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Clinical Records

Giant cell tumour of the temporal bone presenting as vertigo

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Abstract
We report a case of giant cell tumour of the temporal bone arising in a 31-year-old man. The presenting symptoms were unusual, being rotational vertigo, unilateral tinnitus, and hearing loss. A computed tomography (CT) scan showed a large mass within the right temporal bone and the infratemporal fossa. The radiological appearance was suggestive of an aggressive primary neoplasm arising within bone. Biopsy and subsequent resection showed a giant cell tumour of bone. The tumour was histological grade 1. At two-year follow-up, there was no evidence of tumour recurrence or metastasis.

Key words: Temporal bone; Giant cell tumours; Vertigo

Introduction

Giant cell tumour of bone or osteoclastoma is a relatively rare primary neoplasm, constituting approximately five per cent of all primary bone tumours (McGrath, 1972). The tumour is given its name due to the presence of numerous osteoclast-like giant cells. However, it is likely that the giant cells are non-neoplastic and that the mononuclear so-called stromal cells are the basic tumour elements. Giant cell tumours generally occur in patients aged 20 to 55 years and are most common in the third decade of life. Most cases arise *de novo* although occasional giant cell tumours have complicated Paget's disease of bone (Millar *et al.*, 1974). Giant cell tumours usually involve the epiphyseal region of long bones, the sites most commonly affected in order of frequency being the lower end of the femur, the upper end of the tibia and the lower end of the radius. The characteristic radiological appearance is of an entirely lytic, expansile lesion located in the epiphysis, usually without peripheral bone sclerosis or periosteal reaction. Although typical, the changes are not pathognomonic.

Primary involvement of the bones of the skull appears to be uncommon and preferentially involves the sphenoid and temporal bones of the middle cranial fossa (Echols, 1945; Jamieson, 1969; Eeissinger *et al.*, 1970; Gupta *et al.*, 1975; Ohaegbulam and Gupta, 1977; Epstein *et al.*, 1982; Wolfe *et al.*, 1983; Pradhan *et al.*, 1991). These bones arise, as do long bones, through a process of endochondral bone formation. It is speculated that the relative absence of giant cell tumours in other bones of the skull may be related to their genesis in intramembranous bone formation. We report a case of giant cell tumour arising within the temporal bone. We discuss the differential diagnosis and the often difficult histological distinction from other primary bone lesions which may contain abundant osteoclast-like giant cells.

Case history

A 31-year-old man presented initially to the vertigo clinic at the Royal Group of Hospitals, Belfast with a one-year history of rotational vertigo. He also complained of right-sided tinnitus and right-sided hearing loss. Initial examination showed a small red swelling in the attic region of the right tympanic membrane. Cranial nerve examination revealed no abnormality. Audiometric assessment demonstrated a conductive hearing loss of 10 dB in the right ear, with normal thresholds in the left ear. He was

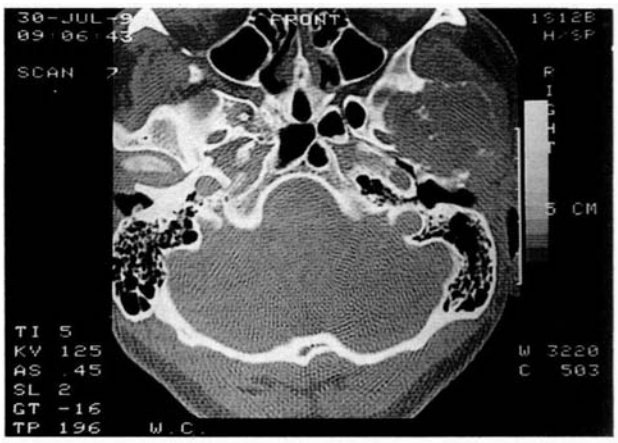

FIG. 1
CT scan showing tumour arising from the right temporal bone.

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admitted eight weeks later for examination under anaesthesia and, by this time, a small painful swelling had developed over the right temporal region, just above the pinna. A computed tomography (CT) scan demonstrated a large, well circumscribed mass arising within the right temporal bone (Figure 1) and extending inferiorly as far as the pterygoid plates in the infratemporal fossa.

Superiorly the mass eroded the base of the skull and posteriorly it impinged on the external auditory meatus. The radiological impression was of an aggressive neoplasm arising primarily within the right temporal bone. Haematological and biochemical blood indices, including bone profile, were normal. There was no clinical or chemical evidence of hyperparathyroidism. Chest X-ray showed no abnormality. Open biopsy was performed and following the histological report, definitive surgery was performed.

The definitive operation was carried out as a joint ENT and neurosurgical procedure. A Fisch type C approach to the right infratemporal fossa was utilized. The right carotid bifurcation was identified and the external carotid artery ligated. The right facial nerve was identified and preserved. Medially dissection was extended as far as the foramen rotundum, which was free of the tumour. The foramen ovale, the lateral wall of which was invaded by tumour, was entered. Radical excision of the temporal bone was performed. A brown coloured tumour mass occupied the whole infratemporal fossa. This was removed piecemeal and was carefully stripped from the dura, to which it was densely adherent. Post-operative radiation therapy was not given.

Following the operation there was an incomplete facial weakness and, as expected, no demonstrable hearing remaining in the right ear. The facial weakness gradually improved leaving a minor residual impairment.

At two-year follow-up there was no clinical or radiological evidence of tumour recurrence or metastasis.

Pathological findings

The open biopsy consisted of small friable fragments of brown coloured tissue. The definitive excision specimen consisted of bone which was distorted and expanded by similar brown tissue.

Histology of the open biopsy and the resection specimen showed similar features. Fragments of normal bone were infiltrated and destroyed by a cellular lesion. Two main components were identified, namely giant cells and stromal cells (Figure 2). The giant cells were numerous and of osteoclast-like type with large numbers of centrally located nuclei and surrounding abundant eosinophilic cytoplasm.

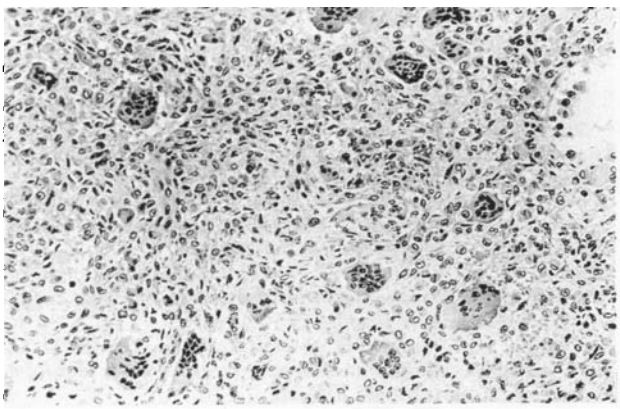

FIG. 2

Cellular tumour composed of a mixture of multinucleate osteoclast-like giant cells and mononuclear stromal cells.

They were evenly distributed and regularly spaced throughout the entire lesion. The other component consisted of ovoid to spindle-shaped stromal cells with a scanty amount of eosinophilic cytoplasm. The stromal cells appeared bland with no evidence of excessive nuclear pleomorphism and few mitotic figures identified. Focal small areas of eosinophilic osteoid were present as well as scattered inflammatory cells and small foci of haemosiderin pigment. No areas of necrosis were identified. The preferred histological diagnosis was of a giant cell tumour of bone. The tumour was classified as grade 1, due to the bland appearance of the stromal cells.

Discussion

The giant cell tumour is generally regarded as an aggressive neoplasm of borderline or low grade malignancy with a marked propensity for local recurrence. Establishing a diagnosis of giant cell tumour of bone is generally straightforward when confronted with the typical clinical picture of a lytic lesion involving the epiphysis of a long bone. Difficulties in diagnosis arise when the tumour arises in an atypical site, as in the present case. Many primary bone lesions may contain abundant osteoclast-like giant cells. Often the histological distinction between these lesions is difficult and may indeed be impossible without knowledge of the exact anatomical site of the lesion and the radiological appearance. Lesions arising primarily in bone which may be rich in osteoclast-like giant cells include reparative giant cell granuloma, cortical fibrous defect and non-ossifying fibroma, ossifying fibroma, fibrous dysplasia, chondromyxoid fibroma, chondroblastoma, eosinophilic granuloma, solitary bone cyst, aneurysmal bone cyst, osteoid osteoma, osteoblastoma, osteitis fibrosa cystica of hyperparathyroidism and the giant cell variant of osteosarcoma. Uncommonly, metastatic carcinoma or sarcoma may contain an abundance of these giant cells. Usually a combination of clinical, radiological and pathological features results in the correct classification of the lesion.

In the present case, the two lesions most considered in the differential diagnosis were giant cell tumour and the so called 'reparative giant cell granuloma'. The latter is considered by many to be a reactive process rather than a true neoplasm (Waldon and Shafer, 1966; Leban *et al.*, 1971) and usually arises within the bones of the jaw, more commonly the mandible than the maxilla. The word reparative is not currently accepted as a suitable descriptive term because of the actual destructive nature of the giant cell granuloma. Both giant cell tumour and giant cell granuloma consist of osteoclast-like giant cells scattered in a background of stromal cells. Qualitatively, the multinucleate giant cells and the mononuclear stromal cells in giant cell tumour are extremely similar to those in giant cell granuloma. Several authors have pointed out that giant cell tumour can histologically simulate giant cell granuloma (Wold and Swee, 1984). It has further been proposed that the two entities are essentially variants of the same disease process (Waldon and Shafer, 1966; Auclair *et al.*, 1988) modified by the age of the patient and the site of occurrence. Be that as it may, frequently described histological differences between giant cell tumour and giant cell granuloma are: (1) the larger more rounded giant cells with a greater number of nuclei in the giant cell tumour; (2) the much more common occurrence of fresh haemorrhage and haemosiderin deposits in the giant cell granuloma; (3) the more uniform dispersal of giant cells in the giant cell tumour; (4) the more frequent production of osteoid or new bone in the giant cell granuloma; (5) the more frequent inflammatory compo-

nent in the giant cell granuloma; (6) the greater tendency for the nuclei to aggregate centrally in the giant cells of giant cell tumour; (7) the presence of foci of necrosis in the giant cell tumour. However these purported differences have been questioned and many believe there is considerable overlap between the two lesions (Leban *et al.*, 1971). One feature in the present case pointing to a diagnosis of giant cell tumour was the relatively even distribution of giant cells throughout the entire lesion. This is in contrast to giant cell granuloma where the giant cells tend to be unevenly dispersed and aggregated around areas of haemorrhage. In addition, the radiological appearance in the present case was suggestive of an aggressive tumour. The radiological appearance of a giant cell granuloma is of a lytic area which only rarely expands the bone.

After establishing a diagnosis of giant cell tumour, an attempt should be made to histologically grade the lesion. Grading is performed with regard to the stromal component of the tumour. Jaffe *et al.* (1940) first emphasized the importance of grading of giant cell tumours. They noted the degree of malignancy increased as stromal cells became more prominent, with increasing nuclear pleomorphism and increased numbers of mitotic figures. They divided giant cell tumours into grades 1, 2 and 3 with grade 1 tumours having a bland stromal component and grade 3 lesions containing an obviously sarcomatous stroma. It is imperative that these tumours be thoroughly sampled with multiple histological sections examined to ensure that the most malignant areas are identified. However, the value of grading has been questioned and many no longer regard grading as valuable in predicting the behaviour of these tumours. Indeed, all giant cell tumours should be regarded as potentially malignant in view of the fact that as many as 30 to 50 per cent recur after currettage and five to 10 per cent give rise to distant metastases. The most common site of metastasis, by far, is the lungs. The type of initial surgical removal is the most significant factor in determining the recurrence rate: in one large series, the recurrence rate was 34 per cent following currettage and seven per cent following wide resection (McDonald *et al.*, 1986). The currently preferred treatment is thorough curettage with bone grafting, or en bloc excision with replacement with allograft or artificial material, depending on the location of the tumour. Special care should be taken to prevent implantation of tumour into the adjoining soft tissue. The value of post-operative radiation therapy in the management of these tumours is controversial. Some believe that radiation therapy should be reserved only for cases in which complete surgical removal is impossible, in view of the risk of malignant sarcomatous transformation following this therapeutic modality (Dahlin *et al.*, 1970; Goldenberg *et al.*, 1970). A single case of post-irradiation fibrosarcoma has complicated a previously diagnosed giant cell tumour of the sphenoid bone (Martins and Dean, 1974). Others believe that because of the seeming inevitability of recurrence with an incomplete resection and because of the apparent safety and effectiveness of modern radiotherapeutic techniques, optimal therapy in the case of a tumour involving the bones of the skull consists of radical resection followed by carefully planned and delivered irradiation (Findlay *et al.*, 1987).

As stated earlier giant cell tumour involving the bones of the skull is relatively uncommon. A correct diagnosis is rarely made pre-operatively. The most commonly affected site appears to be the sphenoid bone. Wolfe *et al.* (1983) reported 10 cases of giant cell tumour of the sphenoid bone. The presenting symptoms were varied and included headache, visual field defects, blindness and diplopia. Several single case reports of giant cell tumour of the temporal bone have also appeared in the literature (Jamieson, 1969; Epstein *et al.*, 1982; Findlay *et al.*, 1987; Pradhan *et al.*, 1991). These have presented with a combination of symptoms, mainly pain, deafness and facial weakness. The hearing loss is often conductive, as in the present case, and is most likely a result of the propensity of these tumours to invade the infratemporal fossa and obstruct the eustachian tube. The patient in the present case had a more unusual presentation. His chief complaint was of rotational vertigo and he initially presented to a vertigo clinic. Vertigo has been mentioned as a presenting complaint in only one reported case of giant cell tumour of the temporal bone (Pradhan *et al.*, 1991). Presumably this symptomatology was due to local tumour activity in the temporal bone although we cannot exclude coincidental pathology in the labyrinth. The development of post-operative facial weakness in the present case in spite of attempts to isolate and preserve the facial nerve was disappointing. However, recovery was good leaving only a minimal residual weakness. Several of the previous reported cases of giant cell tumour of the temporal bone have demonstrated facial nerve weakness at presentation (Epstein *et al.*, 1982; Findlay *et al.*, 1987).

Two-year follow-up in the present case revealed no evidence of tumour recurrence or metastasis. However, long-term follow-up is clearly indicated in view of the fact that tumour recurrence or metastasis has been reported many years following initial surgical removal (Dahlin *et al.*, 1970; Goldenberg *et al.*, 1970).

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