Research Letter

When IL-17 Blockade Backfires: Retropharyngeal Granuloma and Petrous Bone Osteonecrosis in a Patient with Psoriasis

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Introduction

Biologic therapies targeting interleukin-17A (IL-17A) have revolutionized psoriasis management by modulating Th17-driven inflammation [1]. Although adverse events are rare and generally manageable, some paradoxical reactions require prompt recognition. While granulomatous reactions—classically sarcoidosis-like lesions—are well described under TNF-α inhibitors, emerging evidence indicates that IL-17 blockade may provoke similar responses. We report a uniquely severe paradoxical reaction: a retropharyngeal granuloma with osteonecrosis of the clivus and petrous apex during long-term ixekizumab therapy.

Case Presentation

A 61-year-old Caucasian woman with a 20-year history of plaque psoriasis, previously unresponsive to methotrexate, had been on ixekizumab 80 mg monthly for six years, achieving complete clearance (PASI 100). Her history included osteoporosis and dyslipidaemia on cholecalciferol and rosuvastatin. She presented at dermatologic follow-up with new onset left otalgia and conductive hearing loss. No prior ear disease, infection, or trauma was reported. ENT evaluation suggested acute otitis media, and tympanostomy provided only transient relief. Within a month she developed progressive dysphagia and odynophagia. MRI revealed a 32-mm retropharyngeal T2-hyperintense mass eroding the clivus; CT confirmed lytic changes of the clivus and left petrous apex (Figure 1A). PET-CT showed intense peripheral FDG uptake (SUV max 12) with central photopenia. Suspecting fungal infection, isavuconazole was initiated, but after 3 months symptoms persisted. Autologous leukocyte scintigraphy confirmed ongoing inflammation (Figure 1B). CT-guided biopsy showed non-caseating granulomas with multinucleated giant cells and coagulative necrosis. Special stains, cultures, and PCR for *Mycobacterium tuberculosis* were negative; immunohistochemistry excluded malignancy. A paradoxical drug reaction was considered. Ixekizumab was discontinued, and oral prednisone 25 mg daily initiated. Three months later, dysphagia and otalgia had resolved with partial hearing recovery. Follow-up CT showed near-complete regression of the mass and reparative bone changes (Figure 2). At five months, no recurrence was observed. Psoriasis was subsequently controlled with risankizumab.

Discussion

Paradoxical granulomas under IL-17 inhibition may reflect immune imbalance. IL-17A is critical in neutrophil recruitment and granuloma containment; blocking this pathway may enhance Th1-driven granulomatous inflammation. While most reports involve TNF-α blockade, isolated cases link IL-17 inhibitors to sarcoid-like disease [2,3].

Our case is exceptional for concomitant osteonecrosis. IL-17 contributes to bone remodeling through osteoclastogenesis; its inhibition could impair turnover, predisposing inflamed sites to necrosis. Indeed, only one prior case of anti-IL-17-related jaw osteonecrosis has been documented [4]. In our patient, persistent local inflammation likely amplified osteolysis at the skull base. The Naranjo Adverse Drug Reaction Probability Scale scored 7, indicating a "probable" relationship between ixekizumab and the adverse event: temporal association,

resolution after discontinuation, exclusion of alternative causes, objective confirmation, and literature precedent [5].

Conclusion

This case underscores the importance of considering paradoxical granulomatous reactions with osteolytic changes in patients on IL-17 inhibitors who develop atypical ENT symptoms. Comprehensive imaging and histopathology are essential for differential diagnosis. Prompt withdrawal of the suspected biologic and corticosteroid therapy may ensure recovery. Clinicians should remain alert to this rare but serious complication and contribute to pharmacovigilance reporting to refine the safety profile of IL-17 blockade.

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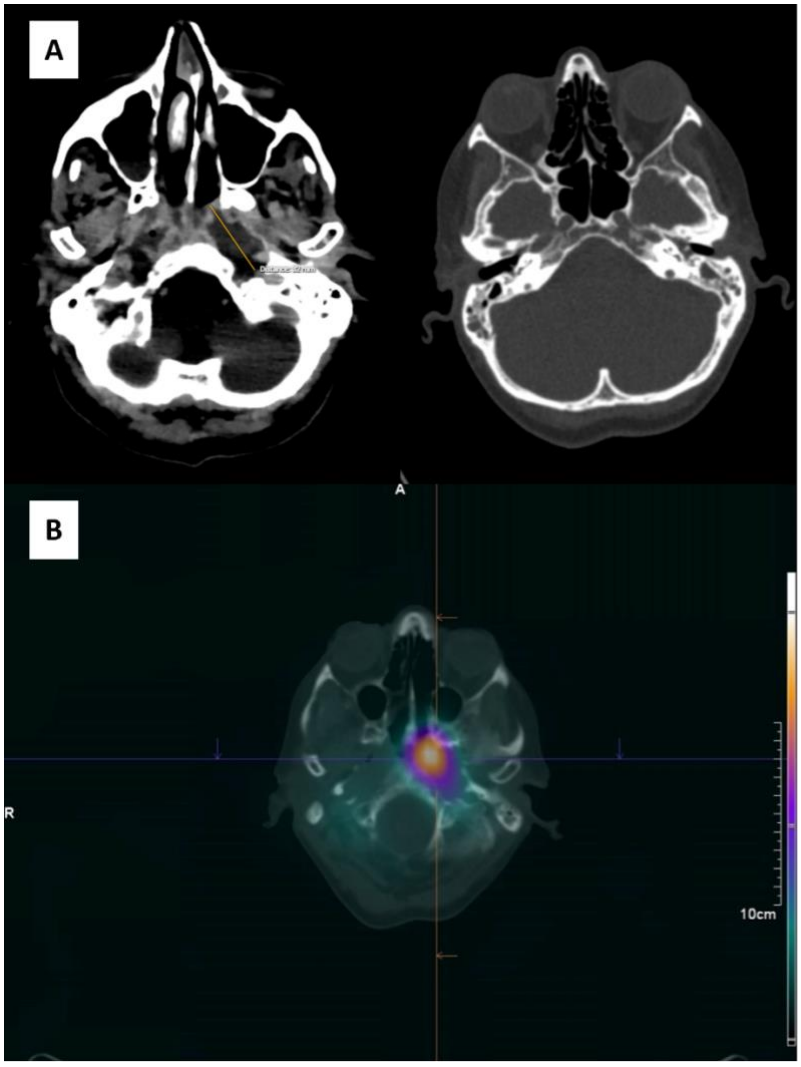

Figure 1. (A) Contrast-enhanced CT scan of the head showing a left-sided retropharyngeal mass with a peripheral hyperdense rim and central hypodensity. Bone window reconstruction demonstrates extensive osteolysis involving the sphenoidal clivus and the apex of the petrous portion of the left temporal bone. (B) Autologous leukocyte scintigraphy demonstrating intense radiotracer uptake in the retropharyngeal region, consistent with focal leukocyte accumulation.

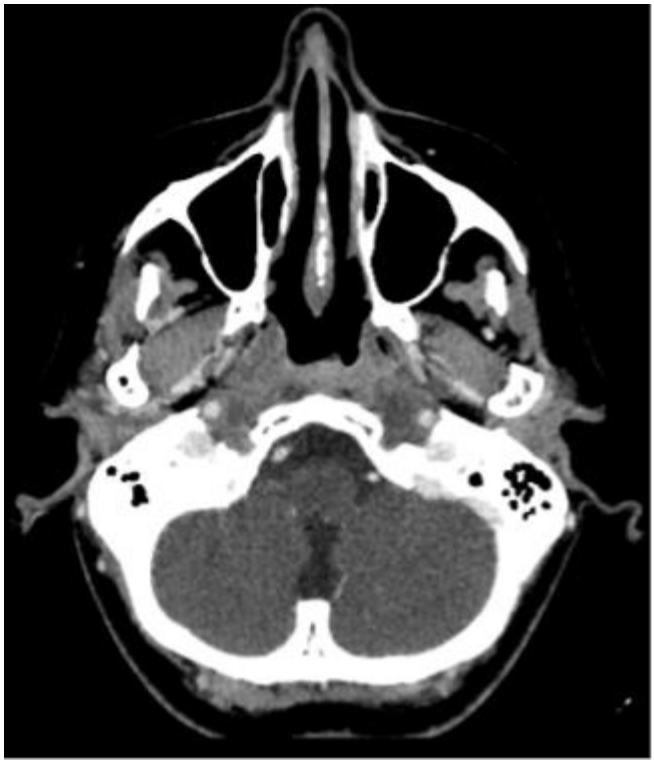

Figure 2. Follow-up CT scan of the head performed in April 2025 showing near-complete regression of the retropharyngeal mass and progressive sclerosis of previously osteolytic bone margins, consistent with reparative changes.