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Giant cell reparative granuloma of the concha bullosa

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Abstract
Giant cell reparative granuloma is a benign lesion occurring most commonly in the bones of the jaw and rarely in the paranasal sinuses. We present an unusual case of giant cell reparative granuloma arising within a concha bullosa. Complete excision was achieved using an endoscopic transnasal approach.

Key words: Granuloma, giant cell; Turbinates; Nose

Case report

A 47-year-old Caucasian woman presented with a six-month history of right-sided nasal obstruction and right-sided frontal headache. There was no significant past medical history but the patient recalled having a piece of crayon removed from her right nasal cavity under a general anaesthetic when she was a child. Clinical examination revealed a large swelling arising from the posterior half of the right middle turbinate causing obstruction to the right nasal airway. The rest of the physical examination was normal. Haematological and biochemical profiles were all within normal limits.

CT scan showed bilateral pneumatized middle turbinates (concha bullosa) and a lesion arising from within the posterior half of the right middle turbinate with a slightly high attenuation edge (Figure 1). The appearance was typical of a benign expanding mass.

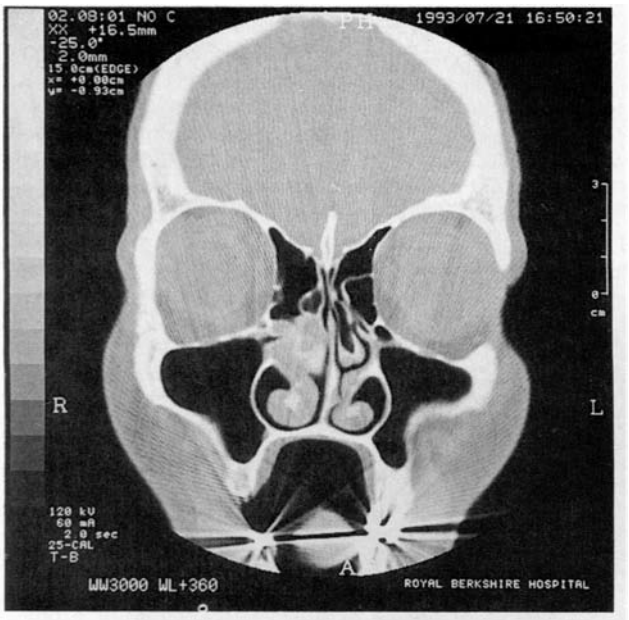

Fig. 1
CT scan demonstrating the giant cell reparative granuloma arising from within the posterior half of the right concha bullosa.

The lesion was excised completely in continuity with the middle turbinate using an endoscopic transnasal approach. The patient was completely relieved of her symptoms with no evidence of recurrence after nine months.

Macroscopically, the lesion measured 2.5 × 1.5 cm with a slightly granular surface (Figure 2). Histology showed areas of increased cellularity within the curetted bony fragments. These foci were composed of cells having ovoid and spindle-shaped nuclei with the appearance of fibroblasts. Admixed were groups of osteoclast-type multinucleated giant cells scattered irregularly through the tumour and associated with areas of both recent and old haemorrhage. Some new bone formation was seen around the periphery (Figures 3 and 4). These features were consistent with a giant cell reparative granuloma (GCRG).

The fibroblast stroma and irregular distribution of giant cells were features against this being a giant cell tumour (GCT). There was no clinical or biochemical evidence to

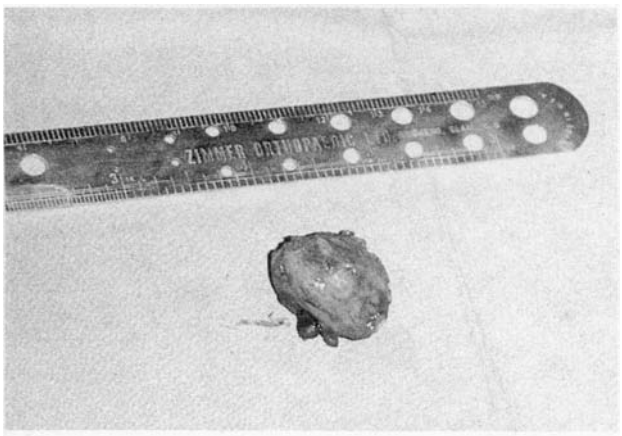

Fig. 2
Macroscopic appearance of the resected lesion.

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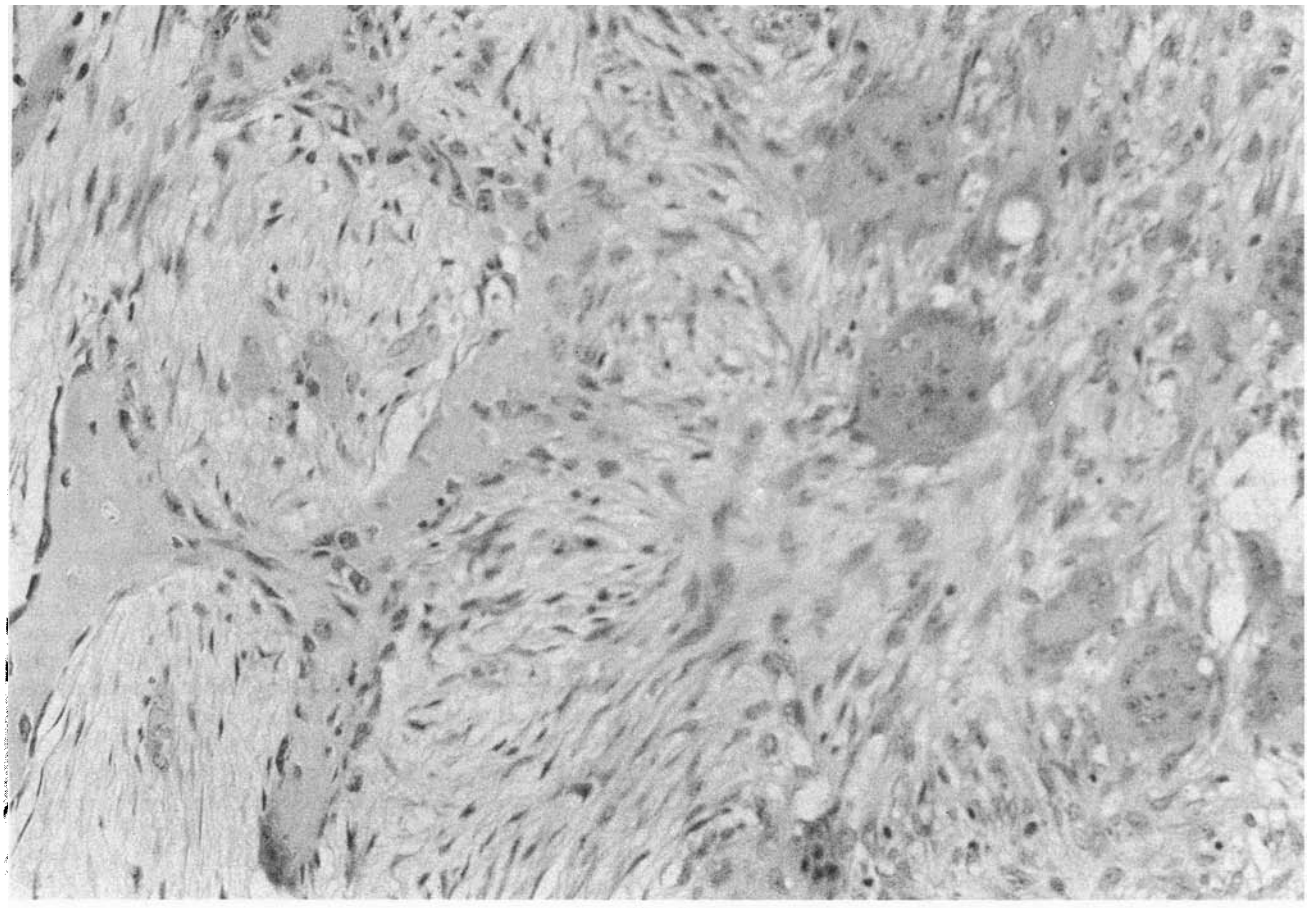

FIG. 3

The tumour is seen to be composed of multinucleated giant cells set in a stroma of spindle cells. Residual bone trabeculae may be seen on one side. (H&E; × 100).

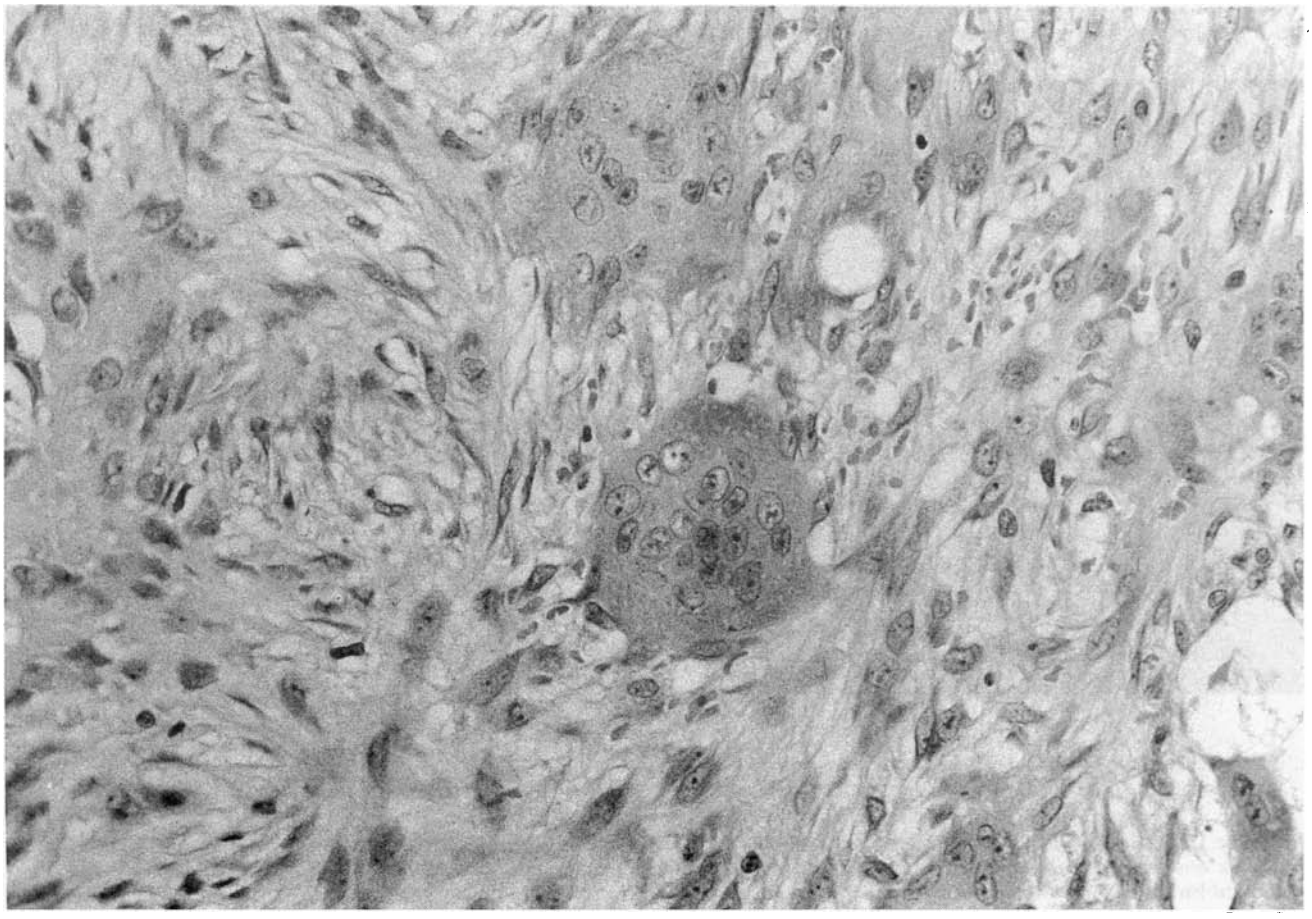

FIG. 4

Enlargement of Figure 3 showing that the multinucleated giant cells are unevenly distributed throughout the tumour. (H&E; × 400).

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suggest this lesion could be due to hyperparathyroidism in which identical histological features might be seen.

Discussion

Giant cell reparative granuloma is a benign lesion which primarily affects the jaw bones. Cases have also been described in the nasal septum (Amin and Samuel, 1990), the skull base (Ciappatta *et al.*, 1990), the temporal bone (Hirschl and Kalz, 1974), the cranial vault (Garza-Mercado *et al.*, 1984) and the paranasal sinuses (Friedberg *et al.*, 1969; Upchurch *et al.*, 1983; Fechner *et al.*, 1984; Weber *et al.*, 1986; Wiatrak *et al.*, 1987; Alappat *et al.*, 1992). Its occurrence outside the craniofacial bone is extremely rare (Oda *et al.*, 1992) and then involves tubular bones of the hands and feet.

Giant cell reparative granuloma of the ethmoidal sinus usually also involves the other adjacent paranasal sinuses and it is often difficult to ascertain the exact site of origin of the lesion. It has a tendency to be locally destructive and may take on an aggressive course despite being a benign disease. Both orbital involvement and intracranial extension are frequent complications (Friedberg *et al.*, 1969; Fechner *et al.*, 1984; Weber *et al.*, 1986; Wiatrak *et al.*, 1987; Alappat *et al.*, 1992). Disease confined to the ethmoidal sinus alone is extremely rare and isolated involvement of the concha bullosa, as in the case described here, has not previously been reported.

Symptoms of ethmoidal GCRG vary depending on the extent and aggressiveness of the lesion. Nasal obstruction, epistaxis, anosmia, facial swelling, headaches, diplopia, proptosis, ophthalmoplegia, blindness and cranial nerve palsy have all been described. The length of symptoms is also quite variable, ranging from a few weeks to a few years. Cases of rapid growth and aggressive behaviour of the lesion during pregnancy have been reported (McGowan, 1969; Geissinger *et al.*, 1970; Fechner *et al.*, 1984).

CT scan findings generally reflect a slowly expansile process with displacement and thinning of bony walls although not infrequently bony destruction and extension through the cribiform plate can also occur. The differential diagnosis on CT scan includes ossifying fibroma, brown tumour of hyperparathyroidism, true giant cell tumour, ossifying fibroma and osteoblastoma (Weber *et al.*, 1986). However, the findings tend to be nonspecific and it can sometimes be impossible to distinguish a GCRG from a malignant lesion or a possible infection. It may also be mistaken for membranous thickening due to density in the sinus with no apparent bone changes (Rhea and Weber, 1983).

Jaffe (1953) first proposed the term 'giant cell reparative granuloma' in an attempt to differentiate it from other giant cell lesions of the jaw bones. He suggested that the term has the advantage of conveying the more precise idea that the lesion is not a neoplasm in the true sense, but represents a local reparative reaction.

Histological differentiation of GCRG from giant cell tumour (GCT) can be difficult. Location of the lesion and its clinical presentation and outcome may play an important role in the final diagnosis. In general, GCRG tends to affect the craniofacial bones, runs a benign course and is usually cured by complete excision. On the other hand, GCT usually affects the epiphysis of the long bones and behaves in a locally aggressive manner with frequent local recurrences and rare distant metastases.

The pathogenesis of GCRG remains controversial. Jaffe (1953) suggested that the lesion represented a local reparative granulomatous reaction to intraosseous haemorrhage caused by trauma which preceded the appearance of the lesion by many years. However, in many cases no previous history of trauma could be elicited. Hirschl and Kalz (1974) therefore proposed that chronic inflammation in the paranasal sinuses in various forms may give rise to local microhaemorrhages which in turn lead to a reactive proliferative process. GCRG also forms a unique clinical entity in association with Paget's disease of the bone (Upchurch *et al.*, 1983).

Concha bullosa is an anatomical variant which occurs in up to 34 per cent of patients with sinusitis (Zinreich *et al.*, 1988). Inflammatory changes within the conchal mucosa may result in conchal sinusitis and conchal mucocele. The location of the conchal ostium near the frontal recess brings it into proximity with the frontal sinus ostium and thus makes it vulnerable to spread of infection from this sinus or *vice versa* (Lidov and Som, 1990). In accordance with the theory proposed by Hirschl and Kalz (1974) the GCRG in our patient may have arisen from inflammatory disease of the concha bullosa although it is also conceivable that the distant history of nasal trauma from crayon removal might have been the initiating factor.

It is generally agreed that the treatment of choice for GCRG is curettage or local excision of the lesion which cures about 80 per cent of the cases. Those that recurred usually require only one additional curettage (Jaffe, 1953; Friedberg *et al.*, 1969; Fechner *et al.*, 1984). In the presence of intracranial extension, a craniofacial resection allows complete resection of the more extensive ethmoidal GCRG (Alappat *et al.*, 1992; Fechner *et al.*, 1984). Although radiotherapy is widely recommended for the nonresectable GCRG, some cases were found to be radioresistant (McGowan, 1969; Wiatrak *et al.*, 1987). Due to the long-term risk of sarcomatous degeneration, it is generally recommended that radiotherapy should be reserved only for those cases not amenable to surgery.

Since GCRG is histologically identical to the brown tumour of hyperparathyroidism, testing for biochemical abnormalities of hyperparathyroidism is essential to establish a final diagnosis before embarking on any definitive treatment. Treatment of the underlying hyperparathyroidism and control of the hypercalcaemia alone will result in gradual healing of the brown tumour and resolution of the local symptoms (Robinson and Woodhead, 1988).

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