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Frontoethmoidal Giant Cell Reparative Granuloma

Gary J. Felsberg, Robert D. Tien, and Roger E. McLendon

Summary: We present a case of a giant cell reparative granuloma of the frontoethmoidal region that had a large intracranial extraaxial component and was studied with MR. Although rare, giant cell reparative granuloma can be suggested in the correct clinical setting and when MR features suggest a fibrous lesion.

Index terms: Paranasal sinuses, diseases; Paranasal sinuses, magnetic resonance; Granuloma

Giant cell reparative granuloma is a rare, benign fibroosseous lesion typically presenting as an expansile mass with cortical bone defect. These lesions are rare in the paranasal sinuses, more typically occurring in the region of the mandible and maxilla. We present the magnetic resonance (MR) findings in a case of giant cell reparative granuloma that involved the frontal and ethmoidal sinuses and had a large extraaxial intracranial component.

Case Report

An 18-year-old woman had right-sided proptosis and increased lacrimation for 1 month. No history of trauma or significant sinus infections was present. Neurologic and ophthalmologic examinations were normal; specifically, extraocular movements were intact and vision was normal. Laboratory studies, including calcium and alkaline phosphatase, were normal. An MR examination (1.5-T Signa scanner; General Electric, Milwaukee) was performed before and after contrast administration (Fig 1A–F). A mass was identified involving the right subfrontal region with extraaxial impression of the right frontal lobe. Involvement of the right frontal and ethmoidal sinuses was seen. MR showed the mass to be isointense with gray matter on T1- and T2-weighted images. Homogeneous enhancement was identified after contrast infusion. Linear regions of hypointensity consistent with fibrous septa were identified within the mass on all sequences. Thickening of the dura at the margin of the mass was consistent with a dural tail. The differential diagnosis preoperatively included a fibroosseous lesion arising from the ethmoidal sinus or wall of the frontal sinus or atypical meningioma. During craniotomy, a hard, bony mass was removed, and the initial frozen-section diagnosis was a giant cell lesion. Histologically, the mass revealed collections of mononuclear mesenchymal cells separated by cords of dense collagen associated with multinucleated giant cells (Fig 1G). Other areas showed well-formed interlacing osteoid with focal regions of mineralization. Mitotic activity could not be identified. A dense fibroblastic response was present in the submucosal soft tissue surrounding the lesion. The final histopathologic diagnosis was giant cell reparative granuloma.

Discussion

Giant cell reparative granuloma was described by Jaffe (1) in 1953. Before this report, any bone lesion containing giant cells was thought to be a giant cell tumor or variant thereof. Giant cell reparative granuloma is not a neoplastic process but is thought to represent a local hyperplastic reparative process after injury (1–3). In those without a history of trauma, giant cell reparative granuloma is likely a secondary granulomatous lesion developing in the setting of infection or inflammation complicated by hemorrhage (2, 4). In our case, the cause of the lesion was not discernible from the patient's history. The lesion is relatively rare, but has been found as a mass lesion in the orbit, paranasal sinuses (most commonly the sphenoidal), temporal bone, calvaria, mandible, and long bones (2, 5, 6).

Reported symptoms are relatively nonspecific and depend on the location of the lesion; local pain, periorbital swelling, and diplopia may be identified when involvement of the bony orbit is present (6). In our case, symptoms resulted from impression of the mass on the orbital soft tissues. Radiographically, the findings are relatively nonspecific. An expansile lesion is seen that may perforate the bony cortex. Occasionally, osteoid is identified within the lesion on computed tomography (6). In our case, al-

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From the Departments of Radiology (G.J.F., R.D.T.) and Neuropathology (R.E.M.), Duke University Medical Center, Durham, NC.

Address reprint requests to Gary J. Felsberg, MD, Department of Radiology, Box 3808, Duke University Medical Center, Durham, NC 27710.

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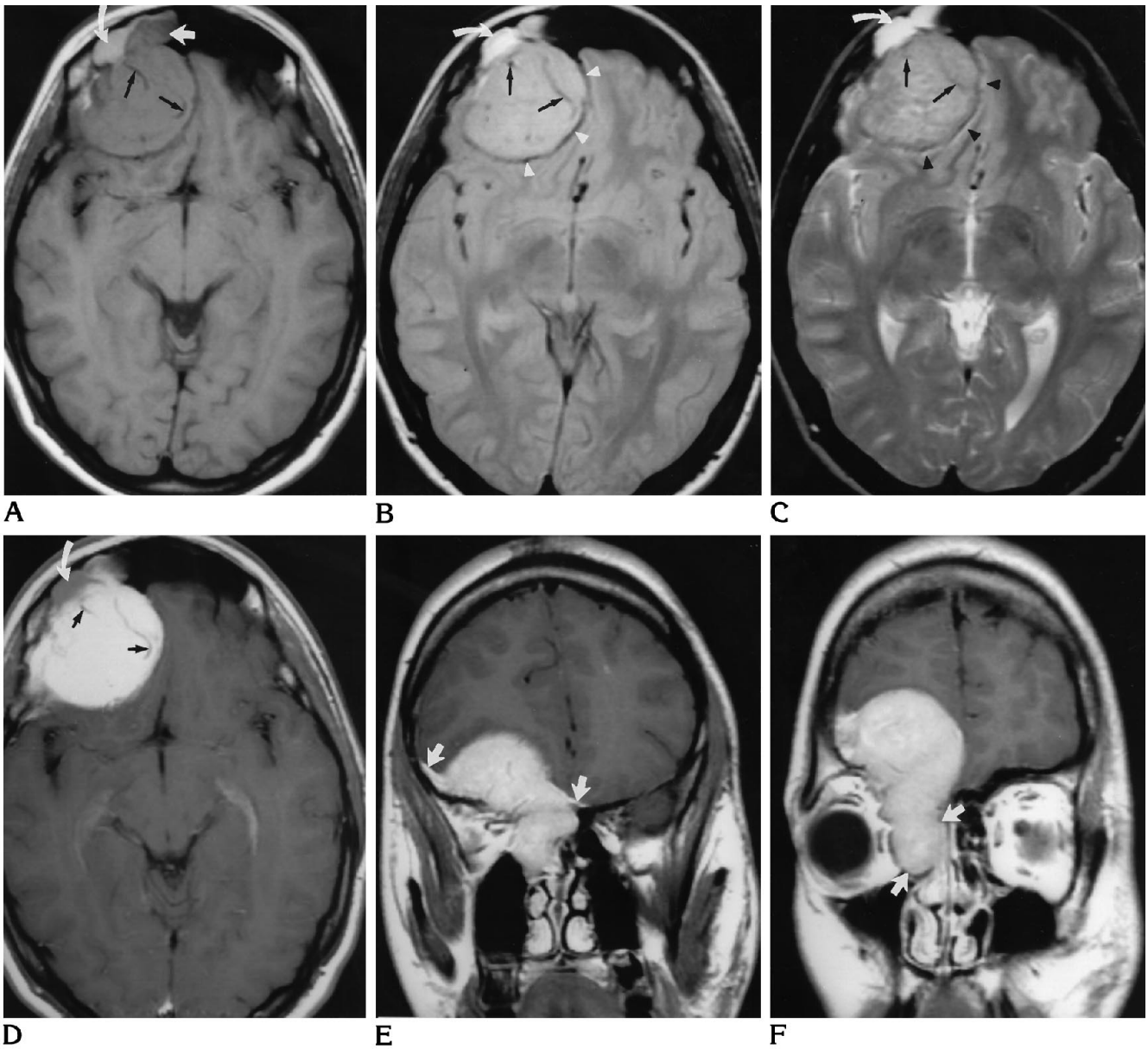

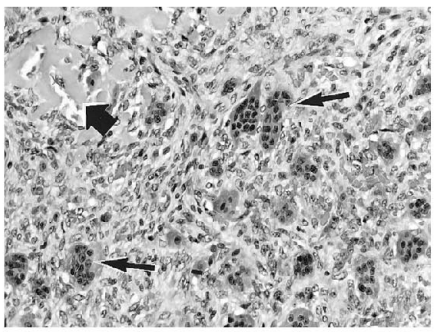

Fig 1. *A*, Axial T1-weighted MR image shows well-defined mass causing impression on the right frontal lobe. Note involvement of the right frontal sinus (*straight white arrow*) and loss of the posterior wall of the frontal sinus. Hyperintense material (*curved arrow*) laterally in the frontal sinus is consistent with trapped sinus secretions. Linear regions of hypointensity are consistent with fibrous septa (*black arrows*).

B, Axial balanced MR image shows mass remains isointense with gray matter. Thin hypointense structure between mass and frontal lobe is consistent with dura and/or thinned posterior wall of frontal sinus (*arrowheads*). Signal abnormality of cerebral parenchyma is absent. Trapped sinus secretions (*white arrow*) and fibrous septa (*black arrows*) are seen.

C, Axial T2-weighted MR image shows mass remains predominantly isointense with gray matter. Peripheral to thin hypointense structure separating mass and cerebral parenchyma is thin hyperintensity consistent with thin cerebrospinal fluid cleft (*arrowheads*). Trapped sinus secretions (*white arrow*) and fibrous septa (*black arrows*) are seen.

D, Axial T1-weighted MR image after contrast infusion shows homogeneous enhancement of the mass. Trapped sinus secretions (*white arrow*) and fibrous septa (*black arrows*) are seen.

E, Coronal T1-weighted MR image at level of orbital apex after contrast infusion shows dural thickening (dural tail sign) along both medial and lateral margins of the mass (*arrows*).

F, Coronal T1-weighted MR image more anterior to *E* after contrast infusion shows extension of the mass inferiorly into the right ethmoidal sinus and superior nasal cavity (*arrows*) with expansion of the sinus structure. The ethmoidal sinus septa are not preserved.

G, Photomicrograph (hematoxylin-eosin, ×250) of surgical specimen shows several multinucleated giant cells (*thin arrows*) and irregular bands of dense collagen (*thick arrows*) separated by sheets of basophilic mononuclear mesenchymal cells.

though extension of the mass through the posterior wall of the frontal sinus was seen along with ethmoidal sinus expansion, areas of calcification or osteoid could not be identified definitely. MR imaging findings have not been reported but should be expected, as in our case, to mimic the MR findings described for giant cell tumors, typified by a large homogeneous lesion similar in signal to gray matter on all sequences with homogeneous contrast enhancement (7). Given the presence of isointensity with gray matter on all MR sequences and the dural tail sign in our case, the possibility of meningioma was also considered.

The main differential diagnosis both radiographically and pathologically is the more common giant cell tumor. Radiographically, the lesions can be indistinguishable, demonstrating an expansile soft-tissue mass with locally invasive characteristics (8, 9). Giant cell tumors, although more common in epiphyseal portions of the long bones, can be seen in the skull, usually affecting the mandible, facial bones, and sphenoidal and ethmoidal sinuses (2, 8, 10, 11). Histologically, giant cell reparative granuloma can be difficult to differentiate from giant cell tumor. Both show multinucleated giant cells in a connective tissue stroma; in the reparative granuloma, however, the giant cells are scattered, mitotic figures are rare, and the stromal cells show a cytoplasmic predominance, as opposed to those seen in giant cell tumors, which show a nuclear predominance (2, 12). A brown tumor can also be difficult to differentiate from a giant cell reparative granuloma. Laboratory values are extremely important in the setting of brown tumors in showing elevated serum calcium, alkaline phosphatase, and parathyroid hormone levels and depressed serum phosphate (2, 6). Clinical information is also useful in differentiation. As in our case, giant cell reparative granuloma usually is seen in the first two decades of life. The usual age of onset of giant cell tumors is in the third and fourth decades (2, 12). The course of giant cell reparative granuloma is also relatively benign when compared with giant cell tumor; surgical resection is curative in most cases and recurrence is rare (1, 6, 13).

The radiologic differential diagnosis of an expansile paranasal sinus lesion should include inflammatory lesions, neoplasia, and other fibroosseous lesions. Frontal sinus mucoceles, the most common expansile lesions of the paranasal sinuses, result from obstruction of the frontal sinus ostium. Intracranial extension can be seen. Although the signal intensity of a mucocele can be highly variable on T1- and T2-weighted sequences, the lesion is usually hyperintense on at least one of these sequences or is of very low signal on both sequences (14). The lack of central homogeneous enhancement in a mucocele also aids in differentiating it from a solid tumor. Sinus expansion is typical with sinus polyposis or a polypoid mucocele; however, radiologic demonstration of preservation of sinus septa and a thin zone of mucoid material at the margins of the expanding polyps helps to distinguish this entity from a more solid tumor mass (15). Intracranial extension can also be seen with polyps. MR shows various degrees of lesional hyperintensity on T1- and T2-weighted images and lesional inhomogeneity on MR images before and after contrast administration (15). Sinus infection, most commonly fungal, can be associated with mild sinus expansion; other signs of infection are usually present, including thickened, sclerotic bone and paranasal mucosal thickening (15).

Although aggressive squamous cell carcinomas typically destroy rather than remodel bone, sinonasal sarcomas including lymphomas, sinus nerve sheath tumors, inverting papillomas, extramedullary plasmacytomas, minor salivary gland tumors, esthesioneuroblastomas, melanomas, granulocytic sarcomas (chloromas), and hemangiopericytomas can be associated with bone remodeling and sinus expansion (15). These lesions can be radiographically indistinguishable from giant cell reparative granulomas, with MR imaging features of intermediate intensity on T1- and T2-weighted images and homogeneous contrast enhancement. These lesions, however, tend to occur in the nasal fossa and ethmoidal and maxillary sinuses; a frontal sinus location of these lesions is rare (15).

Other fibroosseous lesions, including fibrous dysplasia and ossifying fibroma, can be associated with sinus expansion. These lesions arise from the medullary space of the wall of the affected sinus, show low signal intensity on both T1- and T2-weighted images, and show inhomogeneous contrast enhancement (16). As opposed to our case of giant cell reparative granuloma, an intact cortical margin is seen in the majority of cases of fibrous dysplasia and ossifying fibroma (16).

The treatment of choice for giant cell reparative granuloma is surgical resection, although radiation therapy has been advocated in inoperable cases (2, 5). Recurrences are rare but have been described; resection is usually performed in these cases (8). Sarcomatous degeneration after treatment of this lesion with radiation therapy has been described, but de novo malignant transformation is not seen (17). Our patient will continue to have MR follow-up studies after total resection of the lesion.

In conclusion, giant cell reparative granuloma should be considered when an expansile lesion is seen within the paranasal sinuses, bony orbit, or calvaria and MR shows isointensity with gray matter typical of a fibrous lesion, especially in a patient in the first two decades of life.

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