Hemifacial spasm etiology

- Postoperative Dysphagia Management in Hemifacial Spasm: A Case Report of Combined Catheter Balloon and Neuromuscular Stimulation
- Glue-transposition technique for hemifacial spasm involving vertebrobasilar dolichoectasia: a comparative study and literature review
- Identifying associated comorbidities in the development of trigeminal neuralgia: A propensity-matched analysis of the National Inpatient Sample
- Risk factors and Occurrence of Small vessel disease in Acute sensorineural hearing Loss In the Elderly: protocol for a multicentre cross-sectional study
- The silent scream: Unraveling vagal nerve neuralgia; VANCOUVER syndrome and HeLPS
- Arterial Hypertension After Macrovascular Decompression of Vertebrobasilar Dolichoectasia: Rethinking the Cause-Effect Relationship
- Primary Gamma Knife Radiosurgery as a Treatment Option for Hamartoma of Floor of Fourth Ventricle: A Case Report of Pediatric Hemifacial Spasm
- Application of neurophysiological monitoring in differentiation of hemifacial spasm and post-facial paralysis synkinesis

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1. vascular compression syndrome : the most common etiology (much more common than with trigeminal neuralgia)

see Vascular compression in hemifacial spasm.

Hemifacial spasm is usually caused by arterial compression at the root exit zone of the facial nerve. However, other etiologies have been reported.

Purely venous compression is rarely encountered. El Refaee et al. reported for the very first time arachnoid bands strangulating the nerve as a cause for hemifacial spasm without involvement of any vessel [1)].

2. idiopathic

3. tumor compressing the nerve

4. can follow some cases of Bell’s palsy

5. conditions that can mimic HFS

a) blepharospasm (bilateral spasmodic closure of the orbicularis oculi muscles) which is more common in the elderly, and may be associated with organic brain syndrome. Blepharospasm is notorious for disappearing when the patient presents for medical evaluation (an effect of alerting), but may be elicited by asking the patient to gently close the eyes and then rapidly open them, following which a blepharospasm may occur. HFS usually involves more than the ocular muscles

b) facial myokymia: continuous facial spasm which may be a manifestation of an intrinsic

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Rare tumor-related HFS associated with meningiomas, epidermoid tumors, lipomas, and schwannomas in the cerebellopontine angle have been reported. The exact mechanism and the necessity of microvascular decompression for tumor-induced HFS is not clear, especially for vestibular schwannomas.

In clinical pediatric neurosurgery practice, fourth ventricle tumor and cerebellar tumors are not rare. However, reports of secondary refractory hemifacial spasm are very rare. No report is currently available on the treatment of hemifacial spasm secondary to fourth ventricle and cerebellar tumors in China. Zamponi et al. [Childs Nerv Syst 2011 Jun;27(6):1001-5] reported that these lesions can occur in neonates and infants, and surgical resection is effective [2].

Liu et al. retrospectively analyzed 10 patients with vestibular schwannomas out of 5218 cases of hemifacial spasm between 2004 and 2014.

Hemifacial spasm occurred ipsilateral to the vestibular schwannoma in 9 patients and contralateral to the lesion in 1 patient. The mean follow-up period was 86 months (range, 22-140 months). All patients underwent surgery for resection of the vestibular schwannoma. Following the principle of neurovascular compression, offending vessels were found in 7 patients, no offending vessels in 2 patients, and a tumor with the displacement of brain stem contributing to contralateral facial nerve compression in 1 patient. HFS was relieved immediately postoperatively in 9 patients, whereas it improved gradually and then resolved after one month in one patient with a contralateral vestibular schwannoma.

For HFS induced by vestibular schwannomas in this study, the majority of cases are caused by a combination of tumor and vascular co-compression at the REZ. Surgical intervention resulted in resolution of symptoms. For HFS with ipsilateral vestibular schwannoma, exploration of the facial nerve root for vascular compression should be performed routinely after tumor resection. It is critical to check that no vessel is contact with the entire nerve root [3].

During the period from October 1984 to October 2008, Han et al. treated 6,910 HFS patients using a microsurgical procedure. Of these HFS patients, 55 cases were associated with cerebellopontine angle tumors. A small craniectomy was performed in order to excise the tumor. All tumors were found to compress the root exit zone (REZ) of the facial nerve to different extents, but concomitant vascular compression of the facial nerve was observed in a majority of cases, and microvascular decompression of the facial nerve at REZ was conducted in 43 of 55 patients (78.2%) by displacing the co-compressing vasculature away from the REZ and retaining it using a Teflon pad. Intraoperative findings and postoperative pathological examinations suggested that the tumors were epidermoid cysts, meningiomas, and Schwannomas. Follow-up in 48 of 55 patients for 4-230 months after surgery

showed that the clinical symptoms of HFS disappeared in 43 cases, improved in two cases, and recurred in three cases. Ten patients had sequelae associated with the operation. They concluded from this study that the majority of cases of tumor-related HFS are caused by combined tumor and vascular co-compression at the REZ, and tumor removal and microvascular decompression are required in order to relieve the symptoms [4].

Kindling-like hyperactivity of the facial motor nucleus induced by constant stimulation of compressing artery is considered as the predominant mechanism underlying the pathogenesis of Hemifacial spasm (HFS).

Trigeminal neuralgia, hemifacial spasm, vestibulocochlear neuralgia and glossopharyngeal neuralgia represent the most common neurovascular compression syndromes.

In nearly all cases, primary hemifacial spasm is related to arterial compression of the facial nerve at root exit zone (REZ). The offending arterial loops originate from the posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA), or vertebrobasilar artery (VB). In as many as 40% of the patients, neurovascular conflicts are multiple. The cross-compression is almost always seen on magnetic resonance imaging combined with magnetic resonance angiography.

Hemifacial spasm (HFS) associated with type 1 Chiari malformation is particularly uncommon and is limited to isolated case report.

Li et al retrospectively evaluated 13 patients who had simultaneously HFS and type 1 Chiari malformation among 675 HFS patients. Clinical features and radiological findings were collected from each patient and analyzed. All these 13 patients were surgically treated with MVD through retro-mastoid microsurgical approach, and postoperative outcomes were evaluated. A review of literature about this association was also provided. In this study, the frequency of type 1 Chiari malformation in HFS patients was 1.9 %. The clinical profile of this series of patients did not differ from typical form of primary HFS. MVD achieved satisfactory results in 11 patients (85 %) in short- and long-term follow-up. There was no mortality or severe complication occurred postoperatively. Although rare, clinician should be aware of the association of HFS and type 1 Chiari malformation and consider MVD as an effective surgical management [5].

Vascular compression

see Vascular compression in hemifacial spasm.

References

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