Case Report

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Can Facial Paralysis be a Rare Complication of Hypertension?

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Abstract

Facial paralysis is a disorder that can result from a wide spectrum of etiologies including traumatic, infectious, congenital, neurologic, systemic, neoplastic, and iatrogenic causes. It has significant functional, psychological, and social consequences. The most common cause of fascial nerve paralysis is idiopathic facial nerve palsy (Bell's palsy). There is a relationship between facial nerve paralysis and severe systemic hypertension. In this report we present a 43-year-old female patient with fascial paralysis who had a history of hypertension.

Keywords: Facial paralysis, Hypertension, Bell's palsy, Aetiology

Introduction

Facial paralysis is a disorder that can result from a wide spectrum of etiologies including traumatic, infectious, congenital, neurologic, systemic, neoplastic, and iatrogenic causes.[1] It has significant functional, psychological, and social consequences. Functionally diminished lacrimation, ectropion, epiphora, brow ptosis and lagophthalmos can lead to exposure keratopathy with resulting blindness and potential for globe rupture.[2] Nasal valve dysfunction is observed in facial paralysis. Oral competence with resulting difficulties with speech, swallowing, and drooling is also seen.[3]

The most common cause of fascial nerve paralysis is idiopathic facial nerve palsy (Bell's palsy).[4] Bell's palsy (BP) is defined as an acute peripheral facial paralysis, generally affecting one half of the face. The incidence of BP is 15-30/100,000 and it accounts for 60-75% of all unilateral facial palsies.[5] The clinical presentation of BP is usually a rapid onset, unilateral, lower motor neuron-type facial weakness accompanied by symptoms of subjective change in facial sensation, dysgeusia, postauricular pain and hyperacusis. This clinical presentation can be caused by the anatomical construct of the human facial nerve, and especially its mixed nerve profile containing sensory, motor and parasympathetic fibres.[6] It's medical treatment includes corticosteroids and antiviral agents, while physical therapy involves automassage and exercises. Corticosteroids may work best if they are initiated within several days of when the symptoms started.

There is a relationship between facial nerve paralysis and severe systemic hypertension, as was described for the first time by Moxon more than a century ago.[7] In a recently published case-control study it was stated that chronic, nonsevere hypertension may increase the risk of lower motor neuron facial nerve paralysis in patients older than 40 years of age.[8] In this report we present a 43-year-old female patient with fascial paralysis who had a history of hypertension.

Case Report

A 43-year-old female patient presented to our clinic with complaints of headache, facial asymmetry and numbness. Vitals of the patient were found as blood pressure: 155/112 mmHg, SpO_2:96%, and body temperature: 36.7°C. On physical examination of the patient, it was observed that her face was asymmetrical and there was no wrinkle on the right side of her forehead. The patient could not raise her right eyebrow, and could not close her right eye. When she wanted to close her eye, the eyeball was moving up. There was effacement in the right nasolabial groove and sagging

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in the ipsilateral mouth corner. The right half could not accompany laughing, and the patient could not show her teeth. Besides, she described numbness and loss of feeling on the right side of her face. The patient's ECG showed normal sinus rhythm, and there was no abnormality in respiratory and heart sounds. No abnormality was detected in the neurological examination. Although the patient had a history of hypertension, she had not been using any medication for the past year. Laboratory parameters of the patient was found as Hgb: 12.9 mg/dL, Urea: 2.8 mmol/L, creatinine: 52 umol/L, ALT: 68 U/L, AST: 55 U/L, total cholesterol: 4.91 mmol/L, triglyceride: 1.67 mmol/L, HDL: 1.55 mmol/L, HbA1C: 5.6, insulin: 53.7 mcu/mL, TSH: 0.89mIU/L, and FT4: 16.7 pmol/L. The patient was diagnosed with idiopathic Facial Paralysis and was evaluated as Grade 4 according to the House-Brackmann scale. The patient was administered 1 mg/day prednisolone PO tb, acyclovir 800 mg 5x1, synthetic tears and eye closure treatments. In addition, amlodipine 10 mg 1x1 treatment was started and blood pressure was followed up daily and it was controlled in the first week. In the fifth week after the diagnosis, full recovery was observed in all motor and neural functions of the patient. The patient was informed about the objective of this report and gave written informed consent.

Discussion

The facial nerve is a cranial nerve whose function is most impaired due to its long and curved journey in the facial canal between the infratemporal bones.[9] More than 50% of cases of facial paralysis are idiopathic (Bell's palsy).[5] Although the exact etiology of BP is not known, compression of the facial nerve in the canal as a result of edema due to infection/inflammation and vascular ischemia is the most accepted mechanism.[10] Agents such as Herpes simplex virus (HSV), Varicella-zoster virus (VZV), Borrelia burgdorferi have been detected in the cerebrospinal fluids of patients with BP.[11]

When the literature was reviewed to examine the relationship between facial paralysis and hypertension, articles on childhood hypertension and pregnancy-related facial paralysis came to the fore.[12-14] Especially MacArthur and Minson stated that timely diagnosis and treatment of hypertension in facial paralysis improves the prognosis, and blood pressure measurements in children can prevent the disease.[12]

It has been reported that facial paralysis in children is not limited to malignant hypertension and the incidence of facial paralysis increases in benign hypertension cases.[15] When the literature was searched for those over the age of 18, different results were found for the relationship between hypertension and BP. Some studies have reported that hypertension, especially over the age of 40, increases the incidence of BP.[8,16] Our case was 43-year-old and she had a history of uncontrolled hypertension.

Corticosteroids are recommended in the treatment of BP because they reduce edema, increase facial nerve regeneration and improve motor functions.[17] In our case we used prednisolone as corticosteroid.

Although the idea of using antiviral agents in the treatment of BP due to the viral activity shown in the etiology of Bell's palsy, thanks to the increasingly widespread serological tests, has begun to be accepted more and more, in the systematic reviews and meta-analyses conducted to date, definitive and sufficient clinical evidence of the effectiveness of antiviral agents in BP has not yet been obtained.[18] We used acyclovir as the antiviral agent in our patient. Antiviral agents are used as standard treatment in Ramsay Hunt syndrome, which is a disease characterized by reactivation of Varicella zoster virus, which remains latent only in the geniculate ganglion, and subsequent acute peripheral facial paralysis, vesicular rash in the auricle and external auditory canal, and severe ear pain.[19] However, the use of antiviral therapy is still controversial.

Although it has been reported that successful results are obtained when surgical treatment is applied in the early period (within 2-3 weeks after the onset of symptoms) in patients who do not improve despite steroid treatment and who have degeneration over 90-95% in electrodiagnostic evaluations, further studies are needed to determine a common treatment method.[20]

In conclusion, corticosteroids, antiviral agents and antihypertansive medications can be used in the treatment of fascial paralysis caused by Bell's palsy in patients with systemic hypertension. However, since there is no consensus especially on the use of corticosteroids in these cases, further comprehensive studies are needed to enlighten this issue.

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