

Complications of microvascular decompression surgery for hemifacial spasm: A review of 30 operated cases

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ABSTRACT

INTRODUCTION: Microvascular decompression is the gold standard for the treatment of hemifacial spasm. However, this technique is technically demanding, with a steep learning curve and a propensity for complications during the early phase. We review our complications in a series of 30 patients operated in the last seven years.

CASE PRESENTATION: of thirty patients operated in the last seven years were reviewed. The early and late complications were noted and the treatment given for the complications and the outcome was noted.

RESULTS: Delayed facial palsy was the most common complication, occurring in three patients, followed by mild early facial palsy in one. Mild sensorineural hearing loss in one, delayed resolution of the spasms in two and recurrence of spasm in one patient was noted.

DISCUSSION: Hemifacial spasm has an incidence of approximately 10 per 100,000 population with a 2:1 female predominance and a mean age of onset between 45 to 55 years.

CONCLUSION: Microvascular decompression for hemifacial spasm is a safe procedure with a very low rate of complications which recover in a short time.

Key Words: *Microvascular decompression; Hemifacial spasm*

INTRODUCTION

Hemifacial spasm is a gradually progressive disorder characterised by intermittent, tonic, involuntary twitching of the face, usually unilateral. It is caused by an aberrant vascular loop compressing the facial nerve at its point of exit from the brainstem.

Microvascular decompression of the facial nerve, provides the definitive cure for this condition. This procedure is technically more demanding than trigeminal nerve decompression because of its close association with the lower cranial nerves, eighth nerve, flocculus and the choroid plexus. The smallest of technical error may lead to facial nerve palsy, leaving the patient with a cosmetic disfigurement worse than before.

We review the complications in the thirty operated cases in the last seven years.

Aim of the study

To review the complications of microvascular decompression for hemifacial spasm and assess the treatment and outcomes.

CASE PRESENTATION

A total of thirty patients operated by the author in the last seven years were reviewed. All the patients had hemifacial spasm since few years. All had tried medical management in the form of carbamazepine, gabapentin and pregabalin at other hospitals, but the spasms were not controlled. The first patient had a history of treatment by botox injection resulting in a mild facial palsy. MRI of the brain with 3D CISS/FIESTA images was done to identify the vessel compressing the facial nerve root entry zone and to rule out other causes of hemifacial spasm-like tumour, AVM, aneurysm and demyelinating conditions like multiple sclerosis. Only patients with primary hemifacial spasm were included in this study.

Out of a total of thirty patients, six were male, and twenty-four were females. The age distribution was as under (Table 1).

OPERATIVE TECHNIQUE

All the patients were operated on by the retromastoid approach under general anesthesia in the supine position. The head was turned to the opposite side by 90 degrees, and the ipsilateral shoulder was elevated by a sandbag. The vertex of the head was dropped by 10-15 degrees towards the floor [1]. This is important as it improves the visualization of the facial nerve from the inferior angle. A standard retromastoid craniotomy was done, and

TABLE 1

Age distribution of the operated patients

Age (in years)	Male	Female
31-40	2	7
41-50	3	13
51-60	0	4
61-70	1	0

the dura opened. The cerebellum is then gently pressed on the inferior-lateral aspect with the suction cannula over a cottoned patty, to release CSF from the cerebello-medullary cistern. We avoid using self-retaining retractors for cerebellar retraction as this leads to a greater chance of traction injury to the eighth nerve and consequent hearing loss [24]. As CSF is drained, the lower cranial nerves come into view. A small vein drain from the cerebellum to the inferior petrosal sinus may get ruptured. Hence, forceful retraction should be avoided. We usually do not cauterize the vein. The lower cranial nerves are followed back to their origin from the brainstem by cutting the arachnoid adhesions between the nerves and the cerebellum sharply by micro-scissors. Once the brainstem is visualized, attention is directed to the arachnoid between the 8th nerve and the cerebellum. These are cut sharply leading to further visualization of the 7-8th nerve complex. Next, the lower cranial nerves are visualized at their point of entry into the brainstem. Then, the angle of the microscope is turned slightly upwards, and the flocculus and the choroid plexus are visualized. All these steps can be done without using self-retaining retractors. Gentle pressure on the cottonoid with a suction provides sufficient retraction to carry out the dissection. The facial root exit zone is best visualized through the infra-floccular approach lifting the cerebellar rostrally. At this stage, it may be necessary to use the thin 3-mm blade of the self-retaining Leyla retractor to gently retract the flocculus. The root entry zone of the facial nerve comes into view. The vascular loop indenting the facial nerve can now be seen. The loop is gently separated from the facial nerve. Any arachnoid adhesions restricting the movement of the loop are cut sharply with micro-scissors. The goal of the surgery is to reorient the axis of the vessel and change its course away from the facial nerve, rather than simply "push" Teflon between the vessel and the nerve. Soft Teflon felt is now inserted between the nerve and the vessel loop to prevent re-adhesion. After confirming adequate hemostasis, the dura is closed in a watertight fashion. Muscles and skin are closed in standard fashion, and a dressing is applied. Postoperatively, the patient is kept in an ICU for a few hours. Antiemetics, analgesics, and IV fluids are continued for 24 have postoperative transient, vertigo, ataxia, and vomiting may occur. The patient is usually discharged on the second or third postoperative day. Stitches are

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TABLE 2
Different complications in patients

Complication	No of patients
Delayed facial palsy (after 7 days of surgery)	3
Sensory neural hearing loss	1
Early-onset facial palsy	1
Delayed resolution of spasm	2
Recurrence of hemifacial spasm	1

removed on the eighth or ninth postoperative day. Each patient was followed up for six months for the development of complications. Twenty-two patients had no early or delayed complications after MVD for hemifacial spasm, a complication-free rate of 73.33%. Minor complications were noted in eight patients, 26.67% (Table 2).

RESULTS

The patients with delayed facial palsy were put on a short tapering course of prednisone 30 mg three times a day, with facial nerve stimulation. All three patients showed complete recovery within three months. One patient with mild sensorineural hearing loss recovered completely in two months. Two patients showed persistent spasms particularly with the movement of the face, though the spasms were decreased in frequency and severity. These patients were put on carbamazepine 200 mg thrice a day for 15 days. The spasms resolved in both cases. One patient with early-onset facial palsy also completely recovered. One patient had a recurrence of spasms and facial palsy. A patient refused a second procedure and was lost to follow-up.

DISCUSSION

Hemifacial spasm has an incidence of approximately 10 per 100,000 population with a 2:1 female predominance and a mean age of onset between 45 to 55 years. It usually begins with minor twitching of eyelids. Microvascular decompression, first described by Janetta et al remains the safest and the most effective method of treating this condition with a success rate of 80%-100% reported around the world [5-7].

Though botox is being used for the treatment of hemifacial spasms, it is expensive, needs repeated injections, and may lead to asymmetry and atrophy of facial musculature, and does not remove the cause of the disease. Hence, it should be used only when the patient is deemed unfit for surgery. Sensorineural hearing loss and failure of the procedure to relieve the spasm are the two main areas of concern for the operating neurosurgeon.

Hearing loss can be prevented by avoiding self-retaining retractors and forceful cerebellar retraction. The cerebellar retraction initially should be in a rostra-caudal direction and not in the lateral to medial direction. It should be done with the help of gentle pressure by thin no 1 suction cannula over a cottoned to release CSF. This prevents traction injury to the 8th nerve. Failure of surgery is the most common due to inadequate proximal dissection. It is important to look for the loop at the root exit zone of the facial nerve at the brainstem. The distal vessels in contact with the nerve are usually not pathogenic. Studies have shown that in cases of failure, the compression is usually found proximal to the Teflon patch on MRI and confirmed on reexploration [8].

The complications during hemifacial spasms are more likely if the surgeon

is not well-versed with the operative technique. Proper positioning of the patient before surgery, adequate drilling of bone inferiorly, and avoiding lateral to medial cerebellar retraction are important to avoid injuring the eighth and seventh nerve.

During dissection, it is important to keep the suction tip away from the root of the facial nerve. It is the suction pressure that leads to permanent facial nerve palsy, in the majority of cases.

Delayed facial palsy occurs due to facial nerve edema most probably as a result of pressure from the Teflon sponge and always resolves with steroids.

Hearing loss occurs due to stretching of the eighth nerve cerebellar retraction and hence it should be avoided. Electrophysiologic monitoring during the surgery for hemifacial spasms can reduce the incidence of hearing loss, but it may not be available in developing countries due to cost constraints [9].

Persistent spasms are due to intermittent discharges from the nucleus of the seventh nerve and resolve with time. Thus in our series, only one patient had a permanent complication of persistent spasm and facial palsy, a complication rate of 3%.

CONCLUSION

Currently, microvascular decompression remains the gold standard for the treatment of hemifacial spasms, with a success rate of more than 97% with a very low rate of complications.

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