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POSTER PRESENTATION

A Rare Manifestation of Giant Cell Arteritis: Bilateral Scalp Necrosis

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Giant cell arteritis (GCA) is a granulomatous vasculitis of medium and large vessels seen in the elderly. Superficial temporal, vertebral, ophthalmic, and posterior ciliary arteries are commonly involved. It may present with jaw claudication, headache, constitutional symptoms, or scalp tenderness. Rarely, ischemic complications due to intense inflammation and thrombosis may occur [1]. Here, we present a case of GCA presenting with severe scalp necrosis.

CASE REPORT

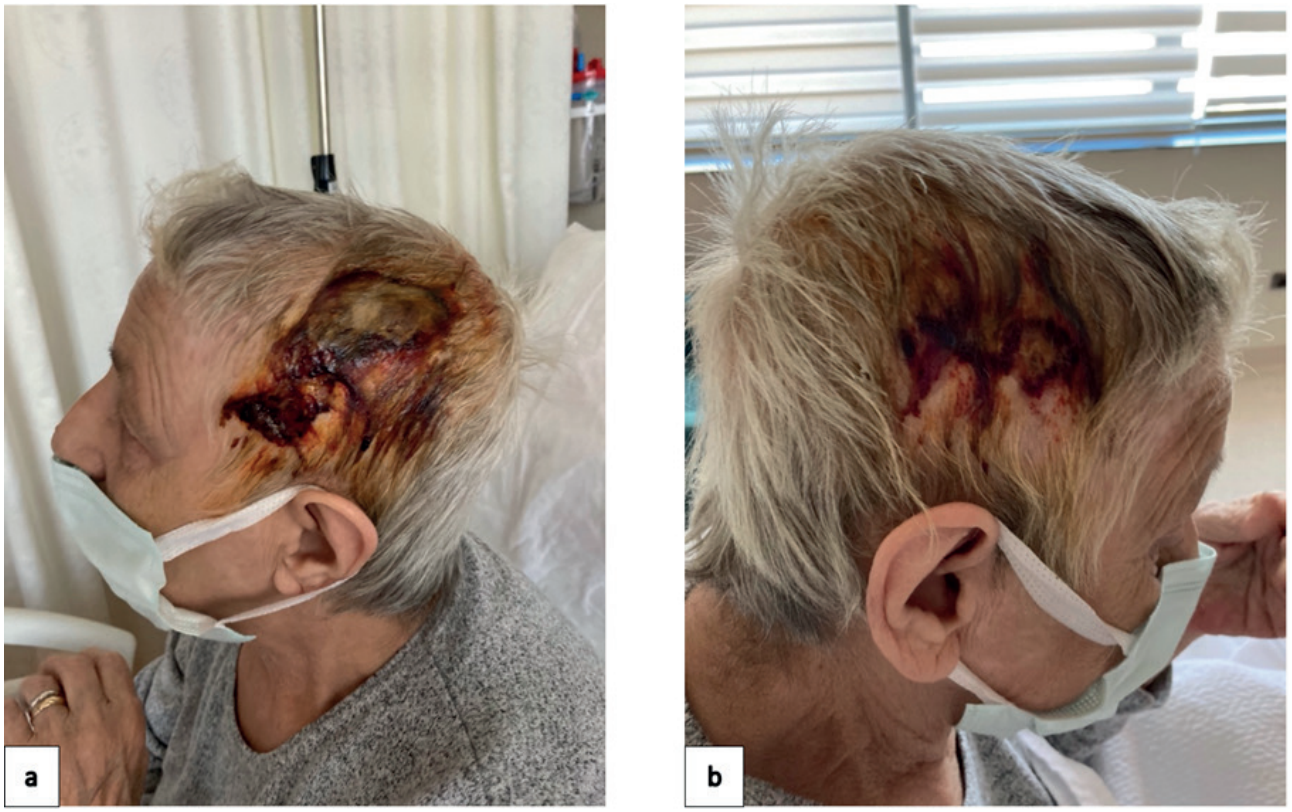
A 73-year-old female patient presented to the Dermatology department with gradually increasing painful lesions on the scalp for about three months and was referred to the Rheumatology department. She also complained of headaches that started simultaneously with skin lesions. The patient did not have symptoms such as vision loss, jaw claudication, and polymyalgia rheumatica. Physical examination revealed diffuse necrotic ulcerations extending to the parietal and temporal regions of the scalp (Picture 1 (a-b)). Temporal arteries were tender bilaterally on palpation, and the left superficial temporal artery pulse was absent. Laboratory tests showed an erythrocyte sedimentation rate of 85 mm/hr and a C-reactive protein level of 44 mg/L. A preliminary diagnosis of GCA was considered, and the patient was transferred to our clinic. Her ophthalmologic examination was unremarkable. Doppler ultrasonography examination of temporal arteries showed hypoechoic thickening, i.e., bilateral halo sign, and mild to moderate luminal narrowing on the left. A biopsy was not considered appropriate due to possible expansion of the ischemic area. Based on the clinical history, physical findings, and pathognomonic sonographic findings, the patient was diagnosed with GCA. Prednisolone treatment at a dose of 1 mg/kg was initiated. Azathioprine 2.5 mg/kg was added to the treatment one week later, and the glucocorticoid dose was tapered over follow-up. A rapid shrinkage was observed in the necrotic lesions within the following days. (Pictures 2 a/b and 3 a/b).

DISCUSSION

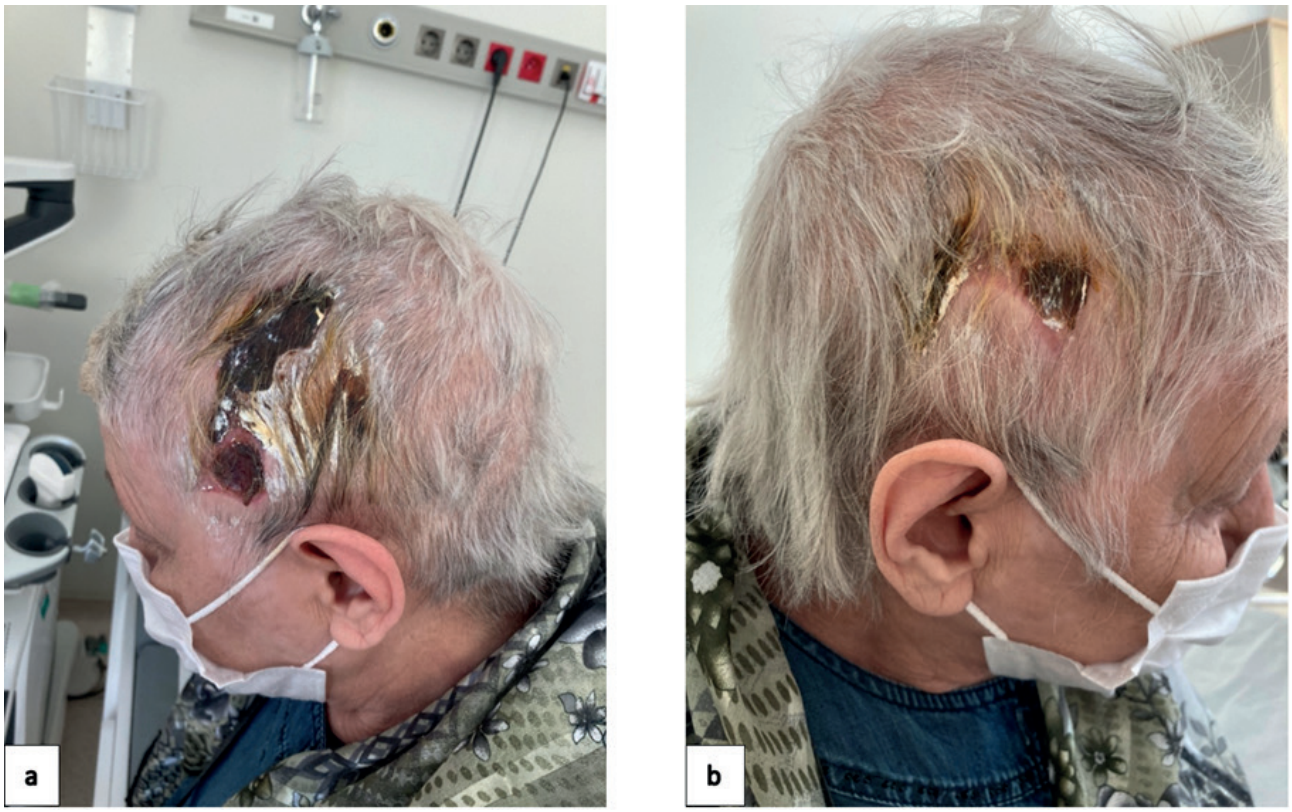
GCA-related scalp necrosis was first described in 1946, and more than 100 cases have been reported since then [1-3]. It is a well-known but rare complication of GCA. Early recognition and early initiation of treatment are significant. The onset is usually insidious, with progression in weeks to months, or acute, within days in up to 20% of cases [4]. Delays in treatment may cause inflammation to spread to all temporal artery branches and more profound tissue loss in the scalp. Lack of early diagnosis and effective treatment may result in skin necrosis, irreversible vision loss, and severe tongue necrosis.

KEY MESSAGES

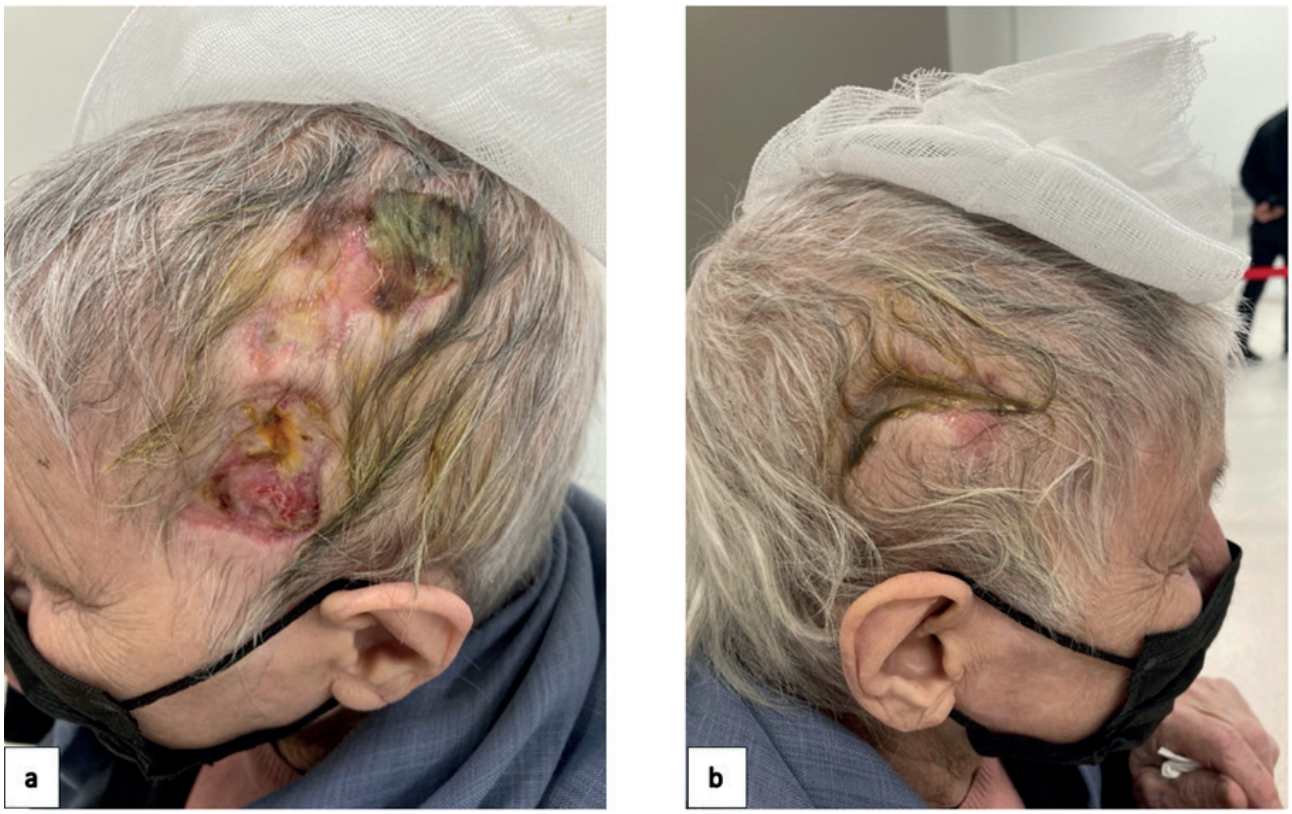
GCA can cause severe tissue and vision loss; early disease recognition is essential. Scalp necrosis is a rare but severe complication of GCA.



Picture 1 a-b. Necrotic ulcerated areas in both temporoparietal regions



Picture 2 a-b. Appearance of the lesions in the 4th week of treatment



Picture 3 a-b. Appearance of the lesions in the 8th week of treatment

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