**Original research article**

**Role of intratympanic dexamethasone injections in post-traumatic facial nerve palsy**

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**Abstract:**

The facial nerve or the seventh cranial nerve has a motor and sensory component which account for 70% and 30% of the fibers respectively. It is affected in 7 to 10% of temporal bone fractures. These injuries typically occur from falls, motor vehicle accidents, and assault, as well penetrating trauma, such as gunshot wounds. Temporal bone fractures are traditionally classified into longitudinal, transverse, or mixed types, depending on their orientation relative to the petrous ridge. (4/5) facial nerve fractures are more common in transverse fractures. Treatment of the injured intratemporal facial nerve is historically controversial, available options for the management includes both surgical and nonsurgical. Up till now non surgical options included giving oral steroids and nerve stimulation by physiotherapy and not much research has been done in the use of Intratympanic Injections of Dexamethasone in such cases especially in Stage 3/4 palsy in patients who denied Surgical options as results of Surgery are also not 100%. In our study we present this option and compare the results of this treatment with simple oral steroids and found encouraging results.

**Keywords:** Post Traumatic Facial Nerve Palsy, Intratympanic Dexamethasone Injections, Facial Palsy, Intratympanic Injection.

**INTRODUCTION**

The facial nerve is most commonly affected nerve in closed head injuries (Turner, 1943). In facial palsy which immediately follows a head injury, the mechanism is obvious, but it is not clear when the facial palsy follows the head injury after many days (Potter and Braakman, 1976). The post traumatic facial palsy can be both immediate and delayed. Traumatic facial palsy has received much attention but few authors distinguish between immediate and delayed palsy. Turner (1944) studied a selected group of war-time head injuries from a military hospital for head injuries, and found an incidence of 2.2 % developing a delayed facial palsy. Potter (1964), however, estimated the incidence of this complication of head injury as about 0.6 %. Michele Rotondo suggest that Computed tomography (CT) is the best procedure for detecting the fracture line at the level of the facial nerve canal and for assessing any associated lesions within the temporal bone. Magnetic resonance (MR) is required if there is a facial nerve paralysis, unexplained by CT findings.

Eli Gordin states that treatment of the injured intratemporal facial nerve is historically controversial. Reanimation of the paralyzed face is an interesting and frequently evolving field. Available options for the management include both Surgical and Nonsurgical. Up till now non surgical options included giving oral steroids and nerve stimulation by physiotherapy and not much research as done in the use of Intratympanic Injections of Dexamethasone in such cases especially in Stage 3/4 palsy in patients who denied Surgical options as results of Surgery are also not 100%. In our study we present this option and compare the results of this treatment with simple oral steroids and found encouraging results

**MATERIAL & METHODS**

# All the cases of Posttraumatic Facial nerve palsy coming to the Department or Casualty of the Institute and did not opt for Surgical treatment whenever indicated, were taken in the study. All the patients who opted for surgery whenever indicated were excluded from the study. All patients with posttraumatic palsy with nerve compression as a cause (bone chip / transaction) who denied for surgery were also given Intratympanic Injections.

# Evaluation of the Temporal bone injury was done using a CT scan and grading of facial nerve palsy was done using the House-Brackmann Classification.

#  C:\Users\E N T\Desktop\POST TRAIUMATIC FACIAL PALSY\Table_1_Harrison.jpg

# Patients with grade 4 onwards facial nerve palsy were taken up for Intratympanic Dexamethasone injection as chances of spontaneous recovery in grades 1-3 is very high. Comparative data presented in the study for both groups is of patients in Grade 4 onwards injury only. All patients of grade 1-3 facial nerve palsy were given routine treatment including oral steroids and had a complete recovery

# Patients (N=40) were divided in two groups

# Group A (N=20) Patients were given oral medication including oral steroids in a dose of 1.5mg/kg body weight) for a period of 2 weeks thereafter tapered to 1mg/kg body weight for 1 week. Results were noted after 1 week and 2 weeks

# Group B (N=20) Patients were given oral medication including oral steroids in a dose of 1.5mg/kg body weight) for a period of 2 weeks thereafter tapered to 1mg/kg body weight for 1 week along with Intratympanic Dexamethasone injection twice a week for 2 weeks. Results were noted after 1 week and 2 weeks of therapy.

# Patients of both the groups were also given Physiotherapy for facial nerve recovery.

# Although Audiological evaluation was done in all the patients but audiological recovery will not be a part of this study

**Method of Injecting Intratympanic (IT) Dexamethasone injection** – (1)

Local anaesthesia of the tympanic membrane was performed by the application of a cottonoid soaked with 4% Xylocaine for 30 min. The IT injection was done with the aid of 0°, 4-mm sinuscope. Patients were positioned in a supine position, with their head rotated to the contra lateral ear. One tiny hole was done at the anterosuperior quadrant of the tympanic membrane as a ventilation hole to let air to escape from middle ear during injection, followed by IT injection just below the ventilation hole using a 25-gauge spinal needle, and Dexamethasone was kept in contact with the round window for 30 min. Patients were instructed to avoid swallowing and speaking for 30 min. The Intratympanic injection was performed twice weekly for two successive weeks with an overall series of 4 injections.



Picture 1: Intratympanic Dexamethasone

Injection being given

**OBSERVATIONS**

**Table 1:** In **Group A** 7 (35%) patients were of Grade 4 palsy, 8 (40%) patients were of Grade 5 palsy whereas 5(25%) patients were of grade 6 palsy. In **group B** 8 (40%) patients were of Grade 4 palsy, 8 (40%) patients were of grade 5 palsy whereas 4 (20%) patients were of grade 6 palsy

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Grade 4** | **Grade 5** | **Grade 6** |
| Group A (n=20) | 7 (35%) | 8(40%) | 5(25%) |
| Group B (n=20) | 8(40%) | 8(40%) | 4(20%) |

**AGE DISTRIBUTION**

**Table 2:**

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Grade 4** | **Grade 5** | **Grade 6** |
| Group A (n=20) | <25yrs = 3 (15%)25-50yrs=2 (10%)>50yrs=2 (10%) | <25yrs = 3(15%)25-50yrs=3 (15%)>50yrs=2(10%) | <25yrs = 3(15%)25-50yrs=2 (10%)>50yrs=0 |
| Group B (n=20) | <25yrs = 4 (20%)25-50yrs=3 (15%)>50yrs=1(5%) | <25yrs = 3(15%)25-50yrs=4 (20%)>50yrs=1(5%) | <25yrs = 3(15%)25-50yrs=1 (5%)>50yrs=0 |

**RECOVERY PATTERN AFTER 1 WEEK**

|  |  |  |  |
| --- | --- | --- | --- |
| **Group A (n=20)** | **Grade 4 (n=7)** | **Grade 5(n=8)** | **Grade 6(n=5)** |
| <25yrs = 3 | Mild recovery of 1 pt. to grade 3 2 Patient No response | <25yrs = 3 | Mild recovery of 1 pt. to grade 42 Patient No response | <25yrs = 3 | No response |
| 25-50yrs=2  | No response | 25-50yrs=3  | Mild recovery of 1 pt. to grade 42 Patient No response | 25-50yrs=2  | No response |
| >50yrs=2 | No response  | >50yrs=2 | No response |  | No response |
| **Group B (n=20)** | **Grade 4 (n=8)** | **Grade 5 (n=8)** | **Grade 6 (n=4)** |
| <25yrs = 4 | Mild recovery of 2 pt. to grade 32 Patient No response | <25yrs = 3 | Mild recovery of 2 pt. to grade 41 Patient No response | <25yrs = 3 | Mild recovery of 3 pt. to grade 5 |
| 25-50yrs=3 | Mild recovery of 2 pt. to grade 31 Patient No response | 25-50yrs=4  | Mild recovery of 2 pt. to grade 32 Patient recovered to Grade 4. | 25-50yrs=1  | Mild recovery of 1 pt. to grade 5 |
|  >50yrs=1 | No response | >50yrs=1 | No response |  |

After 1 week, in group A out of 20 patients, grade IV palsy is observed in 7 patients (35%), grade V palsy is observed in 8 patients (40%) and grade VI palsy in 5 patients (25 %). Most commonly affected age group is <25 yr i.e. 45%.

 In group B out of 20 patients, grade IV palsy is observed in 8 patients (40%) , grade V palsy is observed in 8 patients (40%) and grade VI palsy observed In 4 Patients i.e. 20%. Most commonly affected age group is <25% i.e.50%.

Recovery observed in group A – after week, in grade IV palsy case mild recovery occurs in 1 patient. i.e. 14% in grade V case mild recovery observed in 2 cases i. e. 25% and in grade VI no recovery observed at all.

 While in group B grade IV palsy cases shows, mild recovery of 4 patient i.e. 50% grade V palsy cases shows mild recovery in 4 patients i.e. 50% & in grade VI palsy cases mild recovery observed in all 4 patient i.e. 100%.

**RECOVERY PATTERN AFTER 2 WEEK**

|  |  |  |  |
| --- | --- | --- | --- |
| **Group A (n=20)** | **Grade 4 (n=7)** | **Grade 5 (n=8)** | **Grade 6 (n=5)** |
| <25yrs = 3 | 1 patient recovered to grade 22 patients recovered to grade 3 | <25yrs = 3 | 1 patient recovered to grade 32 patients recovered to grade 4 | <25yrs = 3 | Mild recovery of only1 pt. to grade 52 patients No response |
| 25-50yrs=2  | 1 patient recovered to Grade 31 patient No response | 25-50yrs=3  | Recovery of 2 pt. to grade 31 Pt – No response | 25-50yrs=2  | Mild recovery of only 1 pt. to grade 51 pt. No response |
| >50yrs=2 | Mild recovery of 2 pt. to grade 3 | >50yrs=2 | No response |  |
| **Group B (n=20)** | **Grade 4 (n=8)** | **Grade 5 (n=8)** | **Grade 6 (n=4)** |
| <25yrs = 4 | Complete recovery of all the 4 patients  | <25yrs = 3 | Complete recovery of 3 patientsRecovery of 1 patient to Grade 2 | <25yrs = 3 | Recovery of 1 patient to Grade 1,1 patient to Grade 3,1 patient to Grade 4 |
| 25-50yrs=3 | Complete recovery of all the 3 patients | 25-50yrs = 4  | Complete recovery of 2 patientsRecovery of 1 patient to Grade 2 | 25-50yrs=1  | 1 pt. No response |
|  >50yrs=1 | Complete recovery of the patients | >50yrs=1 | Complete recovery of the patients |  |

On observing recovery after 2weeks patients of Group A shows- in grade IV palsy cases 1 patient recovered to grade 2 (14%) and 5 patients recovered to grade 3 i.e. 71% in grade V palsy cases- 3 patient recovered to grade III (37.5%) & 2 patients to grade IV (25%) and in grade VI cases previously at 1 wk no response seen but at 2 wks 2patients (40%) shows only mild recovery to grade V.

 In group B after 2 wks, Grade IV palsy cases shows complete recovery of all patients, in grade V palsy cases complete recovery observed in 6 cases i.e. 75% & partial recovery observed in 2 patients (25%) and in grade VI palsy cases complete recovery observed in 1 patient, & partial recovery of 1 Patient to grade 3, another patient to grade 4 & 1 patient shows no response.

|  |  |  |
| --- | --- | --- |
| Grade 6 Palsy Day 1 | Grade 4 Palsy Rt. / Grade 2 Left - After 1 Weeks | Grade 3 Palsy Rt. / Grade 1 Left - After 2 Weeks |
| C:\Users\E N T\Desktop\POST TRAIUMATIC FACIAL PALSY\ON ADMISSION.JPG | C:\Users\E N T\Desktop\POST TRAIUMATIC FACIAL PALSY\DAY 1 (15.2.2020) LF INJ..JPG | C:\Users\E N T\Desktop\POST TRAIUMATIC FACIAL PALSY\day 7 a.JPG |

**DISCUSSION:**

The facial nerve or the seventh cranial nerve has a motor and sensory component which account for 70% and 30% of the fibers respectively. The motor root innervates the muscles of facial expression, scalp and ear. The facial nerve is affected in 7 to 10% of temporal bone fractures.(2/3) These injuries typically occur from falls, motor vehicle accidents, and assault, as well penetrating trauma, such as gunshot wounds. Temporal bone fractures are traditionally classified into longitudinal, transverse, or mixed types, depending on their orientation relative to the petrous ridge. (4/5) Approximately 70 to 80% of fractures can be classified as longitudinal, resulting from a temporoparietal impact; 10 to 30% transverse, resulting from an frontal or occipital injury; and 0 to 20% are mixed in nature. (6/7) Facial nerve involvement is seen in anywhere from 10 to 25% of longitudinal fractures and is more common in transverse fractures, occurring in 38 to 50% of cases. (8)

When the facial nerve is involved, the ensuing paralysis is immediate in 27% of cases and has a delayed presentation in 73% of patients.16 Most commonly, injury is localized to the perigeniculate region or, less commonly, the second genu.13 Paresis was noted in 23% of cases in Darrouzet’s series of 115 cases, immediate complete paralysis in approximately 51% of cases, and delayed complete paralysis in approximately 15%. In this study, the onset of facial paralysis was indeterminate in approximately 11% of patients. (8) Our Institute being a tertiary care centre patients are mostly referred from other nearby places so the nature of onset of facial palsy whether immediate or delayed could not be ascertained with surety but some cases in the series are of delayed onset facial palsy and one case is of bilateral delayed onset facial palsy.

 In a general consensus immediate complete paralysis warrants surgical exploration. Cases of complete paralysis in which the onset of paralysis is indeterminate should be treated as immediate in nature. (8) Since we did not consider those patients in our study that underwent Surgery so it will not be discussed further.
Many researchers advocate no exploration for non-penetrating trauma for intra-temporal facial nerve palsy. Patients with delayed onset facial weakness or incomplete facial weakness should be managed conservatively with steroids and vasodilators. Late surgery may be recommended in cases of non-recovery within 6 months after trauma. Hence the management of these patients is still controversial. (9).

The treatment of facial paralysis must be tailored to the individual patient, and the surgeon must select the appropriate course of action based on the circumstances surrounding nerve dysfunction (8) The etiology and severity of the paralysis plays a role in deciding whether treatment should be aimed at long- term restoration of facial function, or if temporary management is appropriate, either when nerve recovery is expected, or as a means of bridging the interval between a surgical reanimation and the onset of neural conduction. Similarly, the duration of paralysis is of paramount importance in selecting treatment, as the complete degradation of motor end plates after 2 years usually renders reinnervation procedures futile. (8)

Another method of treatment Traumatic Facial nerve Palsy is the Modified Stennert’s protocol (11). Stennert’s protocol was initially described for idiopathic facial nerve palsy (Bell’s palsy). Modified Stennert’s protocol was described by Mahesh *et al*., where hydrocortisone was used instead of prednisolone. This was tried in patients with idiopathic facial nerve palsy and in patients with delayed posttraumatic facial nerve palsy. Good improvement was seen equally in both the groups. Modified Stennert’s protocol is a 13 day regimen of tapering doses of dextran, hydrocortisone, and pentoxifylline. Dextran was initially given as 1000 kilodaltons over 16 h, which was tapered after 3 days to 500 kilodaltons over 8 h. Hydrocortisone was given on the 1st day as 200–250 mg/dl (as per patient’s weight) and was tapered every 3 days to stop. Pentoxifylline was continued as 10 mg/kg for 13 days. Serum potassium levels and random blood sugar levels were monitored daily and were corrected accordingly.

Ju Yeon Hwang etal (10) are of the opinion that efficacy of steroids remains controversial in patients with facial paralysis. But, steroids are usually administrated to patients with either traumatic or infectious facial paralysis. Steroids have been used to reduce edema, swelling and scar formation. Marginal benefit of steroid treatment in idiopathic facial paralysis is demonstrated in a randomized double blind controlled study. Also an important management aspect of patient care is the prevention of exposure keratitis with the use of artificial tears and lubricants. In various studies done on posttraumatic facial palsy using only Oral steroids like the one did by Rakesh Kumar etal (12) on management of Posttraumatic facial palsy in a patient with bilateral lower motor neuron facial palsy House/Brackmann grade 5, on follow up of two months weakness on right side of the face had improved to House/ Brackmann grade 2 and on left side the weakness of grade 4 was present. On follow up of four months her left side facial weakness improved to grade 2, hence the recovery patter with oral steroids was slow and incomplete. In our cases of Group A, oral steroids recovery pattern (Table 1 /2) was better than their series.

We could not find ant study in which Intratympanic steroids have been given for the management of Post traumatic facial nerve palsy hence the results could not be compared with others. We found that the results of Intratympanic steroids for the management of Post traumatic facial nerve palsy especially in Grade 3 /4 facial palsy to be very promising and showed marked recovery of palsy as compared to giving oral steroids only, in patients who otherwise refused surgery when ever indicated.

**CONCLUSION**

Posttraumatic facial palsy is a condition of common occurrence, road traffic accidents account for majority of the cases, and treatment guidelines cannot be followed at all the places as the evaluation and management criterias for the palsy may not be available at all the places moreover some patients donot choose to undergo surgery as results of the surgery are also not 100%. Hence we opted for giving another option in the management of Post traumatic facial nerve palsy by giving Intratympanic steroids and found very promising results especially in managing Grade 3 & 4 facial palsy although a similar study with greater number of patients may be needed to support our findings.

**Conflict of Interest**: The authors declare that this study has had no conflicts of interest.

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**Ethical Clearance**: Proper Ethical Clearance from Institutional Ethics committee was received for this study from the Institutional Ethics Committee.

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