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Case Report: Giant cell lesions in the Maxillofacial region: diagnostic points and treatment strategies

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Objective: Giant cell-rich lesions in the maxillofacial region are relatively rare, and comprehensive clinical differential diagnostic protocols are currently lacking. This article aims to provide a reference for the clinical diagnosis and treatment of giant cell-rich lesions.

Methods: This study investigates the distinguishing features of four types of giant cell-rich lesions in differential diagnosis and treatment: giant cell tumor of bone (GCT), aneurysmal bone cyst (ABC), tenosynovial giant cell tumor (TGCT), and giant cell reparative granuloma (GCRG).

Results: Immunohistochemical (IHC) analysis reveals strong p63 positivity in the mononuclear stromal cells of GCT, but not in GCRG. The “fluid-fluid level” observed in magnetic resonance imaging (MRI) is a diagnostic indicator for ABC, reflecting variable signal intensities. TGCT is characterized by the presence of synovial monocytes, multinucleated giant cells, foam cells, and hemosiderin-laden cells.

Conclusion: Accurate diagnosis requires a comprehensive evaluation of clinical, imaging, and pathological data. While complete resection is crucial for GCT to prevent recurrence and malignant transformation, GCRG typically responds well to curettage due to its benign nature. Early surgical intervention is essential for TGCT to control its aggressive progression and minimize complications.

KEYWORDS

giant cell lesions, Maxillofacial region, giant cell tumor of bone, aneurysmal bone cyst, giant cell reparative granuloma, tenosynovial giant cell tumor

Introduction

Giant cell-rich lesions encompass a diverse group of tumors and neoplastic lesions characterized by the presence of varying numbers of reactive, multinucleated osteoclast-like giant cells (1, 2). The World Health Organization (WHO) classifies several osteoclast-rich lesions, including giant cell tumor of bone (GCT), aneurysmal bone cyst (ABC), tenosynovial giant cell tumor (TGCT), and giant cell reparative granuloma (GCRG), alongside non-ossifying fibromas and certain benign conditions (1, 3, 4).

These lesions are relatively uncommon in the maxillofacial region, where accurate diagnosis can be particularly challenging due to significant overlap in the clinical and histopathological features of these disorders (5, 6). Effective diagnosis requires a thorough evaluation of pathological, clinical, and radiological attributes, with special attention to the anatomical location, patient age, and lesion count. Additionally, the identification of elements beyond giant cells is crucial to avoid misdiagnosis (7–9). This article reviews clinical cases of four pertinent disorders, discusses key aspects of their differential diagnosis and

management in routine practice, and offers guidance for the clinical diagnosis and treatment of GCT (6, 10, 11).

Case reports

Case 1

A 43-year-old woman presented with a 4-month history of a left mandibular mass. Computed tomography (CT) scans (Figures 1A–C) revealed expansive osteolytic destruction in the mandibular ramus and body, featuring soft tissue density, cortical thinning, and multifragmented bone with clear soft tissue boundaries. Surgical management involved tumor resection with free fibular flap reconstruction. Hematoxylin-eosin (H&E) staining (Figures 1D, E) demonstrated a giant cell-rich tumor composed of evenly distributed spindle or mononuclear cells and osteoclast-like giant cells, with occasional mitotic figures and sinusoidal cavities, consistent with GCT. IHC analysis (Supplementary Figure 1) yielded the following results: p63(+), S100(-), CD68(+), SMA (+), and Ki-67(+). The

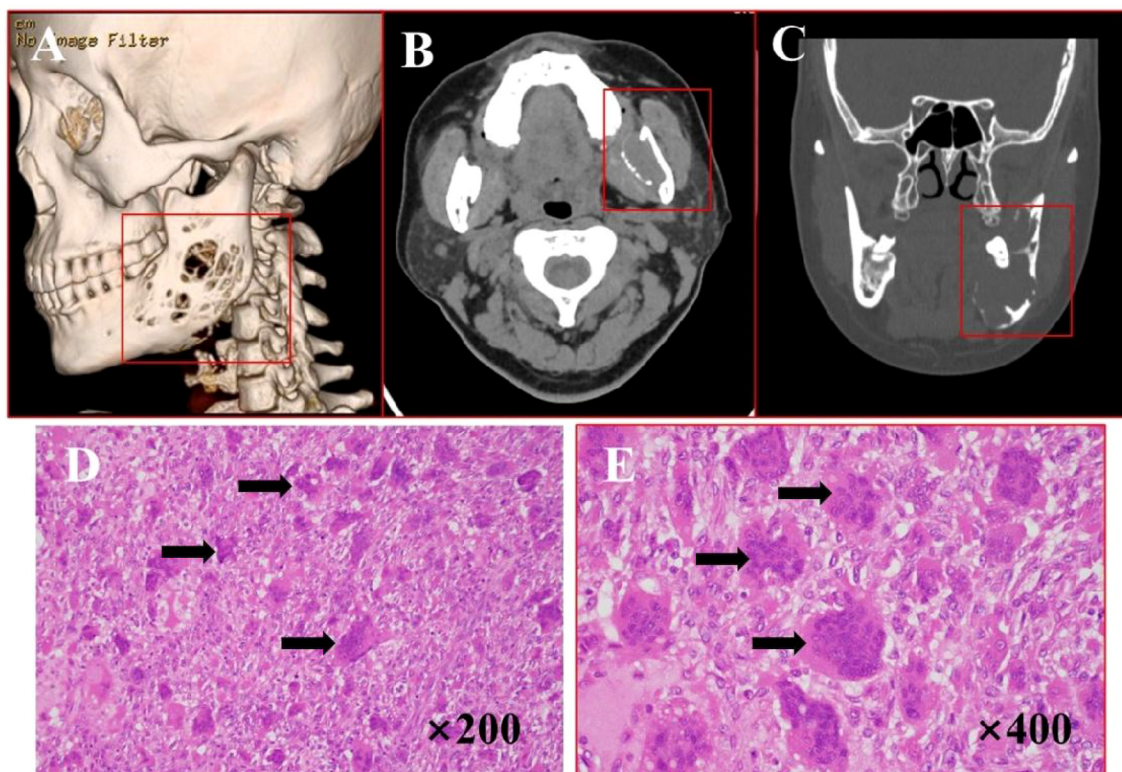


FIGURE 1

Imaging and histopathological features of GCT. (A) Bone CT demonstrates expansive osteolytic destruction in the left mandibular ramus and body with cortical thinning. (B, C) Axial and coronal CT reveal multiple discontinuities in the bone structure, characterized by dense soft tissue, thin cortical bone, multiple bone discontinuities, and clearly demarcated surrounding soft tissue. (D–E) H&E staining (x200, x400): arrows point to giant cells and the cells were oval.

patient remained recurrence-free and asymptomatic during 36 months of follow-up.

Case 2

A 52-year-old woman presented with a 3-month history of a painless right maxillary mass. CT scans (Figures 2A–C) demonstrated extensive alveolar bone destruction with contour loss, well-defined margins, and partial bone thinning, measuring 45×52×39 mm. The lesion, containing a thin bony shell, caused right nasal cavity displacement. Surgical intervention included tumor resection under general anesthesia with titanium plate reconstruction. H&E staining (Figures 2D, E) of the tumor showed areas of active fibroproliferation with abundant reactive trabecular bone structure. Multiple cystic cavities were locally observed, indicative of ABC changes. Postoperative recovery was uneventful, and the patient remained recurrence-free and asymptomatic during 21 months of follow-up.

Case 3

A 19-year-old female presented with a 2-year history of a firm, fixed left temporal mass. CT imaging (Figures 3A–C) revealed an

irregular soft tissue lesion (46×30×37 mm) in the temporal squama and zygomatic region, containing speckled calcifications and associated bone destruction with areas of sclerosis. Surgical management involved complete tumor resection under general anesthesia. H&E staining (Figures 3D, E) revealed that the tumor was rich in giant cells, containing numerous phagocytic, hemosiderin-laden mononuclear cells with abundant cytoplasm and inclusion bodies. Some regions exhibited chondroid differentiation surrounded by pronounced proliferative fibrous tissue, leading to a diagnosis of diffuse TGCT with chondroid metaplasia. IHC results (Supplementary Figure 2) were as follows: SMA(+), S100 (-), p63 (-), Ki-67 (+), CD68 (-). The patient remained recurrence-free and asymptomatic during 21 months of follow-up.

Case 4

A 32-year-old male presented with a 2-year history of a painful right preauricular mass. CT imaging (Figures 4A–C) revealed a 29×45 mm cystic-solid lesion with heterogeneous enhancement, causing bone destruction in the temporal, sphenoid, and zygomatic regions, along with adjacent soft tissue changes. Surgical management included tumor resection via a hemiconal approach, zygomatic osteotomy, and temporal fascia flap

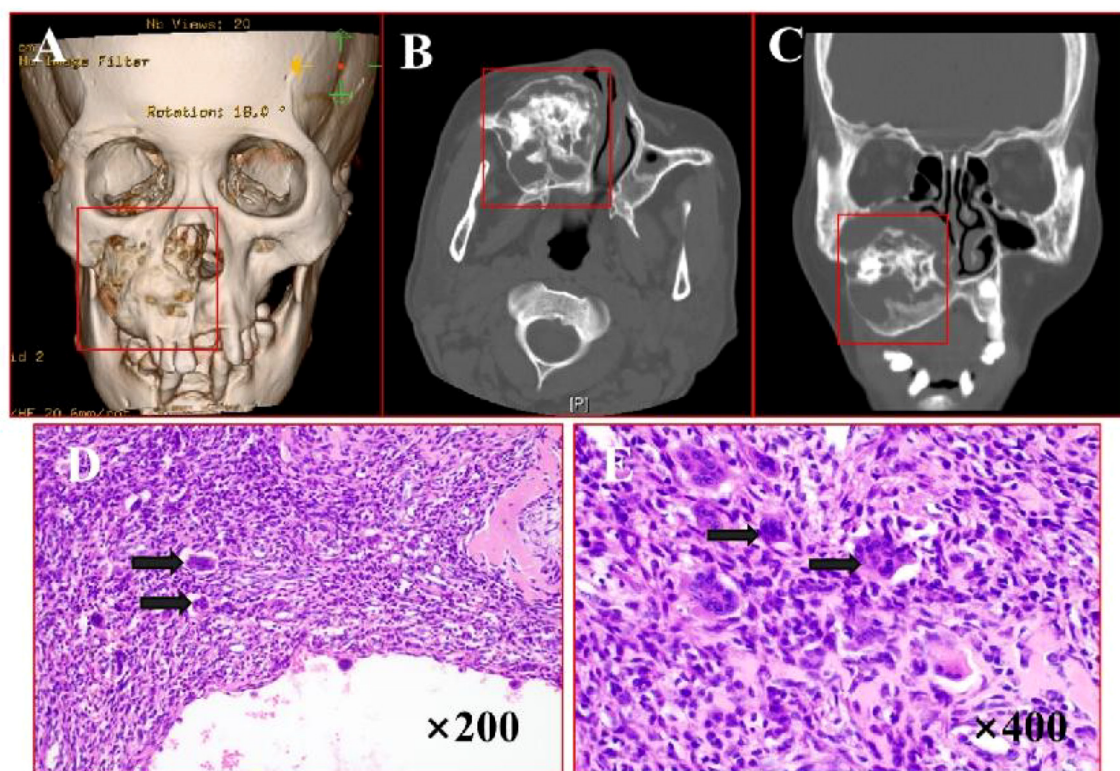


FIGURE 2

Imaging and histopathological features of ABC. (A) Bone CT demonstrates lesion involvement of the right maxilla, maxillary sinus, and palatine bone with irregular surrounding bone thickness. (B, C) Axial and coronal CT reveal osteolytic destruction with well-defined margins, partial bone thinning, and an internal thin bony shell. (D, E) H&E staining (x200, x400): arrows point cystic cavities with reactive new bone formation.

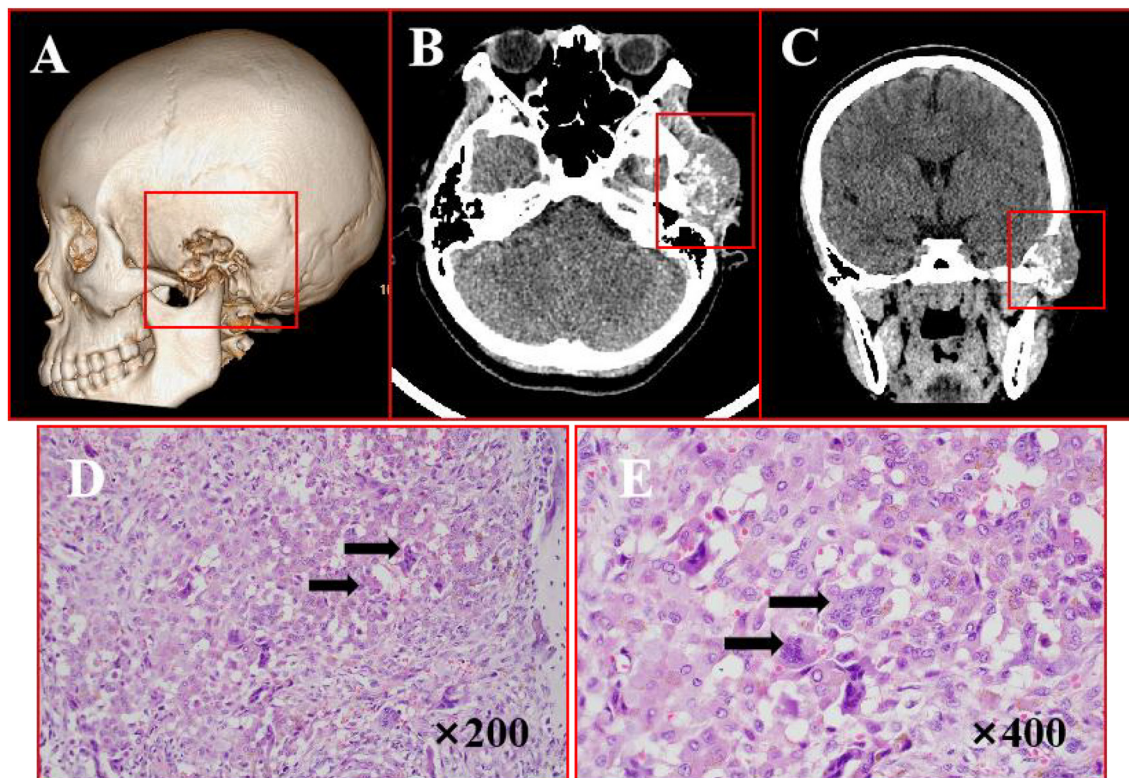


FIGURE 3

Imaging and histopathological features of TGCT. (A) Bone CT demonstrates osteolytic destruction involving the left mastoid air cells, zygomatic arch, and temporal bone with adjacent sclerosis. (B, C) Axial and coronal CT reveal an irregular soft tissue lesion extending into the temporal fossa, featuring poorly defined margins and internal speckled calcifications. (D, E) H&E staining (x200, x400): arrows points actively proliferating monocytes and osteoclast-like multinucleated giant cells.

reconstruction. H&E staining (Figures 4D, E) showed extensive degeneration, fibrous proliferation, hemorrhage, and histiocytic reaction, with areas of mononuclear cell proliferation and osteoclast-like giant cells, consistent with GCRG. IHC results (Supplementary Figure 3) demonstrated: P63 (-), S100 (-), CD68 (+), Ki67 (+), EMA (-) and SMA (+) expression. The patient remained recurrence-free and asymptomatic during 21 months of follow-up.

Discussion

Giant cell-rich lesions, defined by the presence of multinucleated giant cells, encompass various benign, nonneoplastic, and borderline conditions. Accurate diagnosis through histopathological, imaging, and molecular analyses is critical for guiding treatment decisions and predicting recurrence risk (12, 13). This review focuses on four representative lesions: GCT, GCRG, ABC, and TGCT (Table 1).

GCT typically manifests with pain and local swelling, predominantly affecting individuals aged 20–45 years, with a strong preference for long bones and rare cranial involvement (1, 13). GCRG, a non-neoplastic condition primarily seen in children and young adults, commonly involves the jawbones, exhibiting

destructive and locally invasive features that often lead to tumor misdiagnosis (4, 14). ABC presents as a blood-filled cystic lesion containing osseous tissue, mainly affecting adolescents in long bone metaphyses, characterized by distinctive subperiosteal bone deposition (15, 16). TGCT, a benign soft tissue tumor originating from synovial structures, typically occurs in 30–50-year-olds, with female predominance, and presents with joint swelling, pain, and restricted mobility (8, 9).

GCT typically demonstrates expansile bone destruction with adjacent cortical thickening or thinning, showing marked heterogeneous enhancement on MRI (4, 17). ABC is characterized by lytic destruction with multiple fluid-fluid levels on imaging - a diagnostic hallmark - often accompanied by periosteal reaction (15, 18). GCRG presents as nonspecific lytic lesions containing bony septations, typically lacking fluid-fluid levels, which helps differentiate it from GCT and ABC (4, 19). TGCT manifests as a well-defined or irregular soft tissue mass, potentially with calcifications or bone erosion, with MRI being crucial for assessing tumor extent and adjacent tissue involvement (20, 21).

Histopathologically, GCT displays uniformly distributed multinucleated giant cells, with P63 protein expression and occasional ossification/calcification serving as key diagnostic markers to differentiate it from GCRG and ABC (19, 22, 23). GCRG primarily consists of vascular-rich connective tissue

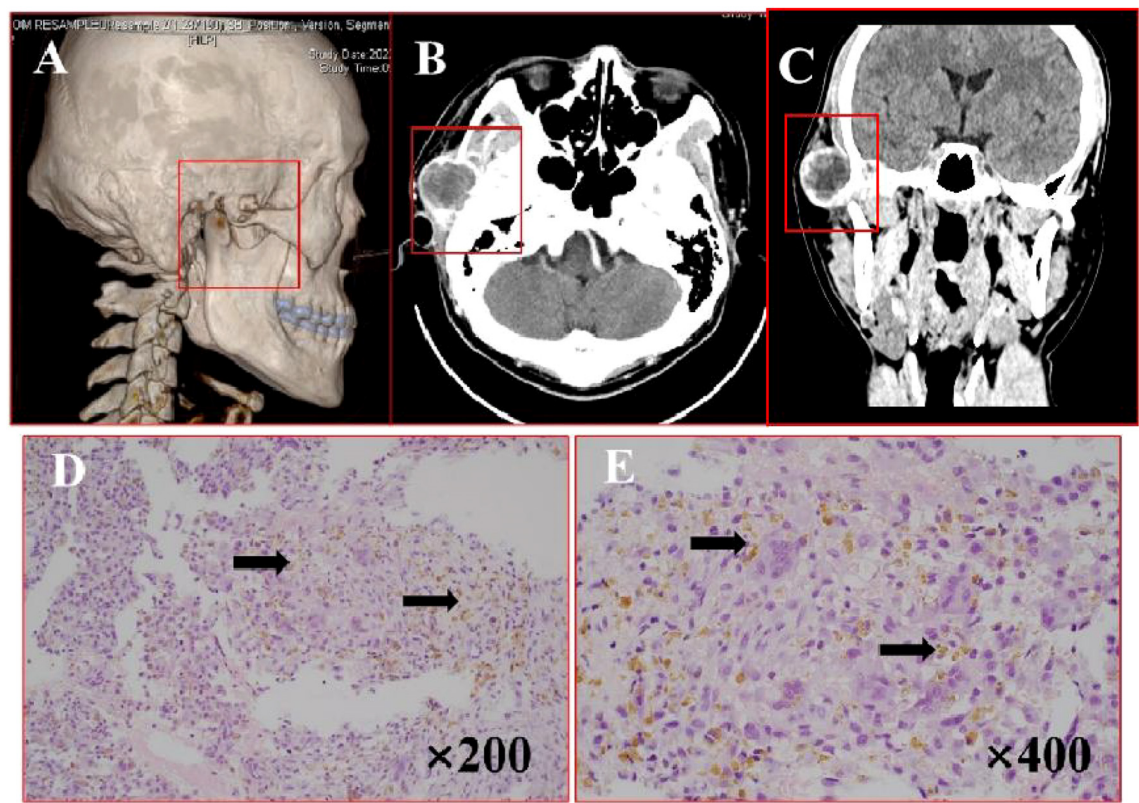


FIGURE 4
Imaging and histopathological features of GCRG. (A) Bone CT demonstrates right temporal expansile osteolytic destruction with cortical thinning and heterogeneous lesion density. (B, C) Axial and coronal CT reveal mastoid bone destruction with soft tissue density and a cystic-solid mass anterior to the right ear. (D, E) H&E staining (x200, x400): arrows points fibrous tissue hyperplasia with multinucleated giant cells, and hemosiderin deposition.

TABLE 1 Differential diagnosis of 4 types of diseases.

	GCT	ABC	TGCT	GCRG
Age	20-40Y	10-20Y	30-50Y	<20Y
Location	Epiphysis of long bones, rare in sphenoid and temporal bones	Metaphysis of long bones	Hands, rarely toes	Maxilla and mandibular bones
Histopathology	Uniformly dispersed giant cells; rare hemosiderin deposits; large, round, multinucleated giant cells; uncommon bone neogenesis	Broad-band cyst wall with osteoclast-type giant cells and histiocytes; large blood cavities with thin walls; hemosiderin deposition with phagocytes and giant cells	Synovial cells, small volume, unclear borders; polygonal or spindle-shaped cells with oval, triangular, or punctate nuclei; vascular fissure structures; stromal collagen fibers may show hyalinization and ossification	Giant cells around hemorrhagic lesions; hemosiderin deposits in long-term lesions; small, irregular, elongated, oligonuclear giant cells; focal osteoid and new bone formation
CT/MRI features	CT: Expansile lytic lesion, thin cortical layer, internal sclerosis. MRI: Low-medium signal on T1WI, medium-high signal on T2WI	CT: Expansile lesion with clear margins, osteolytic bone destruction. MRI: Fluid-fluid levels with multicystic high signal on T1 and T2	CT: Soft tissue mass with intact capsule, adjacent to or enveloping tendons and tendon sheaths. MRI: Low signal intensity on T1WI, mixed signal on T2WI	CT: Non-specific osteolytic lesions. MRI: Low signal on T1 and T2 weighted images
Management	Complete surgical resection; adjuvant radiotherapy when surgery is not feasible	Thorough surgical resection with cryotherapy	Complete surgical resection	Local curettage with total surgical resection

(Continued)

TABLE 1 Continued

	GCT	ABC	TGCT	GCRG
Prognosis	If radical resection is not performed, the recurrence rate is high. The possibility of malignant transformation and metastasis is high	Incomplete surgical resection is prone to recurrence	Recurrence after surgical resection, but not metastasis	The recurrence rate was low. So far, no metastases and malignant transformation have been recorded

containing fibroblasts and mononuclear cells, typically showing hemorrhage, hemosiderin deposition, and occasional osteoid formation. ABC, while also containing giant cells, is characterized by blood-filled cystic spaces lined with hemosiderin deposits and osteoblast-covered reactive bone, sometimes with diagnostic chondroid calcification (14, 15). TGCT exhibits synovial-like mononuclear cells mixed with multinucleated giant cells, foamy cells, and hemosiderin deposits, occasionally demonstrating cleft-like spaces and pseudoglandular structures (24, 25).

Surgical management remains the cornerstone for these lesions, with specific approaches tailored to each condition. For GCT, complete surgical excision is crucial to minimize the 25% recurrence risk and prevent potential malignant transformation or pulmonary metastasis, with adjuvant radiotherapy considered for high-risk cases (25, 26). GCRG treatment primarily involves surgical resection, achieving an 80% cure rate with local curettage, supplemented by calcitonin or bisphosphonate therapy to reduce recurrence (10, 11). ABC management centers on surgical curettage with bone grafting, though its high recurrence risk (10%-60%) may necessitate adjunctive cryotherapy or radiotherapy in complex cases (12, 13, 27). TGCT treatment relies on surgical resection of localized lesions, with postoperative radiotherapy proving particularly effective for diffuse forms, demonstrating improved prognosis despite its inherent recurrence risk. Across all conditions, comprehensive surgical strategies combined with appropriate adjuvant therapies and rigorous follow-up are essential for optimal outcomes (23, 27).

Overall, Surgery is the primary treatment for these lesions, with treatment selection guided by lesion aggressiveness, anatomical location, and patient factors. Postoperative adjuvant therapy and regular follow-up are essential to minimize recurrence and optimize outcomes.

This study highlights the importance of a multidisciplinary approach, integrating clinical, radiological, and histopathological data, to achieve accurate diagnosis and effective management. Emerging tools such as artificial intelligence (AI) in radiology and molecular markers offer significant potential to enhance diagnostic precision and treatment outcomes. Future advancements should focus on integrating multimodal data—clinical, radiological, histopathological, and molecular—to develop comprehensive diagnostic frameworks. Personalized treatment strategies, guided by molecular profiling and AI-driven predictive models, could optimize surgical and adjuvant therapies. By leveraging AI, molecular diagnostics, and multidisciplinary approaches, clinicians can achieve more precise diagnoses and tailored treatments, ultimately advancing patient care in this complex field.

Conclusion

This study explores diagnostic and management challenges of maxillofacial giant cell-rich lesions (GCT, ABC, TGCT, GCRG) through four rare cases. Radical surgical excision yielded excellent outcomes, with no recurrence over 21-36 months of follow-up. A multidisciplinary approach, combining MRI features (e.g., “fluid-fluid levels” in ABC) and immunohistochemical markers (p63, CD68, Ki-67), was crucial for accurate differentiation and pathogenic insights. Complete surgical resection is critical for GCT to avoid recurrence and potential malignant transformation, whereas GCRG, being a benign lesion, generally shows favorable outcomes with curettage. For TGCT, prompt surgical treatment is necessary to manage its aggressive behavior and reduce the risk of complications. The findings provide a comprehensive framework for accurate diagnosis and tailored treatment, enhancing patient outcomes and quality of life.

Data availability statement

The original contributions presented in the study are included in the article/[Supplementary Material](#). Further inquiries can be directed to the corresponding authors.

Ethics statement

As this study is a retrospective analysis based on previously collected data without any direct involvement of patients or interventions, formal approval from an ethics committee was not required. The data used in the study were collected from publicly available sources or previously published cases, ensuring compliance with ethical standards. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s), and minor(s)’ legal guardian/next of kin, for the publication of any potentially identifiable images or data included in this article.

Author contributions

XG: Conceptualization, Data curation, Investigation, Methodology, Writing – original draft. SW: Investigation, Writing – review & editing. YL: Methodology, Project administration, Writing – review & editing. LC: Supervision, Validation, Writing – review & editing. XZ: Formal analysis, Project administration, Writing – review & editing. JS: Formal

analysis, Resources, Writing – review & editing. HX: Project administration, Resources, Writing – review & editing.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Supplementary material

The Supplementary Material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fonc.2025.1572560/full#supplementary-material>

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