ORIGINAL ARTICLE

Surgical Intervention in Traumatic Facial Nerve Paralysis

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Summary

A four years review from June 1998 to June 2002 of traumatic facial nerve paralysis from temporal bone fractures that required surgical intervention is presented. The aim of this clinical presentation was to determine the current pattern of cases with traumatic facial paralysis which required surgical intervention at our center. There were six cases, of which four (66%) were longitudinal fractures, one each (17%) had transverse fracture and fracture over the lateral wall of mastoid. Hearing loss (83%) was the commonest associated clinical symptom. All cases underwent decompression via the transmastoid surgical approach. Intraoperative findings revealed oedema of facial nerve involving vertical segment and horizontal segment in three cases each respectively. Two cases had concomitant bony impingement. The facial nerve functions in four cases (66%) and one case recovered to House Brackmann grade 2 and 4, 12 months and 3 months respectively postsurgery. The case with transverse fracture remained as House Brackmann grade 5 after two years.

Key Words: Facial nerve paralysis, Temporal bone fracture, Surgical intervention

Introduction

Temporal bone trauma can cause significant morbidity and mortality. It is usually due to severe head injury from motor vehicle accident. According to Moffat DA, male patients are more prone and symptoms of temporal bone trauma occur in about 30% of head injuries[1]. Common sequelae of temporal bone trauma include hearing loss, vestibular dysfunction, cerebrospinal fluid leak (otorrhoea or otorhinorrhoea) and facial nerve paralysis which is probably the most distressing complication. Longitudinal fractures (80%) are more common compared to transverse fractures (20%). However, the most serious and those that often require surgery are complications of transverse fractures[1]. Facial nerve injuries occur in approximately 50% of transverse factures and 20% of longitudinal fractures.

Materials and Methods

We reviewed the clinical records of all patients with facial nerve palsy due to temporal bone fracture that were treated surgically from June 1998 to June 2002 at the Department of Otorhinolaryngology, Universiti Kebangsaan

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Malaysia, Kuala Lumpur. Their clinical presentations, radiological findings, surgical treatment and outcome were reviewed and analysed.

Results

Between June 1998 to June 2002, six patients with facial nerve palsy secondary to temporal bone fractures underwent surgical decompression at the Department of Otorhinolaryngology, Universiti Kebangsaan Malaysia, Kuala Lumpur. All were male patients with average age of 30 years (ranging from 15 to 51 years). The commonest cause was motor vehicle accident (83%). Only one case (17%) was due to assault. All cases had immediate complete facial nerve palsies and electroneurography (ENOG) results showed more than 90% degeneration.

Associated clinical symptoms in traumatic facial palsy

The distribution of associated clinical symptoms is shown in Table I. The commonest clinical symptom was hearing loss (83%) followed by vertigo (33%) and ear bleed (33%). Only one case (13%) was associated with cerebrospinal fluid leak (otorrhoea). The summary data of patients with complete traumatic facial nerve palsy is listed in Table II.

Radiologic findings

All patients had High Resolution CT scan (HRCT) performed. Four cases (66%) were reported as longitudinal fractures and one (16%) as transverse fracture. In one case there was no evidence of fracture. Of the four cases noted with longitudinal fractures, the sites of facial nerve injury involved the vertical segment and the horizontal segment in two cases each respectively. There was no documentation regarding the site of injury in the rest two cases.

Operative findings

All patients had facial nerve exploration via the transmastoid approach. Main findings were oedema involving the vertical facial nerve segment in three cases (50%) and horizontal segment in the rest three cases (50%). The rest of the two cases had bone chips compressing facial nerve at the horizontal segment.

Recovery

After surgery, the facial nerve function of four patients (66%) recovered to House Brackmann grade 2 after 12 months, one patient recovered to House Brackmann grade 4 after three months and was expected to have good prognosis. The case with transverse fracture had remained at House Brackmann grade 5, two years following surgery.

Table I: Associated symptoms of patient with traumatic facial nerve paralysis

Symptoms	Number of patients	
	(n=6)	(%)
Hearing loss	5	86
Vertigo	2	33
Ear bleed	2	33
CSF leak	1	16

Table II. Summary data of patients with complete traumatic facial nerve paralysis.

Cases	Mechanisms	CTscan findings	Intraoperative findings	HB grade of recovery
A	MVA	Transverve fracture	Vertical segment of facial nerve oedematous No fracture line seen.	5
B	MVA	No fracture seen	Fracture line over the lateral wall of mastoid. Bone chip compresing facial nerve at the horizontal segment. Oedematous horizontal segment.	2
C	MVA	Longitudinal fracture	Fracture line seen over squamus part of temporal bone posterior to spine of Henle and the antrum. Oedematous horizontal segment and second genu of facial nerve.	2
D	Assaulted	Longitudinal fracture	Fracture line seen over the mastoid. Periosteum of the mastoid elevated and compressing the facial nerve at vertical segment. Oedematous vertical segment of facial nerve.	2
E	MVA	Longitudinal fracture	Fracture line seen over roof of antrum to attic. Oedematous vertical segment.	2
F	MVA	Longitudinal fracture	Fracture line over the mastoid. Bone chip compressing facial nerve at the horizontal segment. Oedematous horizontal segment and second genu of facial nerve	4

Notes: HB- House Brackmann

Discussion

The middle cranial fossa is involved in 60% to 80% of base of skull fractures[1]. The most common group involved are young males. The most common cause of this injury is motor vehicle accident. Thus, five (83%) patients in our study are due to motor vehicle accident and only one case (17%) due to assault. Common sequelae of temporal bone fractures are facial weakness, hearing loss, vertigo and cerebrospinal fluid otorrhoea. However, facial nerve weakness is the only deficit that requires the determination for surgical intervention in the early injury stage[2].

About 80% of temporal bone fractures are longitudinal as a result of blow to the temporal or parietal. The fractures crosses the floor of the middle fossa near the foramen spinosum and courses anterior to the osseous labyrinth, injuring the facial nerve in 10% to 20% of cases[3]. Ear bleed, cerebrospinal fluid otorrhoea and conductive hearing loss are common in this type of fractures[4]. The data showed four patients (66%) with this type of fracture. Two of these patients had three concomitant complications, one patient with facial nerve palsy, conductive hearing loss, cerebrospinal fluid otorrhoea and another patient with facial nerve palsy, conductive hearing loss and vertigo. Two patients had facial nerve palsy and conductive hearing loss. Profound sensorineural hearing loss and vertigo are common in transverse fractures which pass in a plane perpendicular to long axis of temporal bone[5]. One patient in our study with this type of fracture had concomitant facial nerve palsy, vertigo and profound sensorineural hearing loss.

In traumatic facial nerve paralysis, degeneration of 90% or more of the facial nerve within 6 days of the onset of complete paralysis is predictive of poor recovery unless decompression is performed. Most studies have attempted to identify poor prognostic factors in an effort to define a population likely to benefit from surgery. Significant prognostic criteria includes extent of paralysis, timing of onset of paralysis and electroneurography (ENOG). Darrouzet et al, recommended surgical treatment when facial nerve paralysis is total, is of immediate onset and is associated with bad prognostic electromyogram pattern[6]. At our institution, all patients with traumatic facial nerve paralysis undergo audiometry assessment, Schirmer test, axial and coronal high resolution CT scan and facial nerve electrophysiology workup. Surgery is indicated if ENOG showed more than 90% degeneration and absent of voluntary potentials on electromyogram (EMG). All the patients had ENOG of more than 90% before surgical intervention was advocated.

The surgical approach for traumatic facial nerve paralysis depends on the possible site of the injured facial nerve segment. Some advocate limited exploration of facial nerve based on clinical and radiographic information. Darrouzet et al, treated their patients via the middle fossa and the transmastoid (75.3%), the translabyrinthine (10.7%) and the pure transmastoid (14%) approach according to the facial nerve nonmotor branch evaluation, the hearing, the location of fracture and the patients general condition[6]. All our patients underwent transmastoid approach after audiological assessment, Schirmer test and radiological assessment. The radiological assessment revealed the most likely site of trauma to be localized to the vertical and horizontal segments of the facial nerve. Intraoperative findings confirmed all lesions to be confined to the vertical, the horizontal and the second genu of the facial nerve. Two types of facial nerve pathology were noted in our patients, namely neural oedema and bony impingement. In one case there was no evidence of fracture and the most likely site of injury was though to be over the labyrinthine segment, which would have required middle fossa approach for a successful facial nerve decompression.

Good recovery of facial nerve function was observed in 5 patients after surgical decompression. In four patients the function was House Brackmann grade 2, 12 months following the decompression. In one patient the function

was House Brackmann grade 4, three months following the decompression and he is expected to make good recovery. There is evidence of poor recovery of the facial nerve function in the patient with transverse fracture. It was most likely that the injury site was at the labyrinthine segment which could not be accessed via the transmastoid approach without a labyrinthectomy. A middle cranial fossa approach would have been the better approach.

In conclusion, facial nerve decompression is warranted in cases of immediate onset, complete facial nerve palsy after temporal bone trauma if more than 90% neuronal degenerations occurs. Early surgical intervention will improves the final outcome of facial nerve function. Skillful experience in microsurgical techniques of facial nerve decompression is essential.

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