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Vascular cross-compression of the VIIth and VIIIth cranial nerves

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Abstract

A 53-year-old male patient had been suffering from severe aural symptoms (pulsatile right-sided tinnitus and paroxysmal dizziness attacks with nausea) and right hemifacial spasm. Treatment had involved stellate ganglion block with lignocaine and the injection of intravenous sodium bicarbonate solution for attacks of Menière's syndrome and facial nerve block with lidocaine for hemi-facial spasm. Despite these treatments, the dizzy attacks became more frequent, developing into the clustering state. Air CT cisternography and vertebral angiography demonstrated an enlarged and curved vertebral artery. Vascular cross-compression of the VIIth and VIIIth cranial nerves was therefore suspected. Microvascular decompression was performed. After operation, the pulsatile tinnitus, dizziness and hemifacial spasm disappeared. From the present case and a review of the literature, we conclude that vascular cross-compression of the VIIIth cranial nerve should be an indication for microvascular decompression only when symptoms of vascular cross-compression of the VIIth cranial nerve are also seen.

Introduction

Microvascular decompression (MVD) has been widely accepted since the reports by Jannetta (1977, 1980) of neurovascular cross-compression (NVCC) as a possible cause of hemifacial spasm, trigeminal neuralgia and glossopharyngeal neuralgia. There are also some reports (Bertrand *et al.*, 1977; Leclerq *et al.*, 1980; Yeh *et al.*, 1981; McCabe and Harker, 1983; Wigand *et al.*, 1983; Nozue *et al.*, 1987; McCabe and Gantz, 1989) that neurovascular compression also occurs in the VIIIth cranial nerve. However, it is very difficult in clinical practice to differentiate vascular cross-compression of the VIIIth cranial nerve from other aural diseases (Menière's disease, positional vertigo, sudden deafness etc.) This makes the indication for microvascular decompression very difficult. Recently we encountered a patient with hemifacial spasm and aural symptoms (pulsatile tinnitus and dizzy attacks). The patient was confirmed as having the vascular cross-compression of the VIIth and VIIIth cranial nerves by the X-ray examination, and microvascular decompression was successfully performed.

Case report

A 53-year-old male patient had begun to suffer from right hemifacial spasm and ipsilateral pulsatile tinnitus in 1983. The hemifacial spasm was improved by facial nerve block with lignocaine. In December 1986, dizziness with nausea and pulsatile tinnitus suddenly occurred while he was engaged in conversation. Since March 1987, he had attacks of dizziness with pulsatile tinnitus almost daily. A general physician suspected Menière's syndrome and a stellate ganglion block with lignocaine and an intra-venous injection of sodium bicarbonate solution were performed. From December 1987, the dizzy attacks lasted as long as two hours and interfered with the patient's work. At this time, he visited the ENT outpatient department of our university hospital for further examination. Vascular cross-compression of the VIIth and VIIIth cranial nerves was immediately suspected. Vertebral angiography disclosed enlargement of the vertebral artery near the right cerebellopontine angle. Air CT cisternography enhanced within intravenous contrast medium injection also showed right enlarged vertebral artery (Fig. 1).

Otoneurological examinations

Pure tone audiometry showed a fluctuating perceptive hearing disturbance on the right side, ranging from 63.7 (February 2, 1987) to 37.5 dB (March 10, 1988). The recruitment phenomenon indicated inner ear hearing disturbance on the right side (SISI Rt. 95 per cent, Lt. 0 per cent, Bekesy Rt. type II, Lt. type II). Electronystagmographic examination disclosed positional nystagmus toward the right side and bilateral vestibular hypofunction in the air caloric and rotation tests. The righting reflex was slightly disturbed (Mann and stepping tests were positive). Auditory brain stem response showed bilateral slight prolongation of wave V.

Operative findings and clinical course

With a diagnosis of vascular cross-compression of the VIIth and VIIIth cranial nerves, in April 1988 a microvascular decompression by the sub-occipital approach was performed. The posterior inferior cerebellar artery (PICA) compressed the VIIth cranial nerve at its root entry zone and the anterior inferior cerebellar artery (AICA) ran between the VIIth and VIIIth cranial nerves, compressing the VIIIth cranial nerve. The posterior and anterior inferior cerebellar arteries were separated from the VIIth and VIIIth cranial nerves and fixed in a position using collagen sponge so that neither artery would compress the cranial nerves (Fig. 2). The vertebral artery did not compress the VIIth and VIIIth cranial nerves directly (Fig. 2). After operation, pulsatile tinnitus, dizziness and hemifacial spasm disappeared.

Discussion

Neurovascular cross-compression has been found to cause

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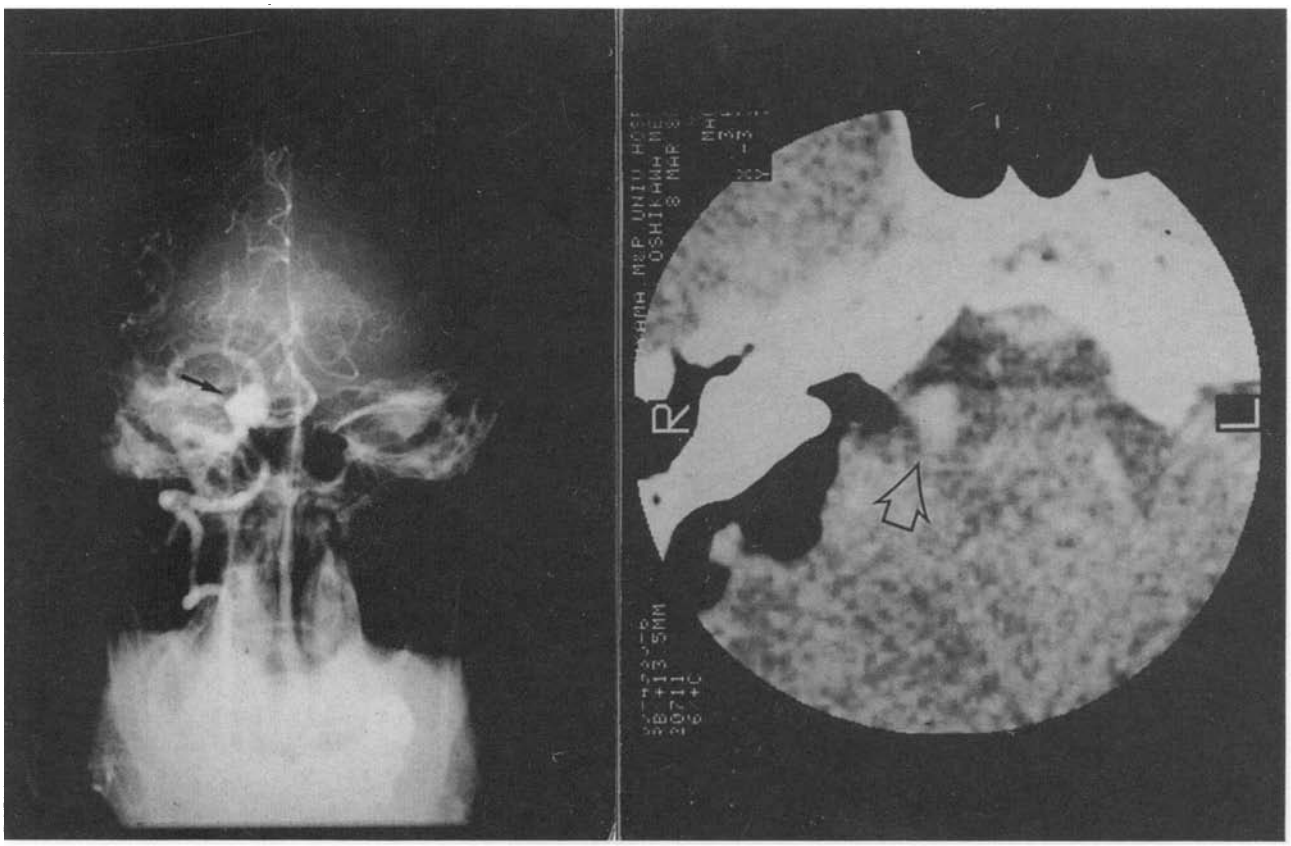

FIG. 1

X-ray findings. Vertebral angiography (left) and air CT enhanced with intravenous contrast medium (right) showed enlarged vertebral artery; however, vascular loop was not detected.

several diseases (trigeminal neuralgia, glossopharyngeal neuralgia and hemifacial spasm—Jannetta, 1977 & 1980). Microvascular decompression is widely accepted as a treatment for these diseases, particularly for hemifacial spasm. The VIIth and VIIIth cranial nerves arise from a very close area of the brain stem and run together through the inner auditory meatus. Therefore, both are easily affected simultaneously if there is any lesion in the cerebellopontine angle (e.g. advanced acoustic neuroma). There are therefore two groups of patients in vascular cross-compression of the VIIIth cranial nerve. The pathology in the first group involves single vascular cross-compression of the nerve, without involvement of the VIIth cranial nerve. The pathology in the second group involves combined vascular cross-compression of both nerves and occasionally the Vth in some patients showing aural symptoms and hemifacial spasm (trigeminal neuralgia in some patients). Studies on the first group have been reported by Jannetta (eight of 117 patients, 1977; 38 of 695 patients, 1980). Wigand (17 patients, 1983). McCabe (eight patients, 1983), Nozue (nine patients, 1987) and McCabe (34 patients, 1989). Studies on the second group have been reported by Bertrand (five patients with the VIIIth cranial nerve symptoms as well as the Vth and VIIth cranial nerve symptoms, 1977) and Yeh (four patients with the VIIIth and VIIth cranial nerve symptoms, 1981).

The common aural symptoms of patients in these two groups were intractable vertigo, tinnitus, hearing loss, (Leclercq *et al.*, 1980; Wigand *et al.*, 1983) or motion intolerance (McCabe and Gantz, 1989). The pre-operative diagnoses were Menière's disease (Jannetta, 1977, 1980), benign paroxysmal vertigo (Jannetta, 1980), vestibular neuronitis (Jannetta, 1980; Wigand *et al.*, 1983), vestibular Menière's disease (McCabe and Harker, 1983; McCabe and Gantz, 1989), or sudden hearing loss (Wigand *et al.*, 1983). In addition, diagnosis should differentiate among perilymph fistula, autoimmune inner ear disease and acoustic neuroma (McCabe and Gantz, 1989). Therefore, vascular cross-compression of the VIIIth cranial nerve may be concealed in patients who have been treated under the above diagnosis, making full investigation very important.

The most common VIIIth cranial nerve symptom in the two groups is vestibular hypoexcitability (Bertrand *et al.*, 1977; Leclercq *et al.*, 1980; Nozue *et al.*, 1987; McCabe and Gantz, 1989). However, Leclercq *et al.* (1980) have stated that good discrimination is useful in distinguishing vascular cross-compression of the VIIIth cranial nerve from hydrops. Further, Bertrand *et al.* (1977) have stated that retrocochlear pathology as assessed by audiometric tests is suggestive of vascular cross-compression of the VIIIth cranial nerve. However, even from other reports (McCabe and Harker, 1983; Wigand *et al.*, 1983; Nozue *et al.*, 1987; McCabe and Gantz, 1989) of MVD for single vascular cross-compression of the VIIIth cranial nerve, we could find no definite findings confirming the single vascular cross-compression of the VIIIth cranial nerve. Therefore single vascular cross-compression of the VIIIth cranial nerve is still controversial (Yeh *et al.*, 1981) and very difficult to prove the need for surgical intervention (Nozue *et al.*, 1987). Therefore, vertebral angiography and air CT with intravenous contrast medium and/or magnetic resonance imaging, which are not always necessary prior to surgery for trigeminal neuralgia and hemifacial spasm, are indispensable in the diagnosis and surgery for single vascular cross-compression of the VIIIth cranial nerve (McCabe and Gantz, 1989). However, it is still very difficult to find the vascular loop as in the present patient, even with air CT. As to the present indications for MVD, we prefer to follow Yeh's suggestion (1981) that microvascular decompression should be performed on patients with suspected vascular cross-compression of the VIIIth cranial nerve only if there is associated hemifacial spasm or other signs of brain stem compression. Accumulated experience, however, may make it possible to

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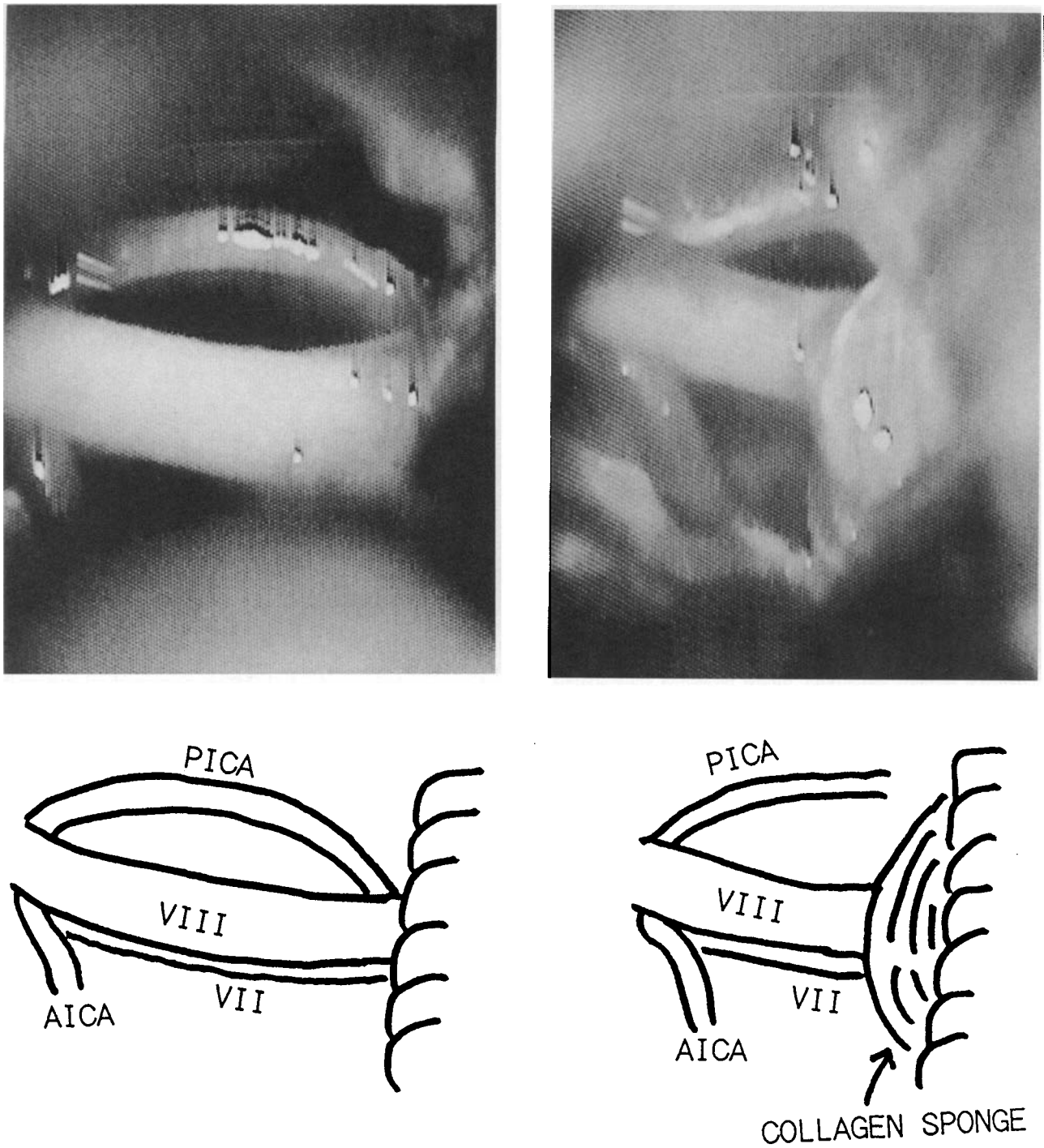

FIG. 2

Finding before MVD (left) and after MVD (right). PICA compressed the VIIth cranial nerve at its root entry zone and AICA ran between the VIIth and VIIIth cranial nerves, compressing the VIIIth cranial nerve (left). PICA and AICA were separated from the VIIth and VIIIth cranial nerves and decompression was performed (right).

select appropriate patients for decompression for single vascular cross-compression of the VIIIth cranial nerve if other forms of therapy have failed and vascular compression is suspected (Yeh *et al.*, 1981).

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