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Tegmental and petromastoid defects in the temporal bone

by

T. R. KAPUR (Stoke on Trent) and WAJAHAT BANGASH (Islamabad, Pakistan)

Introduction

Spontaneous communications between the middle ear, mastoid and the intracranial cavities giving rise to symptoms are rare. In the majority of reported cases, there were communications between a congenitally abnormal labyrinth and the middle ear (MacNab Jones and Fairburn, 1977; Bottema, 1975; Gundersen and Haye, 1970; Gacek and Leipzig, 1979). Some cases have been reported with defects in the footplate of the stapes (Bennett, 1966; Stroud and Calcaterra, 1970; Nenzelius, 1959; Harris, 1978). Lastly, a few cases have been reported with defects in the tegmen (Kline, 1933; Dysart, 1959; Ahren and Thulin, 1965; Neely *et al.*, 1982).

One of us has dealt with two such cases of spontaneous CSF otorrhoea due to tegmental and mastoid defects and this prompted us to investigate the incidence of such defects. This may have an important bearing on the intracranial spread of middle ear infection and spontaneous CSF otorrhoea and rhinorrhoea.

Materials and methods

Fifty temporal bones were examined in 25 cadavers. There were 15 females and 10 males. The skull was cut horizontally about an inch above the supraorbital margin. The pathologists removed the brain, leaving the dura over the base of the skull untouched. The ears were examined to exclude any cases with chronic middle ear disease and cholesteatoma. There were no cases of metastases in the bones examined. A Wild-Heerbrugg microscope for temporal bone dissection was used with some difficulty as the examination was carried out *in situ*. The dura covering the tegmen and petromastoid segments was examined and the two layers of the dura were very careully dissected off the tegmen and petromastoid parts of the temporal bone. Details of any defects were noted for their size, shape and numbers (Table I). It was possible to visualize clearly any bony defects and the thin mucosa of the middle ear cleft was immediately recognized. The defects were confirmed by passing a fine probe into the mastoid air cells or the middle ear.

Results

The size and shape of the defects varied. They were round, oval, triangular or cruciate.

17 bones had defects (34 per cent) (Table II).

6 of the subjects had bilateral defects (24 per cent).

Deficiencies of the tegmen

10 bones were found to have defects in the tegmen (20 per cent).

9 bones had defects in the tegmen tympani.

1 bone had defect in the tegmen antri.

3 subjects had bilateral defects (12 per cent).

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TABLE I
FINDINGS IN 50 TEMPORAL BONES

		Right					Left				
	Sex	Petrous Surface	Tegmen	Post Surface	Size	No.	Petrous Surface	Tegmen	Post Surface	Size	No.
1	F	–	+	–	2 mm. diam.	1	+	–	–	1×1 mm.	1
2	F	–	–	+	3×3 mm.	1	–	–	–	—	—
3	F	V. Cellular	–	–	—	—	V. Cellular	–	–	—	—
4	M	–	+	–	5×5×5mm.	1	–	+	–	2×3 mm:1×2 mm	2
5	F	–	–	–	—	—	–	–	–	—	—
6	F	–	+	–	2×3 mm.	1	–	+	–	2×3 mm.	1
7	F	–	–	–	—	—	–	–	–	—	—
8	F	–	–	–	—	—	–	+	–	2 mm. diam.	1
9	M	Cellular	–	–	—	—	V. Cellular	–	–	—	—
10	F	Cellular	–	–	—	—	Cellular	–	–	—	—
11	M	–	–	–	—	—	–	–	–	—	—
12	M	+	–	–	2 mm. diam.	1	+	–	–	2×1 mm.	1
13	M	+	–	–	1 mm. diam.	1	Cellular	–	–	—	—
14	M	Cellular	–	–	—	—	Cellular	–	–	—	—
15	F	–	–	–	—	—	–	–	–	—	—
16	F	+	–	–	10mm. diam.	1	+	–	–	3 mm. diam.	1
17	M	–	–	–	—	—	–	–	–	—	—
18	F	–	+	–	2 mm. diam.	2	–	–	–	—	—
19	F	V. Cellular	–	–	—	—	V. Cellular	–	–	—	—
20	F	–	–	–	—	—	–	–	–	—	—
21	F	–	–	–	—	—	–	–	–	—	—
22	M	–	+	–	2×2 mm.	1	–	–	–	—	—
23	F	–	–	–	—	—	–	–	–	—	—
24	M	Cellular	–	–	—	—	Cellular	–	–	—	—
25	M	–	+	–	1×1 mm.	1	–	+	–	1×2 mm.	1

Deficiencies of posterior surface of petrous temporal bones

Only one bone had a 2 mm. defect near the fovea. The whole of the temporal bone was removed and was taken to the temporal bone laboratory for dissection. This opening led through a sinuous tract to an internal opening below the promontory—(Hyrtl's Fissure).

Defects of the anterior surface including the petrous apex

Six temporal bones had obvious defects on the anterior surface of the petrous temporal bones (12 per cent). (Extensive pneumatization was not present.) Through some of these defects the superior semicircular canals could be seen.

Pneumatization of the petrous apex

Thirteen temporal bones had cells extending to the petrous apex (26 per cent). 5 of these had extensive pneumatization (10 per cent). In these cases the tegmen plate was so thin that the head of the malleus could be seen through it.

Dural defects

None of the temporal bones had any dural defects.

TABLE II
RESULTS IN 50 TEMPORAL BONES

Defects	Numbers	Percentage
None	33	66
Tegmen	10	20
Posterior petrous surface	1	2
Anterior petrous surface	6	12

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Discussion

Tonybee thought that 5–6 per cent of subjects had congenital dehiscences of the tegmen (Ballance). What has come as a surprise as a result of the investigation, is the significant proportion of defects in the tegmen and petromastoid (34 per cent). It was noteworthy that we found that 20 per cent of the defects were bilateral.

Ahren and Thulin (1965) in their investigations of temporal bone defects reported 21 per cent as such defects.

Lang (1983) found similar defects in 20 per cent of temporal bones examined.

The clinical significance of the findings assume great importance in the intracranial spread of infections from the middle ear and mastoid even in the absence of any bony destruction due to cholesteatoma. This route offers direct access to the extradural space and may be an important cause of extra-dural abscess, particularly in Gradenigo's Syndrome, which occurs only in temporal bones with extensive pneumatization reaching the petrous apex.

Although a high proportion of temporal bones were found to have defects, no defects were found in the overlying layers of the dura.

The ages of the patients varied between 41 and 88 years, with an average of 64 years. The question is whether these defects had been present throughout their life or whether, with increasing age, bone absorption had occurred and resulted in defects.

It is exceedingly difficult to find these defects at the time of operation, possibly due to a number of factors: firstly, the defects are relatively small and communicate with the peripheral air cells and are therefore not visible; secondly, the covering vascular mucosa will almost certainly hide the defects. In contrast, the complete avascularity of the field in the cadaver is an important factor in the easy identification of these defects.

Summary

Fifty temporal bones were examined using the temporal bone dissecting microscope. 34 per cent were found to have defects in the tegmen and petromastoid segments, resulting in communications between the cranial cavity and the middle ear cleft. However, no defects were found in the overlying dura.

This may have an important bearing on the intracranial spread of infection from the middle ear cleft, even in the absence of any bony destruction due to chronic middle ear disease.

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Addresses for correspondence:
T. R. Kapur,
Consultant ENT Surgeon,
North Staffs Royal Infirmary,
Princes Road,
Hartshill,
Stoke-on-Trent
ST11 7LN

W. Bangash,
Consultant ENT Surgeon,
Post Graduate Medical Institute,
Islamabad,
Pakistan.

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