Original Article

Psoriasis Neurodermiformis, Verrucous Psoriasis, and Psoriasiform Keratosis: A Clinicopathological Evaluation

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Abstract

Background: Psoriasis neurodermiformis (PN) and verrucous psoriasis (VP) are two distinct forms of psoriasis characterized by thickened plaques, whose proper description in most dermatologic texts is still lacking. Psoriasiform keratosis (PK) is a recently described clinical entity characterized by a solitary keratotic plaque whose microscopic findings simulate psoriasis. **Aim:** To compare and evaluate the clinical and histological profile of PN, VP and PK, and systematically characterize each of them. **Settings and Design:** This was a prospective, descriptive study done on a total of 51 patients, who were diagnosed with PN, VP and PK based on certain clinical criteria. The study was done at a teaching hospital in eastern India. **Methods and Materials:** The study was carried out on a total of 51 patients presenting with thickened psoriasiform plaques, who visited our outpatient department, over a period of 9 months. They were then carefully evaluated clinically (along with their demographic profile), followed by meticulous microscopic assessment. Each biopsy specimen was then categorically evaluated to enable a precise diagnostic conclusion. **Statistical Analysis:** As all values in our study were qualitative, they were expressed as numeric values and percentages. **Results:** Out of 51 patients, 18 were diagnosed as PN, 19 with VP and 14 with PK. PN demonstrated an equal gender distribution, whereas in VP and PK a male preponderance was apparent. History of past/present psoriasis was positive in only one patient diagnosed with VP. Intensity of pruritus was marked in 88.88%, 21.05% and 14.28% of patients with PN, VP and PK respectively. Dorsa of feet was the commonest site of involvement in PN and VP. In PK, the shin was the predominating site. VP presented clinically as mammillated, verrucous and crateriform phenotypes. PN and PK however, demonstrated single clinical patterns. Microscopically, none of the specimens satisfied all the 7 epidermal criteria set forth by Ackerman. In each slide Trozak's histologic psoriasiform numeric score was >10. **Conclusion:** PN, VP and PK are certainly not as rare as previously considered. Mammillated VP closely mimics PN clinically. Crateriform VP is an extremely rare phenotypic expression encountered. Histological findings of papillomatosis, buttressing and anastomosing rete ridges and a dense dermal lymphocytic infiltrate point more in favor toward VP. Detecting solitary keratotic plaques with a psoriasiform histology should allow clinicians to consider the possibility of PK.

Keywords: Histopathology, psoriasiform keratosis, psoriasis neurodermiformis, verrucous psoriasis

Introduction

Psoriasis is a commonly encountered papulosquamous disorder in all dermatology clinics, throughout the world.

Although chronic plaque psoriasis is the classical phenotype witnessed; there are a number of other clinical presentations of the disease, which the clinician ought to be aware about.

Amongst them, patterns comprising thickened plaques have been specifically identified and studied both clinically and microscopically.

Included within this spectrum, are two variants of psoriasis; namely psoriasis neurodermiformis (PN) and verrucous psoriasis (VP) that have been elaborated in the recent past.[1,2] Of late, another condition referred to as psoriasiform

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keratosis (PK) has been elucidated, which again manifests as a thickened psoriatic plaque.[3] The etiology of PK still remains elusive, and whether it represents a unifocal presentation of psoriasis needs closer evaluation.

Our study was an attempt to analyze PN, VP and PK and determine subtle differences in their clinical and histologic morphologies; thereby allowing a precise conclusion while confronting these entities.

Methods and Materials

This was a prospective, descriptive study conducted over a period of 9 months in the department of dermatology of our institute, after obtaining permission by the institutional ethical clearance committee (Registration number: KMC/IEC/Dept.Res./013/2021-2024DVL).

Only freshly diagnosed patients or patients without any form of treatment for the past 6 months were included in the study.

We were able to get a total of 51 patients during this time frame, of which 18 demonstrated clinical findings of PN, 19 delineated phenotypic features of VP and the remaining 14 expressed characteristics suggestive of PK.

Once patients were enrolled, a written and informed consent was obtained from each of them, following which relevant history concerning the disease was recorded and a complete clinical examination done, which was furthered by a cutaneous biopsy, that was sent for histopathological examination.

Based on certain clinical criteria (that were proposed by the authors of the current study), each patient was designated to one of the three diagnostic entities as stated above; details of which have been elaborated in Table 1. In order to fit a designated entity, fulfillment of each of the aforementioned criterion (as detailed in Table 1), in the respective group was mandatory, the exception being freshly diagnosed cases, wherein the criterion taking into account the responsiveness to topical/intralesional steroids was not required. Table 2 details the number of freshly and previously diagnosed cases (following topical/intralesional steroid therapy and their responsiveness to treatment) of our study subjects.

Biopsy findings were primarily delineated as psoriasiform, based on 7 epidermal criteria originally put forth by Ackerman *et al*,[4] and include neutrophilic microabscesses in the stratum corneum, confluent parakeratosis, hypogranulosis, neutrophilic microabscesses in the spinous layer, slight spongiosis, thinning of suprapapillary plates and regular acanthosis.

This was succeeded by assigning a numeric histologic score as per the grading system designed by Trozak.[5] All slides were then categorically evaluated to determine explicit histological differences in the dermal and epidermal mileu for each of these 3 entities, to enable a precise diagnostic conclusion.

Statistical analysis

As all data in our study were categorical, they were expressed as numeric values and percentages.

Results

Out of the 51 patients studied 18 were clinically diagnosed with PN, 19 with VP and 14 with PK. The salient clinical and demographic profile of each has been outlined in Table 2.

Histologically, we assessed each slide with regard to epidermal and dermal parameters, to identify the existence of major as well as subtle differences in each of the conditions described. These findings have been annotated in Table 3.

Discussion

Our main purpose in conducting this study was an endeavour to delineate the above variants of psoriasis, as their proper description in most dermatologic texts is lacking.

PN, also referred to as lichenified psoriasis, is a variant of psoriasis characterized by accentuated skin surface markings [Figure 1a].[6] It may not be as rare as accounted. More often than not, it could get misdiagnosed as lichen simplex chronicus (LSC) owing to their close clinical approximation. However, there do exist subtle differences between both clinically; meticulous scrutiny of which could be of considerable help [Table 4].

Table 1: Clinical criteria for patient allocation to psoriasis neurodermiformis, verrucous psoriasis and psoriasiform keratosis

Psoriasis neurodermiformis	Verrucous psoriasis	Psoriasiform keratosis
Criteria	**Criteria**	**Criteria**
• Lichenified/leathery pruritic plaques present bilaterally over knees and/or elbows and/or ankle and dorsa of feet	• Verrucous or hypertrophic plaques having an extensor distribution	• Singular lesion or two lesions but unifocal (in a single area)
• Absence of classical scales of psoriasis	• Classical scales of psoriasis may or may not be identifiable	• Absence of classical scaling of psoriasis
• Negative Auspitz sign	• Auspitz sign may be positive or negative	• Auspitz sign negative
• Absence of past or present lesions of psoriasis elsewhere in the body	• History of past or present lesions of psoriasis may or may not be present	• No evidence of psoriasis elsewhere in the body
• Responsive to topical/intralesional steroids	• Responsive to topical/intralesional steroids	• Unresponsive to topical/intralesional steroids

Table 2: Clinical and demographic profile of our study population

Clinical findings	Psoriasis neurodermiformis	Verrucous psoriasis	Psoriasiform keratosis
Gender distribution	Male (9, 50%) Female (9, 50%)	Male (13, 68.42%) Female (6, 31.57%)	Male (10, 71.42%) Female (4, 28.57%)
Age (mean±SD) Range	39.33 ± 22.6 (20 – 60 years)	52.31 ± 23.02 (30 – 74 years)	36.36 ± 28.76 (18 – 60 years)
Duration of lesion (range)	1-5years	6months-5years (in 1 patient 30 years duration)	3months-5years
Number of freshly diagnosed cases	15 (78.94%)	16 (84.21%)	10 (71.42%)
Number of cases who had been treated with topical/intralesional steroids in the past	3 (16.66%) All three patients demonstrated steroid responsiveness, with recurrence of lesions following cessation of steroid therapy. In all these patients lesions recurred after a period of 2-3 months following stoppage of treatment.	3 (15.79%) All three patients demonstrated steroid responsiveness, with recurrence of lesions following cessation of steroid therapy. In all these patients recrudescence of lesions occurred within 3-4 months of stopping treatment.	4 (28.57%) None of these patients responded to topical/ intralesional steroid therapy.
History of past or present psoriasis apart from the lesion described	Negative in all	Negative (18, 94.74%) Positive (1, 5.26%)	Negative in all
Associated comorbidities	Hypertension (2, 11.1%) Obesity (1, 5.5%)	Hypertension (3, 15.78%) Diabetes mellitus (1, 5.26%)	Hypertension (1, 7.14%) Diabetes mellitus (1, 7.14%)
Other associated cutaneous diseases	Nil	Nil	Nil
Associated psychological issues	Nil	Nil	Nil
Intensity of pruritus	Marked (16, 88.88%) Moderate (2, 11.11%)	Marked (4, 21.05%) Moderate (15, 78.94%)	Marked (2, 14.28%) Moderate (7, 50%) Mild (4, 28.57%) Absent (1, 7.14%)
Site(s) of involvement	Dorsa of feet (17, 94.44%) Bilateral knees (2, 11.11%) Ankles (1, 5.55%) Medial malleoli (1, 5.55%) (some patients have involvement of more than one site and so the total number is exceeding 18)	Dorsa of feet (10, 52.63%) Bilateral shins (3, 15.78%) Bilateral shins and dorsa of feet (3, 15.78%) Bilateral knees (3, 15.78%)	Shin (6, 42.85%) Dorsa of foot (4, 28.57%) Knee (3, 21.42%) Lateral malleolus (1, 7.14%)
Auspitz sign	Negative in all	Negative (17, 89.47%) Positive (2, 10.52%)	Negative in all
Nail changes	Nil	Nil	Nil
History of cigarette smoking (no. of patients)	4 (22.22%)	1 (5.26%)	1 (7.14%)
History of alcohol consumption (no. of patients)	3 (16.66%)	1 (5.26%)	1 (7.14%)

Nevertheless, in many cases, this may prove quite challenging, owing to overlapping features that manifest.

Further, absence of Auspitz sign (though not diagnostic, yet a valuable tool) in PN could prevent prompt recognition of the condition. Histopathology therefore becomes mandatory for a precise conclusion.

Despite the presence of overlapping findings in both conditions microscopically, specific features pertaining to both disorders can enable the dermatopathologist in arriving at an end result [Table 4].

Regardless of histological features stated, it has been witnessed that even untreated clinically active psoriatic lesions lack a completely characteristic microscopic architecture.[7] Besides, variable histological findings have been observed even within a single psoriatic plaque, thereby implying that all features of psoriasis may not be viewed very often from just a single specimen.[8] Histological diagnosis therefore relies on an aggregate of attributes; some of which are seen distinctively in psoriasis and others more common to other dermatoses.

Table 3: Histological parameters utilized to evaluate our study subjects

Histological features	Psoriasis neurodermiformis	Verrucous psoriasis	Psoriasiform keratosis
Hyperkeratosis	Marked (11, 61.11%) Moderate (6, 33.33%) Minimal (1, 5.55%)	Marked (13, 68.42%) Moderate (4, 21.05%) Minimal (2, 10.52%)	Marked (3, 21.42%) Moderate (9, 64.28%) Minimal (2, 14.28%)
Parakeratosis	Present: Confluent (10, 55.55%) Focal (3, 16.66%) Absent (5, 27.77%)	Present: Confluent (11, 57.89%) Focal (3, 15.78%) Absent (5, 26.31%)	Present: Focal (5, 35.71%) Absent (9, 64.28%)
Papillomatosis	Present (2, 11.11%) Absent (16, 88.88%)	Present (15, 78.94%) Absent (4, 21.04%)	Absent in all
Hypogranulosis	Present (17, 94.44%) Absent (1, 5.55%)	Present (10, 52.63%) Absent (9, 47.36%)	Present (4, 28.57%) Absent (10, 71.42%)
Acanthosis	Present in all	Present in all	Present in all
Suprapapillary thinning	Present (14, 77.77%) Absent (4, 22.22%)	Present (13, 68.42%) Absent (6, 31.57%)	Present (11, 78.57%) Absent (3, 21.42%)
Spongiosis	Present (11, 61.11%) Absent (7, 38.88%)	Present (15, 78.94%) Absent (4, 21.05%)	Present (12, 85.71%) Absent (2, 14.28%)
Munro's microabscess	Present (13, 72.22%) Absent (5, 27.77%)	Present (14. 73.68%) Absent (5, 26.31%)	Present (2, 14.28%) Absent (12, 85.71%)
Evidence of neutrophils arranged in vertical tiers in the stratum corneum	All negative	All negative	All negative
Spongiform pustules of Kogoj	Present (8, 44.44%) Absent (10, 55.55%)	Present (9, 47.36%) Absent (10, 52.36%)	Present (2, 14.28%) Absent (12, 85.71%)
Pattern of psoriasiform elongation of rete ridges	Regular camel foot elongation of rete ridges (all 18 patients)	Elongated rete ridges with buttressing and anastomoses (all 19 patients)	Regular camel foot elongation of rete ridges (all 14 patients)
Focal excoriations	Absent in all	Absent in all	Present (2, 14.28%) Absent (12, 85.71%)
Dermal inflammatory infiltrates	Predominently lymphocytic in the papillary dermis: -Moderate to dense (5, 27.77%) -Minimal (13, 72.22%)	Predominently lymphocytic in the papillary dermis: -Dense (15, 78.94%) -Moderate (4, 21.05%)	Predominently lymphocytic in the papillary dermis: -Dense (1, 7.18%) -Moderate (7, 50%) -Sparse (6, 42.85%)
Vertical arrangement of collagen bundles in the dermis	Present (10. 55.55%) Absent (8, 44.44%)	Present (7, 36.84%) Absent (12, 63.15%)	No vertical arrangement in all
Evidence of plump fibroblasts	Present (1, 5.55%) Absent (17, 94.44%)	Present (3, 15.78%) Absent (16, 84.21%)	Absent in all
Montgomery's giant cells (clumped endothelial cells)	Absent in all	Absent in all	Absent in all

Likewise, in our study, none of the participants fulfilled all 7 epidermal features as stated above. However, each of these criteria was expounded in significant frequencies amongst our cohorts when carefully taken one by one.

Based on Trozak's histological grading system we scored each slide to obtain a numeric degree of psoriasiform changes.[5]

All our patients with PN demonstrated a numeric psoriasiform grade between 11 and 15 (out of a maximum score of 19), which we consider fitting in making a final diagnosis of psoriasis. Although the apt cut off value has not been determined by Trozak, we regard scores >10 acceptable in making a confirmatory histological diagnosis of psoriasis.

Previous publications regarding the application of Trozak's grading for histopathological evaluation of psoriasis is scant. Literature search revealed only one recent study from India done on classical psoriatic plaques wherein a mean score of 9.44 (in 50 patients) was considered sufficient to label the histological specimen as psoriasis.[9] The reduced utility of Trozak's histologic scoring by most clinicians could be attributed to the fact that in most cases the focus by dermatologists is primarily on clinical assessment scores like the PASI (Psoriasis Area and Severity Index) score, rather than microscopic scoring.

Further the role of histopathology becomes more relevant in atypical phenotypes of psoriasis where clinical diagnosis is not straightforward, thereby warranting scrupulous microscopic assessment in order to obtain a concrete conclusion. In such scenarios, Trozak's grading system could be of substantial value.

As our study dealt with three atypical presentations of psoriasis, histological grading helped us greatly in

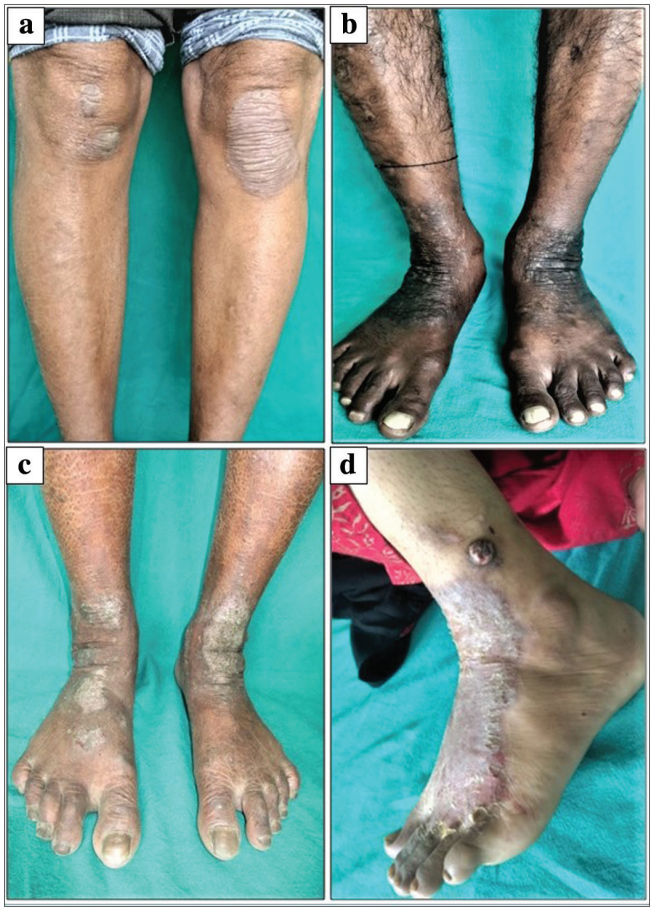

Figure 1: a: Psoriasis neurodermiformis presenting over bilateral knees. The sharper outline and a more keratotic presentation and lack of zonal distribution can be observed. b: Mammillated verrucous psoriasis (VP) over dorsa of feet. c: Classical presentation of VP over dorsa of feet with characteristic verrucous morphology. d: Crateriform VP adjacent to a psoriatic plaque above the left lateral malleoli

evaluating our cohorts. It was only after careful study did we conclude in favor of a score >10 to confer a confirmatory diagnosis of psoriasis microscopically.

Vertically oriented dermal collagen bundles were identified in 10 of our diagnosed patients with PN. Unlike LSC, these bundles were not thickened. This arrangement of collagen bundles has been linked to increased scratching owing to the release of various chemical mediators secondary to activation of the neuro-cutaneous axis involved in psoriasis. Its occurrence therefore should not be an argument to refute a diagnosis of psoriasis.

The mammillated variant of VP can closely mimic PN clinically. VP is a rare variant of psoriasis that requires detailed elucidation. To compound things further, this form of psoriasis presents in four morphological patterns.[2] These include dome shaped papules/plaques, crateriform papules/nodules, annular/oval verrucous plaques and the mammillated phenotype. If the clinician is not acquainted regarding these presentations, there could be a high likelihood of diagnostic discrepancy.

Within our cohorts, 8 (42.11%) presented with the mammillated variant [Figure 1b], 10 (52.63%) were consistent with the verrucous morphology [Figure 1c] and 1(5.26%) displayed features suggestive of crateriform VP [Figure 1d]. In the report by Khalil and colleagues,[2] mammillated VP was the most common presenting morphology and was observed in 67% of their cohorts. The remaining 33% manifested VP in the form of agminated, tan white papules.

Linear verrucous plaques were the characteristic finding in a report from Chennai,[10] and numerous verrucous plaques as the sole morphology of VP was demonstrated in the publication by Wakamatsu *et al.*[11]

Table 4: Distinguishing features (both clinical and microscopic) between psoriasis neurodermiformis and lichen simplex chronicus.

Psoriasis neurodermiformis	Lichen simplex chronicus
Clinical features	
• Plaques are more sharper in outline • Plaques are more keratotic • Plaques do not demonstrate zonal distribution	• Plaques are less sharper in outline • Plaques are less keratotic and more leathery • Plaques usually consist of three zones -a central zone with pronounced skin surface markings -a middle zone composed of multiple small papules -a peripheral zone with pigmentary changes
Histopathologic findings	
Confluent parakeratosis (generally)	Focal zones of parakeratosis
Neutrophilic microabcesses in the horny layer is present in majority of cases	Neutrophilic microabcesses in the horny layer is absent
Hypogranulosis	Hypergranulosis
Thinning of suprapapillary plates is present in majority of cases	Thinning of suprapapillary plates is absent
Elongated rete ridges of even length	Rete ridges more thicker and of less even length
Focal excoriations are absent	Focal excoriations are usually present
Vertically oriented dermal collagen bundles usually absent and if present are not thickened	Vertically oriented thickened dermal collagen bundles are present
Absence of plump fibroblasts in the papillary dermis (usually)	Presence of plump fibroblasts in the papillary dermis
Montgomery's giant cells are absent	Montgomery's giant cells are usually present

Erkek and Bozdogan[12] documented a rare expression of VP in the form of an annular outline of verrucous psoriatic plaques and Garvie *et al.*,[13] reported a phenotype of VP that closely simulated verrucous carcinoma in an 81 year old male.

Crateriform lesions was another rare presentation of VP that was reported by Nakamura *et al.*,[14] in 1994. Following this, to the best of our knowledge no such report of crateriform VP has been published.

Our patient who presented with crateriform VP also displayed lesions of chronic plaque psoriasis. Initially a diagnosis of keratoacanthoma (KA) was contemplated for this lesion, but histology eventually ruled that out.

It would be worthwhile to note that KA has been reported in patients of psoriasis.

In 1961, Vickers and Ghadially reported the first case characterized by multiple KAs emerging over psoriatic plaques treated with coal tar.[15]

A year later, Clendenning *et al.*,[16] in their paper elaborated the occurrence of KAs in a patient of generalized pustular psoriasis.

Almost two decades later, KAs originating at psoriatic treatment sites was again elucidated in two papers; one from India and the other from Canada.[17,18]

Recently, Rehlan and colleagues reported the formation of multiple KAs over healing psoriatic plaques.[19]

Plausible explanations regarding the development of KAs in psoriasis, include tar therapy, immunosuppressive effects of methotrexate, chronic inflammation, phototherapy and epithelial injury as a consequence of psoriasis *per se*.

Interestingly, in all these reports KA developed over a time period that ranged from 4 months to 43 years after the initial presentation of psoriasis, thereby representing a secondary phenomenon.

In our patient, on the other hand crateriform VP and chronic plaque psoriasis presented concurrently.

Further, as crateriform VP is extremely rare, there could be a high likelihood for clinicians to preclude its inclusion in the list of differentials for crateriform lesions. It therefore would be prudent for all dermatologists in being cognizant with this presentation of VP.

Apart from the regular histological findings of psoriasis, epidermal invagination and neutrophilic exocytosis were additional features reported by Nakamura *et al.*,[14] for crateriform VP. In our patient however, neutrophilic exocytosis was not clearly evident. Hyperkeratosis, confluent parakeratosis, scattered neutrophils in the horny layer, a cup shaped epidermal invagination, suprapapillary thinning and elongated rete ridges with buttressing and anastomoses were the predominant findings identified on microscopy [Figure 2a and b].

VP was confined to the lower limbs in all our participants with dorsa of feet (89.94%) being the most common site. In contrast, the study from Tampa, Florida, demonstrated the knees to be the most common site (50%), followed by the elbows (33%) and hands (17%).[2] In the report from Turkey, posterior truncal location of lesions were documented.[12] On the other hand, findings of Rajendran *et al.*,[10] and Wakamatsu *et al.*,[11] closely aligned with our observation of lower limb involvement.

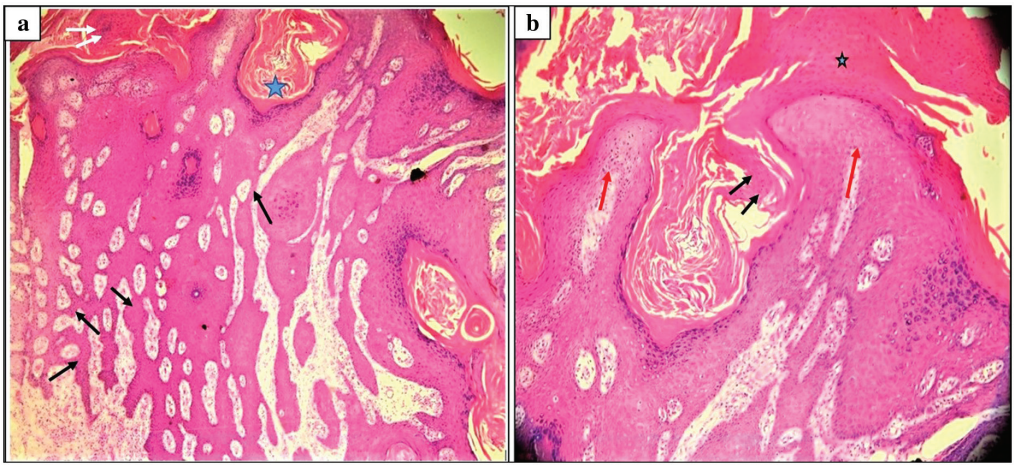

Figure 2: a: Histopathology of crateriform VP demonstrating a cup shaped epidermal invagination (blue star), confluent parakeratosis (white arrows), suprapapillary thinning and elongated rete ridges with buttressing and anastomoses (black arrows) (H&E X100). b: Higher magnification in the histology of crateriform VP illustrating hyperkeratosis, confluent parakeratosis (blue star with black outline), scattered neutrophils in the horny layer (black arrows) and suprapapillary thinning (red arrows) (H&E X200)

Acanthosis, papillomatosis, buttressing of rete ridges, Munro's microabcessess and spongiform pustules of kogoj were seen in 100%, 78.94%, 100%, 52.63% and 47.36% of our VP study subjects respectively. In the study by Khalil *et al.*,[2] similarly acanthosis and buttressing of rete ridges were observed in all their cohorts, whereas munro's microabcessess and spongiform pustules of kogoj were seen in 50% and 92% of their patients respectively.

Further, we observed that spongiform pustules and neutrophills within the stratum corneum in VP were more pronounced than those observed in PN [Figure 3a and b]. Papillomatosis was another significant observation on microscopy in VP [Figure 3c]. Also, unlike the regular camel foot elongation observed in PN, rete ridges in VP demonstrated buttressing and anastomoses as a hallmark finding.

A numeric psoriasiform grade between 11 and 16 was documented in all 19 patients diagnosed with VP.

Diabetes mellitus (DM) has been proposed as a predisposing factor for VP, secondary to the occurrence of microangiopathy and macroangiopathy.[2] Increased susceptibility to the development of VP has also been documented in patients with pulmonary dysfunction and phlebitis, in relation to tissue anoxia.[2] Further, Scavo *et al.*,[20] have reported VP in a patient with chronic hepatitis C treated with interferon. These postulations however need further validation.

Owing to rare reports of VP, a clear pathogenesis remains obscure. It has been propounded that a secondary epithelial response to repeated trauma in patients with preexisting psoriasis or psoriatic diathesis could be the pathogenic mechanism involved.[2]

For PN, constant rubbing over pruritic psoriatic plaques and the development of psoriasis as a consequence of koebnerization secondary to constant friction in patients with LSC are suggested interpretations for its pathogenesis.[1]

PK is a clinical entity that was first illustrated in 2007 by Walsh and colleagues.[3] It is characterized by solitary, unifocal, sharply demarcated keratotic plaques, that demonstrate psoriasiform changes on microscopy [Figure 4a, b, c and d]. Since its initial description, there have been a few more reports reiterating this observation.[21-23] However though, data with regard to PK still remains scant.

The exact etiology of PK remains elusive and has been suggested to be a unique form of epithelial proliferation, culminating in microscopic psoriasiform changes. Further, its inclusion to the list of cutaneous acanthomas has been recommended recently.[21]

Out of our 14 patients, 13 presented with a singular lesion, with one patient having two closely situated keratotic plaques involving the right knee. Majority of our patients were in the 3rd decade, which was in sharp contrast to the observation by Walsh *et al.*,[3] and Mutasim,[21] in which patients belonging to the 7th and 8th decade predominated.

The lower limb was the only site involved in all our participants. This differed from the findings observed by Walsh *et al.*,[3] wherein only 39% of their cohorts demonstrated involvement of the lower limb. In the remaining 61%, sites included the scalp, forehead, neck, shoulder, upper extremities and back. However, with respect to lesional location, findings of Mutasim,[21] Carbone[22] and Pires[23] were in consonance with our findings, allowing us to consider the lower extremity as the commonest site regarding

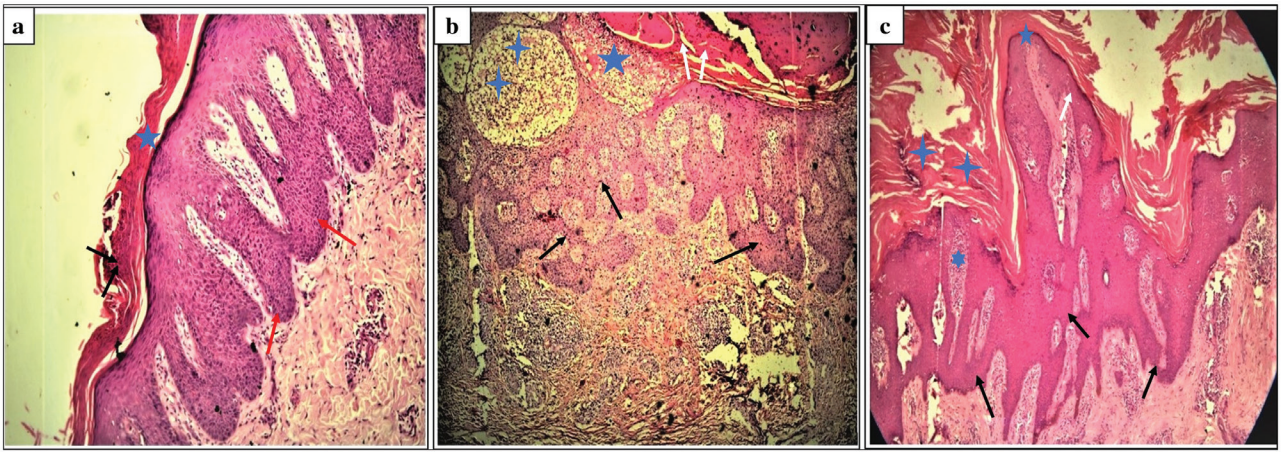

Figure 3: a: Histopathology of psoriasis neurodermiformis showing hyperkeratosis, confluent parakeratosis (five point star), munro's microabscesses (black arrows) and regular camel foot elongation of rete ridges (red arrows) [H&E X100]. b: Histopathology of VP (mammillated variant) showing a more pronounced appearance of confluent parakeratosis (white arrows), munro's microabscesses (five point star) and spongiform pustules of kogoj (four point stars). Rete ridges are showing buttressing and anastomoses with each other (black arrows) [H&E X100]. c: Histopathology of VP (classical verrucous type) showing hyperkeratosis (four point stars), papillomatosis (five point star), suprapapillary thinning (white arrow) and buttressing of rete ridges (black arrows). Dermal lymphocytic infiltrate demonstrate a more dense quality (six point star) [H&E X100]

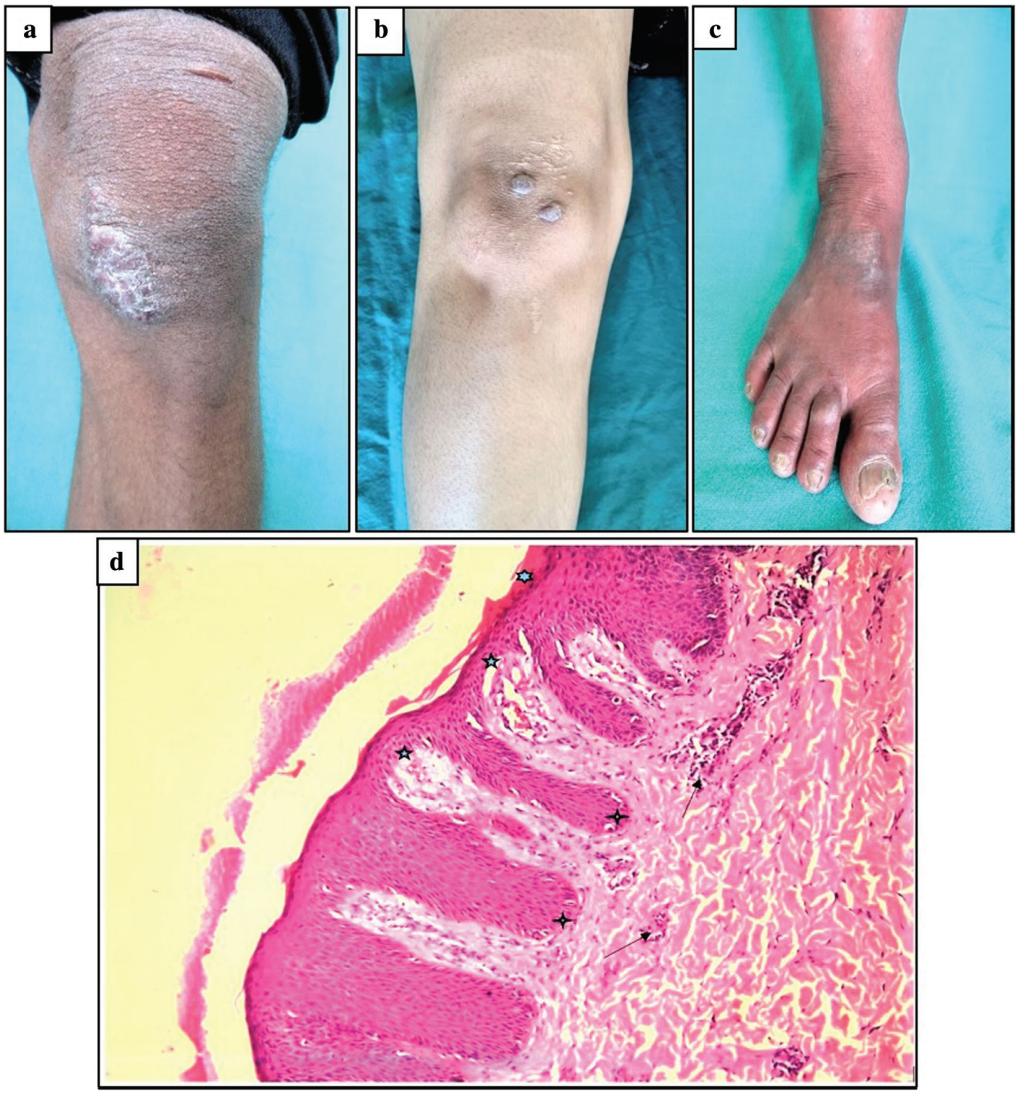

Figure 4: a: Psoriasiform keratosis (PK) over the right knee. b: Psoriasiform keratosis (PK) with two closely situated keratotic plaques involving the right knee. c: Psoriasiform keratosis (PK) with a singular scaly plaque over the dorsa of the right foot. d: Histopathology of PK depicting hyperkeratosis, focal parakeratosis (six point star), suprapapillary thinning (five point stars), camel foot elongation of rete ridges (four point stars) and a mild dermal lymphocytic infiltrate (black arrows) [H&E X100]

the occurrence of PK. Analogous to previous reports, we also considered Bowen's disease, clear cell acanthoma, VP, lichenoid keratosis and psoriasiform eczema as major differentials. In one patient however, owing to a slightly more pronounced thickened texture, possibilities of deep mycosis and tuberculosis cutis were also contemplated, which eventually were ruled out by microscopic examination along with Periodic Acid Schiff staining.

Histologically, parakeratosis was identified in 5 of our 14 patients. This differed from the findings of Mutasim[21] and Walsh *et al.*,[3] wherein parakeratosis was delineated in all their participants. Hypogranulosis was observed in only 28.57% of our patients, in contrast to 69.2% of the subjects in the study from Cincinnati.[21]

Similar to our findings, acanthosis was identified in all histological specimens by Mutasim.[21] Suprapapillary thinning was seen in 78.57% of our participants. This was in contrast to the observation by Mutasim[21] in which none of the slides demonstrated suprapapillary thinning.

The dermal infiltrate in all our patients was composed primarily of lymphocytes. In most of our patients (72.42%), the distribution of inflammatory cells was mild to moderate. This was in consonance with the findings of Walsh *et al.*,[3] and Mutasim.[21]

The numeric histological psoriasiform score ranged from 11 to 13 amongst our PK study subjects.

On comparing PK, with PN and VP, no confusion arose in contrasting it from them clinically.

Histologic overlap though, did occur. Distinguishing VP from PK histologically was not difficult. Certain distinctive findings like anastomosing and buttressing of

rete ridges, papillomatosis, denser dermal inflammatory infiltrate and a more pronounced expression of munro's microabscesses/spongiform pustules of kogoj were more specific for VP and enabled the authors to obtain a concrete histological conclusion.

However, there was considerable overlap between PN and PK microscopically; thereby making clinical correlation essential to arrive at a final/specific diagnosis in these cases.

Conclusion

Although the concept of PN, VP and PK may not be very novel, yet we felt the need to revisit these entities owing to their close similarity with a number of other dermatoses.

Further, because of paucity regarding their representation in medical literature, and their non resemblance to classical psoriatic plaques; often there could be a high likelihood of an incorrect diagnosis.

Moreover, different phenotypes of VP; and the pattern of rete ridges observed under microscopy could further pose a diagnostic challenge to the dermatologist/pathologist if they are not well versed with these findings.

It therefore becomes imperative to adopt a systematic clinicopathological evaluation to arrive at a concrete conclusion.

Besides, we also need to be aware about the fact that it is very unlikely to detect each and every finding of psoriasis in an individual biopsy specimen. Scoring each slide numerically according to Trozak's grading could therefore be of practical significance.

According to our observation, we are of the opinion that any score >10 is acceptable to finalize a histological diagnosis of psoriasis.

Lastly, based on our findings we do not consider PN, VP and PK to be rare entities. This rarity could be attributed to their lower reporting.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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