Clinical Study of Intratemporal Facial Nerve Palsy - The Causes and Outcome

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Abstract: Paralysis of the seventh cranial nerve, the facial nerve, is usually immediately obvious. It results in weakness of the musculature of the face, impacting verbal communication, social interaction with respect to facial expression, oral competence, taste and most importantly, protection of the cornea, ocular globe and vision. Palsy of the facial nerve in its intratemporal course leads to ipsilateral lower motor neuron facial palsy.

Objectives:

1. To study 100 cases of facial nerve palsy due to pathology in the intratemporal course of the nerve.

2. To identify the frequent causes, course of palsy, treatment modality and recovery during a period of 3 months follow-up.

Materials & Methods:

100 patents with facial nerve palsy with an intratemporal pathology were analysed. Detailed history was taken. Patients were subjected to complete clinical evaluation of ear and cranial nerves, radiological evaluation was also done to confirm the diagnosis. Patients were treated appropriately. The facial nerve function was graded again during follow up after 3 months.

Results:

46% patients were diagnosed with Bells palsy, 26% with chronic suppurative otitis media/cholesteatoma, 17% with trauma leading to palsy and 4% patients had tumours that lead to the palsy. Males were more affected (58%) than females. 39% patients were in the age bracket of 21 to 30 years. 25% of cases had Grade 3 palsy at presentation, 40% of cases had Grade 4, 13% had grade 5 and 8% had grade 6 palsy. After 3 months 82% cases showed complete recovery following appropriate treatment.

Keywords: Facial nerve, Palsy, Intratemporal.

INTRODUCTION

The facial nerve is the nerve of the second branchial arch. It serves several functions – motor and sensory. It supplies the striated musculature of the face, neck, and stapedius muscle of the middle ear, parasympathetic fibres to the lacrimal, submandibular & sublingual glands and seromucinous glands of the nasal cavity. It conveys taste sensations from the anterior two-thirds of the tongue. It also has a small cutaneous sensory component [1].

The internal auditory segment is 7 to 8 mm in length [2]. The first part of the facial canal – the labyrinthine segment of the nerve (3–5 mm) is the narrowest part of the facial canal and extends from the fundus of internal auditory canal to the geniculate ganglion [1].When the nerve reaches a point just lateral and superior to the cochlea, it angles sharply forward, nearly at right angles to the long axis of the petrous temporal bone, to reach the geniculate ganglion. At this level, the direction of the nerve reverses itself, executing a hairpin bend so that it runs posteriorly. This is the 'first turn' of the facial nerve. The greater superficial petrosal nerve arises from the geniculate ganglion. The second part or the tympanic segment (10–12 mm) extends from the geniculate ganglion to the second turn of the facial nerve. It passes posteriorly and laterally along the medial wall of the tympanic cavity, perpendicular to the long axis of the petrous bone. Here it lies above the oval window and below the bulge of the lateral semicircular canal. The third part or the mastoid segment (13–15 mm) extends from the second genu to the stylomastoid foramen. Here the nerve assumes a vertical position, dropping downwards in the posterior wall of the tympanic cavity to exit at the base of the skull from the stylomastoid foramen. The nerve to stapedius muscle is a small twig given off from the facial nerve as it descends in the posterior wall of the tympanic cavity behind the pyramidal eminence. The chorda tympani branch originates about 5 mm above the stylomastoid foramen. It exits the mastoid cavity via the stylomastoid foramen and enters the parotid gland [2].

The main branches of the facial nerve are as follows:

1. At the level of the geniculate ganglion, the greater superficial petrosal nerve arises and supplies parasympathetic fibres to the lacrimal gland and mucous membrane of the nose and oral cavity.

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2. Distal to the geniculate ganglion, the facial nerve gives rise to 2 branches. The first is the motor branch to stapedius muscle, which serves to dampen the oscillations of the ear ossicles. The second is the chorda tympani nerve, which joins with the lingual nerve, and carries sensory fibres for taste from the anterior 2/3of the tongue as well as provides parasympathetic innervation to the submandibular and sublingual glands.

3. The facial nerve exits the temporal bone through the stylomastoid foramen and divides into its terminal branches, which supply the motor innervation to the muscles of facial expression [3].

There are various intratemporal causes for the development of facial nerve palsy. Sir Charles Bell, the British Physician, in 1821 described the onset, physical findings and course of idiopathic facial nerve palsy [4]. James Ramsay Hunt (1872 – 1937) an American neurologist described the clinical syndrome of Herpes Zoster Oticus that bears his name [5]. Facial nerve tumours are a very uncommon neoplasm. They may originate from any segment of the seventh nerve, from the cerebellopontine angle to the peripheral branch in the parotid gland [6]. The labyrinthine segment is the shortest segment being 3 to 5 mm in length. However, the facial nerve is particularly vulnerable in this location and is subjected to compromise or complete transection by temporal bone fractures, particularly transverse fractures [7]. Facial nerve paralysis is an uncommon but significant complication of chronic otitis media. Although the incidence of facial nerve paralysis has decreased with the use of antibiotics, prevention thereof remains a challenging problem [8]. Facial nerve palsy may be an iatrogenic complication of mastoid surgery. This seems to have decreased with the introduction of the surgical microscope, better otologic drill and, more recently, instruments for monitoring the facial nerve [9, 10].

In our study we have analysed 100 patients who came to our outpatient department with facial nerve palsy. The etiology, the grade of the palsy and the course of the disease were analysed so as to understand the causative factors, predisposing conditions that make a patient vulnerable to the palsy.

MATERIALS AND METHODS

This study was conducted for duration of 8 years, from 2005 to 2013, at Sri Venkateshwara Institute of ENT, Bangalore Medical College and Research Institute.

Sample Size and Selection of Patients

100 patients with intratemporal facial nerve palsy

Study Design

Prospective study

Those cases in whom, after clinical examination and/or investigations, intracranial or extratemporal facial paralysis was diagnosed and in whom where the site of lesion could not be localized were excluded from the study.

Collection of Data

Detailed history was taken from all the selected cases. In cases of post -operative palsy, details regarding the type of surgery were also obtained. Complete examination of the ear was done. Clinically detailed neurological evaluation was carried out and facial nerve palsy graded according to the House and Brackmann system of classification [11].

Audiometry and radiological investigations (CT scan or MRI) were done to localise the site of lesion. The patients were treated medically or surgically as indicated by their respective diagnosis.

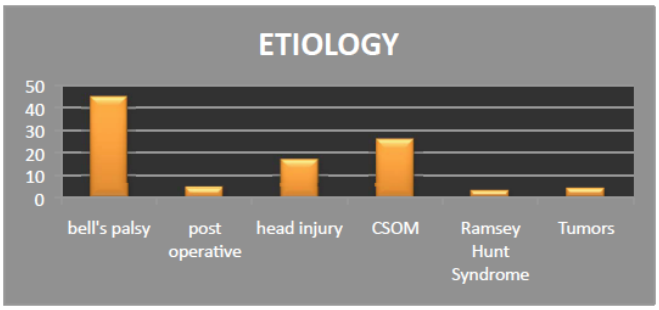
All the cases were followed up for a minimum period of 3 months after which the facial nerve was re-evaluated and any residual palsy present was graded and documented.

OBSERVATION AND RESULTS

The various causes for the facial palsy in our study are tabulated as follows (Table **1** and Graph **1**).

Table1: Showing the Etiological Factors for Facial Palsy

S. No.	Cause of Palsy	No. of Cases
1.	Bell's palsy	46
2.	Post operative	4
3.	Head injury/Temporal bone fracture	17
4.	CSOM / Cholesteatoma	26
5.	Ramsay Hunt syndrome	3
6.	Tumors	4
	Total	100

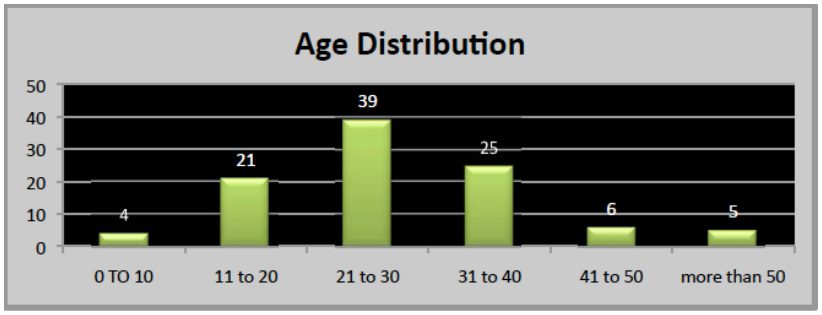


Graph 1: Showing the etiological factors for facial palsy.

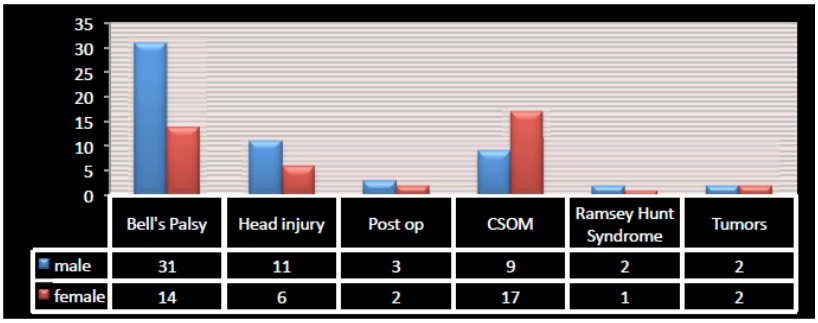
In our study, out of 100 cases 46 patients were found to have Bell's palsy making it the leading cause for palsy. Chronic suppurative otitis media and cholesteatoma was the cause in 26% of patients followed by head injury in 17% of the study population.

Among the cases, 58% were males compared to the 42% females. The tabulation of the age distribution (Graph **2**) of the patients revealed that the maximum number of patients belonged to the young adult category, with 39% falling in the group between 21 to 30 years of age followed by 25% in the 31 to 40 years age bracket. Only 4 of our cases were below 10years of age and similarly only 5% were above 50 years of age.

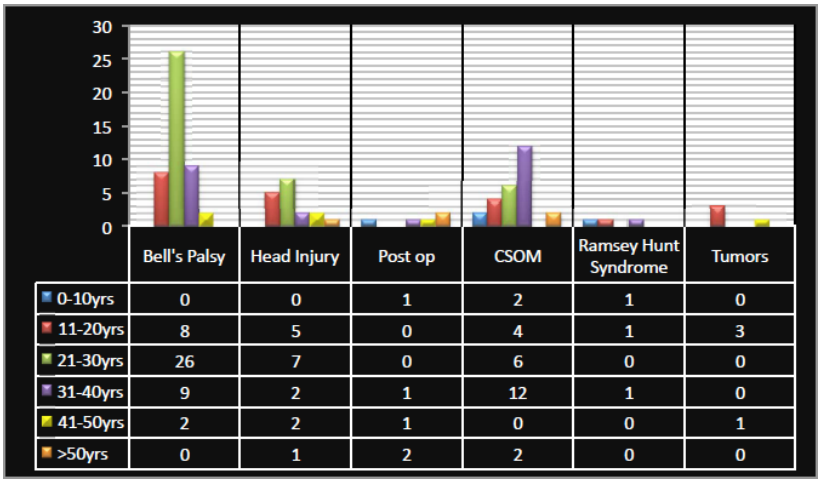
On analysing the gender distribution of the various etiological factors (Graph **3**), we deduced that men were more affected than women by Bell's palsy as well



Graph 2: Showing the age distribution of the patients.



Graph 3: Showing the relationship between the gender and causative factor for facial palsy.



Graph 4: Showing the relationship between the various etiological factors and age of the patients.

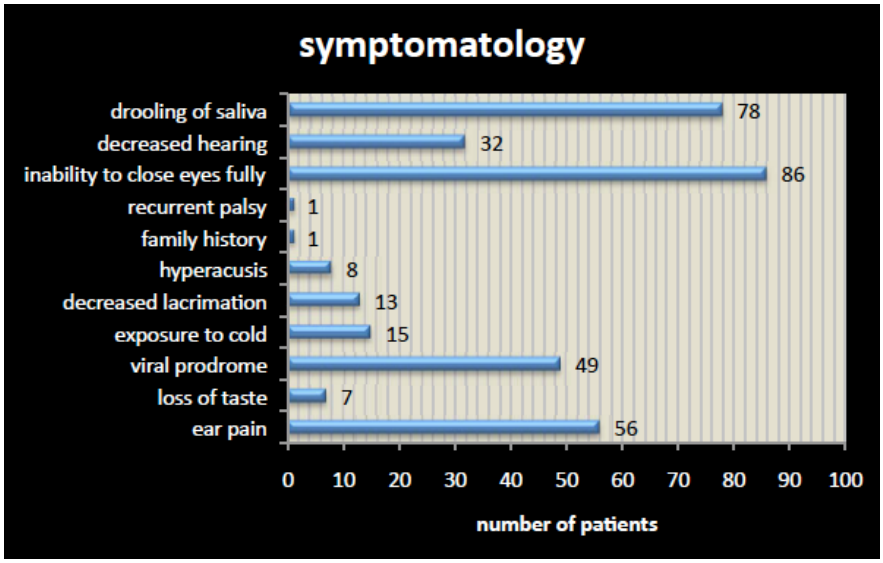
as head injury and trauma, while the leading cause for palsy among the women was found to be chronic suppurative otitis media.

The probable reason for this could be the negligence of early symptoms of cholesteatoma among women therefore leading to the development of complications before the first visit to the otorhinolaryngologist. A similar analysis was done to see the distribution of various etiological factors among the different age groups (Graph **4**).

Bell's palsy and head injury was found predominantly in the young adults in the age bracket between 21 and 30 years. Chronic suppurative otitis media and cholesteatoma with facial nerve palsy was mainly found in the age group of 31 to 40 years. 75% of cases with tumours leading to facial nerve palsy were in the age group of 11 to 20 years. Above the age of 50 years the cause of facial palsy, in this study, was mainly due to chronic suppurative otitis media or mastoid surgery.

The presenting symptoms of the patients were analysed (Graph **5**) and we found that 86% of the patients noticed difficulty in completely closing the eyes. Drooling of saliva was noted in 78% of the cases. Ear pain was the next most common symptom, found in 56% of patients. A viral prodromal symptom was noticed by 49% of cases. Only one patient, who was diagnosed with Bell's palsy, gave previous history of facial palsy. One patient diagnosed with palsy due to chronic suppurative otitis media, gave a family history of similar palsy in his father.

At the time of presentation, the grading of facial nerve dysfunction was done as per the House and Brackmann system of classification and the analysis revealed (Table **2**) that 40 out of the 100 patients had grade 4 facial nerve palsy followed by 25% who had grade3 palsy. Only 8 out of the 100 had complete palsy (grade 6). The patients were treated appropriately and re-evaluated after 3 months duration. The facial nerve function was graded again and our findings are tabulated in Table **3**.



Graph 5: Showing symptoms of facial palsy.

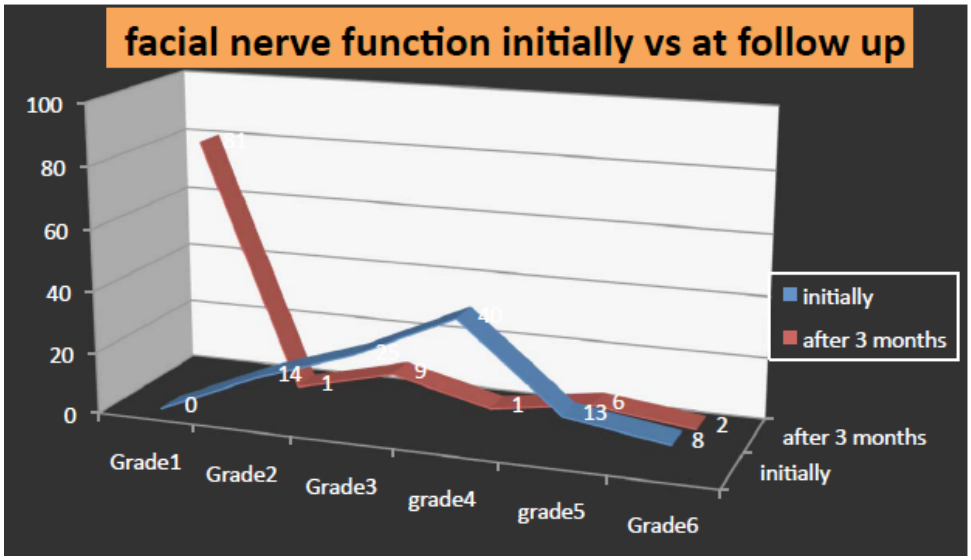
Table 2: Showing the Grading of the Facial Nerve Palsy at the Time of Presentation

Etiology	Grade 2	Grade 3	Grade 4	Grade 5	Grade 6
Bell's palsy	0	10	27	5	4
Post- operative	0	2	2	0	0
Head injury	0	5	7	3	2
CSOM	14	6	3	1	2
Ramsey Hunt Syndrome	0	2	1	0	0
Tumors	0	0	0	4	0
Total:	14	25	40	13	8

Note: Grade 1 is normal

Table 3: Showing the Grading of the Facial Nerve Palsy at Follow up after 3 Months

Etiology	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5	Grade 6
Bell's palsy	38	0	7	1	0	0
Post operative	4	0	0	0	0	0
Head injury	12	1	2	0	0	2
CSOM	24	0	0	0	2	0
Ramsey Hunt Syndrome	3	0	0	0	0	0
Tumors	0	0	0	0	4	0
Total	81	1	9	1	6	2



Graph 6: Showing the comparison of Grade of palsy at presentation versus after 3 months.

On follow up we noted that there was complete recovery and fully functioning facial nerve (Grade 1) among 81 patients. There was no recovery at all among 8 patients who continued to have Grade 5 or Grade 6 as was the Grade when they presented to us. The remaining 11 patients showed partial improvement in symptoms (Graph **6**).

DISCUSSION

John Groves once said, "Otology could be a dull way of life without the seventh cranial nerve arrogantly swerving through the temporal bone to the muscles of facial expression". Facial nerve palsy warrants a detailed history taking and thorough clinical examination of the head, neck, ear and other cranial nerves in order to aid in topographic localisation of the cause of the facial nerve palsy [11].

The facial nerve is composed of 10,000 motor, sensory and parasympathetic fibres [11]. The facial nerve is traditionally divided into the following parts for discussion: supranuclear, nucleus and tracts, cisternal segment, intratemporal segment and peripheral segment [7]. In the current study we have analysed the various causes for facial nerve palsy affecting only the intra-temporal segment and facial nerve palsy graded according to the House and Brackmann system of classification [11].

The intratemporal segment is further subdivided into 3 segments: labyrinthine, tympanic and mastoid. Labyrinthine segment courses antero laterally from the fundus of internal auditory canal to terminate at geniculate ganglion. The greater superficial petrosal nerve containing the pre-ganglionic parasympathetic fibres to the lacrimal glands is the first major branch of VIIth nerve, arising at the geniculate ganglion. The proximal tympanic segment lies along the medial wall of the anterior epitympanic recess. Mid-tympanic segment lies immediately underneath lateral semi

circular canal and superior to oval window. Dehiscence is especially common in this region making the facial nerve vulnerable to injury during middle ear surgical procedure. Distal tympanic segment is found within the pyramidal eminence. Second genu connects tympanic and mastoid segments. From here mastoid segment courses inferiorly and lateral to jugular fossa. Lesions originating from jugular foramen commonly involve the facial nerve at this point [7].

Mastoid segment lies at extreme proximity to the posterior tympanic annulus. Two important branches arise in this segment, the nerve to stapedius and the chorda tympani nerve. The facial nerve enters the stylomastoid foramen and exits the temporal bone to continue in its peripheral course [7]. Here, it immediately gives off branches to the auricular muscles, the posterior belly of the digastric muscle and the stylohyoid muscle. It supplies sensory (vagal) fibres to parts of the external auditory canal and some areas of the auricle, including the lobule [12]. The nerve then courses ventrally and at the posterior edge of the parotid gland, it splits into upper and lower divisions. Within the parotid gland, there is further branching with many individual variations. As a rule, the upper division of the facial nerve gives off temporal, zygomatic and buccal branches, whereas the lower division emits marginal mandibular and cervical branches [7, 12].

There are 23 facial muscles, most of which are paired. In facial expressions, 17 muscles are activated [12]. Temporal bone pathology leads to variable loss of lacrimation, stapedial reflex and taste sensation in the presence of facial asymmetry [7]. The value of topographic localisation lies in the tailoring of appropriate management.

In our study the most common cause for facial palsy was found to be Bell's palsy. Bell's palsy is an acute, peripheral facial paresis of unknown cause [13]. Thirty one out of the 46 patients with Bell's palsy were men unlike the findings of Martha et al [10] who found a female predilection with a female: male ratio of 1.5: 1. 21.7% of patients with Bell's palsy had grade3 palsy, 58.6% had grade4, 10.8% had grade5 and 8.7% had grade6 palsy at presentation. These patients were managed using systemic steroids and antiviral drugs in conjunction with facial physiotherapy. After 3 months we found that 82.6% had completely recovered and remaining patients showed partial recovery. Most authors agree that 75% of Bell's palsy cases regress spontaneously with complete recovery. Approximately 15% of the cases had satisfactory recovery with a slightly detectable neurological deficit and 10% of the cases had permanent paralysis [10]. Gary & Remia suggest that up to 30% of patients with Bell's palsy fail to recover facial function completely [13].

The second most important cause for facial palsy in our study was palsy in association with cholesteatoma and chronic suppurative otitis media. Jin Kim et al have suggested that the frequency of facial nerve paralysis in chronic suppurative otitis media ranges from 0.16 to 5.1%. Osteitis, bone erosion, external compression, oedema and inflammation of the nerve are some of the proposed etiologic factors. Chronic otitis media causing facial nerve paralysis is most frequently due to cholesteatoma [8]. In the present study out of the 100 cases 26 were diagnosed with chronic suppurative otitis media as the cause for palsy. Among these 26 cases 14 (53.84%) had grade2 facial palsy, 23% grade3 palsy, 11.53% grade4, 3.84% had grade5 and 7.6% had complete palsy. The diagnosis of cholesteatoma leading to palsy was established with a high resolution CT scan. The treatment was mainly directed at eradication of disease, as early as possible, through mastoidectomy and facial nerve decompression along with intravenous antibiotics for 1 week followed by oral antibiotics for 3 weeks. During follow up after 3 months 92.30% (24 out of 26 patients) showed complete recovery. Both the patients (7.69%) in whom interrupted facial nerve was observed during the surgery still had grade 5 palsy during follow up.

The next leading cause for facial palsy was following trauma (17 % of cases). All of them were following road traffic accident. Except for one patient, who developed facial asymmetry the following day, all of them developed palsy immediately. The grading of palsy showed that 41.17% (7 patients) had grade 4 palsy, 17.6% (3 patients) had grade 5 and 11.76% had grade 6 palsy at diagnosis. The temporal bone fracture was confirmed by CT scan which showed that 5 cases had transverse fracture, 5 cases had vertical fracture and 5 had developed oedema in the facial canal. These five patients were managed by decompression of facial nerve. All the patients were given a course of systemic steroids. During the follow up after 3 months, we found that 76.47% had recovered completely while 11.76% showed partial recovery with grade3 palsy and 2 patients did not recover.

The final outcome of the facial nerve defect reconstruction found by Dragoljub Popovic et al, by applying the nerve transplant gave the House Brackmann grade III and IV which is statistically worse

than the nerve decompression that resulted in grade II in most cases [14].

Mastoid surgeries are another important cause for facial nerve palsy in the intratympanic segment. In our study, 4 patients developed palsy following canal wall down mastoidectomy. However all of them recovered within 2 to 3 weeks. 50% (2 patients) had grade 3 while the other two developed grade 4 palsy. Intraoperatively no transection of the nerve was noted. The incidence of iatrogenic facial nerve injury is said to be between 1% and 5%. In a study, the overall incidence of iatrogenic facial nerve palsy was 1.7%, while the incidence among surgical trainees was 2% and among consultants was 1.3%. To further avoid iatrogenic facial nerve injury during mastoid surgery, many authors advocate drilling directed more superiorly and anteriorly towards the attic when encountering difficulty in identifying the aditus during mastoid surgery. Cautious drilling with generous irrigation is advocated since the second genu of the facial nerve is located inferior and medial to the aditus and hence it is the most likely site to be injured. Intraoperative facial nerve monitoring has several advantages: it allows mapping of the nerve through soft tissues, tumour and bone; it also predicts dehiscence in the bony canal covering the nerve; and it allows confirmation of the electrical integrity of the nerve before and after surgery [9].

In 1907, James Ramsay Hunt described a clinical syndrome-Ramsay Hunt syndrome that carries his name, in which peripheral facial paralysis, pain and ipsilateral vesicular lesions are caused by involvement of the geniculate ganglion by the Varicella Zoster Virus [15]. In the duration of our study we diagnosed 3 patients with Ramsey Hunt syndrome. Out of them 2 patients had grade 3 palsy while 1 patient had grade 4 palsy at the time of presentation. They were given systemic steroids, antivirals, eye lubricants and physiotherapy and all 3 patients recovered completely. According to Juliana Oliveira et al, Ramsey Hunt syndrome is responsible for 7-16% of unilateral facial palsies of non-traumatic origin [15].

Tumours were responsible for the palsy in 4 patients. Among them two patients were diagnosed with rhabdomyosarcoma of middle ear while two had facial neuroma. Facial nerve tumours are rare neoplasms; the most common of these are schwannomas [6]. Embryonal Rhabdomyosarcoma in middle ear and mastoid is very rare. It is a highly malignant tumour found in paediatric age group [16]. Rhabdomyosarcoma is the commonest soft tissue sarcoma in children under 15 years of age [17]. In our study we found one 11 year old who presented with swelling behind the ear in the mastoid region with facial nerve palsy. FNAC confirmed the diagnosis of rhabdomyosarcoma and CT scan was done. Another child, 13years old, came with facial palsy and axial CT scan revealed a lytic lesion involving middle ear and mastoid. MRI was done and the tumour demonstrated intense homogeneous enhancement after gadolinium administration. The child was taken up for surgery and lesion excised. Histopathology confirmed the diagnosis of rhabdomyosarcoma. Both were treated with a combination of chemoradiotherapy. But the palsy remained unresolved even during follow up after 3 months. The other 2 patients were diagnosed with facial neuroma. Facial neuromas account for 1% of all intrapetrous lesions and 0.15% to 0.8% of all intratemporal tumours. Since the first description of a facial neuroma by Schmidt in 1930, there have been nearly 500 additional reported cases [18]. The patients presented with facial twitching and gradual onset, progressive facial palsy with tinnitus and decreased hearing. One of the two patients presented with recurrent palsy. Both the patients refused to take any treatment.

CONCLUSION

Among the various causes for intratemporal facial nerve palsy the most common was Bell's palsy or idiopathic palsy (46%), followed by chronic Suppurative otitis media (26%). Males were more affected than females in all the various aetiologies for facial nerve palsy except chronic suppurative otitis media were females were affected more. The incidence of iatrogenic palsy due to mastoid surgery has deceased with the introduction of the surgical microscope, better otologic drill and instruments for monitoring the facial nerve.

REFERENCES

[1] Măru N, Cheiță AC, Mogoantă CA. Intratemporal course of the facial nerve: morphological, topographic and morphometric features. Romanian J Morphol Embryol 2010; 51(2): 243-248.

[2] Chandra S, Goyal M, Gandhi D, Gera S, Berry M. Anatomy of the facial nerve in the temporal bone: HRCT. Indian J Radiol Imag 1999; 9(1): 5-8.

[3] Evans AK, Licameli G, Brietzke S, Whittemore K, Kenna M. Pediatric facial nerve paralysis: Patients, management and outcomes. Int J Pediatr Otorhinolaryngol 2005; 69: 1521-1528. http://dx.doi.org/10.1016/j.ijporl.2005.04.025

[4] Ravi NS, Bruce JG. Surgery of the Facial nerve. Surgery of the Ear, 5th ed, Michael EG, Aina JG. BC Decker, Canada 2003; 615-639.

[5] Kai-Wen Z, Zi-Ting S. Microvascular decompression by the retrosigmoid approach for idiopathic hemifacial spasm: experience with 300 cases. Ann Otol Rhinol Laryngol 1995; 104: 610-612.

[6] Ciorba A, Borgonzoni M, Bovo R, Martini A. Intrapetrous facial nerve tumours: Report of 3 cases and literature review. Mediterr J Otol 2007; 3: 108-112.

[7] Chan EH, Tan HM, Tan TY. Facial palsy from temporal bone lesions. Ann Acad Med Singapore 2005; 34: 322-9.

[8] Kim J, Jung G, Park S, Lee WS. Facial Nerve Paralysis due to Chronic Otitis Media: Prognosis in Restoration of Facial Function after Surgical Intervention. Yonsei Med J 2012; 53(3): 642-648. http://dx.doi.org/10.3349/ymj.2012.53.3.642

[9] Long YT, Athar PS, Mahmud R, Saim L. Management of Iatrogenic Facial Nerve Palsy and Labyrinthine Fistula in Mastoid Surgery. Asian J Surg 2004; 27(3): 176-9. http://dx.doi.org/10.1016/S1015-9584(09)60028-2

[10] Keels MA, Long, Jr LM, Vann, Jr WF. Facial nerve paralysis: report of two cases of Bell's palsy. Facial nerve paralysis: report of two cases of Bell's palsy. Pediatr Dentist 1987; 9(1): 58-63.

[11] Bibas T, Jiang D, Gleeson MJ. Disorders of Facial Nerve. In: Michael Gleeson M, Browning GG, Burton MJ *et al*., editors, Scott-Brown's Otorhinolaryngolgogy, Head and Neck Surgery, 7th edition. Great Britain: Edward Arnold Publishers Ltd 2008; 3: 3870-3894. http://dx.doi.org/10.1201/b15118-307

[12] Rodrigues AC,Jesus Carlos Andreo JC, Menezes LF, Chinellato TP, Rosa Júnior GM. Anatomy of the Facial Nerve and its Implication in the Surgical Procedures. Int J Morphol 2009; 27(1): 183-186. http://dx.doi.org/10.4067/S0717-95022009000100031

[13] Gronseth GS, Paduga R. Evidence-based guideline update: Steroids and antivirals for Bell palsy: Report of the Guideline Development Subcommittee of the American Academy of Neurology. Neurology published online November 7, 2012 Published Ahead of Print on November 7, 2012 as DOI: 10.1212/WNL.0b013e318275978c

[14] Popović D, Stanković M, Popović Z, Milisavljević M. Traumatic Facial Palsy. Med Biol 2003; 10(3): 145-147.

[15] Silveira JOF, Schettini de Oliveira F, Bello YB, Melo Reis CH, *et al.* Ramsay Hunt Syndrome: Case Report. Am Med J 2012; 3(2): 237-239. http://dx.doi.org/10.3844/amjsp.2012.237.239

[16] Sinha DK, Sinha PK. Embryonal rhabdomyosarcoma of the middle ear and mastoid. IJO & HNS 1996; 48(2): 157-159.

[17] Shirani F, Alizadeh F, Kashani SS. Rhabdomyosarcoma of the Middle and Inner Ear. Iran J Radiol 2003; 1: 157-159.

[18] Shirazi MA, Leonetti JP, Marzo SJ, Anderson DE. Surgical Management of Facial Neuromas: Lessons Learned. Otol Neurotol 2007; 28: 958-963. http://dx.doi.org/10.1097/MAO.0b013e3181461c8d

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