World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

Sinonasal giant cell granuloma mimicking local recurrence after chemoradiotherapy for nasal cavity squamous cell carcinoma: A case report

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World Journal of Advanced Research and Reviews, 2026, 31(01), 1826–1832

Publication history: Received on 13 June 2026; revised on 18 July 2026; accepted on 21 July 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.31.1.1944

Abstract

Background: Giant cell granuloma is a benign osteoclast-rich lesion that most often involves the jaws and only exceptionally arises in the nasal cavity or paranasal sinuses. In a previously irradiated patient, a new enhancing sinonasal mass may strongly suggest recurrent malignancy, making histopathological confirmation essential.

Case presentation: A 50-year-old man with a history of squamous cell carcinoma of the left nasal cavity treated with definitive chemoradiotherapy four years earlier and followed by an initially favorable course was referred for a newly detected sinonasal mass. Contrast-enhanced computed tomography demonstrated a 47 × 30 × 57 mm tissue process centered on the posterior left nasal cavity, filling the nasopharynx and eroding the inferior wall of the left nasal cavity, without significant cervical lymphadenopathy. The radiological appearance raised concern for local recurrence. The patient underwent surgical resection under general anesthesia. Histological examination showed respiratory mucosa overlying a biphasic giant cell-rich proliferation composed of numerous osteoclast-like multinucleated giant cells and mononuclear spindle-shaped cells, with approximately three mitoses per 10 high-power fields and no atypical mitoses. Immunohistochemistry showed no p63 expression in the giant cells, moderate diffuse membranous and cytoplasmic CD163 expression in the giant-cell population, and a Ki-67 labeling index of approximately 20% in histiocytic cells. The overall morphological and immunohistochemical findings supported the diagnosis of a giant cell granuloma rather than recurrent squamous cell carcinoma. At one year, nasofibroscopic follow-up was normal, without evidence of residual or recurrent disease.

Conclusion: Sinonasal giant cell granuloma is a rare but important benign mimic of recurrent cancer. In patients previously treated for sinonasal malignancy, imaging alone cannot reliably distinguish recurrence from a giant cell-rich reactive or neoplastic lesion. Diagnosis requires careful clinicoradiological correlation, representative tissue sampling, exclusion of hyperparathyroidism, and, when necessary, ancillary testing to exclude giant cell tumor of bone and other osteoclast-rich lesions.

Keywords: Giant cell granuloma; Nasal cavity; Nasopharynx; Squamous cell carcinoma; Post-radiotherapy mass; Osteoclast-like giant cells

1. Introduction

Giant cell granuloma is an uncommon benign lesion characterized by osteoclast-like multinucleated giant cells dispersed within a vascular fibroblastic or histiocytic stroma. The historical term “giant cell reparative granuloma” was

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introduced by Jaffe in 1953, initially to describe jaw lesions thought to represent an exaggerated reparative response to intraosseous hemorrhage [1]. Because trauma is absent in many patients and a purely reparative origin has not been established, the shorter term giant cell granuloma is increasingly preferred.

Most lesions arise in the mandible or maxilla. Extra-gnathic craniofacial involvement is uncommon, and primary lesions of the nasal cavity, paranasal sinuses, orbit, or skull base are particularly rare [2-6]. Despite benign histology, some lesions may grow rapidly, erode bone, extend toward the orbit or cranial cavity, and recur after incomplete removal [2,5,6].

The diagnosis is especially challenging in patients with a previous sinonasal malignancy. A new enhancing mass in an irradiated field is commonly interpreted as recurrent tumor, a second primary malignancy, or a treatment-related complication. However, imaging findings of giant cell granuloma are nonspecific and may overlap with those of malignant neoplasms. Histopathology is therefore decisive.

We report a rare sinonasal giant cell granuloma presenting four years after chemoradiotherapy for squamous cell carcinoma of the left nasal cavity. The lesion formed a large posterior nasal cavity and nasopharyngeal mass that was initially considered radiologically suspicious for local recurrence. This case highlights the diagnostic pitfalls of giant cell-rich lesions in a previously treated sinonasal field and provides a practical discussion of their differential diagnosis.

2. Case presentation

A 50-year-old man was referred to the otolaryngology department for evaluation of a newly identified sinonasal mass. Four years earlier, he had been diagnosed with squamous cell carcinoma of the left nasal cavity and had received combined radiotherapy and chemotherapy. The available records described a favorable post-treatment course before the present episode.

The patient mainly presented with unilateral nasal obstruction associated with rhinorrhea. There was no epistaxis, visual blurring, or facial pain.

On otolaryngological examination, a mass was identified in the left nasal cavity with posterior extension toward the nasopharynx. The remainder of the otolaryngological examination was unremarkable. Cranial nerve examination was normal, and no clinically significant cervical lymphadenopathy was identified.

Contrast-enhanced cervicofacial computed tomography performed on 9 July 2024 demonstrated a tissue process centered on the posterior portion of the left nasal cavity, measuring approximately 47 × 30 × 57 mm and bulging into a completely occupied nasopharynx. Inferiorly, the lesion lysed the floor of the left nasal cavity. Superiorly, the ethmoid cells were preserved; medially, the nasal septum was respected; and laterally, the medial wall of the left maxillary sinus was preserved. No significant cervical lymphadenopathy was identified, and the major cervical vessels remained patent. The examination also showed chronic bilateral sinonasal inflammatory changes and a small right nasal polyp.

Given the patient's oncological history, the lesion was interpreted radiologically as highly suspicious for local recurrence, with an initial radiological classification of T3N0. The patient subsequently underwent surgical resection of the mass under general anesthesia.

The lesion was completely excised through an exclusively endoscopic transnasal approach. Intraoperative assessment showed no macroscopic bone invasion. No significant intraoperative bleeding occurred, and the postoperative course was uncomplicated, without postoperative hemorrhage.

Gross examination consisted of multiple polypoid tissue fragments with a combined weight of approximately 8 g, the largest measuring 2.5 × 1.8 × 1.5 cm. Histological examination showed respiratory-type mucosa containing, in depth, a cellular proliferation with two main components. The first consisted of numerous multinucleated osteoclast-like giant cells with rounded nuclei, fine chromatin, and abundant eosinophilic granular cytoplasm. The second consisted of mononuclear spindle-shaped cells with round to elongated nuclei, focally dense homogeneous chromatin, occasional nucleoli, and abundant eosinophilic cytoplasm. The mitotic index was estimated at approximately three mitoses per 10 high-power fields, without atypical mitoses. Ectatic vascular structures were also present.

Immunohistochemical examination showed absence of p63 expression in the giant cells. CD163 demonstrated moderate diffuse membranous and cytoplasmic expression in the giant-cell population, supporting histiocytic/osteoclastic differentiation. Ki-67 showed nuclear labeling in approximately 20% of histiocytic cells. No morphological evidence of

recurrent squamous cell carcinoma was identified in the examined specimens. The final diagnosis was giant cell granuloma.

Laboratory evaluation performed to exclude a brown tumor showed normal serum calcium, parathyroid hormone, and renal function. Serum phosphate, alkaline phosphatase, and vitamin D levels were not available; this incomplete metabolic assessment represents a limitation of the present report.

At one year of follow-up, nasofibroscopic examination was normal, with no endoscopic evidence of residual or recurrent disease. No additional treatment was required, and the patient remained clinically well.

3. Discussion

Giant cell granuloma is rare in the sinonasal tract. The best-documented sites are the jaws, whereas nasal cavity and paranasal sinus cases are confined largely to isolated reports and small series [2-6]. Patients with nasal lesions commonly present with unilateral obstruction, epistaxis, rhinorrhea, headache, facial swelling, proptosis, diplopia, or visual impairment. Although many reported patients are children or young adults, lesions can occur at any age, including older adults [2,4]. The present patient is unusual because the lesion developed in adulthood within a previously treated sinonasal oncological field.

The relationship between giant cell granuloma and prior irradiation remains uncertain. Trauma, inflammation, surgery, and local hemorrhage have been proposed as triggers, but many lesions arise without an identifiable initiating event [1,2]. Radiotherapy causes chronic vascular injury, tissue hypoxia, fibrosis, mucosal breakdown, and altered bone remodeling; these changes provide a biologically plausible background for a reactive giant cell-rich process. Nevertheless, the available literature does not establish radiotherapy as a recognized direct cause of sinonasal giant cell granuloma. Accordingly, this case should be described as occurring after chemoradiotherapy rather than as definitively radiation-induced.

Imaging is essential for defining extent but is not diagnostic. On computed tomography, giant cell granulomas may appear as expansile soft-tissue or osteolytic masses with variable enhancement, cortical thinning, remodeling, or frank bone erosion. Magnetic resonance imaging may show heterogeneous signal intensity related to fibrosis, hemorrhage, hemosiderin, cystic change, and cellularity [3]. These findings overlap with recurrent squamous cell carcinoma, sarcoma, inflammatory lesions, aneurysmal bone cyst, and giant cell tumor of bone. In the present case, the large enhancing mass, nasopharyngeal extension, and focal bone lysis understandably raised concern for recurrence.

Histologically, giant cell granuloma contains osteoclast-like multinucleated giant cells in a background of spindle or ovoid mononuclear stromal cells, macrophages, capillaries, hemorrhage, and hemosiderin. The giant cells are usually unevenly distributed and may cluster around hemorrhagic areas. Mitotic figures may be present in mononuclear cells, but atypical mitoses and overt malignant cytology should be absent. The morphology in our patient, including numerous osteoclast-like giant cells, spindle-cell stroma, ectatic vessels, limited mitotic activity, and absence of atypical mitoses, was compatible with this diagnosis.

The immunophenotype must be interpreted in its cellular context. CD163 expression in giant cells and histiocytic cells supports a reactive osteoclast/macrophage lineage but is not, by itself, specific for giant cell granuloma. p63 has been studied as a potential discriminator because neoplastic stromal cells in giant cell tumor of bone may express p63 more frequently than giant cell granulomas; however, p63 lacks absolute sensitivity and specificity and should not be used alone [7]. In our patient, the absence of p63 staining in giant cells and the CD163-positive histiocytic population were concordant with giant cell granuloma, but radiological correlation and morphology remained central.

The most important differential diagnosis is giant cell tumor of bone. Giant cell tumor typically occurs in skeletally mature young adults, most often at the epiphyses of long bones, but craniofacial examples occur. Histologically, giant cells are often more uniformly distributed and their nuclei resemble those of the mononuclear neoplastic stromal cells. Giant cell tumor may recur, metastasize, or rarely undergo malignant transformation. Most conventional giant cell tumors harbor an H3F3A mutation, usually p.G34W, detectable by mutation-specific H3.3 G34W immunohistochemistry in mononuclear stromal cells [8,9]. This marker is highly useful when morphology and location create uncertainty. H3.3 G34W staining or H3F3A sequencing was not reported in the present case and could be considered if diagnostic doubt remains.

Brown tumor of hyperparathyroidism is morphologically indistinguishable from other giant cell-rich lesions and must be excluded biochemically. Serum calcium, phosphate, parathyroid hormone, alkaline phosphatase, renal function, and

vitamin D should therefore be assessed. Other differential diagnoses include aneurysmal bone cyst, which usually contains blood-filled spaces and may harbor USP6 rearrangement; cherubism and syndromic giant cell lesions in the appropriate age and distribution; tenosynovial giant cell tumor; foreign-body giant cell reaction; granulomatous infection; osteoclast-rich osteosarcoma; and recurrent or sarcomatoid carcinoma. In a patient with prior squamous cell carcinoma, generous sampling is critical to avoid overlooking a focal epithelial malignancy within a predominantly reactive or giant cell-rich lesion.

The term “reparative” is potentially misleading because a consistent history of trauma is absent and some lesions behave aggressively. Current clinicopathological practice often uses giant cell granuloma or central giant cell granuloma for intraosseous disease and peripheral giant cell granuloma for mucosal/gingival lesions. [12,13] In the sinonasal tract, determining whether a lesion is central or peripheral may be difficult when a large mass involves both mucosa and adjacent bone. In this patient, the lesion was centered in the posterior nasal cavity and caused focal inferior bony lysis, but the precise site of origin should be correlated with operative findings.

Surgical removal remains the most established treatment for accessible sinonasal lesions. Complete excision is preferred when it can be achieved without unacceptable functional morbidity, because residual disease may regrow. Curettage or debulking may be appropriate in selected anatomically constrained cases, but requires close surveillance. Reported nonsurgical treatments for central giant cell granuloma include intralesional corticosteroids, calcitonin, interferon-alpha, bisphosphonates, and denosumab [10,11]. Evidence is based mainly on jaw lesions, heterogeneous case series, and low-level observational data. Denosumab may be effective in extensive or unresectable disease but carries clinically important risks, including disturbances of calcium metabolism and rebound hypercalcemia, and its optimal duration is not established.

Radiotherapy should generally be avoided as a treatment for a benign giant cell granuloma, particularly in a patient who has already received irradiation, because of concerns regarding tissue toxicity and the historical risk of radiation-associated malignant transformation in giant cell-rich bone lesions. It may appear in older reports as an option for unresectable disease, but modern management favors surgery or carefully selected systemic/intralesional approaches.

Follow-up is important because recurrence is principally related to biological aggressiveness and incomplete excision. Surveillance should combine endoscopic examination and cross-sectional imaging, preferably MRI for soft-tissue assessment and CT when bone remodeling or destruction must be evaluated. The interval and duration should be individualized according to residual disease, anatomical extent, and the patient’s previous malignancy. In this case, prolonged follow-up is doubly necessary to detect either recurrence of the giant cell lesion or a true recurrence/second malignancy in the previously irradiated sinonasal tract.

This report has several limitations. The available material does not document the exact primary cancer stage and chemoradiotherapy regimen, the complete metabolic assessment (serum phosphate, alkaline phosphatase, and vitamin D were unavailable), H3.3 G34W status, or detailed pathological margin assessment. Nevertheless, normal serum calcium, parathyroid hormone, and renal function argued against a brown tumor, and one-year nasofibroscopic follow-up showed no residual or recurrent lesion. Moreover, a temporal association with prior radiotherapy does not establish causality.

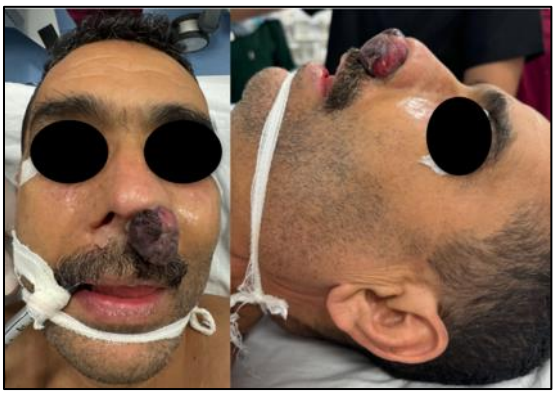

Figure 1 Preoperative clinical findings. Anonymized clinical photograph demonstrating the external or endonasal appearance associated with the sinonasal mass

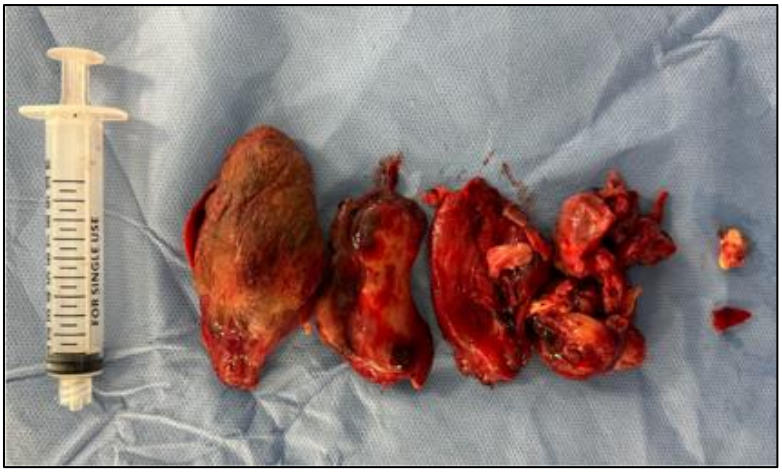

Figure 2 Gross appearance of the resected polypoid mass

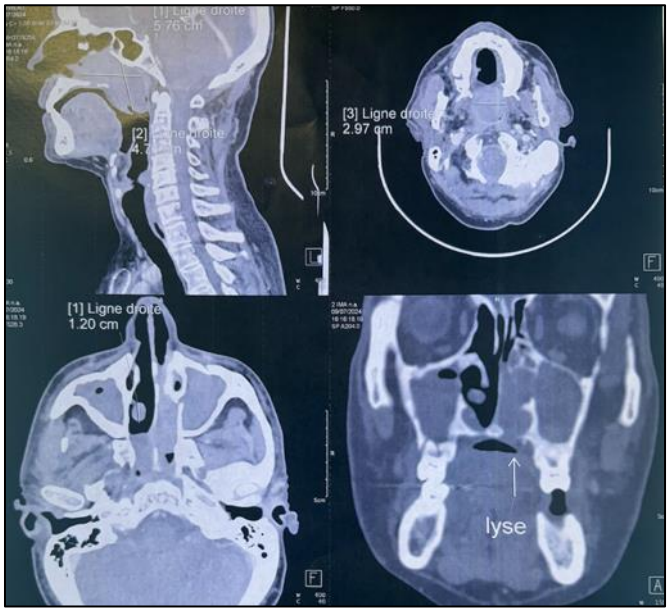

Figure 3 Contrast-enhanced computed tomography of the sinonasal tract. Axial, coronal and sagittal images showing a 47 × 30 × 57 mm tissue mass centered on the posterior left nasal cavity, filling the nasopharynx and associated with focal lysis of the inferior wall of the left nasal cavity. The nasal septum, ethmoid cells, and medial wall of the left maxillary sinus are preserved

3.1. Practical differential diagnosis

Table 1 Main differential diagnoses of sinonasal giant cell granuloma and their distinguishing clinicopathological features

Entity	Helpful clues	Useful ancillary tests	Clinical relevance
Giant cell granuloma	Uneven giant-cell distribution; hemorrhage; spindle-cell stroma; no atypical mitoses	Clinicoradiological correlation; CD68/CD163 supportive; exclude hyperparathyroidism	Benign but may be locally aggressive and recur
Giant cell tumor of bone	More uniform giant-cell distribution; stromal and	H3.3 G34W IHC; H3F3A sequencing	Higher recurrence; rare lung metastasis or malignant transformation

	giant-cell nuclei may resemble each other		
Brown tumor	Histology overlaps completely; may be multifocal	Calcium, phosphate, PTH, ALP, renal function, vitamin D	Treat the endocrine/renal disorder
Aneurysmal bone cyst	Blood-filled spaces, septa, secondary giant cells	USP6 testing in primary lesions	May recur; treatment depends on site and extent
Recurrent SCC / sarcomatoid carcinoma	Atypical epithelial or spindle cells; destructive infiltrative growth	Broad-spectrum cytokeratins, p40/p63, EMA; generous sampling	Requires oncological treatment
Osteoclast-rich osteosarcoma	Malignant cytology and tumor osteoid	SATB2 supportive; molecular work-up as indicated	High-grade malignant bone tumor

4. Conclusion

Sinonasal giant cell granuloma is an uncommon benign lesion that may closely mimic recurrent malignancy, particularly when it presents as a large enhancing and bone-eroding mass in a previously irradiated patient. The diagnosis rests on representative histological sampling and correlation with imaging and clinical history. Hyperparathyroidism must be excluded, and H3.3 G34W immunohistochemistry or H3F3A testing may be valuable when giant cell tumor of bone remains a concern. Complete surgical excision, when feasible, and long-term endoscopic and radiological surveillance are recommended.

Learning points

- A new mass in a previously irradiated sinonasal field is not necessarily recurrent carcinoma.
- Sinonasal giant cell granuloma may be large, enhancing, and locally destructive despite benign histology.
- CD163 confirms histiocytic/osteoclastic differentiation but is not specific; morphology and clinicoradiological correlation remain essential.
- Biochemical exclusion of hyperparathyroidism is mandatory in any giant cell-rich lesion.
- H3.3 G34W immunohistochemistry can help distinguish giant cell tumor of bone from its histological mimics.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of ethical approval

Ethical approval was not required for this retrospective case report in accordance with institutional policy for anonymized case-based reports.

Statement of informed consent

Written informed consent was obtained from the patient for publication of this anonymized case report and the accompanying images.

Guarantor

The corresponding author is the guarantor of this work.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request, with appropriate protection of patient confidentiality.

CRediT authorship contribution statement

- Adam El Hassouni: Conceptualization, Data curation, Formal analysis, Writing - original draft.
- Rajae Borki: Supervision, Validation, Writing - review and editing.
- Aymene Benmansour: Data curation, Investigation, Writing - review and editing.
- Omar Rami: Data curation, Investigation, Writing - review and editing.
- Ilham Rkain: Investigation, Validation, Writing - review and editing.
- Hicham Mimouni: Supervision, Conceptualization, Writing - review and editing.

All authors read and approved the final version of the manuscript.

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