RAMSAY HUNT SYNDROME SECONDARY TO DENTAL EXTRACTION

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INTRODUCTION

Ramsay Hunt syndrome, also known as geniculate neuralgia or nervus intermedius neuralgia is defined as an acute peripheral facial neuropathy associated with erythematous vesicular rash of skin of ear canal, auricle (also termed herpes zoster oticus), and/or mucous membrane of the oropharynx. The varicella zoster virus may involve multiple cranial nerves in addition to the facial nerve such as VIII, IX, V, and VI.

We present a case of Ramsay Hunt Syndrome that developed after upper molar tooth extraction of the same side.

CASE REPORT

An eleven years old child presented to us with history of heaviness of right side of face and inability to close his right eye for the past 01 day. The child had an impacted upper molar tooth extraction on the same side under local anesthesia (2% lignocaine + 1:100000 adrenaline) by the dental surgeon 02 days ago. Approximately 24 hours after the dental extraction his parents noticed asymmetry of the face with deviation of the angle of mouth to left and inability to close the right eye completely and the child was brought to us.

Upon examination the patient was found to have a lower motor neuron type facial nerve paralysis on the right side (Fig). Severity of the facial palsy was assessed as grade IV according to House-Brackmann system. Taste sensations were impaired on right side of tongue and he had hyperacusis in right ear. There was no herpetic rash at that time. The patient was started on tab prednisolone 1mg/kg body weight/day in three divided doses; tab acyclovir 200mg 5 times a day; artificial tear eye drops and eye pad. The parents were counselled and the patient was advised facial excercises. He was also given antacids as a prophylaxis against steroids induced gastric erosions. The patient was requested to follow up on the third day. Examination on his follow-up after 48 hrs revealed a herpetic rash over his right choncha and external auditory meatus. There was no eye or intraoral involvement. It was acquired that the child had no previous history of chicken pox or herpes zoster infection. The dose of acyclovir was increased to 800mg 5 times a day and topical acyclovir was also advised. Pure tone audiometery did not reveal any hearing impairment. Lesions of herpes zoster dried and crusted in another 5 days. Acyclovir was stopped after 10 days whereas steroids were tapered off. His hyperacusis settled in two weeks whereas taste sensations remained absent for another week and then slowly returned. The facial nerve functions took 08 weeks to return to a complete normal.

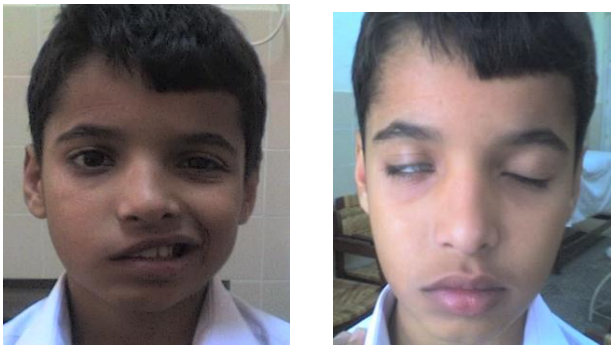

Fig: Lower motor neuron facial nerve palsy (Rt).

DISCUSSION

Facial nerve paralysis following oral surgery has been very rarely reported in the literature. The most common etiology proposed has been due to direct trauma to the facial nerve either as a result of local anesthesia injection needle, osteotomies, or direct trauma to the facial nerve during attempts at removal of cysts, tumors or treatment of facial fractures[1,2]. The paralysis in this case may be partial or complete depending upon the region being operated and the branches involved. The onset is usually rapid and can be easily attributed to surgery.

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In our case the onset was approximately 24 hrs after the surgery and therefore direct trauma was ruled out. Few other proposed etiologies like development of intraneural haematoma or local anesthesia toxicity were also ruled out as the onset in these cases is also rapid[3]. Infection and abscess formation following dental procedure has been reported in literature as a cause of facial nerve paralysis however the fact that an early decompression in these cases did not improve the paralysis supported the fact that the etiology might have been other than infection[1]. Although the exact mechanism is unknown, multiple factors like postoperative ascending edema, infection, lowered cellular immunity as a result of surgery or direct trauma due to a blast of air into the tissue during cleansing of extraction site may be attributed.

Facial nerve paralysis after dental extraction has been proposed to result from direct tissue damage from blast of air into the extraction site for cleaning purposes. Water irrigation is rather preferred as it serves the same purpose and avoids the risk of subcutaneous emphysema and direct injury[3].

Shuaib presented a case of recurrent facial nerve palsy on the same site after dental extraction. The patient developed facial nerve palsy on two different occasions, two years apart, each time after dental extraction. Direct toxicity due to local anesthetic, retrograde compression edema causing ischemia of facial nerve, lowered local cellular immunity and Bells palsy were the proposed etiologies however the exact cause remained unknown[4].

All local anaesthetic agents are neurotoxic to some extent. Procaine and tetracaine cause more nerve damage than bupivacaine or lidocaine, although lidocaine can also be neurotoxic. However these agents are neurotoxic only if they are injected intrathecally in which case one should only expect paralysis of the particular area supplied by the fascicle into which the injection has been given rather than the paralysis of the whole nerve[5].

The varicella zoster virus (VZV) that causes Ramsay Hunt syndrome is the same virus that causes chickenpox. Following resolution of chickenpox, VZV lies dormant in the dorsal root ganglia until reactivation results in herpes zoster (shingles). Exactly why or how VZV reactivate from latency is not fully understood, however, a lowered VZV-specific cell-mediated immunity has been shown to be a major factor in determining reactivation of VZV[6,7]. VZV reactivation causes inflammation in the dorsal root ganglion accompanied by hemorrhagic necrosis of nerve cells. The result is neuronal loss and fibrosis. The distribution of the rash corresponds to the sensory fields of the infected neurons within a specific ganglion. The anatomic location of the involved dermatome often determines the specific manifestations e.g., herpes zoster opthalmicus causing ocular complications when the trigeminal ganglion is involved.

Classical Ramsay Hunt Syndrome, in addition to the vesicular eruption, may be associated with severe otalgia, hearing loss and vertigo. In our case an early diagnosis of Bells palsy was made and the patient was started on acyclovir within the first few hours of the onset of facial paralysis. Probably this was the reason that a full blown Ramsay Hunt Syndrome did not manifest in this case and the recovery was also rapid and full.

The diagnosis of Bell's palsy and Ramsay Hunt Syndrome is mainly clinical as no specific laboratory investigations are available. Structural lesions can be ruled out by CT scan, MRI, or magnetic resonance angiography. Gadolinium enhancement of the vestibular and facial nerves on MRI has been described in Ramsay Hunt syndrome[8]. Audiometry may reveal sensorineural hearing loss however it was normal in our patient. Electrodiagnostic methods, such as facial motor nerve conductions studies (electroneurography), electromyography of facial innervated muscles, the blink reflex, and nerve excitability testing, could add information regarding the extent of seventh cranial nerve involvement but have more of a prognostic value[9].

Inflammation and edema of the facial nerve is thought to be the pathogenesis in Bell's palsy and Ramsay Hunt Syndrome therefore

steroids are thought to be the main stay of treatment[10]. Older studies have shown conflicting results using steroids in treating Bell's palsy[11]. However, 3 recent randomized controlled trials showed significant improvement in outcomes when prednisolone was started within 72 hours of symptom onset[12-14].

Efficacy of antiviral agents in the treatment of Bell's palsy has been debated extensively in the literature however there is evidence to suggest a large percentage of Bell's palsy cases may result from a viral infection. Therefore, antiviral agents may be reasonable in certain situations[12-14]. However in case of Ramsay Hunt Syndrome antiviral agents should always be advised in high doses and an early commencement of treatment is found to be associated with a better prognosis and lesser chances of complications. Labyrinthine sedatives may be required if the patient has got ataxia and carbamezipine is of use in cases where patient develops post herpetic neuralgia.

Facial palsy following tooth extraction is rare and its mechanism is unclear. Though a variety of mechanisms have been linked to this palsy, including direct anesthesia of facial nerve, neurotoxicity of local anesthetic solution, viral re-activation, demyelination, edema, vasospasm, ascending infection and trauma; viral reactivation is proposed to be the most likely one. Therefore, it seems most appropriate to use an antiviral agent combined with steroid for treatment of dental origin facial palsy for a complete and early recovery[1, 3,12-14].

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