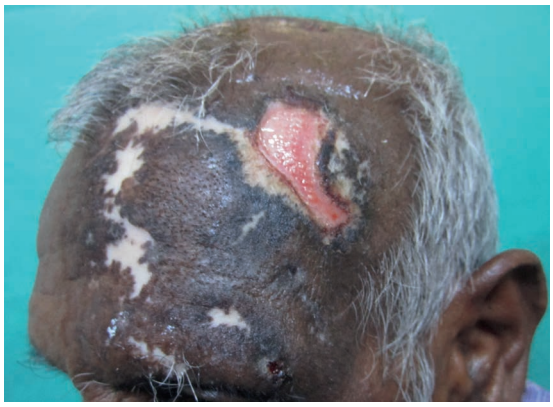

Figure 1: Crescent-shaped ulcer involving the left frontal scalp and a small ulcer above the lateral aspect of the eyebrow

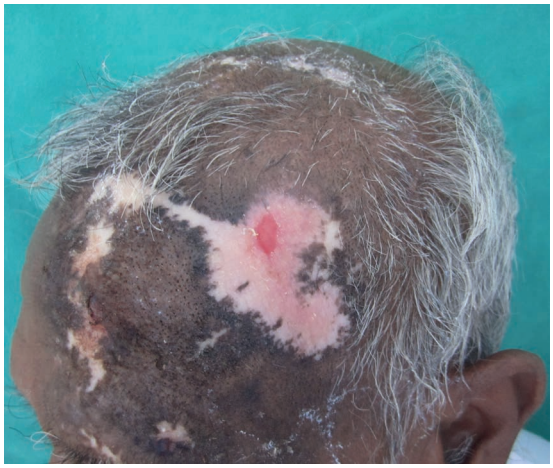

Figure 2: Healed ulcer after three weeks of treatment

Initially, TTS lesions were believed to be a sequel of impaired nerve fibers resulting in the loss of neuronal trophic factors.[6] Later, researchers realized that the condition is caused by self manipulation of the desensate itchy skin in a reflexive action to get rid of the troublesome dysethesias.[3,6] Although TTS characteristically affects the ipsilateral nasal ala, cheeks, and upper lip, involving the V2 or V3 dermatomes, it can appear anywhere in the trigeminal innervation territory.[6] TTS following herpes zoster involving the scalp and forehead is a less common presentation.

Differential diagnosis of TTS includes various diseases manifesting as facial ulcers such as squamous cell carcinoma, basal cell carcinoma, infections, vasculitis, pyoderma gangrenosum, and factitial dermatitis.[1,2,4,5]

Treatment should be centered on behavioral modification intended to reduce self-induced trauma.[7,8] Occlusive dressings can also prevent handling and perpetuation of the skin lesions by the patients. Pharmacotherapy with carbamazepine, amitriptyline, diazepam, chlorpromazine, and pimozide has been used with varying results.[8] Other reported modalities of management include hydrocolloid dressings, transcutaneous electrical nerve stimulation, plastic surgery with innervated flaps, and negative pressure wound therapy.[2,4,5] The present case was successfully managed with counseling, occlusive dressings, and carbamazepine. ❑

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Cutaneous vasculitis: a presentation with endocarditis to keep in mind*

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Dear editor,

Vasculitis comprises a broad and diverse group of diseases defined as an increased number of inflammatory cells in and/or around the vessel wall accompanied by vascular damage. It may be associated with different entities such as infections, malignant disorders, or connective tissue diseases.[1]

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A 47-year-old male patient presented to our department with a palpable purpura on the lower extremities for three weeks. He also reported fever, asthenia, and weight loss in the past eight months. Complementary tests (complete blood count and serum biochemistry) revealed a normocytic anemia, thrombocytopenia, and neutrophilia with left shift. We also detected proteinuria and hematuria. We performed a biopsy of the skin lesions with a histopathological diagnosis of leukocytoclastic small vessel vasculitis. Results of direct immunofluorescence performed on skin showed deposits of IgA with codominant deposition of IgM and C3 at the superficial perivascular level. Blood cultures were positive for *Streptococcus gallolyticus* and an echocardiogram showed the presence of numerous vegetations affecting the aortic, mitral, and tricuspid valves. Therefore, we diagnosed subacute endocarditis. The patient was initially treated with antibiotics, but we detected progression of cardiac involvement after a month of treatment. The patient had a pulmonary embolism and two cerebral embolic strokes. Hence, eventually, we performed heart valve replacement surgery with resolution of the signs and symptoms.

Infective endocarditis (IE) is a disease with high morbidity and mortality, often presented as a multisystem disease. Its heterogeneous features present a diagnostic challenge. According to the literature, the frequency of skin lesions in patients with definite IE who had dermatological manifestations varies widely (5%-25% of IE cases) across investigations.[2] None is pathognomonic for endocarditis.

The classic manifestations of IE include Osler's nodes, Janeway lesions, and petechiae.[3] Osler's nodes are areas of painful nodular erythema on the skin of the palms or soles, typically on the distal phalanges. Janeway lesions are painless, irregular, nonblanchable erythematous macules located on the palms and soles. Histologically, both lesions show septic emboli with inflammatory reactions. Pathologically, microorganisms can grow on skin culture of the lesions. Petechial lesions, which are the most frequent mucocutaneous manifestations, include a large number of clinical manifestations, from isolated conjunctival petechial palatal or subungueal lesions (splinter hemorrhages) to thrombotic thrombocytopenic purpura-like or typical leukocytoclastic vasculitis. Histological findings of these lesions are primarily septic emboli or leukocytoclastic vasculitis. Most cutaneous vasculitis associated with endocarditis are described as leukocytoclastic vasculitis secondary to an infectious process in which either no direct immunofluorescence studies are performed or deposits are nonspecific. Recently, several cases of leukocytoclastic vasculitis with IgA deposits have been described, associated with renal involvement in varying degrees (from proteinuria and hematuria to renal failure). Renal biopsy of these cases, when performed, revealed IgA glomerulonephritis, which, like other manifestations, are resolved with endocarditis treatment.[4] Our patient would correspond to the latter subgroup. Compared with patients with IE without skin lesions, those with dermatological manifestations have more extracardiac complications and are associated with a higher rate of cerebral complications, mainly cerebral emboli.[5]

There is a clear overlap between the classical manifestations described in endocarditis. Therefore, nowadays, it is difficult to differentiate between Osler´s nodes or Janeway lesions and some of the petechial lesions since the underlying mechanism is the same. The original names of these designations are subject to a historical context that has changed with the knowledge of its pathogenesis.[3]

It is important to know and identify these skin manifestations associated with infective endocarditis, given the importance of reaching an early diagnosis and the prognostic implications associated with their appearance. ❑

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