

## Radiology in Focus

# Facial palsy after glomus jugulare tumour embolization

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### Abstract

Facial palsy after pre-operative embolization of glomus tumours is a rare complication. In our case, complete facial palsy occurred within four hours after embolization with polyvinyl alcohol foam. Three days later, embolization material was found in the perineural vessels of the facial nerve in its mastoidal segment. Six months after complete tumour removal, facial decompression with perineural incision, and steroid therapy, facial function recovered completely. In cases of embolization of both stylomastoid and branches of the middle meningeal artery with resorbable material, temporary facial palsy can occur.

**Key words:** Facial paralysis; Glomus tumour; Embolization

### Introduction

Pre-operative super-selective arterial embolization of glomus tumours decreases tumour vascularity and facilitates surgical removal with low morbidity (Murphy and Brackmann, 1988; Young *et al.*, 1988). If surgical removal is planned, microparticles of polyvinyl alcohol (PVA) can be used, as they minimize complication risks. Further, PVA provides excellent embolization of large glomus tumours with multicompartamental vascular composition (Valavanis, 1988). Several tributaries are used in the arterial approach for embolization: the most important inferomedial compartment is supplied by the ascending pharyngeal artery (inferior tympanic and jugular branches), the posterolateral compartment (including the mastoid segment of the Fallopian canal) by the stylomastoid artery (as a branch of the occipital artery in 60 per cent or of the posterior auricular artery in 40 per cent (Djindjian and Merland, 1978). Further, the anterior compartment is supplied by branches of the internal maxillary artery and small branches of the internal carotid artery (caroticotympanic artery) and the superior compartment by branches of the middle meningeal artery.

Facial palsy after embolization is a rare complication. Valavanis (1986) reported two cases of temporary facial palsy, Herdman *et al.* (1993) another one.

### Case report

A 51-year-old female presented with a three-year history of right-sided pulsatile tinnitus and conductive hearing loss. Otoloscopically she had a red, pulsatile mass in the right middle ear with intact ear drum. All cranial nerves were intact, there were no neurological deficits.

An audiogram revealed a moderate sensorineural hearing loss of the high frequencies and an additional conductive block of 40–50 dB through all frequencies.

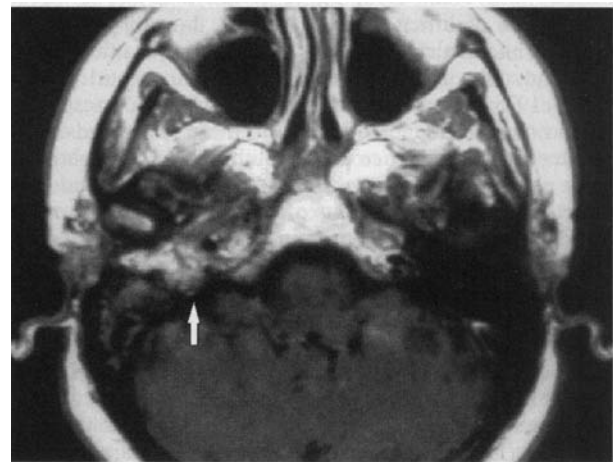


FIG. 1

Pre-operative MRI showing the large tumour of the right jugular foramen (arrow).

The MRI (Figure 1) showed a large mass within the body of the right temporal bone with contact to the posterior fossa dura involving the hypotympanum and the jugular foramen.

A diagnosis of a glomus tumour class De (intracranial extradural tumour extension) according to Fisch' classification (Fisch and Mattox, 1988) was made and surgical removal was decided in view of the fact that she was relatively young and had a large tumour.

One stage angiography and super-selective pre-operative embolization was scheduled. Angiography of the external carotid artery revealed high tumour vascularity suggesting a paraganglioma (Figure 2, left). Both ascending pharyngeal and occipital arteries as well as branches of

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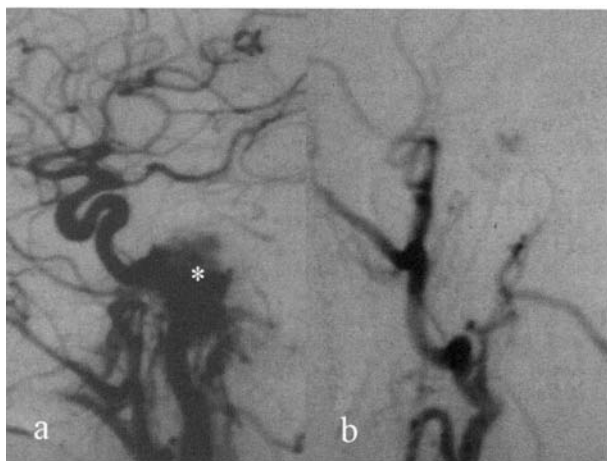


FIG. 2

a. Pre-operative angiography of the common carotid artery with tumour blush (\*) and b. control angiography after embolization with no evidence of tumour enhancement.

the middle meningeal artery (Figure 3) were selectively catheterized with a Tracker 18 microcatheter (Target Therapeutics, Fremont, CA) and embolized subsequently with polyvinyl alcohol microparticles, 50–150 micron particle size. Final check angiography demonstrated no residual tumour blush (Figure 2, right) and there was no neurological abnormality after the procedure.

Four hours after the embolization the patient developed a partial right-sided facial palsy of all branches with no other neurological deficits. In spite of intravenous steroid therapy, facial palsy deteriorated to a total lower motor neurone palsy within 24 hours (House-Brackmann Grade V).

Surgery was performed 72 hours after embolization using an infratemporal approach type A. The vertical segment of the facial nerve was exposed and found to be congested and bluish (Figure 4). The canal was widely damaged by the tumour, which had significant contact with the nerve without infiltration. Perineural vessels were prominent due to intravasal PVA. Decompression was carried out by incision of the perineurium. Total tumour removal was possible after wide opening of the retrofacial cells, removal of the mastoid tip and exposure of the jugular foramen. For this purpose the facial nerve was

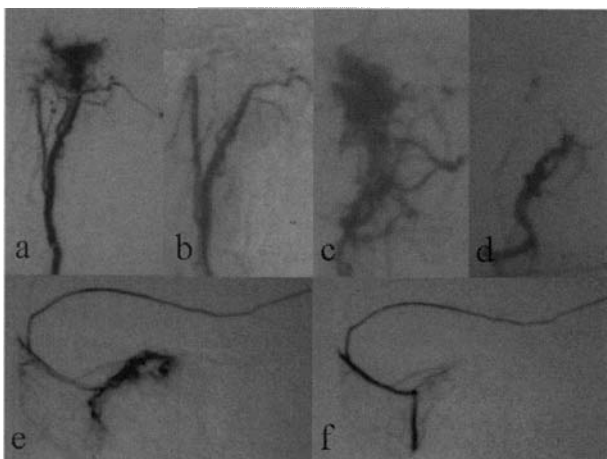


FIG. 3

Ascending, pharyngeal artery before (a) and after embolization (b), occipital artery before (c) and after embolization (d) and middle meningeal artery before (e) and after embolization (f).

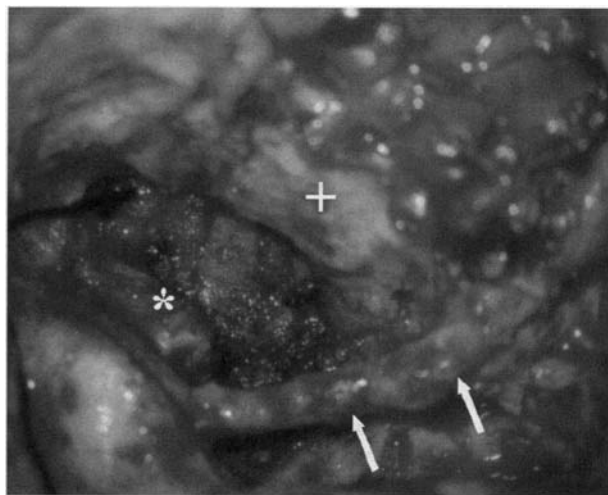


FIG. 4

Intra-operative situation after exposure of the facial nerve (arrows) and removal of the external auditory meatus. The ventral bony wall of the meatus (+) and the promontory (\*) are visible.

mobilized ventrally after exposure of its extracranial segment but repositioned in its normal anatomical position after tumour removal. As the tumour was growing into the internal jugular vein, ligation of the sigmoid sinus and removal of the jugular vein was necessary. The dura of the posterior fossa was not opened. The Eustachian tube was sealed by muscle and fibrin glue and the mastoid cavity with abdominal fat after closure of the external auditory meatus.

The post-operative course was uneventful with normal lower cranial nerve function. Histology revealed a glomus tumour.

Six months after surgery facial function recovered significantly, one year after surgery it recovered completely (House-Brackmann Grade I). There was no evidence of residual tumour using magnetic resonance imaging (MRI).

**Discussion**

Facial nerve palsy is a rare complication of glomus tumour embolization. We observed it in only one out of 15 embolizations (five class B tumours, six class C tumours and four class D tumours), although PVA microparticles under the facial nerve perineurium were detected intra-operatively in two more cases without pre-operative facial palsy (both class C). In all these cases the mastoid segment of the Fallopian canal was destroyed by the tumour, which was adherent to the nerve itself. Valavanis (1986) described another two cases with temporary facial palsy after embolization out of 35 temporal bone paragangliomas and Herdman *et al.* (1993) another one with subtotal recovery.

Herdman *et al.* (1993) suggest that if the stylomastoid artery is occluded, the facial nerve is usually not in danger because of its supply from the middle or accessory meningeal artery, which is evident in 90 per cent of the population (Lasjaunias and Bernstein, 1987a). In the other 10 per cent with no blood supply of the mastoid segment of the facial nerve by the middle meningeal artery, as well as in cases of embolization of middle meningeal artery tributaries (as in our case, Figure 3e and f), facial nerve dysfunction should be expected due to significant occlusion of its supply. A recent experimental study on guinea pigs showed that the blood flow in the geniculate ganglion of

the facial nerve comes not from the stylomastoid artery but mainly from the petrosal artery, so that facial nerve ischaemia at the level of the geniculate ganglion following ligation of the middle meningeal artery or embolization of the maxillary artery can result in facial palsy (Takeda *et al.*, 1997).

However using PVA embolization material, revascularization of initially occluded vessels has been observed; the recovery of the facial palsy in all reported cases as well as temporary palsies of other cranial nerves after embolization with PVA (Russel, 1986; Lasjaunias and Bernstein, 1987b) support this theory. Another possible pathomechanism of temporary neurapraxia of the facial nerve may be haemorrhagic swelling of its vertical portion, which was also treated by surgical decompression during removal of the glomus tumours in the reported cases.

On the other hand, permanent embolization with liquid embolic agents may cause permanent palsy of the facial or other cranial nerves; if permanent embolization is indicated (when surgery is not possible), the provocation test with pre-embolic injection of lidocaine may assess the risk of lasting palsies.

The use of intra-operative facial nerve monitoring is not possible after facial palsy due to pre-operative embolization. However, the surgeon should avoid additional damage to the nerve, which could diminish the good prognosis of the palsy after embolization.

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