

Background

Tophaceous gout (TG) is an advanced manifestation of gout characterized by monosodium urate crystal deposition forming mass-like lesions in soft tissue and bone¹. Although tophi typically involve peripheral joints, axial and craniofacial involvement is rare and may present as an expansile, destructive-appearing lesion that closely mimics neoplastic processes^{2, 3}.

Skull base involvement is exceptionally uncommon, with only a limited number of cases reported³⁻⁶. Lesions may arise from adjacent structures, most notably the temporomandibular joint (TMJ), with secondary extension into the skull base, and can demonstrate bony erosion and mass effect despite minimal or nonspecific symptoms^{2, 7, 8}. The absence of a prior history of gout may complicate recognition and often necessitates tissue diagnosis.

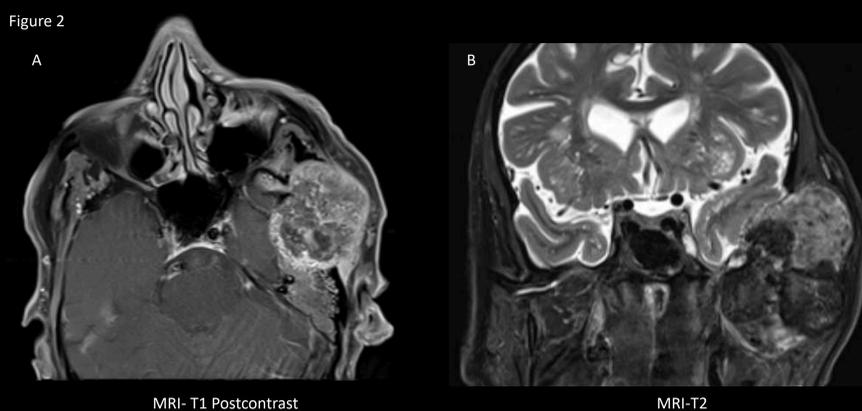
We report a rare case of TG presenting as a large temporal skull base mass with intracranial mass effect in an elderly patient. The precise anatomic site of origin could not be definitively established, underscoring the deceptive and potentially aggressive appearance of this entity and highlighting the importance of considering TG in the differential diagnosis of destructive skull base lesions.

Objective

Present a rare case of skull base TG manifesting as an expansile temporal skull base mass with intracranial mass effect in an elderly patient, and highlight its relevance in the differential diagnosis of temporal bone lesions.

Patient

81-year-old male who presented with a large temporal skull base mass involving the infratemporal fossa and mandible and producing intracranial mass effect. Despite the size and extent of the lesion, the patient was minimally symptomatic. His clinical history was notable only for progressive enlargement of the mass over a period of approximately 6–12 months, associated with dull discomfort, without focal neurological deficits. Imaging demonstrated an expansile skull base lesion, raising concern for neoplastic pathology (Figures 1A, B and 2A, B).



Intervention

Operative incisional biopsy was obtained for diagnostic clarification, with limited debulking of the lesion also performed. The pathology revealed findings consistent with a gouty tophus with associated calcium deposition. The patient had no prior history of gout. The patient was referred to rheumatology for further metabolic evaluation and management.

Discussion

In this rare case of TG, the extensive involvement of the temporal skull base, infratemporal fossa, and mandible strongly raised suspicion for a neoplastic process. Biopsy was performed to establish a definitive diagnosis while minimizing morbidity. Histopathologic confirmation of TG resulted in a fundamental shift in management.

The absence of a prior diagnosis of gout further complicated recognition and underscores the need for a broad differential diagnosis when evaluating destructive skull base lesions. A review of the literature identified only five reported cases of gout with skull base involvement (Table 1)⁴⁻⁸, all of which demonstrated TMJ involvement with secondary extension, suggesting the TMJ as a likely site of origin. Although the distribution in the present case raises similar suspicion, primary skull base origin cannot be definitively excluded.

Several case series have also described calcium pyrophosphate deposition disease (pseudogout) involving the skull base and TMJ^{9,10} with clinical and radiographic features closely resembling those observed here. Despite these similarities, pseudogout is pathologically distinct, characterized by calcium pyrophosphate rather than monosodium urate crystal deposition, and should remain an important consideration in the differential diagnosis.

The cornerstone of management of TG is pharmacological therapy aimed at controlling hyperuricemia and reducing crystal burden¹¹. However, in cases of TG with skull base involvement, surgical resection or debulking is reserved for diagnostic purposes or for patients with neurological deficits or significant mass effect, in whom surgical intervention may provide symptomatic relief.

This case underscores the importance of tissue diagnosis, judicious surgical decision-making, and interdisciplinary collaboration, as accurate recognition of this rare entity can in specific cases prevent unnecessary extensive skull base surgery in favor of pharmacologic disease-targeted therapy.

Table 1. Summary of reported cases of tophaceous gout with skull base involvement, including cases with undetermined primary versus secondary origin.

Author(s), Year	Number of Cases	Involved anatomic Location	Treatment
Suba et al., 2009 ⁴	1	TMJ, skull base, ITF	Surgical resection
Ott et al., 2009 ⁵	1	TMJ, ITF, TB, GWS	Subtotal resection + pharmacological therapy
Barthélémy et al., 2010 ⁶	1	TMJ, TB, GWS	Surgical resection
Birk et al., 2022 ⁷	1	TMJ, EAC, TB, MCF, ME	Pharmacological therapy
Hng et al., 2022 ⁸	1	TMJ, skull base	Pharmacological therapy
Present case	1	TMJ, TB, ITF, MCF	Diagnostic biopsy with limited debulking, medical therapy pending
Total	6	—	—

Abbreviations: external auditory canal (EAC); infratemporal fossa (ITF); middle cranial fossa (MCF); middle ear (ME); temporal bone (TB); temporomandibular joint (TMJ); greater wing of the sphenoid (GWS). "Skull base" indicates involvement without a more specific anatomic location specified.

Conclusion

Skull base TG is exceedingly rare but should be considered in patients presenting with expansile temporal bone and skull base masses, even in the absence of a prior history of gout, particularly when the clinical course is indolent. As illustrated in this case, these lesions may demonstrate aggressive radiographic features despite minimal clinical symptoms, resulting in diagnostic uncertainty. Recognition of this entity is critical, as accurate diagnosis directly guides management and may prevent unnecessary extensive surgical intervention in favor of appropriate therapy.

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