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Post Traumatic Facial Paralysis Treatment Options and Strategies

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Abstract: Facial palsy is a socially incapacitating sequel to head injuries, occurring in 1.5% of such patients. This ugly aftermath of middle cranial base fractures is seen in 70% of transverse fractures and 30% of longitudinal petrosal fractures. A thorough knowledge on diagnosis, treatment and prognosis of facial nerve injuries is essential. The saving face (pun intended) with regard to post injury facial paresis is that 75% of facial injuries recover spontaneously.

Treatment of facial nerve injuries depends on two principal factors : onset of facial weakness (acute or delayed) and extent of facial weakness. Early onset and/or complete facial palsy indicate disruption of continuity of the nerve, ruling out the possibility of spontaneous recovery. A delayed onset and/or partial paresis of the nerve suggests secondary swelling or compression of the nerve which is more likely to recover spontaneously. In the former category, early facial nerve exploration is mandated. Facial EMG is unhelpful in the acute phase as denervation potentials take at least 3 weeks to develop. The facial muscles are the most active of all the muscle groups in the body and when denervated, they rapidly atrophy and involute. Hence, every patient of facial nerve injury should be started on percutaneous facial stimulation and vigorous passive facial physiotherapy.

Surgical treatment options for facial nerve injuries include direct repair or repair with a nerve graft when both ends are available. Dynamic facial reanimation procedures include facio-hypoglossal or facio-phrenic anastomosis, and static reanimation procedures like fascia lata sling and temporalis muscle transposition, or gold coin implantation into the upper lid. These are reserved for facial nerve injuries more than two years old where there is no hope of natural regeneration

This paper is based on our experience with 10 patients of traumatic facial palsy following petrous fractures.

Keywords: head injury, post-traumatic facial palsy, temporal bone fractures

INTRODUCTION

Facial palsy is a common condition with an estimated incidence of 20-25 cases per 100000 population[1,2] which may arise due to various reasons viz. idiopathic (Bell's palsy), trauma, inflammation tumors, and others[3]. The facial nerve palsy following trauma, is an uncommon condition which occurs in 1.5% patients of skull base fractures[5], majority of them due to road traffic accidents and missile injuries[4] causing temporal bone fractures. Two types of petrous temporal bone fractures have been described on the basis of direction of fracture in relation to the long axis of the petrous pyramid, viz., longitudinal and transverse. Facial paralysis is seen in about half the transverse fractures and in about 20% of longitudinal fractures which most commonly involve the region of the geniculate ganglion[6]. In most of the cases spontaneous recovery follows but still surgical exploration and even neurosuture may be needed for a complete coaptation and recovery.

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MATERIAL AND METHODS

In a retrospective study, records of 10 cases of traumatic facial paralysis treated at Bombay Hospital Institute of Medical Sciences, Mumbai, India by us between 1987-2003 were analyzed. Out of 10 patients, nine were males and one female. Mean age was 37 years. The mode of injury was road traffic accidents in 8 patients (80%), blow on head, and fall from slight height in one patient each respectively. Nine patients developed immediate facial palsy and one patient had delayed facial palsy following head trauma. Six patients had haemotympanum with conductive deafness, whilst three presented with sensorineural deafness. Two patients showed ecchymosis over the mastoid (Battle sign). All patients were investigated by using high resolution CT scan brain and temporal bone, and electrophysiological studies. Out of 10 patients, nine had petrous bone fractures (longitudinal fracture - 3, transverse fracture – 6) and one patient did show any evidence of temporal bone fracture.

Three patients had associated temporal lobe contusion and one patient had traumatic SAH that later on resulted in right hemiparesis due to left MCA stenosis.

RESULTS

All patients were treated conservatively. Conventional EMG did not help in diagnosing severity of facial nerve injury when done immediately after trauma. Out of 10 patients, four had very severe facial paralysis (group 1-grade5) and six had very mild to moderate facial paralysis (group 2-grade 2 to 3). All these patients were followed by conventional EMG at interval of 3-6 months. Group 2 patients recovered completely in 6 to 8 months whilst group 1 patients converted to moderate facial paralysis in 3 to 4 years after facial exercise and nerve stimulation.

DISCUSSION

Facial nerve is called the 'master organizer of facial orchestra' because it innervates all the mimic muscles of the face, carries taste sensation from anterior 2/3 of tongue and provides secretomotor fibres to submandibular, sublingual and lacrimal glands. Post traumatic facial paralysis has been seen in 1.5% of skull base fractures[5]. Fractures of temporal bone constitute approximately 20% of all skull fractures which are most commonly produced by motor vehicle accidents (50%), blunt trauma (40%), and gun shot injuries (<10%)[6]. These fractures have been grouped into longitudinal and transverse fractures in relation to the long axis of petrous pyramid[1]. Longitudinal fractures are clinically more common than transverse fractures[6]. Facial paralysis is very uncommon with longitudinal fractures and when it occurs, is usually delayed in onset; on the other hand transverse fracture produces severe immediate paralysis. The facial paralysis can be managed conservatively or by surgery. Controversy exists with regard to the timing of surgical intervention in the management of immediate onset of facial paralysis. Fortunately, most cases recover spontaneously. Electrophysiology which is positive only after 8-10 days of nerve injuries, may show axonal degeneration or reinnervation. Lack of recovery indicates exploration and this can be done in few days, weeks, or even months after injury, often with good results, even in delayed cases. Prognosis depends on onset of paralysis[2], degree of paralysis[3] and site of injury[7].

The onset of facial palsy may be immediate or delayed. Immediate facial palsy occurs due to stretching/ compression/crushing/division[8] of the nerve. Compression injury does not produce any interruption in nerve conduction and is akin to neurapraxia which recovers promptly in 2-3 weeks. Crushing or stretching injury causes interruption in nerve electric conduction and is similar to axonotmesis, which recovers in a period of 3-6 months. Stretching injury recovers slower than crushing injury as they differ in range and degree. Divisional injury shows complete interruption of nerve conduction and are akin to neurotmesis which recovers after six months of injury[8]. Delayed facial paralysis occurs usually after 1-10 days of trauma and occurs more commonly due to entrapment of nerve within fibrous tissue contiguous to fracture and less commonly due to nerve edema within fallopian canal. Delayed injuries have more favorable prognosis than immediate injury. Similarly incomplete injuries have better prognosis than complete injuries.

CONCLUSION

Longitudinal fractures, incomplete and delayed facial nerve injuries bear the best prognosis. Most patients of posttraumatic facial nerve injuries recover with conservative treatment and time. Surgery and reanimation are, in fact, rarely required.

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