Temporosphenoid Giant Cell Tumour

Vikas Inder Nath Nehru, Assistant Professor
Jaimanti K, Senior Resident
Postgraduate Institute of Medical Education and Research,
Chandigarh 160 012

Abstract

Only a few cases of Giant Cell tumours of temporal and sphenoid bones have been documented in English literature. Concurrent temporosphenoid location is extremely rare for a Giant Cell Tumour. These locally aggressive bonedestructive tumours usually have a fairly predictable pattern of spread. We present here a case of Temporosphenoid Giant Cell Tumour that exhibited an unusual pattern of spread. This tumour was removed in a single stage without any untoward sequelae. A brief review of literature is included.

Key Words

Giant Cell Tumor Temporosphenoid

INTRODUCTION

Representing 3.7% of all primary neoplasms of bone, Giant cell tumors or Osteoclastomas of bone commonly involve epiphyses of long bones. Only 2% of them occur in the Head and Neck. Cranial Giant Cell Tumours usually involve sphenoid, ethmoid and temporal bones independently. A concurrent temporosphenoid Giant Cell Tumour is perhaps extremely uncommon as we could find mention of only five cases in English literature prior to this report. It is a locally aggressive tumour and destroys bone indiscriminately. At present surgery appears to be the most effective therapeutic modality. In the past local recurrences have been reported but the modern developments in surgery of the cranial base may alter the scenario.

Case Report

An otherwise healthy 40 years old man was admitted under ENT Service who complained of steadily progressive hearing loss in left ear since 5 years, whistling non-pulsatile tinnitus since 2 years and an increasing fullness in front of

his left ear for previous six months but it was a discharging mass lesion in the external auditory canal of recent onset that urged him to seek medical help. His general physical examination, baseline parameters, serum biochemistry and endocrine evaluation were normal. Local examination showed a diffuse subcutaneous fullness in the preauricular and temporal area which was firm and nontender. Pinna was normal. There was a pinkish firm polypoidal mass completely filling the external auditory canal that bled on touch. Fistula test was negative and facial nerve was intact. Pure Tone Audiometry showed 50 dB conductive hearing loss on left side. Rest of the examination was normal. Contrast Enhanced High Resolution CT of the temporal bone showed a moderately enhancing soft tissue shadow obliterating the external auditory canal with erosion of anterior and posterior bony walls and scutum. The mastoid air cell system was partly invaded (Fig.1). The squamous temporal was destroyed anterosuperior to linea temporalis (Fig. 2). The Great Wing of Sphenoid was destroyed with intracranial extension of the le-

Fig 1 Axial CT temporal bone showing a mass in EAC with destruction of anterior and posterior walls. Note air space lateral to promontory

sion. There was a large smooth walled extension of the lesion in the Infratemporal Fossa with evidence of intratumoral tissue breakdown (Fig.3).

A biopsy taken from the external auditory canal revealed a Giant Cell Tumour. Patient was

Fig 2 Coronal CT temporal bone showing destruction of squamous part of temporal bone.

operated via a large C shaped postaural incision similar to the one employed for Type A Infratemporal Fossa Approach. A soft reddish brown tumour was seen in the bony canal with destruction of both anterior and posterior canal walls. The tympanic membrane was de-

Fig 3 Coronal CT temporal bone showing destruction of Great wing of Sphenoid and mass in Middle Cranial Fossa and Infratemporal Fossa

Fig 4 Microphotograph showing regularly placed Multinucleate Giant Cells in a stroma of diffuse sheets of plump pvoid cells (H&E staining 40x)

stroyed. The tumour was also seen in the mesotympanum surrounding an intact Malleus, epitympanum, aditus and antrum. Incus was absent. The stapes superstructure was intact, the tumour having miraculously stopped short of it with actually an airspace, albeit small, lateral to the promontory. The mastoid air cells were only slightly invaded. A 3 cm. area of squamous temporal bone anterosuperior to linea temporalis and adjacent floor of middle cranial fossa was destroyed. The dura was exposed but not invaded. From here the tumour spread anteriorly destroying root of zygoma. More anteriorly, lateral aspect and undersurface of Great Wing of Sphenoid were destroyed up to Foramen Ovale. This allowed an upward extradural extension of the tumour into the middle cranial fossa as well as a downward extension of a large soft tissue component of the tumour which had acquired a pseudocapsule around it and had a central cystic degeneration. The maxilla, pterygoid plates and the muscles in the infratemporal fossa were not invaded. A piecemeal but complete excision of the tumour was performed extracranially exploiting the space afforded by the destroyed bone itself.

The cartilaginous external auditory canal was oversewn for permanent blind sac closure. Eustachian tube was closed off with a muscle plug and bone chips. The mastoid cavity and the extradural dead space were obliterated by rotating a pedicled temporalis muscle flap and the wound closed.

Postoperatively the patient had mild giddiness for a week. He had a permanent 60 dB conductive hearing loss but no cranial nerve palsy. There was no other morbidity and the patient left the hospital on tenth postoperative day. A histopathological examination of the specimen showed multinucleate Giant Cells (Fig. 4) in a stroma comprising diffuse sheets of plump ovoid cells (Fig. 5). It was reported as Giant Cell Tumour.

Discussion

There is an ongoing controversy about an appropriate system of classification for Giant Cell lesions, which may, in part, be due to their infrequent occurrence as well as similarities in histological characteristics. Simth and Ward (1978) subdivided Giant Cell lesions into three groups viz., Brown Tumour of Hyperparathyroidism which may be diagnosed on metabolic, endocrine and renal evaluation; Giant Cell Granuloma which occur in the alveolus and the palate, particularly in the young and the true Giant Cell Tumours.

Giant Cell Tumours comprises 3.7% of all bony tumours and 90% of these involve ends of bones. The tumour is thought to arise from the stroma of the bone marrow of cartilaginous and membranous bones which has an osteogenic potential. Until 1994 only 88 cases of cranial Giant Cell tumours had been reported of which 43% arose from the sphenoid bone and 23% from the temporal bone (Rock et al, 1994). Subsequently sporadic cases were reported by Nemoto (1995) and Buter and Chilla (1997). However a concurrent location in the temporosphenoid region has been reported extremely rarely. Rock et al in 1994 mentioned only two cases as having been reported until then and described a third case of their own. Mc Cluggage et al reported another case in 1995.

Giant Cell tumour occurs in third and fourth decades affecting women more frequently than men. Most common presentations include headache and cranial nerve palsies.

Headache is due to dural stretch at the floor of middle cranial fossa. Compression of Cavernous Sinus may result in cranial neuropathy in Sphenoidal tumours. In these cases deafness can result from obstruction of the eustachian tube in the infratemporal fossa due to destruction of the Great Wing of Sphenoid. Temporal bone tumours may present with deafness and facial weakness.

Grossly these tumours are soft and reddish brown due to intratumoural haemorrhage. Histologically they consist of multinucleate Giant Cells interspersed in a stroma of ovoid or spindle shaped cells. Sarcomatous features occurring hand in hand with Giant Cell Tumours have been described (Barnes and Verbin 1983). They cause extensive bone destruction as is illustrated by the case of our patient in whom the local spread pursued an unusual course.

Hunt and Glasscock (1974) described combined intracranial and extracranial access to these tumours chiefly for dural protection. In our

case, however, a cautious piecemeal approach succeeded as the extensively destroyed bone itself allowed sufficient room for manoeuvre. The risks of late-onset sarcomatous changes in the event of irradiating cranial base are too well known although radiotherapy has been mentioned in the past as a therapeutic alternative. However, recent strides made in accomplishing surgery of the cranial base should not only make a wide field excision the treatment of choice but also diminish risk of local recurrence reported in the past.

We have not seen recurrence 11 months after surgery in our case.

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