SURGICAL MANAGEMENT OF TRIGEMINAL NEURALGIA BY MICROVASCULAR DECOMPRESSION

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ABSTRACT

Objective: To find out the results of Microvascular Decompression for trigeminal Neuralgia, not responding to conservative treatment.

Material and Methods: Between January 1998 and December 2001. Fifty patients underwent Microvascular decompression for trigeminal neuralgia in the Department of Neurosurgery, Hayatabad Medical Complex, Peshawar. There were 36(72%) male and 14(28%) female patients. Among them 48(96%) were unilateral cases of trigeminal neuralgia. Right side was involved in 18(36%) and left side in 32(64%). All patients underwent Microvascular Decompression.

Results: During surgery, Superior Cerebellar Artery was found to be the offending vessel in 27(54%), Anterior Inferior Cerebellar Artery in 10(20%), Posterior Inferior Cerebellar Artery in 01(02%), Unnamed vessel in 04(08%), Vein in 03(06%). In 03(06%) no vascular compression could be detected during the surgery. During the follow-up period 40(80%) patients had excellent results. Good results were achieved in 06(12%) and no response in 04(08%) of patients. After surgery 04(08%) patients developed transient vomiting & vertigo, 01(02%) patient developed wound infection and 01(02%) had Cerebrospinal Fluid Leak from wound. Cerebrospinal Fluid Rinorrhoea occurred in 02(04%) patients. One patient (02%) developed permanent ipsilateral Facial nerve weakness. There was 01(02%) postoperative death.

Conclusion: Trigeminal neuralgia is disease of different etiologies. Vascular compression of the trigeminal nerve roots at its entry into pons is one of the most accepted aetiology. Microvascular decompression has become one of the primary treatment of trigeminal neuralgia. We believe that in patients where medical treatment fail to respond, Microvascular decompression is the treatment of choice in trigeminal neuralgia.

Key words: Microvascular Decompression. Trigeminal Neuralgia. Vascular Compression.

INTRODUCTION

Trigeminal neuralgia has affected human being for centuries. Although different effective treatment has been devised, the basic pathology of the disease is still poorly understood. The characteristic feature of trigeminal neuralgia is severe pain of sudden onset, lasting for few seconds on one side of the face, rarely on both sides[1]. The different types of treatments available for trigeminal neuralgia include pharmacotherapy by membrane stabilizing drugs, injection of sclerosing agent along trigeminal pathway, trigeminal branch avulsion, Retrogasserian neurotomy, Percutaneous trigeminal radio-frequency thermocoagulation and Microvascular decompression of trigeminal nerve roots at brain stem.

Arterial Contacts with the dorsal root of trigeminal nerve was first described in 1929 by Dandy[2]. In 1950s Gardner and Miclos[3], and Taarnhoj[4] reported the beneficial effects of decompressing the trigeminal nerve for tic douloureux, since then Microvascular decompression has become one of the primary treatment for trigeminal neuralgia.

MATERIAL AND METHODS

This retrospective and perspective study was conducted in the Department of Neurosurgery, Postgraduate Medical Institute, Hayatabad Medical Complex Peshawar from January 1998 to December 2001. Fifty patients of trigeminal neuralgia were included, who underwent Microvascular decompression. In all cases complete relevant history was taken with special emphasis on previous medical therapy and surgical procedure for trigeminal neuralgia. Before being considered for surgery, all patients underwent a full course of medical therapy. Those who were found refractory to the medical treatment were considered for Microvascular decompression. To determine the results of surgery out come was divided into three categories. "Excellent" denoted patients that were free of medication, "Good" denoted patients with infrequent pain control with low dose medication and "poor" described patients with no post surgical relief on or off medication. (Table 1).

CHARACTERISTICS OF PATIENTS OPERATED FOR TRIGEMINAL NEURALGIA

Features			No. of patients
Total Patients			52
Unilateral Pain	Right	18	48
	Left	30	
Bilateral Pain			02
Operated MVD			50
Cerebellopontine angle Tumor			02
Mean Duration of Symptoms			8.3 years
Mean age at operation			49 years
Male /Female ratio			36/14

TABLE–1

Operative Procedure

A complete neurlogic examination was carried out in all cases. The functional status of the fifth cranial nerve was recorded. CT scan of brain was done to exclude a space occupying lesion. MRI was done in three cases due to doubt in CT scan findings. All patients were operated in lateral position with affected side up. Approximately 3cm square area behind the ear was prepared. A vertical incision 5-7 cm long was drawn 1 cm medial to mastoid process. The soft tissues were dissected. A small craniectomy was performed until the junction of transverse and sigmoid sinus was exposed. The dura was incised in "C" shaped manner at the junction of the transverse and sigmoid sinus and the dural flaps were sutured away. Under the operative microscope, drainage of Cerebrospinal fluid was done and the table was slightly rotated away from the surgeon.

A self-retaining brain retractor over apiece of cottoniod was placed on the superior surface of cerebellum. The retractor was advanced gently to facilitate more Cerebrospinal Fluid drainage. Arachnoid was dissected with micro dissector. The petrosal vein complex was identified and coagulated. On entering the Cerebellopontine angle, the seventh-eight nerve complex was visualized and the fifth nerve inferolateral to it. The fifth nerve was exposed up to its entry zone into pons. Any offending vessel compressing the fifth nerve root was identified and noted. A piece of muscle, taken from the wound edges was placed between the nerve and vessel. The retractor was removed. The dura was closed watertight. The incision was closed in layers. The patients were kept in Neurosurgical Intensive care for 24-48 hours. In all cases carbamazipine was stopped after surgery.

RESULTS

Immediate excellent postoperative pain relief was achieved in 40(80 %) of patients undergoing Microvascular decompression for trigeminal neuralgia. Good relief was archived in 06(12%) and 04(08%) had no response. During the surgery vascular compression was seen in 47(94%) cases. In 3(6%) cases no vessel could be identified. The Superior Cerebellar Artery (SCA) was compressing the nerve roots in 27(54%) patients. Anterior Inferior Cerebellar Artery (AICA) in 11(22%) cases, Posterior Inferior Cerebellar Artery (PICA) in 02(04%) cases, Unnamed vessel in 04(08%) and Vein in 3(6%) of cases were identified. (Table 2).

Follow-up

The patients were followed for 1 to 2 years after surgery. Four patients had re-currence of pain. Three were the cases in which no vessel was detected during the surgery. One patient was reoperated at the interval of one year. He improved after the second surgery. The offending vessel was missed in first operation. Carbamazipine was started in rest of the patients.

OFFENDING VESSEL/LESION IN MICROVASCULAR DECOMPRESSION FOR TRIGEMINAL NEURALGIA

Vessel/Lesion	No. of patients	Percentage
SCA	27	54%
AICA	11	22%
PICA	02	04%
Unnamed Vessel	04	08%
No Vessel Seen	03	06%
Vein	03	06%

TABLE–2

Complications

Cerebrospinal Fluid Rinorrhoea occurred in 2(4%) patients. Repeated lumbar drainage of Cerebrospinal Fluid was done and the Rinorrhoea stopped. One (2%) patient developed Cerebrospinal Fluid leak from the wound and one (2%) case developed wound infection. They were treated conservatively by intravenous antibiotics and lumbar drainage. There was one (2%) postoperative death. Six (12%) patients had transient vomiting & vertigo after surgery, which improved. One (2%) patient developed permanent ipsilateral Facial nerve weakness. (Table 3).

COMPLICATIONS OF MICROVASCULAR DECOMPRESSION

Complications	No. of patients	Percentage
Transient vomiting & Vertigo	06	12%
Wound Infection	01	02%
CSF Leak from wound	01	02%
CSF Rinorrhoea	02	04%
Facial Nerve Weakness	01	02%
Death	01	02%

TABLE–3

DISCUSSION

A large number of surgical options are available for the management of medically resistant trigeminal neuralgia. The exact aetiology, excluding tumors and demyelinating disease, has not fully clarified. Two divergent viewpoints, Central verses Peripheral were presented to explain the possible mechanism of trigeminal neuralgia. In theory of central mechanism, a tactile stimulus on face evokes a repetitive self-exciting discharge at the brain stem, which is not inhibited at central level. The peripheral theory suggest that there is compression of trigeminal nerve roots at entry into the pons by a vessel or tumor, resulting in demyelination of the nerve due its pulsatile compression. This demyelination results in short-circuiting of neuronal flow and hence trigeminal neuralgia. Microvascular decompression of the trigeminal nerve root at its entry zone has become an accepted surgical technique for the treatment of trigeminal neuralgia. This operation is based on the original observation of Dandy[2], developed by Gardner & Miklos[3] was perfected and popularized by Jannetta[5]. In our series of fifty patients, who underwent Microvascular decompression, favorable results were obtained. The mean duration of symptoms was 8.3 years, which is comparable to the 7.9 years mean duration of symptoms reported by Barker et al[6]. In our study mean age of the patient at the time of operation was 49 years. It is less than mean age reported by Bederson & Wilsons[7], which is 59.9 years. Pollack and Jannetta[8] reported 53 years. The reason of low age at operation in our study could be the selection criteria of patients regarding their fitness for general anaesthesia. In our series male patients were predominant, which is quite opposite from international studies. Bederson and Wilson reported female to male ratio of 5.5:2. In Pollack and Jannetta series it was 1.4: 1. The reason for this difference is probably the social setup in our region where females are not allowed to visit hospital in early stage of there disease. Superior Cerebellar Artery was the commonest offending vessel (54%), Which is comparable to 61% reported by Mendoza and Illingworth[9]. The detailed com-

OPERATIVE FINDINGS IN DIFFERENT SERIES OF MICROVASCULAR DECOMPRESSION

Author	Artery	Vein	Artery & Vein	Other Lesions	No Abnormality
Mandoza & Illingworth (1955)	Cases: 134 SCA 82 AICA 05 SCA+AICA 07 BA 01	06	SCA+Vein 20 SCA+AICA+ Vein 01	Tumor 06	04
Jannetta[12] (1983)	Cases: 103 SCA 82 AICA 09 PICA 01 SCA+AICA 03 Unnamed 04	19	Artery 22	19	—
Our Study (1998-2000)	Cases: 52 SCA 27 AICA 11 PICA 02 Unnamed 04	03		02	03

TABLE–4

parison of different series is given below. In our study 04(08%) patients had recurrence of pain, which is almost equal to 4.5% recurrence rate reported by Apfelbaum[10] in one year, while Klaun B[11] reported recurrence of 6% in his study. (Table 4).

CONCLUSION

In our small series of 50 patients, the results confirm that the basic aetiology of trigeminal neuralgia is highly related to vascular compression of the nerve roots at brain stem. Radiology like CT scan and MRI of brain is only helpful in identifying any other organic lesion and planning surgery. In majority of patients excellent postoperative results were achieved with no recurrence after one year follow-up. Male patients were predominant in our study. The most common vessel was Superior Cerebellar Artery, as mentioned in other studies. Those where definite compression was present responded well as compared to cases where no vascular compression was seen. The complications associated with the procedure can be reduced by meticulous surgical procedure and more experience.

On the basis of above these observation, we believe that the patients in whom medical treatment fail to respond and who are medically fit for intracranial surgery, Microvascular decompression should be the treatment of choice in trigeminal neuralgia.

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