

Verrucous Psoriasis: An Integrative Review of Pathogenesis, Diagnosis, and Treatment Landscape

Zhixuan Guo, Menger Guo, Guangji Gui, Yuhua Liu, Xiangyun Li*

Department of Dermatology, The Seventh Affiliated Hospital of Sun Yat-sen University, Shenzhen, China

ABSTRACT

Psoriasis, a prevalent chronic inflammatory disorder, exhibits diverse clinical manifestations, among which Verrucous Psoriasis (VP) stands out as a rare and distinctive variant. Characterized by warty, hypertrophic lesions, VP often poses significant diagnostic and therapeutic challenges due to its overlapping features with other skin conditions. This review aims to provide an updated synthesis of the pathogenesis, clinical presentation, histopathological features, and treatment modalities for VP, highlighting the need for increased awareness and research in this area.

Keywords: Verrucous psoriasis; Pathogenesis; Clinical manifestations; Histopathological features; Treatment strategies

INTRODUCTION

Verrucous Psoriasis (VP), a rare and intriguing subset of psoriasis, is distinguished by its hyperkeratotic, warty lesions that can masquerade as other dermatological conditions, including verruca vulgaris, squamous cell carcinoma, hypertrophic lichen planus and other conditions. The histopathological features of VP, which include acanthosis, papillomatosis, and hyperkeratosis, overlap with those of common warts, complicating the diagnostic process. Furthermore, the response of VP to traditional psoriasis therapies has been inconsistent, with some cases showing resistance to topical medications, phototherapy, systemic agents, and even biologics. Due to the rarity of VP, knowledge of its pathophysiology, clinical and histopathological presentation, and optimal management strategies has been limited. Our review of the literature aims to provide a synthesis of the current understanding of this rare condition.

LITERATURE REVIEW

VP is a rare and complex variant of psoriasis which typically presents with symmetric, hypertrophic, and verrucous plaques, often localized on the extremities (Table 1). However, it can range from localized to extensive involvement, with a higher prevalence in middle-aged and elderly males. Notably, VP can manifest unilaterally, or following surgical trauma, as illustrated in a case

post-coronary artery bypass surgery [1,2]. Although a small percentage of patients are newly diagnosed, most of the verrucous psoriasis have a previous history of psoriasis vulgaris (Table S1). Besides the erythematous squamous plaques, the wart-like appearance of VP often leads to misdiagnosis with conditions like verruca vulgaris, squamous cell carcinoma, hypertrophic lichen planus and other skin diseases. In addition to the usual presentation, VP can also present with erythrodermic lesions [3-5]. The diversity of clinical manifestations makes further laboratory tests particularly important.

Histopathologic examination of VP lesions reveals a spectrum of features that, while overlapping with classic psoriasis, also exhibit unique characteristics [6,7]. The epidermis in VP demonstrates psoriasiform hyperplasia, with elongated and tortuous rete ridges, creating a papillomatous surface. This is accompanied by marked parakeratosis and hyperkeratosis, resulting in the formation of the thick scale that is a hallmark of the verrucous phenotype. Beneath the parakeratotic layer, there is often a thinning of the granular layer and the suprapapillary plates, which is a feature commonly observed in psoriasis. However, VP also displays focal spongiosis, indicative of intercellular edema, and a superficial perivascular inflammatory infiltrate that may contain a mixture of lymphocytes and neutrophils. The presence of Munro's microabscesses and spongiform pustules of Kogoj, while not invariant, adds to the histologic diagnosis when observed. The absence of koilocytic

Correspondence to: Xiangyun Li, Department of Dermatology, The Seventh Affiliated Hospital of Sun Yat-sen University, Shenzhen, China, E-mail: lixiangyun@sysuhs.com

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changes and the negative immunostaining or polymerase chain reaction for Human Papillomavirus (HPV) help to differentiate VP from verruca vulgaris and other HPV-related conditions. Furthermore, the histopathologic features of VP may show variations, reflecting the dynamic nature of the disease process and the potential influence of external factors such as trauma and inflammation.

The pathogenesis of VP remains elusive, but is hypothesized to involve factors such as repeated trauma, lymphatic obstruction, microangiopathy, diabetes, and obesity. In the dermatologic literature, a series of case reports have shed light on the intriguing phenomenon of VP, which may be associated with various comorbidities. Sanyal et al., described a case of unilateral VP emerging after coronary artery bypass surgery [2]. Baba et al., presented a case of psoriasis with verrucous appearance, suggesting a potential link between local lymphatic abnormalities and the development of such lesions [8]. Savoia et al., described a case of Elephantiasis Nostras Verrucosa (ENV) co-occurring with psoriasis, proposing a multifactorial pathogenesis involving chronic venous insufficiency, obesity, and congestive heart failure, and responding to a comprehensive treatment approach [9]. The simultaneous occurrence of VP, along with the aforementioned conditions, suggests a multifactorial etiology. These comorbidities may induce microenvironmental changes that predispose to the verrucous transformation of psoriatic lesions. Besides, the potential exacerbating role of interferon therapy in hepatitis C patients developing VP and emergence of VP post secukinumab therapy, underscores the influence of systemic treatments on cutaneous manifestations. These cases collectively suggest a complex interplay between local tissue alterations, systemic conditions, and therapeutic interventions in the pathogenesis of VP [10,11].

The discourse on the therapeutics of VP has evolved, with an

emerging body of literature underscoring the heterogeneity in treatment responses and the exploration of novel biologic agents. Traditional therapeutics, such as topical corticosteroids, methotrexate, and acitretin, have demonstrated variable efficacy, with some cases illustrating successful outcomes, as reported by Moesch et al., with topical steroids and Shivers and Montanez-Wiscovich with methotrexate and acitretin combination therapy [12,13].

Notably, Kenya Wakamatsu et al., emphasized the role of oral etretinate and compression bandaging in achieving remission, highlighting the necessity for tailored treatment strategies. In the realm of biological therapy, a paradigm shift has occurred, with an array of case reports attesting to the remarkable responses observed with agents targeting specific immune pathways. Duarte et al., showcased the therapeutic potential of adalimumab, an anti-Tumor Necrosis Factor-Alpha (TNF- α) agent, in a case of unilateral VP [14,15]. Similarly Maejima et al., reported the efficacy of adalimumab, reinforcing the significance of TNF- α inhibition in recalcitrant VP [16].

Apremilast, a phosphodiesterase 4 inhibitor, has also garnered attention, with successful treatment reports by Xenopoulou et al., Okazaki et al., suggesting its utility in refractory cases. The advent of Interleukin-17A (IL-17A) inhibition has further expanded the therapeutic arsenal, with ixekizumab demonstrating efficacy in both adult and pediatric populations, as illustrated by Sherkin et al., [17-19]. The discontinuation of ustekinumab, an IL-12/23 inhibitor, has been linked to the emergence of VP, as reported by Ger et al., [20]. However, Guo et al. delineates the emergence of VP in a patient following secukinumab therapy for psoriasis vulgaris, which was relieved by combination therapy of oral etretinate and ustekinumab following the standard regimen [11]. All of these reports underscored the intricate relationship between biologic therapy and atypical psoriasis presentations.

Table 1: Clinical, histopathologic features and treatments of VP.

Articles	Age/sex	Comorbidities	Lesion localization	Lesion manifestations	Histopathologic features	Effective treatments
Nakamura, 1994	60/M	Diabetes, hypertension, hyperlipemia, renal dysfunction, prostatic hypertrophy	Trunk, extremities, fingers	Erythematous squamous papules and plaques, verrucous papules (fingers)	Acanthosis, Munro's microabscess, spongiform pustule of Kogoj	Topical corticosteroids, 24r-dihydroxy cholecalciferol ointment, 2% coal tar ointment, puva, cryotherapy
Sato, 1996	41/M	Impaired glucose tolerance, obesity, local lymphatic disturbances	Trunk, buttock, extremities	Erythematous squamous plaques, papillomatosis (lower legs)	Hyperkeratosis, papillomatosis, proliferation of capillaries in the upper dermis, lymphectasia in the lower dermis	Topical betamethasone dipropionate or tacalcitol
Erkek, 2001	22/F	None	Scalp, back	Erythematous squamous plaques, annular verrucous plaques (back)	Hyperkeratosis, parakeratosis, hypogranulosis, acanthosis, papillomatosis, Munro's microabscesses, dermal papillae capillary ectasia, perivascular lymphohistiocytic infiltration	5% crude coal tar, topical corticosteroids of moderate potency

Scavo, 2004	44/M	Chronic hepatitis C	Trunk, extremities, glans penis, scalp, external auditory canals,	Erythematousquamous and hyperkeratosis plaques	Parakeratosis, hypogranulosis, acanthosis, papillomatosis, Munro's microabscesses, Spongiform pustule of Kogoj, inflammatory infiltrates of the dermis	IFN treatment was suspended, emollients, systemic antihistamines
Khalil, 2005	Average age of 61.8/ 7M, 5F	UK	6 on the knees, 4 on the elbows, 2 on the hands	Hyperkeratotic plaques and papules	Acanthosis papillomatosis, Munro's microabscesses, Spongiform pustule of kogoj, dermal papillae capillary ectasia, perivascular lymphohistiocytic infiltration	UK
Okuyama, 2006	60/M	Obesity, renal dysfunction	Trunk, extremities	Erythematousquamous and wart-like lesions	Hyperkeratosis, parakeratosis, acanthosis, papillomatosis, Munro's microabscesses, neutrophils and lymphocytes infiltrates of the dermis, collagen bundles appeared sclerotic	oral etretinate (30 mg/day)
Wakamatsu, 2010	39/M	Obesity	Legs	Erythematousquamous and verrucous lesions	Parakeratosis, acanthosis, papillomatosis, hypogranulosis, munro's microabscesses, neutrophils and lymphocytes infiltrates of the dermis	Topical vitamine D3, oral etretinate (30 mg/day), compression bandaging on the legs
Monroe, 2011	84/F	Nonmelanoma skin cancer, atrial fibrillation, hypothyroidism	Trunk, extremities	Erythematousquamous plaques, hyperkeratotic verrucous papules	Hyperkeratosis, parakeratosis, hypogranulosis, Munro's microabscesses, Spongiform pustule of Kogoj, perivascular lymphohistiocytic infiltration	None
Curtis, 2012	46/M	Colon cancer	Trunk, extremities, face, scalp, genital area	Erythematous and verrucous plaques	Hyperkeratosis, epidermal hyperplasia, papillomatosis, Munro's microabscesses, dermal papillae capillary ectasia	Ustekinumab
Maejima, 2012	55/M	Obesity, atypical psychosis	Trunk, extremities	Erythematousquamous plaques, verrucous plaques	Hyperkeratosis, parakeratosis, acanthosis, papillomatosis, hypogranulosis, Munro's microabscesses, lymphocytes infiltrates of the dermis	Adalimumab (40 mg every other week)
Savoia, 2013	60/M	Obesity, heart failure, venous insufficiency, lymphedema, hypothyroidism	Trunk, extremities	Erythematousquamous plaques, verrucous plaques (trunk and the upper limbs)	UK	Oral acitretin(50 mg/d), compressive bandages
Lawrence, 2013	44/M	UK	Trunk, extremities, scalp	Hyperkeratotic papules and plaques	Acanthosis, papillomatosis, hypogranulosis, Munro's microabscesses, perivascular and interstitial infiltrate with eosinophils and perivascular plasma cells	Topical and intralesional corticosteroids, methotrexate

Inani Kawtar, 2014	43/F	Autoimmune hepatitis	Trunk, extremities, face	Erythematousquamous plaques, verrucous plaques (extremities) & pigmented plaques	Parakeratosis, epidermal hyperplasia, papillomatosis, lymphocytic infiltrate band	None
Baba, 2016	58/M	Renal dysfunction, heart failure	Lower legs	Erythematousquamous plaques, verrucous plaques	Hyperkeratosis, acanthosis, papillomatosis, Spongiform pustule of Kogoj,	Topical vitamin D3, topical corticosteroid, compression bandaging
John Moesch, 2016	14/F	None	Scalp	Erythematousquamous plaques, verrucous plaques	Hyperkeratosis, parakeratosis, epidermal hyperplasia, dermal papillae capillary ectasia, perivascular mixed inflammatory-cell infiltrate	Topical corticosteroids, 3% salicylic acid shampoo
Sarra Ben Rejeb, 2017	63/F	None	Legs	Erythematousquamous plaques, verrucous plaques	Hyperkeratosis, acanthosis, perivascular lymphohistiocytic infiltration	UK
Iinuma 2017	72/M	Diabetes, venous insufficiency	Lower legs	Erythematousquamous and papillomatous plaques	Hyperkeratosis, parakeratosis, hypogranulosis, papillomatosis, Munro's microabscesses, Spongiform pustule of Kogoj, dermal papillae capillary ectasia, perivascular lymphohistiocytic infiltration	Oral acitretin (20 mg/day), topical vitamin D3, topical corticosteroids, compression bandaging
Artem Sergeyenko, 2017	53/F	Hypertension, asthma, arthritis	Extremities	Hyperpigmented verrucous papules and plaques	Psoriasiform hyperplasia, parakeratosis, hypogranulosis, Munro's microabscesses, dermal papillae capillary ectasia, perivascular lymphocytic infiltrate	UK
Tzong-Yun Ger, 2018	43/F	UK	Trunk, extremities	Erythematousquamous plaques, warty-like plaques (extremities)	Hyperkeratosis, parakeratosis, acanthosis, papillomatosis, hypogranulosis, Munro's microabscesses, lymphocytes and neutrophils infiltrates of the dermis	Ustekinumab
Okazaki, 2019	67/M	Obesity, venous insufficiency	Trunk, lower legs	Erythematous papules, papilloma-like surfaced, verrucous nodules	Hyperkeratosis, parakeratosis, acanthosis, hypogranulosis, Munro's microabscesses, lymphocytes and neutrophils infiltrates of the dermis	Apremilast (60 mg/day)
Garvie, 2019	81/M	Nonmelanoma skin cancer	Lower legs, forearms, and feet	Verrucous plaques	Hyperkeratosis, hypogranulosis, carcinomatous epithelial hyperplasia, glassy keratinocytes, mixed inflammatory-cell infiltrate of the dermis	None

Shivers, 2019	76/F	Venous insufficiency	Legs, back	Hyperkeratotic papules and plaques	Epidermal hyperplasia, parakeratosis, papillomatosis, hypogranulosis, Munro's microabscesses	Oral acitretin (25 mg/d), methotrexate (10-20 mg/week), topical corticosteroids
Atul Dongre, 2019	80/M	UK	Legs	Erythematous squamous plaques, verrucous nodules	Epidermal hyperplasia, parakeratosis, papillomatosis, hypogranulosis, Munro's microabscesses, dermal papillae capillary ectasia	Methotrexate (7.5 mg/week), topical corticosteroids and emollients
Sunantawanich, 2020	35/M	UK	Trunk, extremities	Erythematous squamous plaques, verrucous plaques (legs)	Epidermal hyperplasia, hyperkeratosis, parakeratosis, papillomatosis, hypogranulosis, Munro's microabscesses, Spongiform pustule of Kogoj, mixed inflammatory-cell infiltrate of the dermis	Ixekizumab 80 mg every two weeks
Sanyal, 2021	80/M	Hypertension, coronary artery disease (coronary artery bypass surgery with a saphenous vein graft)	Legs, trunk, left leg	Erythematous squamous plaques, verrucous nodules	Hyperkeratosis, acanthosis, Munro's microabscesses, dermal papillae capillary ectasia, neutrophils infiltrates of the dermis	None
Hernández, 2021	63/M	Epilepsia	Trunk, extremities	Erythematous plaques, verrucous plaques (legs)	Hyperkeratosis, parakeratosis, papillomatosis, Munro's microabscesses, dermal papillae capillary ectasia, perivascular lymphohistiocytic infiltration	UK
Sherkin, 2022	14/M	obesity, alopecia	Trunk, extremities, scalp	Verrucous plaques	Hyperkeratosis, parakeratosis, acanthosis, papillomatosis,	Ixekizuma, topical corticosteroids
Xenopoulou, 2023	64/F	UK	Extremities	Hyperkeratotic plaques	Hyperkeratosis, parakeratosis, epidermal hyperplasia, Munro's microabscesses, dermal papillae capillary ectasia, perivascular mixed inflammatory cell infiltrate	Apremilast
Sugioka, 2024	61/M	Stable Hodgkin lymphoma, renal dysfunction	Right legs	Erythematous lesions, verrucous lesions	Hyperkeratosis, parakeratosis, hypogranulosis, papillomatosis, Munro's microabscesses, edema of dermis, and dilated lymphatic vessels	Risankizumab
Duarte, 2024	31/F	Obesity	Left trunk	Erythematous squamous plaques, verrucous crusts	Hyperkeratosis, acanthosis, Munro's microabscesses, Spongiform pustule of kogoj, dermal papillae capillary ectasia	Adalimumab (40 mg every two weeks)
Zhixuan Guo, 2024	61/M	None	Limbs and trunk	Erythematous squamous patches, verrucous plaques	Hypogranulosis, papillomatosis, dermal papillae capillary ectasia, perivascular lymphohistiocytic infiltrate	Oral etretinate (30 mg/day), ustekinumab

Note: M: Male; F: Female; UK: Unknown.

DISCUSSION

VP, a rare and clinically distinct variant of psoriasis, presents with pronounced epidermal hyperplasia and a complex interplay of cellular, molecular, and signaling pathways. The pathophysiological underpinnings of VP are posited to involve an intricate interplay of cytokines, growth factors, and signaling cascades that collectively dictate the epidermal proliferative response. The dysregulation of this network, potentially instigated by genetic predispositions, immunological perturbations, or environmental triggers, culminates in the exaggerated hyperkeratotic phenotype characteristic of verrucous lesions.

The convergence of these case reports on the theme of VP in the context of various comorbidities enriches our understanding of the disease's pathophysiology. It also emphasizes the necessity for clinicians to maintain a high index of suspicion for atypical presentations of psoriasis, particularly in patients with underlying systemic disorders or undergoing specific treatments, to ensure timely and effective intervention.

The heterogeneity in therapeutic response observed in VP may be attributed to the disruption of a delicate balance within a sophisticated network of regulatory factors that govern keratinocyte proliferation and differentiation. Given the multifactorial nature of epidermal hyperplasia regulation, it is plausible that a one-size-fits-all therapeutic approach may not be efficacious across the patient spectrum.

The variability in treatment outcomes suggests that the perturbed inflammatory milieu in VP may not uniformly respond to a single modality of intervention. However, the condition's embedment within the broader inflammatory framework implies that alternative anti-inflammatory therapies, customised to the specific derangements present, may yield superior outcomes. The therapeutic heterogeneity observed in VP highlights the need for a personalized medicine approach, wherein treatment strategies are informed by a comprehensive understanding of the molecular aberrations underpinning each case. Future research endeavors should focus on delineating the specific cytokine and signaling pathway imbalances in VP to identify novel therapeutic targets and biomarkers that predict treatment response. Moreover, the exploration of combinatorial therapeutic regimens that address the multifactorial aspects of VP may offer a more efficacious approach to management. The potential synergistic effects of combining targeted biological agents with traditional systemic therapies warrant investigation to enhance the therapeutic armamentarium against this recalcitrant dermatosis.

The treatment of VP is confounded by its complex pathophysiology and the heterogeneity of patient responses. The therapeutic landscape for VP is diversifying, with a convergence of evidence supporting the role of both traditional and biological agents. A deeper understanding of the cellular and molecular mechanisms driving this condition is imperative to develop more effective, personalized treatment strategies. The prospect of harnessing the complexity of the inflammatory network in VP to inform precision therapeutics holds promise for improving clinical outcomes in this challenging subset of patients.

The heterogeneity in treatment responses underscores the need for individualized therapeutic approaches and continued exploration of novel agents. The emergence of VP following biologic therapy or discontinuation highlights the complexity of disease management

and the potential for immunologic rebound phenomena. As our understanding of VP's pathophysiology deepens, so too will the precision of our therapeutic interventions, ultimately enhancing the dermatologic care for patients afflicted with this rare and challenging variant of psoriasis.

CONCLUSION

VP represents a rare and therapeutically challenging variant of psoriasis. Its diagnosis relies on a combination of clinical suspicion, histopathological examination, and exclusion of other conditions with similar presentation. While traditional treatments offer limited benefits, the use of systemic therapies and biological agents has shown potential. Further research is warranted to better understand the pathogenesis of VP and to develop targeted treatment strategies.

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