I J Pre Clin Dent Res 2015;2(1):94-98
January-March

International Journal of Preventive & Clinical Dental Research

Corticosteroids: Still a Cornerstone in Treatment of Bell's Palsy - A Case Report

Abstract

Bell's palsy accounts for almost three quarters of all acute facial palsies, with the highest incidence in the 15 to 45 year old age group. The most alarming symptom of Bell's palsy is paresis; up to three quarters of affected patients think they have had a stroke or have an intracranial tumour. The palsy is often sudden in onset and evolves rapidly, with maximal facial weakness developing within two days. Treatment is probably more effective before 72 hours and less effective after seven days. A case of Bell's palsy reported in a twenty-seven year old male patient with no other neurological defect, was treated with systemic corticosteroids within first 24 hours of onset of the disease. Further we outline the recent developments in Bell's palsy and current best evidence in its management.

Key Words

Facial palsy; corticosteroids

Rachninder Kaur[1], Trisha Rastogi[2], Sonam Kohli[3], Sugandha Sharma[4]

[1]Senior Lecturer, Department of Oral Medicine and Radiology, DJ College of Dental Sciences and Research, Modinagar, Uttar Pradesh, India
[2]Senior Lecturer, Department of Oral Medicine and Radiology, DJ College of Dental Sciences and Research, Modinagar, Uttar Pradesh, India
[3]Senior Resident, ASCOMS Batra Hospital, Jammu, Jammu & Kashmir, India
[4]Senior Lecturer, Department of Oral Medicine and Radiology, DJ College of Dental Sciences and Research, Modinagar, Uttar Pradesh, India

INTRODUCTION

Facial Palsy is a self-limiting, non fatal and spontaneously remitting disorder of acute onset due to non-suppurative inflammation of the facial nerve within the stylomastoid foramen.[1-3] The disease does not appear to result from specific infection, but has been reported to arise in cases of both local and systemic disease. The occurrence of occasional cases following extraction of teeth has suggested a possible relation to trauma, although the role of the injection of local anaesthetic solution might also be considered in the aetiology of the disease. Some evidence indicates that the disease may be caused by ischemia of the nerve near the stylomastoid foramen resulting in oedema of the nerve, its compression in the facial canal and finally paralysis.[4] Different modalities of treatment have been tried with varying degree of success. One of the oldest attempts was the use of cortisone has advocated by Robinson and Moss in 1954.[5] Here by presenting a treated case report of Unilateral Bell's Palsy-(idiopathic paralysis of facial nerve) with administration of intramuscular corticosteroid.

CASE REPORT

A 27 year old male patient reported to the department of Oral Medicine and Radiology with a chief complaint of muscle weakness on right side of face since 1 day. History of present illness revealed that patient suddenly experienced some weakness on the right side of his face 1 day back. He gave a history of exposure to cold one day back when he drove his motorcycle early in the morning without covering his face. Since then he was unable to close his right eye and his mouth was deviated to left side while speaking, eating or laughing. He was unable to hold water in his mouth. He was not able to tolerate loud voice.There was no history of similar recurrent episodes, loss of taste sensation with no history of fever and trauma. On extra oral examination (Fig. 1), the right side of his face showed signs of paralysis. The right side of face showed loss of facial expressions and tone, widening of the palpebral fissures, diminished blinking of the eye causing dryness of eye, drooping of the corner of mouth along with drooling of saliva. On palpation, the right pre-auricular region was tender. Following signs were noted on right side of face: (a) Frontalis corrugator activity showed failure to raise the eyebrow, along with absence of forehead wrinkling (Fig. 3). (b) Orbicularis oculi sphincter function showed inability to close the eye, rather the eyeball turned upward in an attempt to

Fig. 1: Extra-oral picture

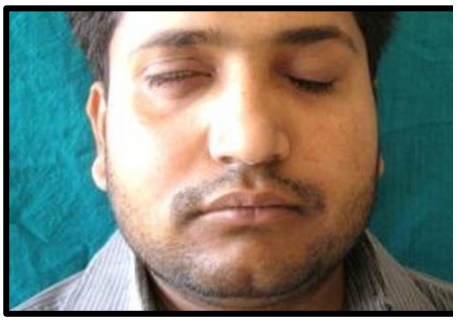

Fig. 2: Bell's sign

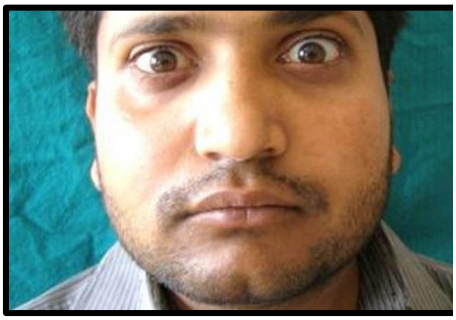

Fig. 3: Patient is unable to raise the eyebrows

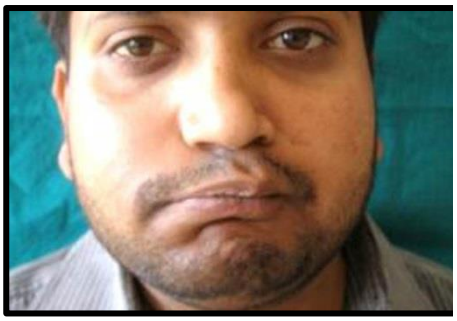

Fig. 4: Patient is unable to puff the cheeks

Fig. 5: Face turned to left side while smiling

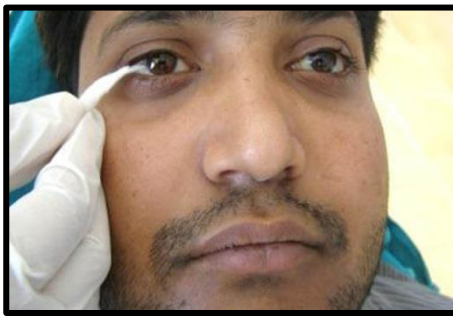

Fig. 6: Loss of corneal reflex

Fig. 7: Post-treatment extra-oral picture

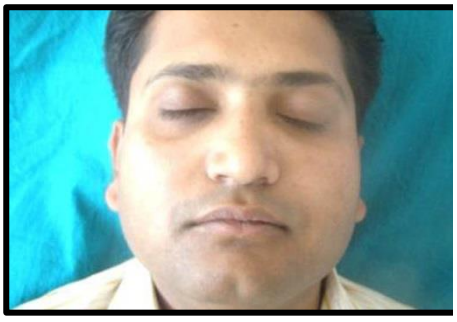

Fig. 8: Patient is able to close his right eye

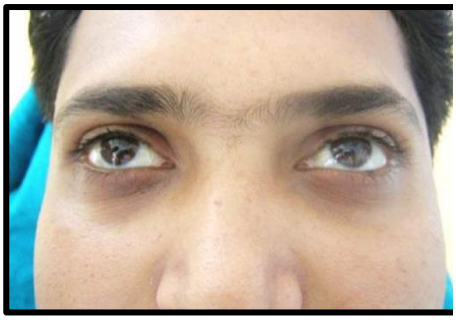

Fig. 9: Patient is able to raise the eyebrows

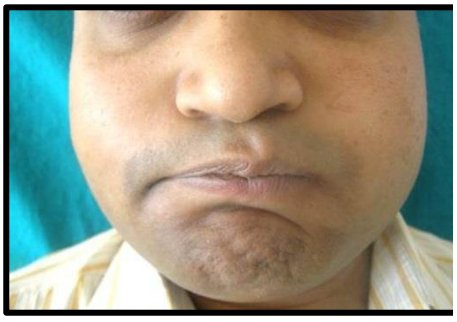

Fig. 10: Patient is able to puff his cheeks

close eye, hiding the pupil, while leaving only the white sclera visible (Bell's sign) (Fig. 2). (c) When patient was asked to blow his cheeks, he was not able to do so (Fig. 4). (d) When a cotton pointer was touched to his cornea, there was absence of corneal reflex (Fig 6). When patient was asked to smile the face was drawn to left side (Fig 5). (e) Taste testing (chorda tympani nerve) was performed by placing a small amount of salt and sugar on the tongue and it was found that patient was able to appreciate the taste. (f) Balance test (vestibulochoclear nerve test) was performed and it was found that patient was able to balance and co-ordinate his movements properly. Intra-orally, no abnormality was detected. With the above history given by patient and clinical findings, working diagnosis of complete facial paralysis of the right side (lower motor neuron lesion of facial nerve) was put forthe. A differential diagnosis of Transient facial paralysis (facial nerve anesthesia), Lyme's disease, Ramsay hunt syndrome (Geniculate Neuralgia) were given. Routine hematological investigations were carried out and were found to be within normal range. As there were no active lesions of Herpes Simplex viral infection, but to rule out dormant stage, Polymerase chain reaction (PCR) was carried out for rising antibody titres to herpes virus which turned out to be negative. Further, patient was referred to ENT specialist to rule out any middle ear infection and report was found to be negative. With the above investigations, final diagnosis of Bell's palsy (idiopathic paralysis of facial nerve i.e. the lesion of lower motor neuron) was given. Supportive measures were advised: To wear an eye pad or goggles to protect the eye, Eye drops (lubrex 0.5%) to lubricate the eye and ciprofloxacin (Ciplox 0.3%) to avoid any secondary infection were prescribed. Single dose of Dexamethasone (4mg/ml) 2ml subcutaneously was administered on the same day. Patient was followed up after three days and improvement in muscles of facial expression was observed (Fig. 7 - Fig. 11).

Fig. 11: Patient is able to smile properly

DISCUSSION

Bell's palsy is named after Sir Charles Bell (1744-1842) who was responsible for the initial description. He mentioned that the palsy was essential due to acute inflammation or oedema involving the nerve, leading to compression of nerve fibres within the stylomastoid foramen in turn resulting in paralysis. An infective aetiology was suggested by Leibowitz after his careful epidemiological survey. In some cases no predisposing cause was found, but not uncommonly there is history of exposure to chill or cold as in riding a vehicle with window open as reported in the present case or sleeping with one side of the face to the air conditioner. Rarely Facial paralysis may follow acute infection of the nasopharynx as seen in Herpes Zoster (Ramsay Hunt syndrome).[3] Ballenger says that primary vasospasm of the blood supply of facial nerve or a viral mononeuritis leads to oedema of nerve causing secondary interference with capillary and lymphatic supply to the nerve and partial or complete loss of function. This inevitably leads to a fibrous change in the tissue surrounding the nerve as it leaves the stylomastoid foramen.[6] Some evidence indicates that disease may be caused by ischaemia of the nerve near the stylomastoid foramen resulting in oedema of the nerve, its compression in the facial canal and, finally, paralysis.[4] The diagnosis of the lesion is of utmost significance as the probable course and the type of treatment will depend on this. Bell's palsy must only be diagnosed when all other types of facial palsy have been excluded. In the diagnosis of facial nerve palsy of idiopathic type, the following points should be noted:[7]

1. A detailed history should be taken including history of exposure to cold, ear discharge, trauma to the orofacial region, major surgical procedures around face, viral fever, or even familial tendencies.
2. Accurate assessment of involvement of the facial muscles and the severity of involvement by electromyographic procedures is required.

In general, Bell's palsy should be confirmed only if most or all of the following symptoms are present:[8]

1. Mostly unilateral involvement and rarely bilateral.
2. Sudden onset with or without pain in the mastoid region, within the ear or around the angle of the jaw. There can even be stiffness of the muscles of facial expression.

3. Paralysis of the muscles of facial expression. Both upper and lower facial muscles are affected in lower motor neurone palsy and only lower facial muscles are affected in upper motor neurone palsy. The muscles are paralysed to an equal extent for voluntary, emotional and associated movements.
4. The eyebrow droops and the wrinkles of the eye are smoothed out. Frowning and raising of the eyebrow is impossible owing to paralysis of the orbicularis oculi.
5. The palpebral fissure is wider on the affected side than on the normal side and closure of the eye is impossible.
6. Demonstration of Bell's sign (eyeball rolling upwards and slightly inwards).
7. Eversion of the lower eyelid and lack of approximation of the punctum to the conjunctiva impair absorption of tears which tend to overflow the lower lid.
8. The naso-labial furrow is smoothed out, the mouth is drawn over to the normal side in unilateral palsy, and the face will become mask like in bilateral palsy.
9. As the buccinators is also paralysed, the cheek is puffed out in respiration and food tends to accumulate between cheek and teeth.
10. When the inflammation has expanded up to involve the nerve about the point at which the chorda tympani leaves it, there is loss of taste on the anterior two-thirds of the tongue and if the branch to the stapedius is also involved, the patient might have hyperacusis.

Bell's palsy causes a peripheral lower motor neuronepalsy, which manifests as the unilateral impairment of movement in the facial and platysma muscles, drooping of the brow and corner of the mouth, and impaired closure of the eye and mouth. Bell'sphenomenon - upward diversion of the eye on attempted closure of the lid - is seen when eye closure is incomplete. A central upper motor neurone deficit causes weakness of the lower face only. More complex segmental deficits may be caused by peripheral facial nerve lesions.[9] The main aims of treatment in the acute phase of Bell's palsy are to speed recovery and to prevent corneal complications.

Eye Care

Eye care of patients with Bell's palsy focuses on protecting the cornea from drying and abrasion due to problems with lid closure and the tearing mechanism. The patient is educated to report new findings such as pain, discharge, or change in vision. Lubricating drops should be applied hourly during the day and a simple eye ointment should be used at night.[10]

Corticosteroids

Two recent systematic reviews concluded that Bell's palsy could be effectively treated with corticosteroids in the first seven days, providing up to a further 17% of patients with a good outcome in addition to the 80% that spontaneously improve. Other studies have shown the benefits of treatment with steroids; in one, patients with severe facial palsy showed a significant improvement after treatment within 24 hours. Recovery rates in patients treated within 72 hours were enhanced by the addition of acyclovir. The rationale for the use of corticosteroids in acute phase of Bell's palsy is that inflammation and oedema of the facial nerve are implicated in causing Bell's palsy and corticosteroids have a potent anti-inflammatory action which should minimise nerve damage and thereby improve the outcome.[8]

Antiviral Agents

Treatment with antivirals seems logical in Bell's palsy because of the probable involvement of herpes viruses. Aciclovir, a nucleotide analogue, interferes with herpes virus DNA polymerase and inhibits DNA replication. Because of aciclovir's relatively poor bioavailability (15% to 30%), newer drugs in its class are being trialled. Better bioavailability, dosing regimens, and clinical effectiveness in treating shingles have been shown with valaciclovir (prodrug of aciclovir), famciclovir (prodrug of penciclovir), and sorivudine.[8]

CONCLUSION

A case of 27-year old male patient with unilateral Bell's palsy is reported. The possible etiological & treatment aspects have been discussed. The rationale for the use of corticosteroids in acute phase of Bell's palsy is that inflammation and edema of the facial nerve are implicated in causing Bell's palsy and corticosteroids have a potent anti-inflammatory action which should minimise nerve damage and thereby improve the outcome. It was noted that corticosteroids proved efficacious in the initial stages of Bell's palsy especially when treated within 24 hours of onset of the disease with a single subcutaneous injection of long-acting corticosteroid.

REFERENCES

1. May M. Trauma to the facial nerve. Otolaryngol Clin North Am 1983;16:661-70.

2. May M, Klein SR, Taylor FH. Idiopathic (Bell's) facial palsy. Natural history defies steroid or surgical treatment. Laryngoscope 1985;95:406-9.
3. Walton JN. Brain's disease of the nervous system. 8th edn. (First Indian impression.) Delhi: Oxford University Press, 1980:182-5.
4. Shafer WG, Hine MK, Levy BM. A text book of oral pathology. 4th edn. Philadelphia: WB Saunders, 1983:859-60.
5. Robinson WP, Moss BF. Treatment of bell's palsy with cortisone. JAMA 1954;154:142-3.
6. Ballenger JJ. Disease of nose, throat, ear, head and neck. 13th edn. Philadelphia Lea & Febiger, 1985:1278-9.
7. Oslen KD. Facial nerve paralysis. I. General evaluation bell's palsy. Postgrad Med 1984;75:2219-22, 224-5.
8. Muralidhar M, Raghavan MRV, Bailoor DN. Bilateral Bell's palsy: current concepts in a aetiology and treatment. Case report. Aust Dent J 1987;32(6):412-6.
9. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg 1985;93:146-7.
10. Holland JN, Weiner GM. Clinical review: Recent developments in Bell's palsy. BMJ 2004;329:553-7.