGICAL REFERENCE
Hemoglobin (g/dl)	11.5	13.0-17.0 g/dl
RBC (millions/cumm)	4.3	4.0-5.5 millions/cumm
WBC (cells/cumm)	5,500	4,000-10000 cells/cumm
Platelets (lakhs/cumm)	3.8	1.5-4.1 lakhs/cumm
Neutrophils	46%	40-80%
Lymphocytes	42%	20-40%
Eosinophils	02%	01-06%
Monocytes	10%	02-10%
Basophils	00%	01-02%
ESR	25 mm1st hr	Females[10-12]mm1st hr
AEC	110	40-440 cells/cumm

VITAMIN LEVELS

PARAMETERS	RESULTS	BIOLOGICAL REFERENCE
Vitamin -D	18.2	21-116 nmol/L
Vitamin -B12	180.8	180.0-914.0 pg/ml

SERUM IgE

PARAMETERS	RESULTS	BIOLOGICAL REFERENCE
Total IgE	546.78	<150 IU/mL

BLOOD GLUCOSE

PARAMETERS	RESULTS	BIOLOGICAL REFERENCE
RBS	95	<200mg/dl

SPECIAL INVESTIGATIONS

Punch Biopsy

A 4 mm skin punch biopsy taken from the lesion over the left leg revealed epidermal hyperkeratosis, parakeratosis, acanthosis with elongation of rete ridges, and focal spongiosis. The dermis showed fibrocollagenous thickening with perivascular lymphocytic and eosinophilic infiltrate. These features were consistent with Prurigo Nodularis.

TREATMENT CHART

TRADE NAME	GENERIC NAME	DOSE	ROA	FREQUENCY	INDICATION
Tab. TECZINE	Levocetrizine Dihydrochloride	10mg	P/O	OD	To Treat various allergic conditions
CEBHYDRA LOTION	Cebhydra lotion		E/A	TID	Used for moisturization and hydration of dry skin
T BACT OINT	Mupirocin		E/A	BD	To treat and prevent bacterial skin infection
Cap.UPRISE D3	Cholecalciferol	60K IU	P/O	Weekly once	To treat and prevent vitamin D deficiency
Tab.BETNESOL FORTE	Betamethasone sodium phosphate	1mg	P/O	Weekly twice	To manage the associated inflammation, swelling, and severe itching
Tab. PAN	Pantoprazole	40mg	P/O	OD	To decrease acid reflux which is caused by other drugs
SUNCROS SOFT SUNSCREEN	Suncros soft sunscreen		E/A	BD	Used for protecting skin from harmful UV radiations
Tab. METHOTREXATE	Methotrexate	5mg	P/O	Weekly once	To control severe itch and inflammatory nodules,used for PN
Tab. FOLIC ACID	FOLID ACID	5mg	P/O	OD	Used as a supplement to reduce the side effects of methotrexate
Tab. TFCT-NIB	Tofacitinib	11mg	P/O	OD	Used often in refractory or severe Prurigo Nodularis
Tab.AZEE	Azithromycin	500mg	P/O	OD	To treat secondary bacterial skin infection caused by scratching
Inj. AVIL	Pheniramine maleate	2cc	IV	STAT	Indicated for symptomatic relief of severe itching
CREAM. TENOVATE	Clobetasol		E/A		To help shrink the nodular skin lesions

DISCHARGE MEDICATIONS

TRADE NAME	GENERIC NAME	DOSE	ROA	FREQUENCY
Tab. TECZINE	Levocetrizine Dihydrochloride	5mg	P/O	OD× 1 Week
CEBHYDRA LOTION	Cebhydra lotion		E/A	TID× 1 Week
Cap.UPRISE D3	Cholecalciferol	60K IU	P/O	Weekly once × 1 Month
Tab.BETNESOL FORTE	Betamethasone sodium phosphate	1mg	P/O	Weekly twice × 1 Week
Tab. METHOTREXATE	Methotrexate	5mg	P/O	Weekly once× 1 Week
Tab. FOLIC ACID	FOLID ACID	5mg	P/O	OD× 1 Week
Tab. PAN	Pantoprazole	40mg	P/O	OD× 1 Week
SUNCROS SOFT SUNSCREEN	Suncros soft sunscreen		E/A	BD× 1 Week

REVIEW

Patient was advised follow-up after 1 week for lesion assessment and side-effect monitoring.

DISCUSSION

Prurigo Nodularis (PN) is a chronic, inflammatory dermatosis characterized by severe pruritus and hyperkeratotic nodules resulting from a persistent itch-scratch. In the presented case, the 22-year-old female exhibited generalized itching for over one and a half years, with progressive development of multiple hyperpigmented, excoriated nodules over bilateral limbs and trunk. This clinical picture is typical of PN, where chronic itching leads to repeated scratching, causing lichenification, ulceration, crusting, and eventually nodular lesion formation.

The patient's biopsy findings revealed hyperkeratosis, parakeratosis, acanthosis, elongated rete ridges, and perivascular inflammatory infiltrates, which are consistent with classical histopathological features of PN. These features support the hypothesis of chronic inflammation with neuro-immune dysregulation, where inflammatory cells and altered nerve fibers perpetuate

the itch response.

Laboratory evaluation revealed low serum Vitamin D (18.2 nmol/L) and elevated total IgE (546.78 IU/mL). The vitamin D deficiency may have contributed to impaired skin barrier function and heightened immune reactivity, both of which are associated with chronic pruritic dermatoses. Elevated IgE suggests atopic or hypersensitivity background, which aligns with current evidence linking PN to Th2-mediated immune pathways involving cytokines such as IL-4, IL-31, and IL-13.

Management of PN is often challenging as it responds poorly to standard antihistamines alone. In this patient, treatment involved a multimodal approach, including oral antihistamines, topical corticosteroids, moisturizers, vitamin D supplementation, and methotrexate as an immunomodulator. Methotrexate was chosen due to its anti-inflammatory and T-cell suppressive effects, which help reduce pruritus and lesion progression in refractory PN. The patient showed significant symptomatic relief and reduction in lesion severity, demonstrating the benefit of incorporating immunosuppressive therapy when conventional measures are insufficient.

Additionally, patient education regarding avoidance of scratching, use of emollients, sun protection, and adherence to follow-up played an essential role in preventing further skin damage and recurrence. This case underscores that addressing nutritional deficiencies and immune dysregulation simultaneously can lead to significant improvement in refractory PN.

CONCLUSION

Prurigo nodularis remains a therapeutic challenge due to its multifactorial etiology and chronic relapsing nature. Early diagnosis, correction of contributing factors such as vitamin D deficiency, and combined use of topical, systemic, and immunomodulatory agents are key to achieving remission. Patient education to avoid scratching, maintain skin hydration, and adhere to treatment significantly improves outcomes.

This case emphasizes the importance of early recognition, nutritional correction, and combined immunomodulator therapy to achieve sustained remission in prurigo nodularis.

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