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Safety and effectiveness of microvascular decompression for treatment of hemifacial spasm through mini craniotomy

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Abstract: *Background:* The hemifacial spasm (HFS) defined as involuntary intermittent twitching of the muscles of the face (usually unilateral). The spasms characteristically begin around the eye and then extend to affect other muscles of the ipsilateral face. It is caused by vascular element compressing the facial nerve that may be either the anterior or the posterior inferior cerebellar arteries in most cases. *Objective of our work:* to describe the operative technique (pearls and common mistakes), the efficacy and morbidity of microvascular decompression technique for hemifacial spasm through mini craniotomy, determine the prognostic factors affecting success rate of the surgery. *Material & method:* A retrospective study of 23 cases of hemifacial spasm treated by mini craniotomy retro sigmoid approach and microvascular decompression at neurosurgery dept., Mansoura University Hospital in last 10 years was investigated. This include Epidemiological, clinical and imaging details, selected treatment options and patients' outcome. *Results:* complete resolving of symptoms was conducted in 19 cases 82.6% while reoperated in 2 cases with improvement in one case. Facial palsy appeared post-operative in 6 cases 4 of them improved in 3 months, transient hearing loss in 4 cases 17.4% which improved later, cerebrospinal fluid leak appeared in 3 cases 13% which managed conservatively. *Conclusions:* MVD relieves symptoms of HFS in about 80% of patients while recurrence still in low percentage. The study reported low permanent Complications and generally transient.

Key words: Hemifacial spasm, vascular decompression, microvascular, face muscle twitches, micro vascular surgery

Introduction

Hemifacial spasm (HFS) usually defined as an involuntary, unilateral in most cases,

paroxysmal, tonic-clonic facial musculature contraction that is innervated by ipsilateral facial nerve “the seventh cranial nerve”. In

majority of the cases these twitches begin around the eyes “orbicularis oculi muscle “then spread progressively to the cheek, mouth, and neck “platysma “inferiorly & frontalis (i.e., muscles of the forehead) superiorly muscles in severe cases. [1]

The incidence of HFS is around 0.8 per 100,000 persons. [2] While others reported higher prevalence of HFS as 9.8 per 100,000 persons [3] or even higher in worldwide as 14.5 per 100,000 women and 7.4 per 100,000 men [2, 4]. Maybe this due to underdiagnoses lead to reduction of the cases discovered [5]

The aetiology of the primary HFS is vascular element compressing the facial nerve at root (exit or entry) zone. this vascular element may be the anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), vertebral artery (VA), veins ,perforators or even un-named vessels ,especially if there was lateral deviation of one or both vertebral arteries that play as major risk factor [6,7]. Usually the compression occurred as a result of the vessel loop indenting the facial nerve or arachnoid thickening between the brainstem and the offending vessels stretching the nerve during pulsations[8] .some cases showed more than one offending vessel and may be this one of causes of microvascular decompression [9].in some case unfortunately did not have any recognisable cause [10].

The pathophysiology of HFS hypothesis speculate demyelination of the facial nerve at (entry -exit) nerve zone that is termed as the alteration site between central zone myelination by (oligo-dendrocytes) and peripheral zone myelination by (Schwann) cell myelination which is enclosed by

arachnoid membrane without any inter-fascicular connective tissue or epineurium isolating the fibres leading to more susceptibility to injury by compression [11] the second hypothesis of HFS stated the ectopic excitation and ephaptic transmission along the facial nerve due to demyelination lead to emerging an abnormal impulses at lower threshold level that spread through the facial nerve making short circuit at the site of the lesion[12] . the last but not the least of HFS theory conducted that central demyelination with subsequent connection reorganization of the affected facial nucleus making it hyper excitable because of dendritic spike formation [13] Sympathetic hypothesis suggested the sympathetic endings in adventitia of the offending vessels induce abnormal action potential at neuromuscular junction inducing hemifacial spasm [14]

The diagnosis of primary HFS by presence of “brow-lift sign” that consist of eyebrow lifting with the same side eye closure, triggering the activity of the frontalis in addition to orbicularis oculi muscles in the exact time during HFS. [15] Radiological investigation especially T2-weighted MRI imaging and ultra-resolution thin sections are most frequently used to demonstration possible vascular compressive elements [11]. Other new techniques in MRI like combination between steady-state MR images and three-dimensional time-of-flight MR angiography may show the approximate actual anatomy of the facial nerve at root exit zone (figure 1). [16] EMG can assist in diagnosis of HFS by appearance of high-frequency synchronized ignition [12]



Figure 1- MRI Brain T2WE thin axial cuts showed AICA compressing the facial nerve in ONE of HFS cases

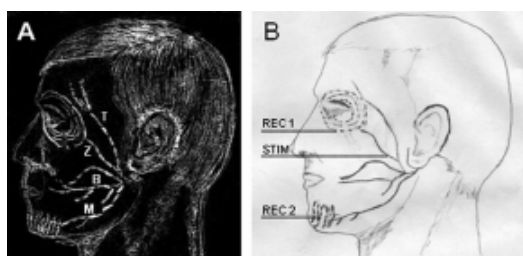


Figure 2 - A, Branches of the facial nerve (CN VII); without cervical branch. B, Monitoring for the abnormal lateral spread response (LSR). The facial nerve zygomatic branch is excited by electricity and the produced EMG response in the orbicularis oculi muscle could be recorded. In HFS patients, an abnormal induced response appeared as what is called LSR in the mentalis muscle, temporal; zygomatic; buccal; marginal mandibular. [22]

The traditional medical treatment to HFS botulinum neurotoxin injections that can relief the manifestation of HFS in approximate 85% of cases [17] but it must be repeated every 3-6 months and other drawback is tolerance beside atrophy of the injected muscles in long run [18] while medical treatment by anticonvulsive drugs show failure in most case except early and very mild HFS case who may show infrequent improvement.

The alternative curative treatment is surgical micro vascular decompression

(MVD) that give a long duration relief of manifestation of HFS .this management offering a solution for those patents who did not give response or had tolerance to botulinum toxin injections or could not withstand lifetime frequent facial nerve injections [18].The procedure principle depend on separation of the offending vessel from the facial nerve exit zoon by isolator material after sharp dissection of the neurovascular arachnoid. The corner stone depend on identification of the vessel that compressing the nerve intraoperative by neurophysiological monitoring [19]. This intraoperative neurophysiological monitoring includes monitoring of brainstem auditory evoked (BrAI) responses is usually applied to discover eighth nerve dysfunction [20]. It also includes monitoring and recording of lateral spread response (LSR) which can intraoperatively give the surgeon a proof of adequate neurovascular decompression. LSR provoked by electrical stimulation of facial nerves branches that led to electrophysiological specific waves denoting HFS which disappear after adequate neurovascular decompression. The value of LSR as predictor of surgical outcome still controversial [20-22]

Objective of our work

To describe the operative technique (pearls and common mistakes), the efficacy and morbidity of microvascular decompression for HFS through mini craniotomy, determine the prognostic factors affecting success rate of the surgery.

Material & method

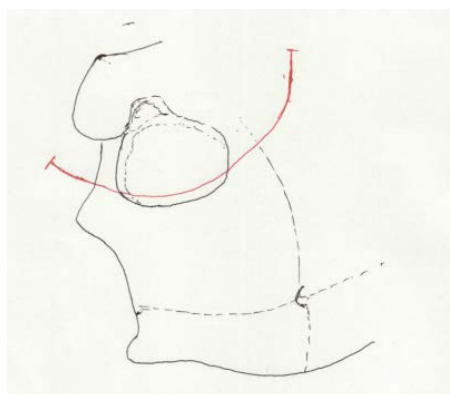
A retrospective study of 23 cases of HFS treated by mini craniotomy retro sigmoid approach and microvascular decompression at neurosurgery dept., Mansoura University Hospital in last 10 years was investigated. This include Epidemiological, clinical and imaging details, selected treatment options and patient's outcome. All patient in our study suffering from primary HFS. The patients had secondary HFS due to tumors, vascular aneurysms besides arteriovenous malformations AVM in the cerebello-pontine angle site as discovered by standard head CT or MRI investigation were excluded. Also, patients with severe systemic sickness and patients with HFS due trauma viral infections have been disregarded.

The surgical technique of lateral suboccipital approach:

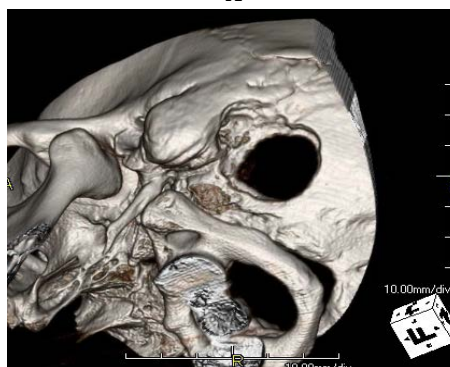
- General anaesthesia
- The lateral position. “military position” with the affected side up
- the neck is slightly in flexed position with turning the head a few degrees to the ipsilateral side of the proposed craniotomy
- Linear or curved skin incision. (figure 3: showed intraoperative picture anatomical landmark of the mastoid process and the position of planned mini craniotomy)
- burr hole at the junction of the transverse and sigmoid junction
- quarter coin craniectomy or craniotomy (figure 3)



Figure 3 - Showed intraoperative picture anatomical landmark of the mastoid process and the position of planned mini craniotomy



A



B

Figure 4 - A showed the planned curved skin incision & mini craniotomy of HFS case to do MVD. B: showed CT 3D of operated MVD OF HFS case revealed the retro mastoid quarter coin craniectomy

- the medial edge of the sigmoid sinus is exposed by drilling the mastoid bone
- The opened mastoid air cells during craniotomy were closed by bone wax
- inferolateral cerebellar approach using the microscope
- slowly release of CSF in the cerebellopontine cistern
 - superior petrosal and supra cerebellar veins injury
- opening the basolateral cisterns by elevating the flocculuscerebelli carefully
- in some cases, we adopt Jannetta technique who has suggested a method for protecting the cerebellar cortex during the advancement of the cottonoid around the cerebellum by lining it with a piece of glove (called rubber dam)
- we used Fukushima teardrop suction tube and a micro scissors for dissecting the arachnoid
- The arachnoid membranes just inferior to superior petrosal and supracerebellar veins is sharply fenestrated and an additional amount of CSF released. The arachnoid that surround both VII/VII cranial nerves is usually left intact. The retractor blade is placed barely below the superior petrosal vein
- Special attention was given to labyrinthine artery which enters the internal auditory canal beside the VII/VIII cranial nerves complex
- Retraction is parallel to the glossopharyngeal course. The retractor blade is then placed on the cerebellum just superficial to the visible part of cranial nerve IX
- Aggressive manipulation of the nerve should be avoided
- The root exit zone of the facial nerve appears grayish, anterior and slightly inferior to eighth cranial nerve that have whit color appearance and may be directly visualized upon gentle elevation of nerve VIII using a fine dissector
- Adequate exposure of the root entry zone of the facial nerve to see the offending vessels that compressing the facial nerve.
- The technique of decompression was by interposition of Teflon sponges between the nerve and the offending blood vessel (ball-shaped). Overzealous use of the Teflon implant should be avoided.
- Not only using Teflon as interposition but also making loop with Teflon & fibrin glue to pull the vessel away from the Facial Nerve “sling retraction method”
- filling the surgical cavity by saline to release air bubbles
- tight closure of the dura the continue closure in anatomical layers

Results

Average patient age was 51.5 ± 11.3 years (range, 23 – 64 years), with ratio female to male was 1.55:1. The mean follow-up period was 38.4 ± 14.3 months (range: 7 – 76.2 months)

TABLE 1
Summary of Demographic Data and Clinical Features of 23 HFS Cases

Variable		
Number of patients	23	
Gender	female	male
	14	9
Side	left	right
	19	14
Botox usage	12	
H-B classification of facial weakness	Grade II	16
	Grade III	5
	Grade IV	2
Preoperative symptom	Diminished corneal reflex	2
	Tinnitus	3
	Reduced hearing	5
	Tonus	14
	Platysmal involvement	11
	Specific triggers	8
Compressing Vessel	AICA	10
	PICA	8
	VA	3
	Multiple or veins	2

AICA, anterior inferior cerebellar artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery.

Complete resolving of symptoms was conducted in 19 cases 82.6% while reoperated in 2 cases with improvement in one case. Facial palsy appeared post-operative in 6 cases 4 of them improved in 3 months, transient hearing loss in 4 cases 17.4% which improved later, cerebrospinal fluid leak appeared in 3 cases

13% which managed conservatively. Table 2 shows the success rate in relation to the time following surgery.

The complications of these surgery presented in the following diagram (figure 5).

TABLE 2
The Success Rate of MVD Surgeries in Relation to Post-Operative Periods

	NO SPASM	SPASM
Immediate Post-Operative	13	10
On Discharge	15	8
On Follow Up	19	4

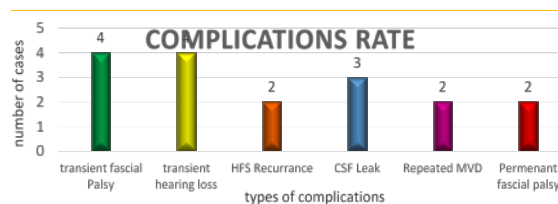


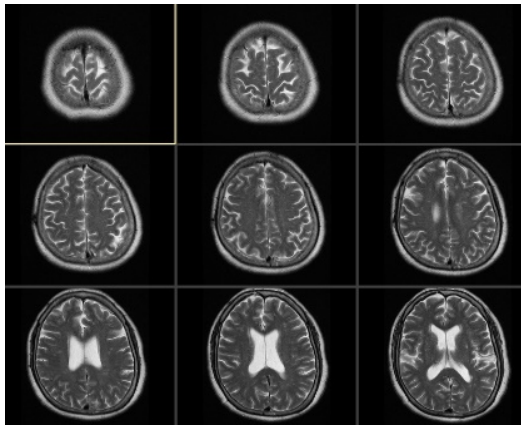
Figure 5 - showed MVD complications of our 23 HFS cases

Cases of presentations

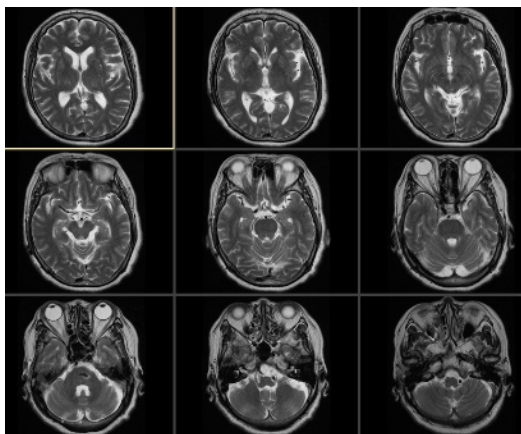
- Case 1: 62 years female complain of mild persistent left facial spasm not responding to medication and she had been injected with Botox. (Figure 6: MRI Brain Axial cuts showed no significant secondary pathology in left cerebellopontine angle). On the other hand (figure 7 which is MRI Brain T2WE topography thin axial cuts revealed compression of the left facial nerve at (root exit zone) by left PICA & Vertebral Artery).

The plan of surgery steps as described earlier in our paper (figure 8 A-H).

The actual operation steps demonstrated in figure 10 (A-Q).



A



B

Figure 6 A & B - MRI Brain Axial cuts showed no significant secondary pathology in left cerebellopontine angle

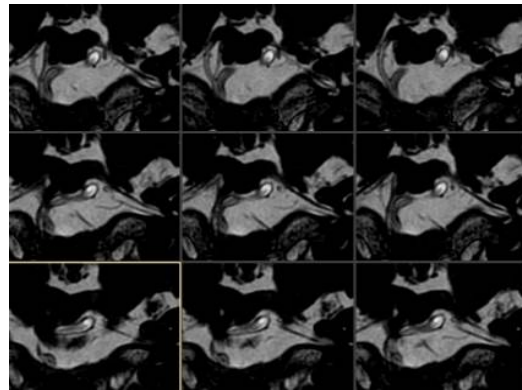


Figure 7 - MRI Brain T2WE topography thin axial cuts revealed compression of the left facial nerve in root exit zone by left PICA Artery

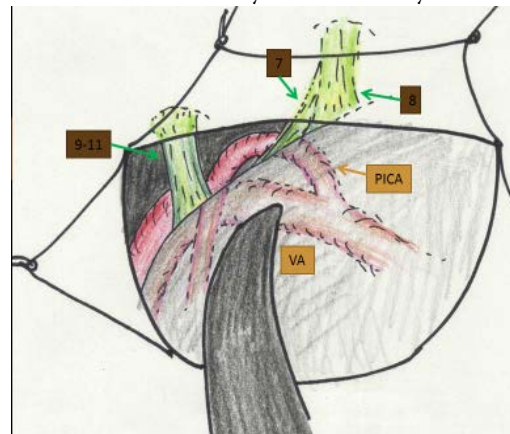


Figure 9 - The root exit zone of nerve VII (grayish in color) is anterior and slightly inferior to nerve VIII (more whitish in color) and may be directly visualized upon gentle elevation of nerve VIII using a fine dissector. Adequate exposure of the root entry zone of the facial nerve to see the offending vessels that compressing the facial nerve which is left PAICA & Vertebral artery

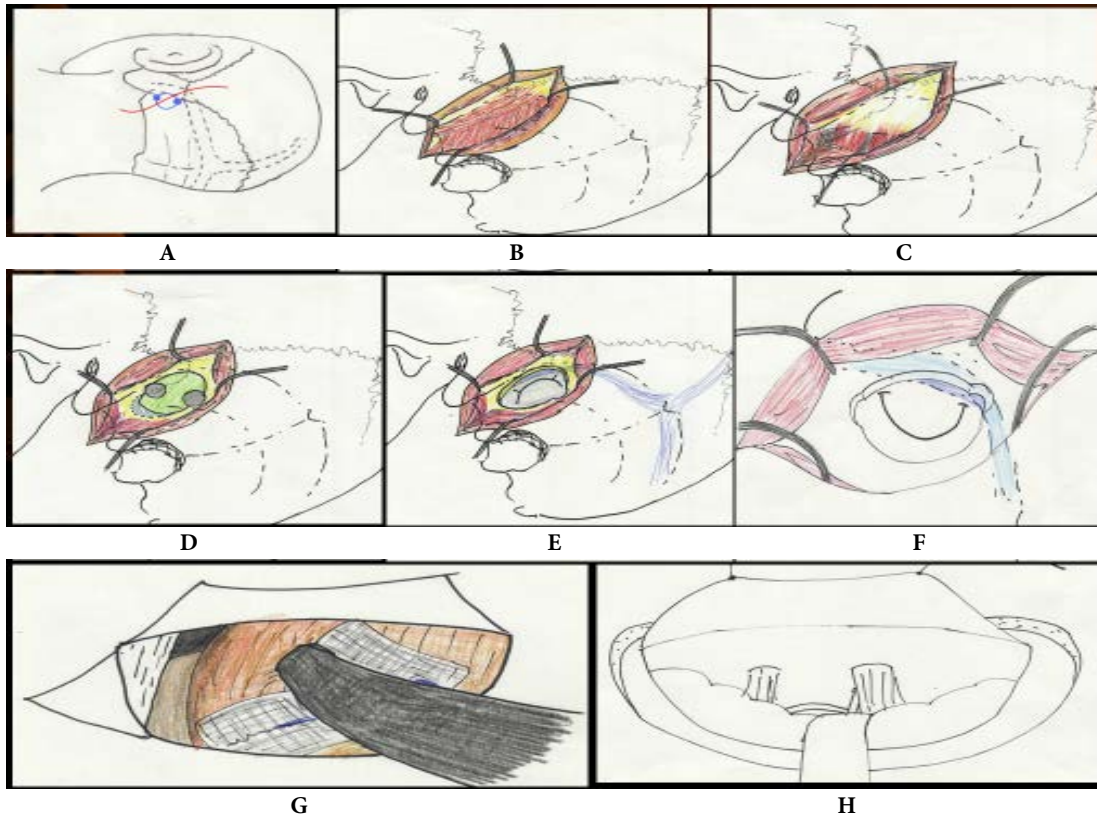
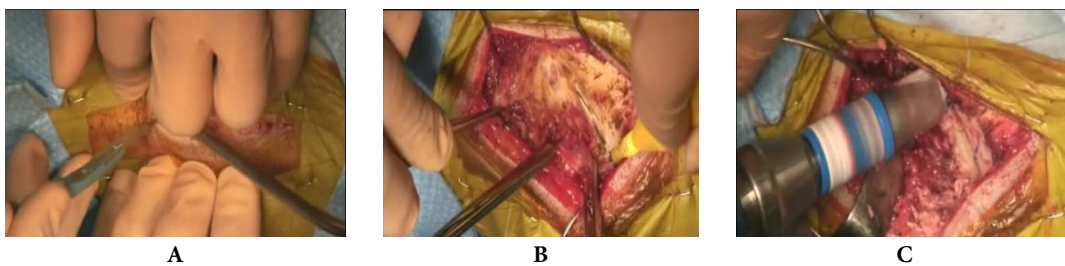


Figure 8 - A Linear or curved skin incision. B dissection of the first muscles layer .C dissection of the second muscles layer. D mini craniotomy flap in retro mastoid area E removal of the mini craniotomy free bone flap and exposure of the dura. F the medial edge of the sigmoid sinus is exposed by drilling the mastoid bone. The opened mastoid air cells during craniotomy were closed by bone wax. G infralateral cerebellar approach using the microscope. H opening the basolateral cisterns by elevating the flocculus cerebelli carefully



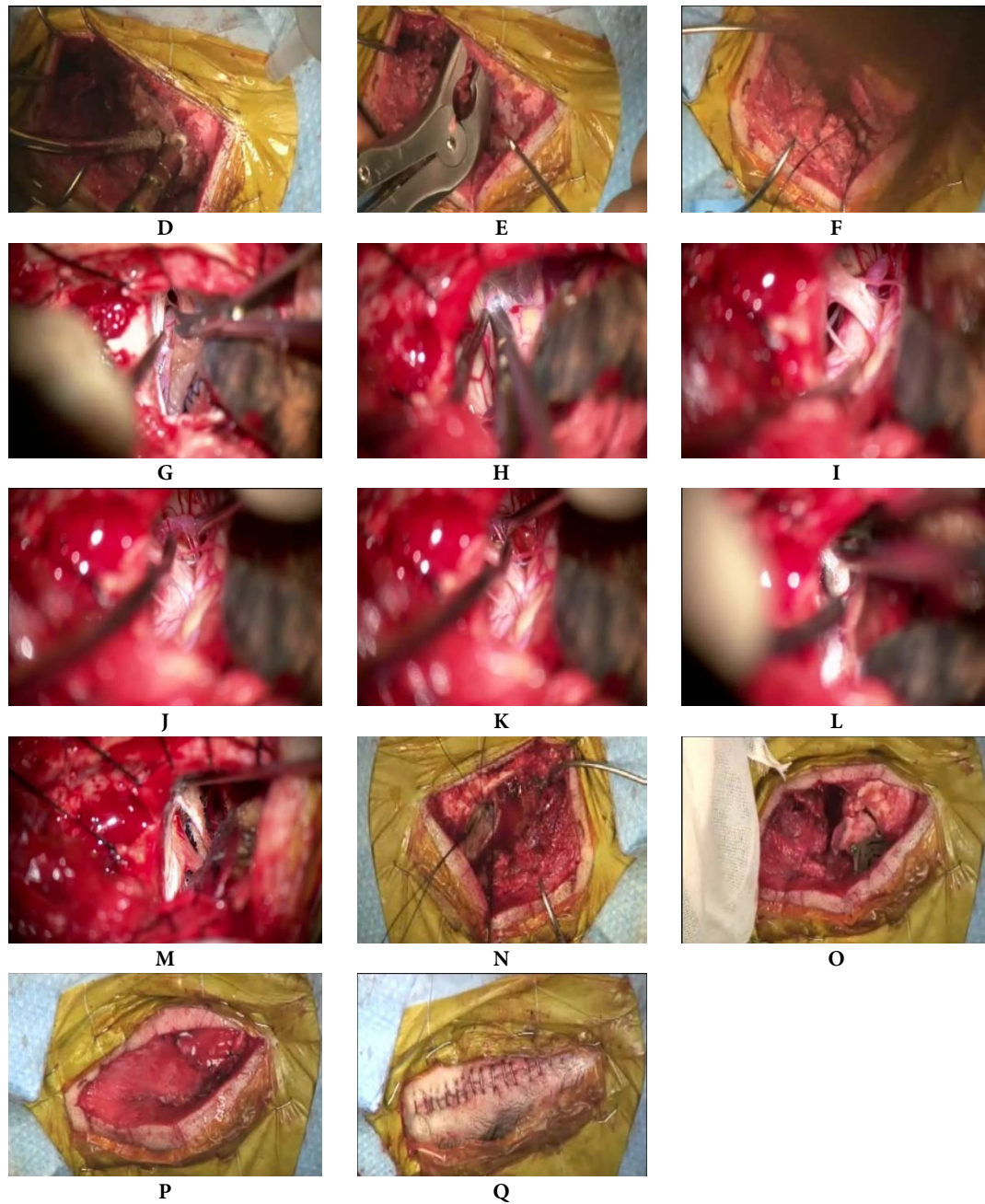


Figure 10 - A Linear skin incision retro mastoid. B dissection of the muscles layers. C making retro mastoid burr hole. D mini craniotomy flap in retro mastoid area. E the medial edge of the sigmoid sinus is exposed by removing some bone of the mastoid & surrounding bone. The opened mastoid air cells during craniotomy were closed by bone wax. F opening the dura & making fixing stitches to keep it opened. G inferolateral cerebellar

approach using the microscope. H opening the basolateral cisterns by elevating the flocculus cerebelli carefully. I The root exit zone of nerve VII (grayish in color) is anterior and slightly inferior to nerve VIII (more whitish in color). Adequate exposure of the root entry zone of the facial nerve to see the offending vessels that compressing the facial nerve which is left PAICA & Vertebral artery in this case. J microscopic picture show the offending vessel that compress the left facial nerve which is PAICA in this case and dissecting all the arachnoid surrounding it. K microscopic picture revealed retracting left PAICA away from the left facial nerve without injuring any branches. L interposition of Teflon sponges between the nerve and the offending blood vessel (ball-shaped). M making loop with Teflon & fibrin glue to pull the vessel away from the Facial Nerve “sling retraction method”. N tight dural closure after filling the cavity with saline. O fixing the mini craniotomy flap by bur hole plate. P muscles layer closure by stitches anatomically. Q skin closure in two layers

The patient improved on discharge.

- Case 2: 50 years male had left facial spasm 13 years ago. He had history of Botox injection with some weakness in orbicularis oculi 5 years ago. MRI Brain T2WE topography thin cuts showed compression of the left facial nerve by AICA “Anterior Inferior Cerebellar Artery “(figure 11).

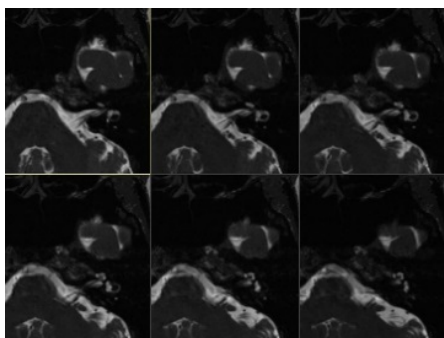
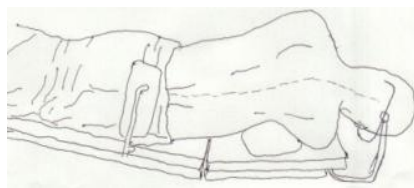


Figure 11 - MRI Brain T2WE topography thin axial cuts revealed compression of the left facial nerve in root exit zone by left AICA Artery

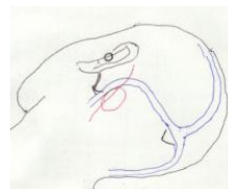
The plan of surgery steps as described earlier to relief the Aica artery pressure on left facial nerve (figure 12 A-C: An Operative patient position. B retro mastoid surgical skin incision & mini craniotomy target. C interposition of Teflon sponges between the nerve and the offending AICA artery (ball-shaped)

shaped) & making loop with Teflon & fibrin glue to pull the vessel away from the Facial Nerve “sling retraction method”).

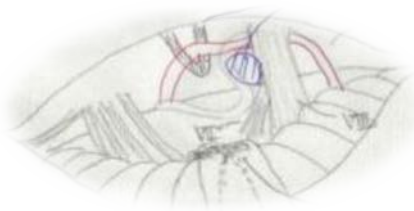
Operative steps present in (figure 13 A-I).



A



B



C

Figure 12 A-C - A Operative patient position. B retro mastoid surgical skin incision & mini craniotomy target. C interposition of Teflon sponges between the nerve and the offending AICA artery (ball-shaped) & making loop with Teflon & fibrin glue to pull the vessel away from the Facial Nerve “sling retraction method”

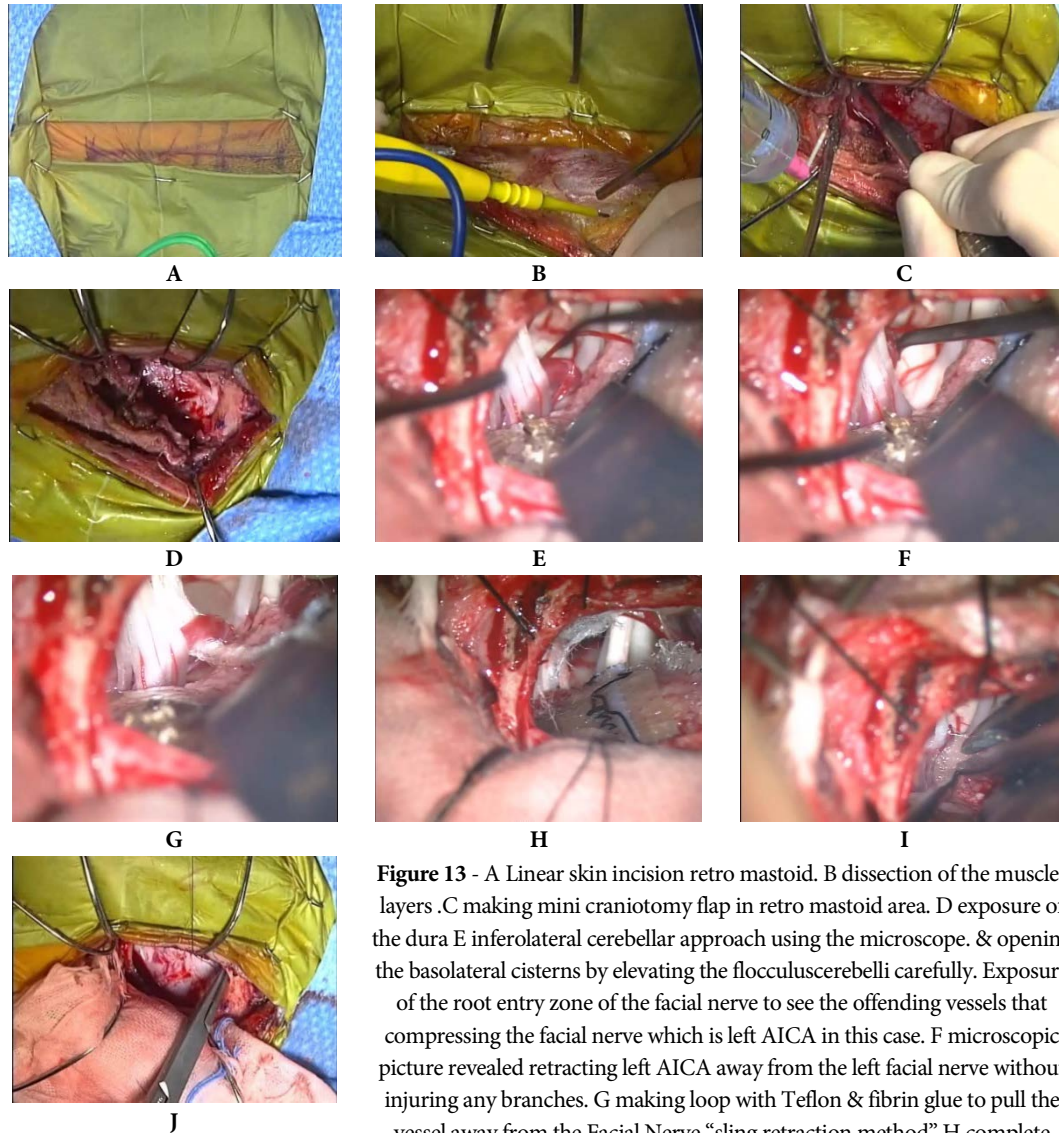


Figure 13 - A Linear skin incision retro mastoid. B dissection of the muscles layers .C making mini craniotomy flap in retro mastoid area. D exposure of the dura E inferolateral cerebellar approach using the microscope, & opening the basolateral cisterns by elevating the flocculus cerebelli carefully. Exposure of the root entry zone of the facial nerve to see the offending vessels that compressing the facial nerve which is left AICA in this case. F microscopic picture revealed retracting left AICA away from the left facial nerve without injuring any branches. G making loop with Teflon & fibrin glue to pull the vessel away from the Facial Nerve “sling retraction method” H complete relieving the compression on the left facial nerve I interposition of Teflon sponges between the nerve and the offending blood vessel (ball-shaped). J tight dural closure after filling the cavity with saline.

The patient HFS diminished immediate post-operative & Follow up MRI Brain T2WE topography thin axial cuts revealed relief all

the compression of the left facial nerve at (root exit zone) caused by left AICA Artery.

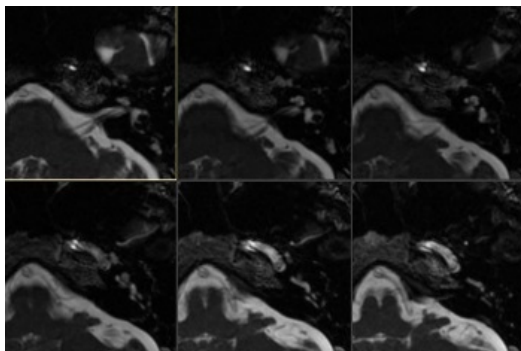


Figure 14 - Follow up MRI Brain T2WE topography thin axial cuts revealed relief all the compression of the left facial nerve in root exit zone by left AICA Artery

Discussion

In our research HFS female patients represented about 60.9% of all cases and represent three of four failed MVD cases. The offending vessels in our cases was AICA 10 cases, PICA 8 cases, VA 3 cases and multiple vessels & veins 2 cases which like what conducted by many previous researches [7, 23, 24].

MVD surgery has become more popular as definitive treatment for symptomatic primary with increasing its safety and effectiveness. Despite its postulation by J. Gardner in 1962 in limited number & result, and its modification by Jannetta and others but the following forty years made it with better reputation by improved outcomes, less complications and preservation of facial & auditory nerve structure & function. [25]

Our success rate of MVD of facial nerve to relief HFS on follow up was 82.6%. In reviewing the previous papers about the effectiveness of MVD for HFS as definitive management we found its cure rates results

extending roughly from 70.00% to 94.70%. Maybe we did not reach the optimum as over 90% in the cases or more due to small number of cases that make the statistics not accurate, absence of endoscopic usage to see the area before of the facial nerve REZ (2-3 mm) which cannot be visualised by microscope due to limited craniotomy bone window and condensed anatomical strictures in posterior fossa. Many literatures stated that the angled lens endoscope can assists the surgeons in finding the compressing vessels, checking the Teflon material position and appropriate volume in 100% of the cases. [26] The cases in our series showed success rate rise in follow up period. This may be due to period needed for remyelination of the impaired portions of facial nerve, in addition to the reappearance of normal facial moto nucleus excitability [21]

The previous application of Botox injection in 12 cases did not influence MVD operations success rate in 23 HFS cases statistically which conducted by several papers. [22] We can suggest the cause of that, absence of sever muscle atrophy in territory of affected facial nerve beside reversible of its damage.

The corner stone in this operation type is identification of the offending vessel that have many features indicate its involvement like presence of its loop compressing the facial nerve at REZ, presence of brown atherosclerotic plaque at this area, presence of depression of facial nerve opposite this offending vessel especially due to operative positioning and brain shift after opening of the dura may making the offending vessel away from the facial nerve. [26, 27] the MVD success rate did not show any statistically

significant between young and elderly patients that goes with statement of Sekula et al. [25]

The failed 4 cases had many characteristics: one of them was reoperated upon and HFS was improved post 2nd MVD operation. The reason of failure can be explained by inadequate facial nerve decompression, missed another offending vessel or not shredded Teflon sponge migration against the involved facial nerve as conducted by many studies. [24] in the light of the previous causes, in our case who improved after 2nd MVD, the cause was inadequate facial nerve decompression as a sequela of Teflon implant migration while the other three cases despite adequate MVD decompression they did not show any HFS improvement including the other case who had 2nd MVD exploration. Many reasons can clarify this result. One of it: all 3 cases had Platysmal involvement that indicate sever HFS [22]. This also strength the theory conducted that central demyelination with subsequent connection reorganization of the affected facial nucleus making it hyper excitable because of dendritic spike formation [13] so the cause may be related to monotonous active facial nerve nucleus. 2 patients from the three cases have facial palsy grade 4 and the last one was grade 3 that also indicate severe facial nerve impairment that might be not reversible despite adequate MVD.

The permanent facial nerve palsy appeared in two cases post MVD operation. The aetiology may be facial nerve stretching in the step of cerebellar retraction or dissection in cerebellopontine angle area. Heat may produce damage to nearby vessels and nerves after bipolar coagulation was applied in

cerebellopontine angle critical area. Inappropriate stimulation of nerves during dissection in addition to stripping in surgery can cause internal auditory artery spasm or rupture. [26]

The rest of complications in our series had limited patients' numbers (Table 3) which comparable with the complications in 5700 cases who have been treated by MVD presented in 22 studies summarized in the following diagram (figure 15). [28]

TABLE 3
Complications of MVD in 23 HFS cases

Complications	Number of cases
Transient facial palsy	4
Transient hearing loss	4
Recurrent HFS	2
CSF Leak	3
Repeated MVD	2
Permanent facial palsy	2

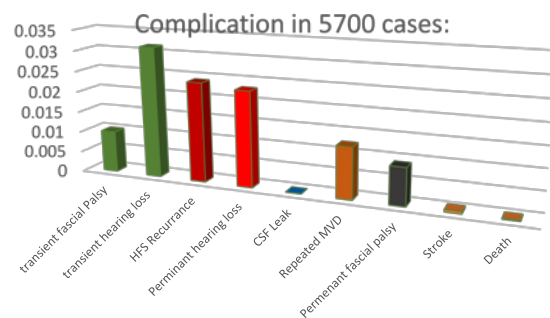


Figure 15 - The complications in 5700 cases who have been treated by MVD presented in 22 studies [28]

Other complications were transient and managed conservatively i.e. transient facial palsy and transient hearing loss that improved in 6 weeks as a result of resolving the edema of facial& auditory nerve while CSF Leak cases

improved in two weeks by medical treatment & lumbar drain. No cases in our series showed hearing deficit postoperative in spite its presence in other papers [28]. Thus, could be as a result of little patients' number and absence of audiometry for all cases pre- & post MVD surgeries. We depended on the complaint of the patients about their hearing postoperative.

Conclusions

MVD relieves symptoms of HFS in about 80% of patients while recurrence still in low percentage. The study reported low permanent Complications and generally transient.

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