in adults.[6] The metabolism of both drugs is still unknown.[5]

Biologic drugs can trigger immediate transfusion reactions and rarely induce the formation of cellular autoantibodies or even autoimmune conditions, such as lupus erythematosus, usually of late onset. The frequency of tocilizumab's infusion reactions is around 7%; in a recent study with 226 infusions in individuals with autoimmune conditions, no immediate infusion reactions were observed. Nonetheless, it is still a medication with limited use.[7]

There are no reports of biologic drug-induced pemphigus until now. We highlight the importance of the dermatologist in the pharmacovigilance phase for new drugs. ❑

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Trigeminal trophic syndrome*

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Dear Editor,

An 80-year-old male presented with chronic non-healing ulcers involving the left side of the scalp and forehead for three months. The lesions were associated with intractable itching and vague crawling sensation. Following that, he started picking and rubbing his skin, which resulted in ulcers. His medical history was suggestive of herpes zoster involving the left ophthalmic (V1) branch of the trigeminal nerve for nine months. Physical examination revealed a 4x3-cm ulcer involving the left frontal aspect of the scalp and two other small ulcers, one involving the center of the scalp and the other above the lateral aspect of the left eyebrow, corresponding to the ophthalmic (V1) branch of the trigeminal nerve. The latter lesion showed areas of post inflammatory depigmentation (Figure 1). Routine hematological and biochemical investigations were within normal limits. Head MRI revealed age-related cortical atrophy and was otherwise normal. Systemic examination revealed no abnormality. A diagnosis of trigeminal trophic syndrome (TTS) was entertained. The patient was treated with occlusive dressings, topical antibiotics, and carbamazepine with complete resolution of the lesions within three weeks (Figure 2).

TTS is a rare clinical entity characterized by unilateral facial ulceration involving the trigeminal nerve (TN) territory following damage to its central or peripheral nerve structure. The classical clinical triad of TTS consists of trigeminal anaesthesia, facial paraesthesia, and crescent shaped ulcers.[1] The presenting features will be that of picking, rubbing, or scratching sensations on the affected areas secondary to hypoesthesia, paraesthesia, or pain resulting from damage of the sensory trigeminal fibers.[2] Adolf Wallenberg was the first to describe TTS in 1895 in a patient with lateral medullary infarction.[3]

TTS is frequently triggered by iatrogenic causes, usually following procedures for pain management in trigeminal neuralgia.[4] Other causes include stroke, acoustic neuroma, post-infectious encephalitis, trauma, amyloid deposits in the TN, and infections.[1,4,5] Herpes zoster and leprosy are also major dermatological causes for TTS.[5]

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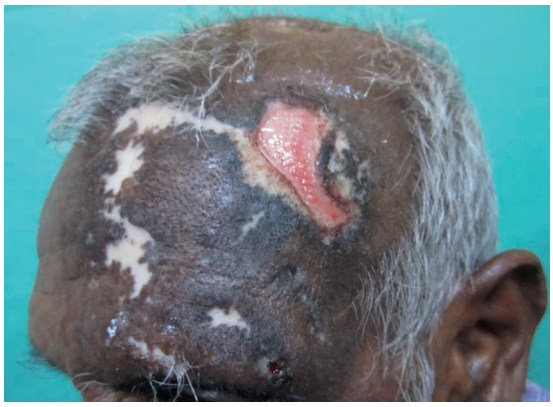

Figure 1: Crescent-shaped ulcer involving the left frontal scalp and a small ulcer above the lateral aspect of the eyebrow

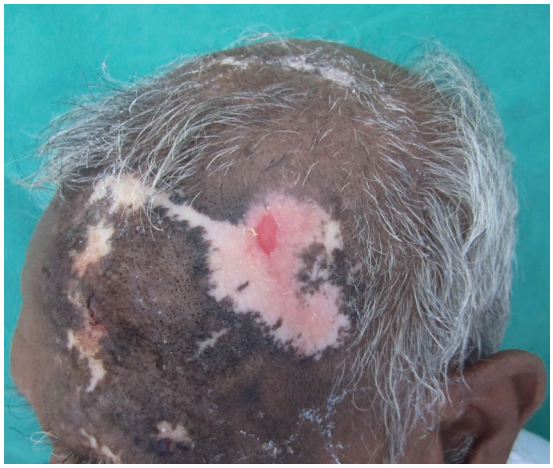

Figure 2: Healed ulcer after three weeks of treatment

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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