Mycotic aneurysm of the petrous portion of the internal carotid artery

J. SAMUEL*, M.D. and C. M. C. FERNANDES*, F.C.S. (Otol) (Durban, South Africa)

Abstract
A case of a mycotic aneurysm of the petrous portion of the internal carotid artery is presented. This entity is extremely rare and the problems associated with the diagnosis and clinical management are discussed.

Introduction
Mycotic aneurysms of the petrous portion are extremely uncommon and only five cases have been recorded in the literature.

The term “mycotic aneurysm” is used for the description of an inflammatory destruction of the arterial wall. This can be due to embolisation from bacterial endocarditis, direct extension from the adjacent infection, or a primary mycotic aneurysm in the absence of obvious infection (Karsner, 1947).

Case report
A 40-year-old black female presented with a history of purulent discharge from the right ear since childhood and deafness

FIG. 1
A-P tomographic view of the mastoid shows the destruction of the (R) hypotympanum and cochlea compared with the normal left side.

* From Department of Otorhinolaryngology, University of Natal, Durban, South Africa.
FIG. 2

Computerised tomographic scan shows a large defect in the region of the right jugular foramen.

FIG. 3

The right carotid angiogram shows the aneurysm of the internal carotid artery in the petrous portion.
Coronal section of the petrous bone illustrating the erosion caused by the aneurysm of the internal carotid artery (continuous line) and the granulation (dotted area) filling the tympanic cavity.

for many years. No history of tinnitus or dizziness was elicited. On examination the right ear showed a total perforation with granulation tissue filling the tympanic cavity. There was no pulsation and no suggestion of an underlying tumour.

The audiogram showed a severe sensorineural hearing loss in the right ear and normal hearing on the left. Radiographic examination of the mastoid showed opacification of the mastoid cells in the right side, with evident destruction and a diagnosis of possible cholesteatoma was made. The patient was planned for a mastoid exploration.

At operation, the superficial granulation tissue was removed and revealed deeper destruction of the promontory and the cochlea. A pulsating mass was encountered in the depths of the operation cavity. The operation was interrupted and the patient was further investigated to diagnose the nature of the vascular tumour.

Histological examination showed the presence of a chronic inflammatory infiltrate.

Tomography and a CT scan showed a large defect in the region of the jugular foramen, compatible with a glomus tumour (Figs. 1 and 2). A right carotid angiogram and jugular venogram were performed and showed an aneurysm of the internal carotid artery in the petrous portion (Fig. 3). The established diagnosis was mycotic aneurysm.

The patient was planned for a second operation. Granulation tissue was stripped from the aneurysm through a tympanic approach (Fig. 4) and the cavity was filled with temporalis muscle and packed with B.I.P.P. The latter was removed after 10 days. The patient was given intravenous ampicillin for two weeks.

Eight months later, the patient was well and no pulsation could be seen in the tympanic cavity and the cavity was clean and dry. The patient would not agree to undergo a repeat angiogram.

Discussion

Although mycotic aneurysms of the internal carotid artery are occasionally seen with a reported incidence of 2.5 to 4.5 per cent (Suwanwela et al., 1972), those affecting the petrous portion of the temporal bone are extremely rare and only five cases have been recorded in a review of the literature of the last 30 years.

Mycotic aneurysms are classified according to the pre-existing arterial disease (arteriosclerosis, aneurysm or arterial prosthesis) or to the source of infection which may be intravascular (embolism, septicaemia) or extravascular sources (adjacent infection) (Patel and Johnstone, 1977). The cases summarised from the literature are of an extravascular origin, chronic otitis media with granulation being the source of infection.

Most of the supraclinoid mycotic aneurysms are of an embolic type, as compared with the intracavernous and petrous portions which are of an extravascular type. The adjacent bacterial infection invades the arterial wall, the adventitia being the first layer to be involved. Thrombosis of the artery then follows and the toxins from the inflammatory process thin and weaken the wall, resulting in a mycotic aneurysm.

### TABLE I

<table>
<thead>
<tr>
<th>Author</th>
<th>Sex/Age</th>
<th>Clinical features</th>
<th>C.N. Involvement</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barrett and Lawrence, (1959)</td>
<td>M/26</td>
<td>Chr. otitis media since childhood</td>
<td>None</td>
<td>Ligation of common carotid artery and packing the ear</td>
<td>Alive and well.</td>
</tr>
<tr>
<td>Hiranandani et al., (1962)</td>
<td>M/40</td>
<td>Otitis media for 6 years. Bleeding from (R) ear</td>
<td>5, 7, 8</td>
<td>None</td>
<td>Died</td>
</tr>
<tr>
<td>Cook and Fernandes, (1983)</td>
<td>F/50</td>
<td>Discharge, deafness R ear for 6/12</td>
<td>3, 5, 7, 8</td>
<td>Dott clamp of internal carotid artery</td>
<td>Alive and well.</td>
</tr>
<tr>
<td>M/56</td>
<td></td>
<td>Chr. otitis media since childhood, deafness and pulsatile tinnitus. P.T.B.</td>
<td>8</td>
<td>Ligation of carotid artery</td>
<td>Died</td>
</tr>
<tr>
<td>Present case</td>
<td>F/40</td>
<td>Discharging (R) ear since childhood and deafness</td>
<td>8</td>
<td>Temporalis muscle packing</td>
<td>Alive and well.</td>
</tr>
</tbody>
</table>

P.T.B. = Pulmonary tuberculosis.
tions from the pharynx, sphenoid sinus and cavernous sinus thrombosis have been reported as sources of mycotic aneurysm (Suwanwela et al., 1972).

To our knowledge, only 37 cases of aneurysm of the petrous portion have been reported (Cook and Fernandes, 1983; Brandt et al., 1986), five of which were mycotic aneurysms (Barrett and Lawrence, 1960, Hiranandani et al., 1962; Stallings and McCabe, 1969; Cook & Fernandes, 1983), (Table I).

The clinical presentation of a mycotic aneurysm includes long-term discharge from the ear, pulsatile tinnitus and deafness; bleeding from the ear can occasionally occur. With lateral erosion of the arterial wall, the neurological deficit is not extensive and if it occurs, the VIIth and VIIIth cranial nerves are those most commonly involved. A medial extension of the aneurysm may often involve the cranial nerves with or without Horner’s syndrome.

Only two out of five patients were found to have a neurological deficit, consisting of IIIrd, Vth, VIIth and VIIIth cranial nerve palsy. The proximity of the artery to the IIIrd and Vth cranial nerve explains their involvement. Loss of cochannel function occurred in all cases.

The most commonly accepted theory (Brandt et al., 1986), is that the lesion is secondary to a congenital aneurysm which thinned or destroyed the wall of the petrous bone by bone resorption. The longstanding infection of the middle ear was the additional causative factor. The other possible theory is that the local chronic infection weakened the arterial wall, leading to the formation of the aneurysm.

The differential diagnosis of a pulsating mass or pulsating tinnitus would include glomus jugulare, glomus tympanicus, prominent jugular bulb, persistent stapedial artery, carotid aneurysm, aberrant internal carotid artery of normal morphology.

The initial radiological investigations are lateral mastoid, Towne’s and base of skull views in which the destruction of the petrous bone can be easily identified. A-P polytomography at the cochlear and vestibular level will show erosion of the floor of the tympanic cavity and carotid canal. The definitive diagnostic procedure is carotid arteriography.

Various methods of treatment for mycotic aneurysm of the petrous portion have been described, ligation of the internal carotid being the most common and this was used in three patients. Temporalis fascia laid over the aneurysm was used in one case (Stallings and McCabe, 1969) with a good result. The treatment preferred in our case was stripping the granulations from the aneurysm and packing the lateral wall of the aneurysm with temporalis muscle. Intravenous ampicillin was administered for two weeks.

In our opinion, removing the inflammatory causative factor and strengthening the weakened wall with muscle was the simplest and least debilitating procedure.

One month after the operation, the patient is well and no pulsation can be seen in the tympanic cavity.

Antibiotic therapy was successfully used in three patients with a mycotic aneurysm of the intracavernous portion of the carotid artery (Suwanwela et al., 1972; Adeloye et al., 1973).

We have had experience of two previous mycotic aneurysms (Cook and Fernandes, 1983), which were handled by carotid artery ligation. Ligation in these two cases was essential because both patients presented with episodic bleeding from the ear and there was a very real danger of an exsanguinating haemorrhage. However, one of the two patients developed a hemiplegia and died after ligation.

In the patient presented in this paper, the arterial wall was clearly exposed and there did not appear to be any danger of imminent rupture. We felt that a muscle pack to strengthen the vessel wall would be the safest treatment because of the dangers of carotid ligation and because of the experience of Stallings and McCabe (1969) with this technique.

**References**


Address for correspondence:

J. Samuel, M.D.,
Dept. of Otorhinolaryngology,
P.O. Box 17039,
Congella,
4013 South Africa.