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Hearing outcomes after microvascular decompression for trigeminal neuralgia: an institutional experience

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# Abstract

**Context:** Microvascular decompression is a safe and effective treatment for trigeminal neuralgia. Hearing loss is a rare but dreadful complication after MVD with significant impact on patient's life.

**Aims:** Our aim was to evaluate the hearing outcomes after MVD surgery for trigeminal neuralgia. Analysis of factors responsible for hearing loss and measures to reduce the incidence was also done.

**Methods and Material:** A Retrospective analysis of records of all patients who underwent MVD for trigeminal neuralgia at Bantane hospital, Fujita health University from 1<sup>st</sup> January 2014 to 31<sup>st</sup> December 2018 was conducted. This included patients with at least 3 months of follow up. Routine perioperative audiogram was not done. Audiogram was done in patients with subjective hearing loss.

**Statistical analysis used:** Quantitative data will be described in mean and qualitative data will be described in proportions. Factors responsible for good outcome will be analysed as well as those for complications.

**Results:** Total number of patients included in study was 35(n=35). Mean age was 64.1 years (range=33-83 years, SD=14.2 ). Mean follow up period was 11.4 months. Complete pain relief (E0) was achieved in 27 patients(77.1%) at the time of discharge. Postoperative hearing loss was found in one male patient which was confirmed by audiometry. Postoperative audiogram showed left side profound SNHL and right side mild to moderate SNHL.

**Conclusions:** MVD is safe and effective surgical technique for trigeminal neuralgia. Hearing loss is rare but one of the most disastrous complications, which should be minimized by using all possible measures.

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**Keywords:** Hearing loss, Microvascular decompression, Trigeminal neuralgia

**Key message:** Hearing outcome is a disastrous complication after MVD surgery for trigeminal neuralgia. All the possible efforts should be made to minimize the incidence of hearing loss after MVD.

### Introduction

Microvascular decompression (MVD) is considered as an effective and safe treatment option for trigeminal neuralgia. Medical treatment, botulinum toxin, Gamma knife Radiosurgery (GKRS) are less invasive alternates, however, Microvascular decompression had been proven as more effective and long lasting option.<sup>[1]</sup> Complications of MVD include facial numbress, facial paresis, aseptic meningitis, wound infection, CSF leak through wound or CSF rhinnorhoea and hearing loss.<sup>[2]</sup> Hearing loss is one of the most dreadful complications following MVD. Sensory Neural Hearing Loss (SNHL) can be caused due to stretching of CN 8 while cerebellar retraction, direct manipulation of CN 8, injury or vasospasm of labrynthine artery and AICA acoustic trauma due to drill noise. <sup>[3–5]</sup>Fluid and entering the mastoid air cells may cause middle ear effusion leading to conductive hearing loss.<sup>[6]</sup>

Hearing loss may have very significant impact on individual's social and professional life. The aim of our study was to evaluate the incidence and severity of hearing loss following microvascular decompression in trigeminal neuralgia. We also tried to analyze the factors which might be responsible for this complication, so that it can be further minimized in future. Role of intraoperative adjuncts like Neurophysiological monitoring (BAER), Dual image angiography (DIVA) and endoscope was video evaluated in terms of outcome.

## **Subjects and Methods**

A Retrospective analysis of records of all patients who underwent MVD for trigeminal neuralgia at Bantane hospital, Fujita Health University from 1<sup>st</sup> January 2014 to 31<sup>st</sup> December 2018 was conducted. This included patients with at least 3 months of follow up. Preoperative evaluation included MRI brain SPGR sequence and CT angiography. MRI and CT angiography images were fused to clearly demonstrate the neurovascular conflict.(Figure 1) Pure tone audiometry was not performed routinely in all patients. Surgical technique: (Figure 2)

All patients were operated in lateral decubitus position. Small retromastoid craniotomy was made. Dura opened in curvilinear fashion based on sigmoid sinus and laterally tented with tack-up sutures. Cisterns were fenestrated to release CSF and relax the cerebellum. Sharp arachnoid dissection was performed around cranial nerves except CN 8. Endoscope was used at this time for identification and better anatomical orientation of offending vessel loop. Machida right angle endoscope were used which is easy to introduce deep through narrow working corridor. Teflon pledget was placed between the vessel loop and V nerve. Transposition of the vessel loop was preferred method, whenever feasible. Primary dural closure was performed in all cases.

Intraoperative Brainstem auditory evoked potential:

BAEP monitoring was performed using Nihon Kohden system. Auditory stimulus was delivered at an frequency and intensity 11.1 Hz and 105 db respectively through earphones. Baseline recordings were done. Increase in wave V latency by >1.0 ms and fall in amplitude by more than 50 % was considered significant. Postoperatively, Outcome was assessed using scoring system given by Kondo et al.<sup>[7]</sup> Hearing was assessed subjectively, however, PTA was performed in patients who complained hearing loss.

## Statistical analysis

Quantitative data were described in mean and qualitative data were described in proportions. Factors responsible for good outcome were analysed as well as those for complications.

### Results

## **Patient demographics**

Total number of patients included in study was 35(n=35). Mean age was 64.1 years (range=33-83 years, SD=14.2). There were 13 (37.1%) male and 22 female patients (62.8%). All the patients presented with unilateral typical trigeminal neuralgia pain. (Table 1)

### **Operative findings**

Most commonly encountered offending vessel was SCA (62.8%) followed by AICA(11.4%). In one case, no vessel loop was found intraoperatively. In three cases (8.5%), two loops were found compressing the nerve. Vein was found culprit in two cases (5.7%). Out of total 38 vessel loops, Interposition and transposition was done in 22 (57.8%) and 16 (42.1%) cases respectively. Endoscope and dual image video angiography (DIVA) was used in all cases.

Age range	33-83	years
	(Mean=64.17,SD=14.29)	
Sex distribution	Male :Female = 13:22	
	SCA-	
	22 (62.85%)	
	AICA-	
	4 (11.42%)	
	AICA,SCA-	

Table 1: Patient demographics and operative findings.

Offending vessel	2 (5.71%)	
	VA-	
	2 (5.71%)	
	PICA,AICA-	
	1 (2.85%)	
	PERFORATOR-	
	1 (2.85%)	
	LATERAL PONTINE VEIN-	
	1 (2.85%)	
	PETROSAL VEIN-	
	1 (2.85%)	
	NO VESSEL-	
	1 (2.85%)	
Procedure done	Interposition= 22(57.8%)	
(Interposition/tran	Transposition=16(42.1%)	
sposition)		
	1	

#### Outcomes

**Freedom from pain:** Mean follow up period was 11.4 months. Complete pain relief (E0) was achieved in 27 patients(77.1%) at the time of discharge. Moderate pain persisted(E2) in 6 patients(17.1%) which was controlled with medicines. There was no relief from pain in 2 (5.7%) patients.

**Hearing loss:** Postoperative hearing loss was found in one male patient which was confirmed by audiometry. Offending vessel in this case was SCA which was transpositioned during surgery. This patient was completely pain free postoperatively.

Postoperative audiogram showed left side profound SNHL and right side mild to moderate SNHL (Figure 3)

Retractor was not applied during surgery. During arachnoid dissection there was increase in wave V latency by 1 millisecond and drop in amplitude by more than 50%,after which dissection was stopped till wave V latency and amplitude came back to normal.(Figure4) At the end of procedure, V wave latency and amplitude was same as baseline.

### Discussion

Typical trigeminal neuralgia is characterized by unilateral, paroxysmal, severe, stabbing or lancinating pain in distribution of one or more branches of CN V, <sup>[8-10]</sup>usually provoked by sensory stimuli like chewing, brushing, shaving etc. <sup>[11]</sup>Annual reported incidence of TN is 4 per 1,00,000.<sup>[12,13]</sup> Peak incidence lies between 50 to 60 years of age and prevalence gradually increases with age.<sup>[13]</sup>It is rarely seen in paediatric population.<sup>[14]</sup>

Amidst all controversies regarding the pathophysiology of disease, most commonly accepted etiology is segmental demyelination of sensory branch of trigeminal nerve at root entry zone or brainstem, with chronic compression when it comes out of pons. <sup>[11,13,15,16]</sup>This chronic compression leads to an ephaptic transmission at the site of microinjury.<sup>[16]</sup> Peripheral myelin is more resistant to compression as compared to central or transition.<sup>[17]</sup> Common cause of compression is an arterial loop (Figure 5) or a vein, however focal arachnoid thickening, aneurysm, AVM, cerebellopontine angle tumours can also cause TN.<sup>[18-20]</sup> Bioresonance theory proposed by Jia and Li states that when the vibration frequency of a structure surrounding the trigeminal nerve becomes close to its natural frequency, the resonance of the trigeminal nerve occurs.<sup>[21]</sup> This bioresonance can damage nerve fibers and lead to the abnormal erratic transmission of the impulse, which may finally result in facial pain. Devor et al proposed ignition hypothesis which states that TN results from specific abnormalities of trigeminal afferent neurons in the trigeminal root or ganglion. Injury renders axons and axotomized somata

hyperexcitable. The hyperexcitable afferents, in turn, give rise to pain paroxysms as a result of synchronized after discharge activity.<sup>[22]</sup>

Treatment options include medical therapy, ablation techniques, Gamma knife percutaneous radiosurgery and Microvascular decompression. Medical treatment is first line of treatment. <sup>[2,23]</sup>Patients refractory to medical treatment are candidates for other options. Carbamazepine (CBZ) and oxcarbazepine (OCZ) are the preferred agents.<sup>[2]</sup> Although CBZ is more effective,<sup>[24,25]</sup>OCZ has got better safety profile. <sup>[26]</sup>Other drugs like Baclofen, phenytoin, gabapentin, levetiracetam, valproate, pregabalin, topiramate, clonazepam and lamotrigine are used as alternate treatment. <sup>[27–32]</sup> Botulinum toxin can be tried in patients who are unfit or not willing for surgery and in postoperative cases with recurrence of pain.<sup>[33,34]</sup>

Percutaneous ablative procedures include Radiofrequency thermocoagulation(RFT), Balloon compression(BC) and percutaneous glycerol rhizolysis(PGR) of gasserion ganglion. Facial sensory loss is the most common side effect followed by dysaesthesias, anaesthesia dolorosa and keratitis. [35-<sup>37]</sup>In view of these distressing side effects and shorter pain free period in comparison to MVD, these procedures are generally reserved for elderly patients with multiple comorbidities, multiple sclerosis patients, recurrent pain after MVD and patients with impaired hearing contralateral side. Gamma knife on Radiosurgery (GKRS) delivers a focused beam of radiation to trigeminal nerve root in CP angle cistern. Regis et al evaluated long term safety and efficacy of GKRS in 497 patients with trigeminal neuralgia and they found rate of maintaining pain relief was 67.8 % at 10 years. Facial hypoaesthesia rate was 20.4 % at 5 years and 21.1% at 7 years and then remained stable till

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14 years. Very bothersome facial hypoaesthesia was seen in 0.6% patients.<sup>[38]</sup>

MVD is the most effective method of treatment. In experienced hands, immediate postoperative pain free condition can be achieved in around 90% of the patients. <sup>[1,2]</sup>Most commonly encountered offending vessel is SCA followed by AICA and veins.<sup>[39–41]</sup> Postoperative complications, although not common, include aseptic meningitis, CSF leak, sensory loss and hearing impairment.<sup>[2]</sup>Hearing loss can be conductive or SNHL. Conductive HL are usually transient and caused due to middle ear effusion. Major causes of SNHL are cochlear nerve damage due to cerebellar retraction and vasospasm of labrynthine artery.<sup>[3,4]</sup>

In the literature, incidence of hearing loss after MVD for trigeminal neuralgia is variable. In the study conducted by Leo et al, incidence of hearing loss was 1.7%<sup>[42]</sup> whereas Thirumala et al reported incidence is around 30 %.<sup>[3]</sup> This variation is largely because of different methods of hearing assessment. Some studies use subjective method where as some routinely use perioperative audiogram. As per the meta-analysis done by Bartindale et al, which included 7093 patients operated for TN, the overall prevalence of hearing loss was 5.58%. Incidence of hearing loss was higher in studies using intraoperative BAER.<sup>[43]</sup> In our study, BAER was used in all the cases and incidence of hearing loss was 2.8%.

Intraoperative Brainstem auditory evoked potential (BAEP) is an useful monitoring adjunct in posterior fossa MVD surgery.<sup>[43,44]</sup> As per American society of Neurophysiology monitoring recommendations, surgeon should be alerted when wave V amplitude decreases by >50% or latency increases by >0.1 ms. <sup>[44]</sup>However, Dannenbaum et al reported 114 cases of

MVD without using BAEP monitoring, in which they found comparable results with those studies using monitoring.<sup>[45]</sup>

Intraoperative use of rigid retractors especially for longer duration is one of the important causes of hearing loss after MVD and it should be avoided.<sup>[46,47]</sup> Suction cannula can be used for gentle retraction of cerebellum. Early CSF drainage also helps in relaxing cerebellum and minimizing the need for retraction. Even in the cases where retractors are required, it should not be applied along the VIII nerve as it may produce tension on the REZ of nerve, leading to hearing disturbances.

Lee et al studied the correlation between cerebellar retraction time and intraoperative BAER changes and emphasized the greater distance between the cerebellar surface of petrous temporal bone and point of neurovascular conflict as an important risk factor for SNHL.<sup>[5]</sup>Some authors also advocate inferolateral retraction of cerebellum to avoid stretching of cochlear nerve.<sup>[48]</sup>

Spasm of labrynthine artery is another major cause of hearing loss after MVD. Morawski et al demonstrated use of topical papaverine in reversal of internal auditory artery vasospasm and its imact on cochlear nerve functions in animals.<sup>[49]</sup> Scavo et al also reported prophylactic effect of diluted papaverine in preventing hearing loss during MVD for trigeminal neuralgia.<sup>[50]</sup>

Intraoperative use of endoscope is quite helpful in visualization of vessel loop near REZ. It also helps in minimizing cerebellar retraction and related complications.<sup>[42,51]</sup> In the study by Teo et al, 113 patients underwent endoscopy assisted MVD. Freedom from pain was achieved in 99.1% patients and 1.7% developed hearing loss.<sup>[42]</sup> Indocyanine green angiography can be helpful intraoperatively for

delineating neurovascular conflict in cerebellopontine angle cistern.<sup>[52]</sup> we routinely use dual image video angiograpgy(DIVA) during MVD.

## Limitations

We acknowledge certain limitations of our retrospective study. Most important is the small number of cases as compared to other studies. Perioperative audiogram had not been performed routinely which might have changed our results.

### Conclusion

MVD is safe and effective surgical technique for trigeminal neuralgia. Hearing loss is rare but one of the most disastrous complications having significant impact on patients life. Incidence can be further decreased using proper surgical techniques and various intraoperative adjuncts like BAER monitoring, use of endoscope and Indocyanine green (ICG) or dual image video angiography (DIVA).

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## **Legend Figure**







Figure 2

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Figure 5