The Journal of Laryngology & Otology
February 2004, Vol. **118,** pp. 165–166

Radiology in Focus

Pulsatile tinnitus as a rare presenting symptom of residual cholesteatoma

M. Falcioni, M.D., A. Taibah, M.D., Rohit, M.S.

Abstract
Pulsatile tinnitus is an uncommon otological symptom, which often presents a diagnostic and management dilemma to the otolaryngologist. This symptom always deserves a thorough evaluation to avoid disastrous consequences from potentially life-threatening associated pathology. In most of the patients a treatable underlying aetiology can be identified. Frequent causes mentioned in the literature responsible for pulsatile tinnitus are benign intracranial hypertension syndrome, temporal bone paragangliomas and arteriovenous fistulae. Pulsatile tinnitus as a consequence of sigmoid sinus compression by a cholesteatoma has not been reported previously in the literature. Here a case of residual cholesteatoma with pulsatile tinnitus is presented, nine years after the first surgery.

Key words: Tinnitus, Cholesteatoma

Introduction

Pulsatile tinnitus may be caused by various aetiologies.[1] Venous causes of pulsatile tinnitus include a jugular megabulb,[2] dural sinuses stenosis[3] and other abnormal venous anomalies.[4] In this paper a case of residual cholesteatoma that presented with pulsatile tinnitus nine years after the surgery is reported. The cause of the pulsatile tinnitus was compression of sigmoid sinus by the residual cholesteatoma. To the best of our knowledge this cause has not been reported in the literature previously.

Case report

A 23-year-old male underwent a left canal wall down mastoidectomy in 1990 for an epitympanic cholesteatoma with a large erosion of the posterior canal wall. After the operation a 35 dB hearing threshold was preserved and the cavity remained free of disease for two years, after that the patient was lost for follow-up. In 1999 the patient was again referred to us by a radiologist with a high resolution computed tomography scan (HRCT) of the temporal bone showing cholesteatoma in the ipsilateral mastoid. At the time of examination, the patient's main complaint was pulsatile tinnitus on the operated side especially in the supine position. It was intermittent in the beginning, but now has become continuous. On otoscopy the cavity was perfectly skin-lined, without any sign of recurrence of cholesteatoma or inflammation. Under the microscope a swelling was seen in the postero-inferior portion of the external auditory canal (EAC). On palpation the swelling was soft and non-tender. The HRCT scan showed a lytic lesion at the level of the mastoid tip, with sharp bony margins. On its medial aspect the lesion was in contact with the sigmoid sinus and posterior fossa dura (Figure 1 and 2). The lesion also appeared to be in contact with the skin of the EAC, so producing the bulging which was visible on microscopy (Figure 1). The mass was also in close proximity with the mastoid portion of the facial nerve (Figure 3). The radiological images were strongly sugges-

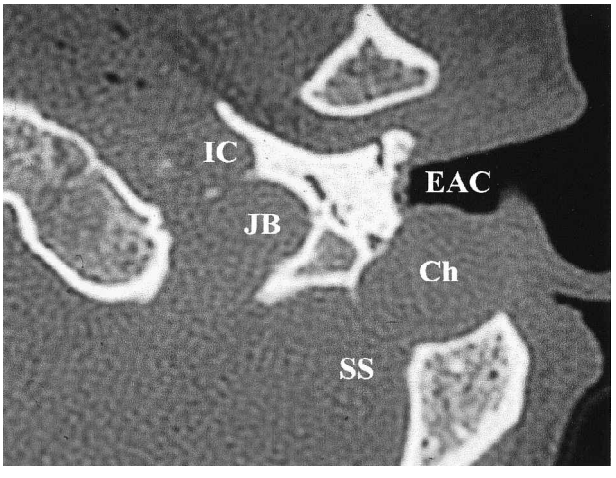

Fig. 1
CT scan (axial plane): A lytic lesion is present at the level of the mastoid tip. The bony shell of the sigmoid sinus is eroded and the lateral surface of the sinus medially dislodged. The lateral extension of the lesion produces a moderate bulging of the posterior wall of the meatoplasty into the cavity. Ch: cholesteatoma; SS: sigmoid sinus; JB: jugular bulb; EAC: external auditory canal; IC: internal cartoid artery.

From the Gruppo Otologico (Piacenza – Rome), 29100 Piacenza, Italy.
Accepted for publication: 14 November 2003.

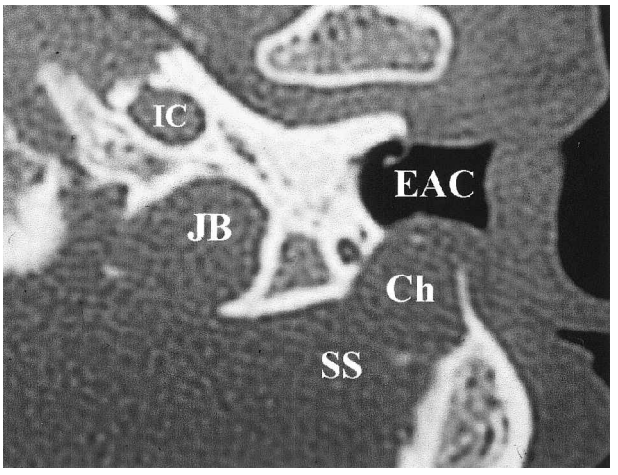

FIG. 2

CT scan (axial plane): Showing sharp bony margins at the mastoid tip, due to erosion of the cholesteatoma. Ch: cholesteatoma; SS: sigmoid sinus; JB: jugular bulb; EAC: External auditory canal; IC: internal carotid artery.

- **Pulsatile tinnitus is associated with a number of conditions including arteriovenous fistulae, paragangliomas and benign intracranial hypertension**
- **In this case the symptom was associated with residual cholesteatoma discovered nine years following an initial surgical exploration**

tive for a residual cholesteatoma and the patient was scheduled for revision surgery. On October 26, 1999, he underwent an exploration of the previous cavity through a retroauricular incision. The skin lining of the posterior portion of the cavity was carefully elevated as a single piece, in order to keep the surgical field separated from the mastoid cavity. At the level of the mastoid tip a large cholesteatomatous cyst was identified. Once the contents of the cyst were evacuated the blue tinge of the sigmoid sinus appeared through the cholesteatoma matrix. After careful removal of the matrix, a large exposed area of the sigmoid sinus and posterior fossa dura, the whole digastric ridge and a small adjacent segment of the mastoid portion of the facial nerve were clearly visible and bone over these structures was eroded by the disease process. In order to reduce the risk of recurrence the bony margins were saucerized with a diamond drill and the exposed part of the sigmoid sinus and the posterior fossa dura were cauterized by means of bipolar forceps.[5] The surgical defect was obliterated with abdominal fat, the skin repositioned and the original radical cavity packed with Spongostan® (Johnson & Johnson, Gargrave, Skipton, UK). The patient has not experience any further pulsatile tinnitus since the first post-operative day.

Discussion

Pulsatile tinnitus is a clinical symptom common to a variety of conditions, such as benign intracranial hypertension syndrome, temporal bone paragangliomas and arteriovenous fistulae. Among the venous causes of pulsatile tinnitus the most frequently reported cause is jugular megabulb, an anatomical variant. Pulsatile tinnitus originating from venous aetiologies is considered to be due to the turbulence in the blood flow. This turbulence can be caused by either an increased venous flow or stenosis of the vessel lumen. The sound produced due to the turbulent

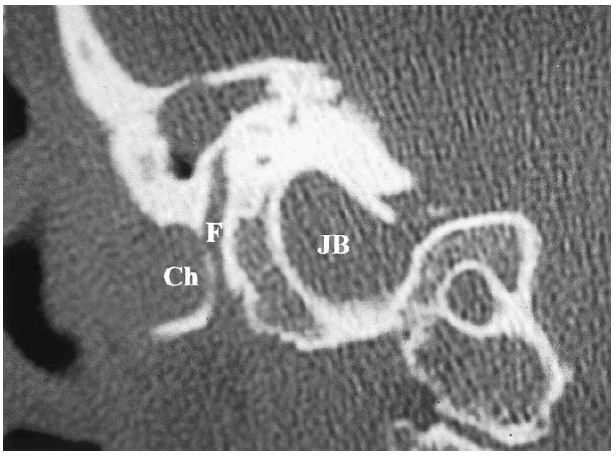

FIG. 3

CT scan (coronal section): the close relationship between the lesion and the mastoid portion of the facial nerve is clearly visible. Ch: cholesteatoma; F: facial nerve; JB: jugular bulb.

flow of the blood is transmitted directly to the otic capsule and hence causes the pulsatile tinnitus. Pulsatile tinnitus as a consequence of sigmoid sinus compression by a cholesteatoma has never been reported in the literature until now. The mastoid tip is rarely involved by common middle-ear cholesteatoma, and so residual disease in this location is not frequent. In our case the pulsatile tinnitus was the only symptom of the patient affected by residual cholesteatoma. On micro-otoscopy it was very difficult to detect the slight protrusion of the skin on the postero-inferior aspect of the EAC. Our hypothesis is that the growing cystic lesion caused the compression of the sigmoid sinus with consequent alteration of the venous flow dynamics. This possible explanation is supported by the fact that in the beginning, the tinnitus was precipitated only in the supine position, as a consequence of the increased compression on the sigmoid sinus related to the head position. Further confirmation came by disappearance of the pulsatile tinnitus following the surgical removal of the lesion.

References

1 Sismanis A. Pulsatile tinnitus. A 15-year experience. *Am J Otol* 1998;**19**:472–7
2 Buckwalter JA, Sasaki CT, Virapongse C, Kier EL, Bauman N. Pulsatile tinnitus arising from jugular megabulb deformity: a treatment rationale. *Laryngoscope* 1983;**93**:1534–9
3 Mathis JM, Mattox D, Malloy P, Zoarski G. Endovascular treatment of pulsatile tinnitus caused by dural sinus stenosis. *Skull Base Surg* 1997;**7**:145–50
4 Lambert PR, Cantrell RW. Objective tinnitus in association with abnormal posterior condylar emissary vein. *Am J Otolaryngol* 1986;**7**:204–7
5 Sanna M, Zini C, Gamoletti R. N Frau, A Taibah, A Russo, *et al.* Petrous bone cholesteatoma. *Skull Base Surgery* 1993;**3**:201–13

Address for correspondence:
Maurizio Falcioni, M.D.,
Gruppo Otologico,
Via Emmanueli 42,
29100 Piacenza, Italy.

Fax: ++39-0523-453708
E-mail: maurizio.falcioni@gruppootologico.it

Dr M. Falcioni takes responsibility for the integrity of the content of the paper.
Paper supported by a grant from Associazone Studio Aggiornamento Basicranio, (ASAB)