Hemifacial Spasm caused by Vascular Compression in the Cisternal Portion of the Facial nerve

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Although primary hemifacial spasm (HFS) is mostly related to a vascular compression of the facial nerve at its root exit zone (REZ), its occurrence in association with distal, cisternal portion has been repeatedly reported during last two decades. We report two patients with typical HFS caused by distal neurovascular compression, in which the spasm was successfully treated with microvascular decompression (MVD). Vascular compression of distal, cisternal portion of the facial nerve was identified preoperatively in the magnetic resonance imaging (MRI). It was confirmed again with intraoperative findings of compression of cisternal portion of the facial nerve by the meatal loop of the anterior inferior cerebellar artery (AICA) and absence of any offending vessel in the REZ of the facial nerve. Immediate disappearance of lateral spread response (LSR) after decompression and resolution of spasm after the operation again validated that HFS in the current patients originated from the vascular compression of distal, cisternal portion of the facial nerves.

A 50-year-old male patient presented with a 2-year history of left-sided typical HFS. Painless irregular clonic contraction of the facial muscles began initially in the orbicularis oculi muscle of the lower lid. It gradually spread to other muscles innervated by the facial nerve on the left side of the face, including platysma. The paroxysm was induced or aggravated by emotional tension, stress, and voluntary and reflexive movements of the face. He had significant difficulty in his work and social life despite of 2 times of bolutinum toxin injection. Medical treatment with carbamazepine (up to 600 mg) and baclofen (30 mg) was not effective. He was referred for surgical treatment. His medical history was unremarkable. His physical and neurologic examinations were normal, including hearing. No tinnitus or discernible noise heard in his left ear was found. Only typical nature of clonic hemifacial spasm was evident. Abnormal synkinesis between the orbicularis oculi and orbicularis oris muscles was found by the electromyographic examination of the blink reflex. Despite of typical HFS, there was no discernible vascular structure in the REZ of left facial nerve (Fig. 1a). However, a meatal loop of AICA abutting to the cisternal portion of the facial nerve was found.

Under the impression of HFS caused by neurovascular compression of distal facial nerve, standard microsurgical procedure was performed described previously.In addition to intraoperative monitoring of BAEPs, LSR, which is an abnormal muscle response demonstrated by EMG recordings from mimic muscles that are innervated by a different branch of the facial nerve, were also monitored throughout the operation. The entire course of the facial nerve and offending arteries were exposed under microscopic vision. Upon exposure of the REZ of the facial nerve, there was no offending vessel in the REZ as expected. The distal, cisternal segment of the facial nerve was found to be bent by a meatal loop of the AICA. A small piece of Teflon felt was interposed between the facial nerve and the meatal loop of the AICA with extreme care not to stretch the internal auditory artery and the distal facial nerve. After interposition of Teflon felt, LSR immediately disappeared and BEAP was stable also. The closure of the dura and wound was performed in routine manner. The HFS resolved completely following the surgery. The postoperative course was uneventful with no signs of facial weakness or hearing impairment by pure-tone audiometry. No recurrence of HFS or neurologic sequele was evident at a 12-month follow-up.

According to our literature review of 64 patients with HFS caused by distal neurovascular compression, distal compression can be classified by pure distal neurovascular compression (31 cases, 48.4%) and double compression (both distal segment and the REZ of the facial nerves, 33 cases [51.6%]) according to the presence or absence of simultaneous offender in the REZ. Eight-four percent of 64 identified distal offenders was the AICA, especially its meatal and postmeatal segments. Before awareness of distal neurovascular compression causing HFS and sophisticated MRI imaging (before 2000), the rate of reoperation was high (58%). Preoperative MRI and intraoperative monitoring of LSR seems to be an essential element in determination of real offending vessel in MVD caused by distal offender.