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Case Series

Extracranial Bell's Palsy: A Case Series

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Abstract:

The facial nerve which is the seventh cranial nerve, comprising of motor, sensory and parasympathetic components is responsible for voluntary facial movement, taste to the anterior two-thirds of the tongue; and control of salivary and lacrimal gland secretions. Bell's palsy obtains its name from Sir Charles Bell, (1774-1842). Sir Charles Bell first described the syndrome along with the anatomy and function of the facial nerve. Bell's palsy is a common, idiopathic, acute, cranial mononeuropathy, affecting males and females equally. It has a slightly higher incidence in men above 40 years of age and in women younger than 20 years of age. The incidence ranges from 11.5 to 40.2/100,000. Individuals with Diabetes Mellitus, arterial hypertension, immunocompromised patients, patients who have had a viral upper respiratory tract infection; and pregnant woman are at greater risk of Bell's palsy. We present here two unusual cases of Bell's palsy. The first case was in a 45 year old lady who presented with grade IV House Beckmann facial nerve palsy. The likely aetiology was local anasthesia injection trauma for removal of 16 impacted molar tooth due to aberrant facial nerve pathway but was unlikely due to the delayed presentation. She was managed with oral corticosteroids, valacyclovir and lubricants to prevent eye dryness. The second case was in a 17 year renal transplant recepient on immnuosuppressants, who presented with painful swelling of parotid gland. MRI revealed parotid necrosis with the swelling extending into the retropharyngeal space. He was managed with injectable antibiotics, valacyclovir and reduction in dose of immunosuppressants. Both cases were managed successfully in our tertiary care centre and were discharged after complete recovery.

Keywords: Bell's palsy, Facial Nerve, Extracranial, Steroids, Valacyclovir.

Introduction

The complex anatomy of the facial nerve which is the seventh cranial nerve, comprising of motor, sensory and parasympathetic components is relevant to understanding its function. [1,2] It is responsible for voluntary facial movement, taste to the anterior two-thirds of the tongue; and control of salivary and lacrimal gland secretions. [1]

The facial nerve runs along an intracranial, intratemporal and extratemporal path. The terminal facial branches of the facial nerve traditionally conceptualized into temporal, zygomatic, buccal, marginal mandibular and cervical branches. These terminal motor branches control all facial expression and other functional tasks such as eye and mouth closure and nasal patency during inspiration. All along its course, the facial nerve forms several communications between its own branches as well as with adjacent cranial nerves. [1]

Bell's palsy obtains its name from Sir Charles Bell, (1774-1842), who was an accomplished Scottish anatomist, artist, surgeon and teacher. [1,3] Sir Charles Bell first described the syndrome along with the anatomy and function of the facial nerve. [4] Bell's palsy is a common, idiopathic, acute, cranial mononeuropathy, affecting males and females equally. It has a slightly higher incidence in men above 40 years of age and in women younger than 20 years of age. [1,2,4] The incidence ranges from 11.5 to 40.2/100,000. [1,5] Individuals with Diabetes Mellitus, arterial hypertension, immunocompromised patients, patients who have had a viral upper respiratory tract infection; and pregnant woman are at greater risk of Bell's palsy. [1,4,5]

Autoimmune disorders, infections, ischaemia and hereditary predisposition are some of the various aetiologies proposed for Bell's palsy. [6,7] The herpes simplex virus inducing facial nerve oedema is the most generally accepted etiology. [2,5] Some studies have demonstrated seasonal variation, with a slightly higher incidence in winter; and slight preponderance in arid climates. [1,8,9]

Case presentation

Case 1: A 45-year-old woman with chronic periodontitis had developed an impacted molar. On the left no 16. She was under treatment for Periodontitis with antibiotics and had undergone root canal treatment for the same. She was planned for removal of her 16 molar tooth under local anesthesia. Her removal of the tooth was uneventful. She reported after 48 hours with complaints of asymmetry of her left nasolabial fold and left lip and partial inability to close her left eye at rest. There was no evidence of any conjunctival inflammation. Her perioral examination revealed a healing dental mucosa and no evidence of inflammation.

She was diagnosed as a case of Grade IV House-Brackmann Bell's Palsy syndrome. [10] She underwent relevant investigations in the form of HSV Antibodies, Varicella Zoster Virus antibodies, serum ACE levels, complete blood counts and metabolic profile which were all normal. A possible etiology considered was a local anasthesia injection trauma due to aberrant facial nerve pathway but was unlikely due to the delayed presentation. She was managed with oral pprednisolone 40 mg for 05 days and empirical tab valacyclovir 1 gm three times a day pending her varicella zoster antibody results. She had 80% recovery within the first week. She was also symptomatically treated with eye lubricants to prevent dryness.

Case 2: A 17 year old renal transplant recipient with native kidney disease being posterior urethral valve related secondary vesicoureteral reflex leading to chronic interstitial disease. He had undergone a live related transplant with his mother being donor and was having a stable graft function with urea- 18 mg/dl, serum creatinine- 0.8 mg/dl, AST- 18 IU/L, ACT – 12 IU/L, Hb- 12.8 g/dl, TLC- 8100 mm3, platelets- 160000 mm3 at one year. He presented with 05 days history of painful swelling of parotid gland, difficulty in opening of the mouth and high grade fever of temp 102˚F. On examination, there was enlargement of the parotid gland with raised temperature and local tenderness. There was an incomplete right facial nerve palsy House- Brackmann grade III in the form of loss of nasolabial fold, facial asymmetry and difficulty in complete closure of the left eye. [10] He underwent MRI of parotid which revealed non enhancing hypointense lesion in the right parotid gland with severe parotid necrosis and the swelling extended into the retropharyngeal space. He was managed with injectable antibiotics, tab valacyclovir 01 gm three times a day for five days, reduction in dose of his mycophenolate mofetil to 500 mg BD from 01gm BD; and careful observation for an invasive disease. His virological markers were negative for herpes simplex virus, varicella-zoster virus, paramyxovirus and serum ACE levels. He gradually improved with reduction in size of the swelling without requiring any incision and drainage. He was discharged after 02 weeks of antibiotics with complete improvement in the facial palsy. A follow up MRI scan showed complete resolution of the hyper intensity in the parotid.

Discussion

Bell's palsy is one of the commonest aetiology of acute, unilateral facial paralysis. Though the exact etiology is yet unknown to medical science, it is believed that HSV-1 virus mediates facial edema, consequently leading to facial paralysis. [2] Other infectious agents such as influenza virus, mycoplasma, syphilis, human immunodeficiency virus, and Lyme disease have also been postulated. [11,12,13] Traumatic facial nerve palsy due to local anaesthesia has also been reported by Lubszczyk et al. [14] Extracranial aetiology of facial nerve palsy in immunocompromised renal transplant patients has also been reported by Fustes et al and Hartley et al. [15,16]

In 70% of cases of Bell's palsy recovery is spontaneous and complete. Remission usually commences within 3–4 weeks; and complete recovery occurs within 6 months. [17] In a study of 2570 cases of facial nerve palsy, Petersen observed that, 85% of patients' facial nerve functionality was restored within 3 weeks, while in the remaining 15% it was restored after 3–5 months. [12] Majority of Bell's palsy cases recover facial function spontaneously. Initial management with oral corticosteroids to improve facial function recovery is recommended, along with measures to protect ophthalmic desiccation. [2] Heckmann et al recommended corticosteroid treatment for the idiopathic form of the disorder. [18]

The underlying cause, expected duration of nerve dysfunction, anatomical manifestations, symptoms severity; and objective clinical findings are multiple factors which determine the appropriate treatment of patients of Bell's palsy. Patients with new-onset Bell's palsy should be managed with systemic steroids to increase the likelihood of recovery of facial nerve function; and reduce synkinesis. [18] However, optimal treatment for Bell's palsy still remains controversial. Early institution of corticosteroids for 10 days is strongly recommended. Some schools of thought often also recommend the simultaneous use of antiviral therapy, albeit with less supporting evidence. [19] Peregrino et al demonstrated that electrical stimulation treatment in the early phase of Bell's palsy led to a recovery. [5]

A multidisciplinary team plays a pivotal role in the evaluation and rehabilitation of these patients. [17,19] Other management strategies recommended in literature include observation, physical therapy,

pharmacological therapy, chemo denervation, facial nerve exploration, decompression, repair, and the entire gamut of static and dynamic surgical interventions. [20] Further management to improve facial function; and manage secondary sequelae such as synkinesis, hypertonicity or facial asymmetry may be required in patients who do not resolve completely. [2]

Conclusion

In this case series we present clinical profile of two atypical cases of Bells' palsy with extracranial aetiology, who were successfully managed in our tertiary care centre. Because 25–40% of cases of facial nerve palsy are not idiopathic, differential diagnosis is of prime importance. Key diagnostic methods include a clinical neurological examination, otoscopy, and a lumbar puncture for cerebrospinal fluid examination. Idiopathic Bell's palsy being a frequent clinical encounter, diagnosis has to be made carefully in all age groups, excluding other causes of peripheral facial nerve dysfunction. Our case series highlights the importance of considering Bell's palsy as a potential diagnosis for facial nerve palsy across all age's age group, which thus guides further clinical care plans accordingly. Comprehensive diagnostic investigation is mandatory in atypical cases, and thorough management must be multidisciplinary.

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