FACIAL NERVE PARALYSIS : A THREE YEAR RETROSPECTIVE STUDY

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Abstract: *Of all the cranial nerves, the facial nerve is the one which is most commonly involved in disease. Facial paralysis leaves the patient severely disfigured. Timely diagnosis and treatment can lead to considerable recovery. 16 consecutive patients of facial paralysis of all age groups and due to different causes diagnosed and treated in a tertiary referral hospital have been studied retrospectively. The frequency of aetiological factors, the various factors governing the management of these cases and their actual outcomes after a minimum period of one year are discussed .The causes of facial nerve palsy included cholesteatomas, Bell's Palsy, iatrogenic, traumatic, neuroma and others. In general, early reporting, diagnosis and surgical intervention wherever indicated have resulted in better recovery. Surgical decompression of the facial nerve traditionally advocated have been questioned. Rare causes of facial nerve paralysis like postoperative BIPP pack allergy and bilateral congenital agenesis of facial nerve are also included.*

Key words *: Facial Nerve paralysis, Cholesteatoma, Trauma, Iatrogenic, BIPP*

INTRODUCTION:

The facial nerve (FN) innervates all the muscles of facial expression. Facial expression is the most valued possession human beings have. Paralysis of facial muscles can cause severe disfigurement in affected persons. It causes psychological and emotional trauma. Therefore , treatment of facial nerve palsy (FNP) should be energetic and with concern for the possible outcomes at the very outset.

This nerve has the distinction of being the commonest cranial nerve to be paralysed. This is due to the fact that it has a long intracranial route , the major part (3.75 cm) is within a bony canal. This makes it susceptible to a whole range of injuries and insults as the consequent oedema compresses the nerve within the bony canal.

The primary objective is to assess the degree, site and possible causes of the injury. Wherever a reversible insult is suspected the aim is to prevent further degeneration and wherever possible to aid regeneration. Whenever an irreversible damage to the FN is suspected no effort should be spared to establish anatomical and functional continuity of the nerve at the earliest possible opportunity.

The management outcomes vary as it depends on a host of etiological factors, the degree of injury and modality of management. The common etiological causes of FNP are shown in Table I. It is clear that management decisions should be based on sound surgical logic and mandates a certain degree of urgency in infective cases. This paper is a retrospective review of the management of FNP by the authors over a three year period.

MATERIALS AND METHOD:

A total of 16 consecutive patients of facial nerve palsies treated in a tertiary care hospital over a period from Jan 2000 to Jan 2003 have been studied. All the patients have undergone detailed otologic and head and neck clinical examination and investigations deemed appropriate in a busy hospital. Bell's palsy cases have undergone impedance audiometry and stapedial reflex testing along with topognostic tests. Electrophysiological testing were not routinely done. Those suspected to have intracranial complications or where specifically indicated have undergone imaging studies. Steroids were displayed only after excluding common contraindications. Canal wall down mastoidectomy has been performed by the standard method in all cases of cholesteatoma. During surgery the course of the facial nerve has been identified in all cases. Where an erosion due to cholesteatoma was found, the nerve underwent decompression a few mm proximal and distal to the site of erosion. Where the site of injury could not be made out , the entire mastoid and tympanic portion was decompressed. Facial nerve monitor was not used routinely as it has not been the practice at our hospital. Patients age, sex, clinical presentation, diagnostic evaluation, management and outcome have been analysed . The recovery of facial palsy has been assessed at least 8 weeks and at the end of 12 months after the onset by the end stage House Brackman (HB) scale. Recovery has been classified as good (HB-1) , satisfactory (HB-2 to 3) and unsatisfactory (HB-4 to 6)

RESULTS

The various causes of FNP treated and sex distribution are shown in Table II. There were 16 cases in all. The youngest patient was 7 years old and the oldest was 65 years with a mean age of 29 years. The various points of interest concerning FNP due to cholesteatomas is shown in Table III.

Three cases of Bells palsy occurred in females and one in a male. The average age at presentation was 36 years. The average delay in presentation was 5 days. Three cases who reported after 3 days or had complete palsy was treated only with acyclovir 200 mg five times a day for five days. Of these two showed complete recovery. The other case, a 54 year old man who had

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early complete palsy showed unsatisfactory recovery at 12 months. One case who presented early received steroids alone. She showed full recovery after one year.

3 cases sustained iatrogenic injury. One case was due to section of the nerve at the parotid segment. One case underwent acoustic neuroma surgery elsewhere and had total nerve palsy. Both these cases did not show satisfactory recovery. The third case had transient nerve palsy possibly due to vibration and heat of the drill and recovered fully. Two of three cases of iatrogenic trauma were caused by experienced surgeons and only one by less experienced surgeon.

One case showed bilateral facial agenesis. Patient had no other abnormality in the head neck region or deafness. One case had a temporal bone trauma with the fracture line passing through the mastoid segment. This case underwent decompression. Nerve was apparently stretched and not severed. Recovery was unsatisfactory. One case was of a facial nerve neuroma. Neuroma was arising from the mastoid segment and could be excised without damaging the nerve. Paralysis remained at the preoperative level, but there was no progression of the palsy after surgery. One case had facial transient paralysis due to BIPP allergy.

DISCUSSION

Treatment of facial nerve paralysis is perhaps one of the most challenging decision making processes for any otolaryngologist . Many issues involving the diagnosis and treatment of facial nerve disorders continue to engender controversy and debate. This article discusses certain causes of FNP encountered at our institution and their management.

It will be generally agreed that Bell's palsy is the commonest cause of FNP in everyday otologic practice. Recent work by Murakami S, Miyamoto N, Watanabe N and Matsuda F[1] has established that Bell's palsy is caused by alpha Herpes Simplex Virus. Since complete recovery occurs in 85% of the patients , conservative management is indicated in most cases. We have used acyclovir in three cases having early complete FNP. Two cases recovered fully and one did not. One patient who presented early with incomplete palsy received steroids and recovered fully. The numbers are not enough to say whether steroids or acyclovir have any advantage over the other. It has been observed that any FNP that becomes complete in 72 hours has delayed and incomplete recovery. Because Bell's palsy is not always the cause physicians should be able to identify critical findings on history and clinical examination that indicate an alternate diagnosis. The clinical picture of all patients with peripheral facial palsy is identical, irrespective of the etiology. So they are often wrongly classified and treated as Bell's palsy[2] . In most cases, the cause of FNP can be determined on the basis of the clinical evaluation and expensive diagnostic tests can be avoided. Once identified, these findings can lead to a specific and directed evaluation [3].

Cholesteatomatous ear disease is a well recognized cause of FNP. We came across 5 cases of FNP due to chronic otitis media. We did not come across a single case of FNP due to acute otitis media. Probably they would have been managed by the primary care physicians. It has been observed that the disease has to be present for several years before a FNP occurs. Paralysis was always gradual and incomplete [4,5]. The facial canal was dehiscent mostly in the tympanic and pyramidal segment [6] . It was naturally dehiscent in only one case and eroded by the cholesteatoma in four other cases. In all the cases that we operated the facial nerve sheath was intact. It was the direct pressure and the inflammation around the nerve that was responsible for the palsy [7]. This is the reason why the palsy is gradual and incomplete. In our series all patients had good recovery. The facial nerve sheath, like the dura, resists invasion by cholesteatoma for a considerable period. FNP due to acute otitis is usually incomplete and occurs if the nerve is exposed by congenital dehiscence or when the adjacent mastoid air cell becomes inflamed .

The most devastating complication of otologic surgery is the inadvertent injury to the facial nerve[8]. During the period of study, 187 mastoidectomies, 231 tympanoplasties / ossiculoplasties / cochlear implantations and 33 stapedectomies were done in this institution. The incidence of iatrogenic FNP due to mastoid and middle ear surgery was 2 out of 451 (0.44%).

We came across 3 cases of iatrogenic nerve injury. Two occurred at our institution due to tympanomastoid surgery and one occurred during acoustic neuroma surgery elsewhere. One case was due to section of the nerve due to a extended postaural incision in an adult. This was quite unusual. On exploration , the FN within the parotid was found to be very superficial. End to end nerve suturing was done but recovery was unsatisfactory. Predisposing factors for operative nerve injury are congenital abnormality of the intratemporal course of the nerve (rare , as in our cases) or lack of surgical landmarks within the ear. Landmarks may be unidentifiable from advanced disease or previous surgery. The other causes of iatrogenic nerve injury would include parotid surgery per se. Immediate complete palsy and delayed but progressive palsy beyond twelve hours calls for immediate re-exploration of the nerve. Incidence of iatrogenic FNP varies in different studies.

Jones PH[9] has reported FNP due to BIPP allergy for the first time in 1985. We use gelfoam or ribbon gauge pack impregnated with BIPP in all cases of tympanomastoid surgery. We came across one case having bilateral chronic otitis who developed delayed but progressive FNP in right ear following BIPP packing after mastoid surgery. The nerve was explored but no injury was found. Palsy recovered completely within two weeks. Six months later she underwent surgery in the left ear. She again developed delayed palsy. This time nerve was not exposed as we were sure there was no operative injury . BIPP packing was immediately removed .She recovered fully with conservative management. This was the only FNP due to BIPP allergy (0.22 %) in 451 consecutive tympanomastoid surgery. Lim PV, Hughes RG and Oates J[10] have reviewed case notes of 185 patients who underwent BIPP packing after ear surgery and he reported allergic reaction in 5.9% cases. He also reported higher rates of graft perforations in allergic

Table I

COMMON CAUSES OF FACIAL NERVE PARALYSIS

1.	**Intracranial**
	(a) Brain stem lesions : tumors, vascular accident, poliomyelitis, multiple sclerosis.
	(b) Cerebellopontine angle lesions: acoustic neuroma, arachnoidal cysts, meningitis.
2.	**Intratemporal**
	(c) Otitis media-acute or chronic
	(d) Trauma- surgical or accidental
	(e) Viral infections-Herpetic facial palsy (Bell's), Herpes zoster oticus.
	(f) Temporal bone tumors
3.	**Extratemporal**
	(g) Parotid tumors
	(h) Trauma-surgical or accidental
4.	**Miscellaneous**
	(i) Sarcoidosis
	(j) Melkerson-Rosenthall syndrome
	(k) Polyneuritis
	(l) Infectious mononucleosis
	(m) Leukaemia

patients than non allergic patients. There were no cases of FNP in his study. It will be interesting to note that Vrabec JT [11] postulated that delayed FNP occurring 72 hours after otologic surgery could be due to viral reactivation by varicella zoster and in his series of 486 patients 1.4% had delayed FNP.

Even though RTA and head injuries are common occurrences, FNP due to head injuries were not so common. We came across 18 temporal bone fractures during the study period of which only 1 (5.5%) had traumatic FNP in the mastoid segment. Transmastoid decompression was done. Fracture line was found to be crossing the mastoid segment but the nerve was not found to be transected .Inspite of this the recovery was not satisfactory even after one year. Surgery is indicated if FNP is immediate and total [12]. In all cases early decompression is indicated for restoration of function [13]. In many cases of head injury, neurosurgical intervention takes priority and immediate decompression is not always possible. Quaranta A, Campobasso G, Piazza F et al obtained good functional result in two of three cases operated three months after trauma [14].

Facial nerve neuromas are rare causes of FNP. It should be suspected in slowly progressive paralysis. They may be associated with a variety of other symptoms depending on the site of the tumor. In the only case that we treated we were able to resect the tumor without sacrificing the nerve [15]. There was no improvement in the palsy but there was no worsening either. Jervis PN and Bull PD [16] reported, possibly the first case of congenital facial nerve agenesis diagnosed incidentally for an unrelated condition. We came across one case of bilateral FN agenesis in a 9 year old girl , who had no deafness or any other congenital anomaly. She had no features of Moebius syndrome. Agenesis was confirmed by imaging studies. Here the facial nerve could not be traced after the tympanic segment in both ears. Bilateral tarsorraphy was done. Facial reanimation surgery has been planned at a later date . Similar findings were observed by Saito H, Takeda T and Kishimoto S [17] in nine of forty two temporal bones of infants born with various congenital anomalies.

CONCLUSION

The facial nerve has been very closely associated with the otologist. No other cranial nerve has invited so much attention and concern because FNP is a devastating consequence for any patient. While controversies in the management of FNP have always been and will always remain, most surgeons have devised their own strategies for their management. The outcome of FNP is not very gloomy as it was in the past, provided the patients report early. Incomplete palsies almost always recover. A FNP that is total or becomes complete within two to three days may not recover fully. During surgery, the fear of the facial nerve has resulted in more iatrogenic palsies than when the nerve and its landmarks have been routinely identified . It is the responsibility of the otologist who sees the patient for the first time to think of the possible outcomes at the very outset and institute the appropriate treatment.

Table II

ETIOLOGY OF FACIAL PALSY (n=16)

	Males	Females	Total	Percentage
Cholesteatoma	1	4	5	31.25
Bell's palsy	1	3	4	25.00
Iatrogenic	2	1	3	18.75
Traumatic	1	0	1	6.25
Agenesis	0	1	1	6.25
BIPP Allergy	0	1	1	6.25
Neuroma	1	0	1	6.25
Total	6	10	16	100 %

Table III

FACIAL NERVE PALSY DUE TO CHOLESTEATOMAS (n=5)

	Number of cases	%
Males	1	20
Females	4	80
Age< 12 Years	3	60
Age>12 Years	2	40
Complete recovery	3	60
Residual palsy	2	40
Tympanic part involvement	3	60
Pyramidal part involvement	2	40

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Corrigendum

- Vol: 58, No.2, April-June 2006 issue page no. 137, The main author has to be read as Kapil Dua of DMC & H., Ludhiana.
- Vol: 58, No.2, April-June 2006 issue page no. 202, The list of authors has to be read as Dr. (Mrs) K.S. Das Gupta, Dr. S.V. Joshi, Dr. Kanchan Lanjewar, Dr. Neeraj Murkey.