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Case Report

Hemifacial Spasm Caused by Epidermoid Tumor at Cerebello Pontine Angle

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Hemifacial spasm (HFS) is almost always induced by vascular compression but in some cases the cause of HFS are tumors at cerebellopontine angle (CPA) or vascular malformations. We present a rare case of hemifacial spasm caused by epidermoid tumors and the possible pathogenesis of HFS is discussed. A 36-year-old female patient presented with a 27-month history of progressive involuntary facial twitching and had been treated with acupuncture and herb medication. On imaging study, a mass lesion was seen at right CPA. Microvascular decompression combined with mass removal was undertaken through retrosigmoid approach. The lesion was avascular mass and diagnosed with an epidermoid tumor pathologically. Eventually, we found a offending vessel (AICA : anterior inferior cerebellar artery) compressing facial nerve root exit zone (REZ). In case of HFS caused by tumor compression on the facial nerve REZ, surgeons should try to find an offending vessel under the mass. This case supports the vascular compression theory as a pathogenesis of HFS.

KEY WORDS : Hemifacial spasm · Facial nerve · Epidermal cyst.

INTRODUCTION

Hemifacial spasm (HFS) is a most common hyperactive cranial rhizopathy and presented with involuntary facial twitching of unilateral facial muscles²⁾. HFS is almost always caused by vascular compression on the facial nerve root exit zone (REZ) closely located at the brain stem. The hypothesis of vascular compression is established by the fact the involuntary facial movement disappears just after microvascular decompression (MVD). However, the relationship between the abnormal condition of nerve signals and the facial muscles have not been clearly verified.

HFS can also be induced by benign tumors developed at cerebellopontine angle (CPA) or in 4th ventricle, glioma in brain stem¹⁷, vascular malformations¹², cysticercosis¹⁵ or lipoma⁸. And, it has been also reported that the remote lesion such as parotid gland tumor^{6,14} and remote meningiomas⁴ or contralateral lesions^{13,16} can induce the facial

involuntary movement. HFS caused by unusual cause can be helpful to understand the mechanism generating the facial symptom. We report a rare case that had a facial symptom caused by an epidermoid tumor.

CASE REPORT

A 36-year-old female patient presented with a 27-month history of progressive involuntary facial twitching. Until admission, the patient tried with acupuncture and herb medication. On admission, she had no neurological deficit on physical examinations. Magnetic resonance (MR) images showed a mass lesion on right CPA (Fig. 1A, B). She underwent mass removal by retromastoid suboccipital approach and the tumor was located anterior to 7th, 8th nerves and lower cranial nerves. The mass was avascular and easily removable. We could define the 6th nerve, which was displaced anteriorly by the mass. And, between the tumor and 7th, 8th nerve complex there was a compressing vessel, anterior inferior cerebellar artery (AICA), but the arachnoid band was adhesive between facial nerve and offending vessel and thus dissected cautiously (Fig. 2). Teflon was then inserted. Post-operatively the facial involuntary movement was relieved but the facial palsy was

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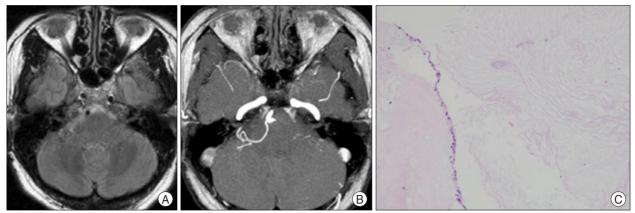


Fig. 1. Fluid-attenuated inversion recovery magnetic resonance (MR) image showing the iso-signal intensity lesion displacing brain stem at right cerebellopontine angle (A) and three-dimensional short-range reconstruction MR image demonstrating the offending vessel, which is displaced by the mass (B). Pathologically, the tumor contains laminated keratin material lined by stratified squamous epithelium (Hematoxilyn-Eosin stain, × 100) (C).

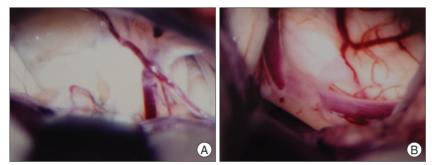


Fig. 2. A: On operative finding, the lesion is avascular and whitish mass. B: After the mass removal, anterior cerebellar artery is detected under the tumor as a compressing vessel but arachnoid band between offending vessel and facial nerve is found (B).

noticed that was relieved at 17 days after the surgery.

DISCUSSION

HFS is a hyperactive cranial rhizopathy which is generated at the facial REZ at pontomedullary junction¹⁰⁾. The generally accepted hypothesis of HFS is that elongated vessels as a result of aging process irritate facial REZ. At the point where the vessel is compressing, atypical neuronal signal is generated and conducted to facial muscles¹⁾. But whether the actual site of the ephapsis is at the site of the lesion or at a nuclear level due to hyperexcitability of the facial motor nucleus is still controversial. Another hypothesis is that hypersensitivity of facial nucleus can cause the facial involuntary movement and it is supported by the phenomenon that HFS could be induced by brainstem glioma³⁾.

As for HFS induced by benign tumors, many authors have suggested the mechanisms, which might induce the facial symptom. In many cases, it was suggested that the vascular compression under the tumor is the cause of the HFS^{5,7,9)}. And, several authors suggested that in spite of the remote meningioma⁴⁾ or contralateral lesions^{13,16)}, there was a possibility of the offending vessel by the distorted brain structure with large mass lesion. In this case, we found the definite offending vessel, which was displaced by epidermoid cyst, and compressing the facial REZ.

However, others had reported that there were no compressing vessels under the tumor^{8,9)}. Nagata et al.¹²⁾ suggested that there was another cause inducing the symptom. He had found

that there was no artery compressing the facial nerve at the REZ and in three cases, the HFS disappeared after removal of the tumor in contact with the facial nerve and proposed that the cause of HFS was the compression or encasement of the facial nerve by the tumor was the pathogenesis.

Another possible hypothesis of facial symptom is by the changes of arachnoid membrane. Some authors have reported that arachnoid adhesion can be a possible evidence of a prior inflammatory process and may force the pulsatile arterial branches into constant contact with the 7th and 8th nerve complex but these hypothesis were not verified¹⁸⁾. Kobata et al.¹¹⁾ reported the large cases of unusual causes of HFS by 30 epidermoids but there were only 2 cases of HFS patients. He proposed that arachnoid adhesion might be a cause as one of the recurrence of symptom. In our case, after the mass removal, the arachnoid band between AICA and facial nerve was partially remained because it was very adhesive and had a possibility of facial nerve tearing when removed. It may be possible that arachnoid bands between the offending vessel and facial nerve can be an aberrant conduction. But, we decompressed

the facial nerve REZ as possible as we could to detach from the facial REZ.

CONCLUSION

This case can support the vascular compression hypothesis in case of HFS caused by tumors lesion at CPA. And, it is suggested that surgeons should try to find the underlying offending vessel displaced by the tumor mass.

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