Clinical Neurology and Neurosurgery 117 (2014) 102–106

Contents lists available at ScienceDirect

Clinical Neurology and Neurosurgery

journal homepage: www.elsevier.com/locate/clineuro

Case report

Microvascular decompression of the eighth cranial nerve for unilateral pulsatile tinnitus

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ARTICLE INFO

Article history:
Received 12 September 2013
Received in revised form
17 November 2013
Accepted 28 November 2013
Available online 9 December 2013

Keywords:
Microvascular decompression
Tinnitus
Vestibulocochlear nerve
Facial nerve
Cranial nerves

1. Introduction

Tinnitus is a common symptom of various underlying diseases. The severity of tinnitus varies from an occasional awareness of noise in one or both ears, to an unbearable sound that markedly interferes with the patient's daily activities and sleep, causing considerable psychological distress. It may be classified as pulsatile (PT) or continuous. PT is puls-synchronous (coinciding with the patient's heartbeat) and usually suggests a vascular etiology ranging from dural arteriovenous fistulae (DAVF), intracranial aneurysms, atherosclerotic disease of the craniocervical arteries, vascular tumors of the temporal bone, and cerebello-pontine angle (CPA), to conditions associated with high cardiac output (e.g., hypertension, anaemia, and thyrotoxicosis) [1–9].

Based on few reports, vascular compression of the eighth cranial nerve (CN VIII) has been recognised as an additional cause of incapacitating tinnitus [10–25]. However, microvascular decompression (MVD) of CN VIII solely for relieving tinnitus yielded poor results compared to those achieved for trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia by decompression of the corresponding cranial nerves. This discrepancy is mainly attributable to the lack of sufficient diagnostic criteria for tinnitus caused by neurovascular compression (NVC) and inadequate patient selection.

We report two patients with unilateral PT that subsided following MVD of CN VIII to illustrate exemplarily the key criteria for identifying patients suffering from tinnitus due to NVC.

2. Case reports

2.1. Case 1

A 73-year old man presented with a 20-year history of a constant unilateral PT in the left ear. Tinnitus was intensified by physical strain, interfering with the patient's every-day activities, and severely diminishing his quality of life. His physical examination was remarkable for a sensorineural hearing loss in the left ear (−80 dB in 1–2 kHz hearing range, Fig. 1), vertigo and a coexistent peripheral facial nerve palsy (grade II according to the House–Brackmann scale). Otoscopic and neurological examinations were otherwise normal. T2-weighted cranial MRI demonstrated a vascular loop crossing the left CN VIII in its cisternal segment (Fig. 2). Vascular pathologies such as DAVF and arterial wall diseases (e.g., atherosclerosis and aneurysms) were ruled out by complementary digital subtraction angiography. The latter furthermore demonstrated an elongated V4-segment of the vertebral artery and a dominant anterior inferior cerebellar artery (AICA) on the left side (Fig. 3).

With a presumptive preoperative diagnosis of NVC of CN VIII the patient underwent MVD surgery according to Jannetta [26]. The left

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0303-8467/$ – see front matter
http://dx.doi.org/10.1016/j.clineuro.2013.11.031

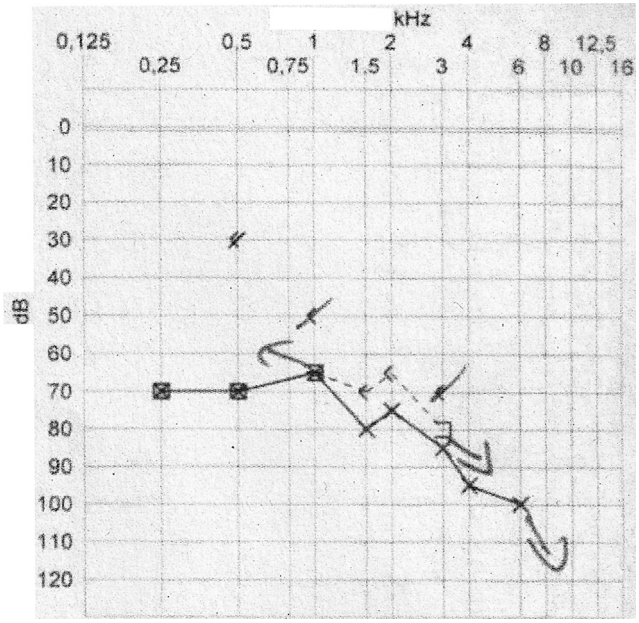

Fig. 1. Case 1. Preoperative audiometry depicting a severe high-frequency sloping hearing loss in the left ear.

CPA was explored under the intraoperative microscope through a retrosigmoid craniotomy, with the patient in the modified Concorde position. Intraoperatively, CN VIII was found to be impinged in its cisternal segment by both a loop originating from the left AICA and a bridging vein. Once the exposure was complete, the AICA loop was mobilized away from the nerve and a small Teflon cushion was inserted between the offending vessel and CN VIII. The bridging vein was coagulated and transsected.

Postoperatively the patient reported about an immediate resolution of the PT. The afunctional preoperative hearing ability of the left side had deteriorated to anacusis. On 1-year follow-up the patient presented with an unaltered facial nerve palsy (grade II according to House–Brackmann scale), and anacusis on the left side. The vertigo was alleviated, PT had not recurred.

2.2. Case 2

A 57-year old woman was referred to our clinic for definitive medical treatment of a hemifacial spasm (HFS) on the left side, which had commenced 11 years previously. Response to repeated botulinum neurotoxin injections into the affected facial muscles had attenuated over time and the patient suffered a heavy

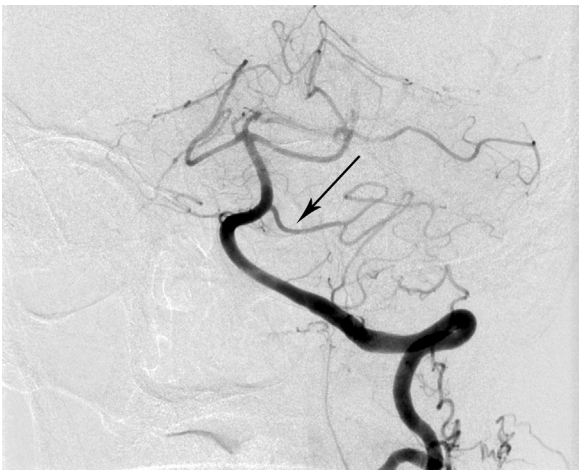

Fig. 3. Case 1. Digital subtraction angiography depicts an elongated V4-segment of the left vertebral artery and a dominant AICA (arrow) on the left side.

emotional burden of the society's response on her progressive symptoms. In addition to this, an ipsilateral low pitched PT with associated hypacusis that was synchronized with her facial spasms and aggravated by physical strain was reported. Her physical examination was remarkable for a slight peripheral facial nerve palsy (grade II according to the House–Brackmann scale) and typical bursts of tonic and clonic contractions of all facial muscles on the left side. T2-weighted cranial MRI demonstrated an indentation of the root of the left CN VII/VIII complex by a loop of the ipsilateral posterior inferior cerebellar artery (PICA), that was originating from an elongated vertebral artery (Fig. 4). Retrosigmoid CPA exploration was carried out. At surgery, the left CN VII/VIII complex was found to be impinged in its root exit zone by a loop of the ipsilateral AICA and PICA, respectively, and the elongated left vertebral artery. All three vessels were gently displaced away from the nerves and small Teflon felts were placed between the involved vessels and the CN VII/VIII complex to maintain decompression.

The patient awoke from surgery completely free from both the spasm and tinnitus. Her improvement has now been maintained at 3-month follow-up.

3. Discussion

The concept of NVC was first described in 1875 with a patient suffering from attacks of hemifacial spasm due to compression of the facial nerve by a vertebral artery aneurysm [27]. Dandy revived the concept in 1934 by attributing trigeminal neuralgia to NVC and sparked off extensive studies on the fundamental concept of

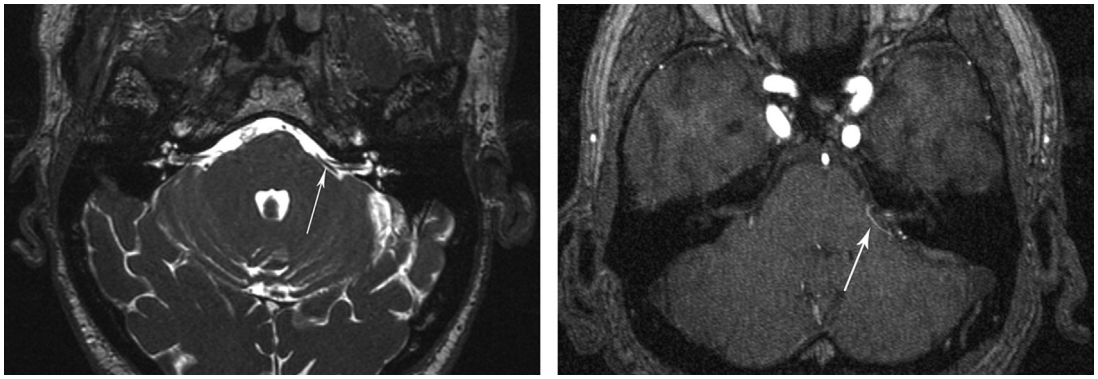

Fig. 2. Case 1. Preoperative MRI T2-weighted CISS sequences (left) and 3D-TOF sequences (right) demonstrating a vascular loop (arrow) crossing the left CN VIII in its cisternal segment.