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Bell's Palsy- A brief Review

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ABSTRACT

Bell's palsy is an acute, ipsilateral, facial nerve (CN VII) paralysis of unknown etiology that results in weakness of the platysma and muscles of facial expression. Peripheral facial nerve palsy is the commonest cranial nerve motor neuropathy. The causes range from cerebrovascular accident to iatrogenic damage. Bell's palsy is currently considered the leading disorder affecting the facial nerve. The literature is replete with theories of its etiology, but the reactivation of herpes simplex virus isoform 1 (HSV-1) and/or herpes zoster virus (HZV) from the geniculate ganglia is now the most strongly suspected cause. Despite the advancements in neuroimaging techniques, the diagnosis of Bell's palsy remains one of exclusion. In addition, most patients with Bell's palsy recover spontaneously within 3 weeks.

1. Introduction

Orofacial Pain is a complaint that around the world affects millions of people on a daily basis (1,2). It constitutes any symptom that occurs from a large number of disorders and diseases that result in a sensation of discomfort or pain felt in the region of the face, mouth, nose, ears, eyes, neck, and head. When pain occurs in the Orofacial region however, it often sparks an immediate attention consisting of a significant level of concern and worry (3).

Orofacial pain is the field of dentistry devoted to the diagnosis and management of chronic, complex, facial pain and oromotor disorders(4). Orofacial pain, like pain elsewhere in the body, is usually the result of tissue damage and the activation of nociceptors (noci-is derived from the Latin for "hurt"),(5) the relatively unspecialized nerve cell endings that initiate the sensation of pain which transmit a noxious stimulus to the brain. However, due to the rich innervation of the head, face and oral structures, orofacial pain entities are often very complex and can be difficult to diagnose (4).

In this century, the concept of pain has evolved from that of a one-dimensional sensation to that of a multidimensional experience encompassing sensory, discriminative, cognitive, motivational and affective qualities. The most recent definition of pain, produced by the Task Force on Taxonomy of the International Association for the Study of Pain (IASP) is, "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."(6) (Burkitt's text book - 10th edition)

Facial nerve paralysis (FNP) is the most common cranial nerve disorders and it results in a characteristic facial distortion that is determined in part by the nerves branches involved. With multiples etiologies, these included trauma, tumor formation, idiopathic conditions, cerebral infarct, pseudobulbar palsy and viruses.(7)

Nicolaus Friedrich, an 18th century professor of medicine at Wurzburg, may have been the first to publish a case report of a facial nerve paralysis of unknown origin. He gave an account of 3 middle aged adults who had a similar onset of acute or sub-acute unilateral facial paralysis, which gradually improved over a period of weeks to months. His clinical findings, *De paralysis Musculorum Faciei Rheumatica*, were first published in 1798 in the German medical literature. Two years later, this paper was reviewed in English and published in the Annals of Medicine in Edinburgh. Around that time, a young Charles Bell was studying medicine at the institution, and may have read Friedrich's paper. Bell later studied the function of the facial nerve in animals while practicing surgery in London, he encountered many cases of unilateral facial nerve paralysis and published his first report in 1821. Interestingly, his most famous and widely quoted account of facial paralysis was published in 1828, in which he told the story of a man who had been tossed by a bull. The ensuing puncture wound led to a lasting paralysis of his facial nerve. Although Bell's initial publication on facial paralysis came after Friedreich's account by 23 years, Grzybowski argues that Bell deserves the credit for differentiating peripheral from central facial nerve paralysis. Today, the term *Bell's palsy is synonymous* with *Idiopathic Peripheral Facial Paralysis*.

Bell's palsy or idiopathic facial paralysis is the most common cause of unilateral facial paralysis, accounting for approximately 50% of the cases.(8) The reported incidence of Bell's palsy ranges from 13 - 34 per 100,000 population annually.(9) The second most common cause of facial paralysis is infection (15% of cases), may be odontogenic; followed by neoplasms (13.5%) such as acoustic neuromas on the base of the brain, parotid tumors of the side of the face, and glomus jugular tumors of the neck. Bell's palsy affects people of all ages, but, most commonly, individuals 15 - 45 years old. Its onset is sudden, with facial muscle weakness pro- gressing over hours to days.(8)

In 85% of the cases recovery of facial muscle function begins within the first 3 weeks after onset. Of the remaining 15% of the patients, return of facial muscle function does not begin until after 3 months. Approximately 70% of all patients recover completely. The remaining 30% may experience residual weakness, hyperkinesia, contracture, or synkinesis.

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The degree of recovery bears some relation to age, with older patients having a poorer recovery.(9)

A thorough medical history and physical examination are the first steps in making a diagnosis. It is essential to rule out other causes of facial paralysis before making the definitive diagnosis, which implies the intervention.(8)

Recent advances in neuroimaging techniques such as MRI have equipped modern physicians with a considerable advantage over their predecessors in respect to visualizing the facial nerve. However, to our knowledge, there is no consensus on the etiological explanation or the preferred long-term treatment option for Bell's palsy. In this review, we aim to provide the most comprehensive understanding of Bell's palsy to date, with emphasis on clinical implications and the preferred management strategies based on reports from the recent literature.

2. Discussion

Teeth are a common and obvious source of Orofacial pain. Ninety percent of orofacial pain arises from the teeth and oral structures.(2) As dentists, we are trained to diagnose and treat often acute dental pain problems. After ruling out dental problems, musculoskeletal and neuropathic pain conditions are the most common causes of facial pain.(2, 9, 10) Due to the diversity of manifestations and different mechanisms of pain transmission, the differential diagnosis is crucial for the establishment of a successful management strategy.(4)

Persistent and chronic pain is more common in the head and neck region than in any other part of the body; therefore, dentists are more likely to encounter these rather complex cases in their practices.(11) Causes of unilateral facial nerve paralysis are varied and include multiple possibilities (idiopathic, infectious, traumatic, and neoplastic). Bell's palsy is the most common cause of unilateral facial nerve paralysis. (12)

Facial nerve is a mixed nerve with special visceral efferent, general visceral efferent, special visceral afferent and general somatic afferent functions.(13) The pathway of the facial nerve is long and relatively convoluted, and so there are a number of causes that may result in facial nerve paralysis.(14) Facial nerve paralysis may be central or peripheral in origin, complete or incomplete and results in a characteristic facial distortion that is determined in part by the nerves branches involved. Its cause is varied and included idiopathic conditions, infections, tumor formation, iatrogenic problems, trauma, cerebral infarct, pseudobulbar palsy and viruses.(7)

Tissue response to infection involves cytokine release and edema which cause local metabolic disturbances, intraneural swelling and ischemia with vasa nervorum (small arteries that provide blood supply to peripheral nerves), pre- venting normal axonal conduction.(15, 16) Neuropraxia of the facial nerve caused by compression is the most likely cause of the patient's hemi-facial paralysis. Minor compression causes temporary conduction block without axonal degeneration, and the recovery is full and rapid. Removal of the offending tooth with endodontic treatment of next tooth resulted in improvement, confirming that a temporary conduction block is more likely than axonal disruption.(16)

The review of literature confirms that lower motor neuron palsy of the facial nerve in conjunction with infections of dental origin is rarely reported. Hamlyn et al. reported the case of 12 years old child who developed acute hemiplegia attributable to a fractured infected lower incisor tooth. The mechanism was unclear but the possibility of local infection crossing internal carotid arteries and subsequent central nervous symptoms appeared most likely.(16)

Bobbitt TD, et al. reported a case in which an 18 years old man presented with an infected lower third molar and palsy of the frontal branch of the left facial nerve in the presence of left parotid and left posterior auricular swelling. Resolution occurred within 6 months. The authors concluded that the exact mechanism was unclear, but likely to be a mixed picture of toxicity and compression neuropraxia. (16, 17)

Vasconcelos BC, et al. reported a case of a 21 years-old black woman who developed a Bell's palsy after an impacted third molar surgery under local anesthesia. The treatment was based on prescription of a citidine and uridine complex; one tablet twice per day and a close follow up. Three months later, the patient recovered her normal facial muscle activity.(7)

Bell's palsy typically presents with a sudden and rapid onset of unilateral facial weakness, often within a few hours [20]. In fact, the symptoms can be so startling that most of the affected individuals either think they have had a stroke or a serious brain lesion [21]. It is important to note that up to 60% of these patients report a preceding viral illness [23]. Initially, partial palsy is reported by most patients, with maximum facial weakness often seen within 2 days [20,21]. Patients may also complain of ipsilateral earache as well as numbness of the face, tongue, and ear [22]. Moreover, cases of hyperacusis (possibly from stapedial muscle dysfunction), tinnitus, taste disturbances (most likely from injury to nervus intermedius proximal to geniculate ganglion), and decreased lacrimation have also been reported [18,19,21]. The motor deficit is almost always unilateral in Bell's palsy [19], with both the upper and lower parts of the face affected. This helps to distinguish the disorder from a central supranuclear lesion, in which paresis occurs only in the lower facial muscles [19]. An often overlooked psychological component may also exist in patients presenting with acute Bell's palsy, in which the acute onset of the condition can drastically affect social activity [24].

Bell's palsy is typically diagnosed by exclusion, and a thorough history and physical exam is needed to rule out other treatable or intracranial lesions [18]. Ear function should routinely be tested using tuning forks and standard pneumatic otoscopes. Clinical evidence of herpes zoster infection may help aid in the diagnosis of Bell's palsy. However, vesicular lesions may be absent in the presence of pre-herpetic neuralgia in a clinical condition termed *Zoster sin herpete* [21]. The modified House-Brackmann scale (1985) is a clinical tool used to document the degree of facial paralysis and to predict probability of recovery. It assesses gross facial features and symmetry, both at rest and during motion. The grading is from 1

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to 6, with the latter being total paralysis [20]. Patients who present with some observable facial movement and incomplete paralysis are expected to have uniformly good recovery. Patients with a House-Brackmann score of 6 may have prolonged or incomplete recovery. The Sunnybrook facial grading system, Yanagihara grading system, and Sydney grading system represent regional alternatives to the House-Brackmann scale and have similar reliability, although reported ease of use varies. The House-Brackmann scale is currently the most widely used and accepted.

Electromyography (EMG) and motor nerve conduction studies of the facial nerve can yield useful information on the viability of the affected nerve, thus aiding in the decision making process regarding treatment and/or surgical management of disease [25]. These electrodiagnostic studies yield information on the amount of evoked action potentials in affected muscles. Using this data, clinicians can estimate the amount of axonal loss. Patients exhibiting greater than 90% axonal degeneration should be considered for surgical decompression, while axonal degeneration of less than 90% has a favourable prognosis [26]. These studies are clinically useful within 2 weeks of complete facial paralysis. After 3 months of onset of symptoms, needle electromyography may be employed to confirm any subclinical signs of re-innervation, thus acting as a prognostic indicator for the possibility of recovery [25]. Up to 5% of all lower motor neuron facial paralysis may be due to benign and malignant neoplasms. If there is clinical suspicion, imaging studies such as CT with contrast or gadolinium-enhanced MRI are useful in ruling out neoplasms [18]. It is suggested that any case of BP without resolution within 4 months or first presenting 4 months after symptom onset undergo contrast-enhanced imaging of the parotid gland, temporal bone, and brain. Repeat imaging is indicated if symptoms persist at 7 months without a readily identifiable cause. Biopsy of affected tissue adjacent to the facial nerve may then be considered if imaging is negative at 7 months.

Because the exact cause is still unknown, Bell's palsy has no prevention or cure. Thus, attempts at management over the years have been geared toward reducing inflammation to the facial nerve and/or preventing corneal complications stemming from paresis of the facial muscles [20]. Protecting the cornea from excessive dryness and abrasions should be addressed by the clinician through proper patient education. The cornea of patients with BP is especially at risk of drying because of improper lid closure and decreased tear production. Prescription of hourly lubricating eye drops and eye ointment during sleep are recommended [21]. In addition, the clinician should be prepared to provide psychological support in the early stages of management [21].

A meta-analysis by de Almeida et al. on 10 trials, assessing the efficacy of Prednisolone versus placebo in the management of Bell's palsy, also reported an overall significant benefit to treatment using Prednisolone, with the number needed to treat (NNT) at 11 [27]. These findings were corroborated by a recent Cochrane review of 8 RCTs with over 1500 patients (2010), which validated the benefit of treating Bell's palsy patients with corticosteroids. Of the 754 patients who were treated with corticosteroids, the authors noted that only 23% had incomplete recovery of facial motor function at 6 months, compared to 33% of patients who were given the placebo. In addition, they noted that the patients who had received corticosteroids had a significant reduction in motor synkinesis during follow-up compared to their counterparts [29].

Antiviral agents against HSV have also been extensively used to treat Bell's palsy, and their effectiveness has been increasingly disputed based on recent studies. A 2009 Cochrane systematic review of 7 high-quality trials with 1987 participants concluded that anti-herpetic anti-virals provide no significant benefit compared with placebo in generating complete recovery from Bell's palsy [30]. Although anti-virals alone do not provide a clear benefit, the efficacy of corticosteroids plus anti-virals is less clear. Several studies have found no significant benefit from using combined antiviral and glucocorticoids treatment [31]. Recently, a network meta-analysis of 6 studies with a total of 1805 patients from 1996 to 2008 showed that a combination of corticosteroids with anti-virals had a marginal benefit over corticosteroids alone [28]. However, since the findings did not reach statistical significance, the authors concluded that Prednisone remains the single best evidence-based treatment for Bell's palsy [28].

3. Conclusion

In conclusion, the cause of Bell's palsy remains elusive, but herpetic reactivation is now the most strongly suspected cause. Its clinical presentation is well-documented in the literature. However, the diagnosis is still one of exclusion. While prior systematic reviews showed no difference between medical therapy and placebo in treating Bell's palsy, results of recent RCTs strongly recommend corticosteroids as the treatment of choice. Antiviral therapy is of no significant help according to most reliable studies. Surgical treatment of Bell's palsy is still controversial, and should only be used for refractory cases. Although odontogenic infection rarely presents with facial nerve paralysis and it should be considered, particularly before more significant complications of submandibular or submassetric abscess formation becomes established.

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