Facial Nerve Venous Malformation Presenting As Bell's Palsy

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Abstract

Objectives: Facial nerve venous malformations (FNVMs) are slow-growing, benign vascular lesions that arise from perineural capillary networks. They may involve any segment of the facial nerve and most commonly involve the geniculate ganglion. Methods: A 8-year-old boy presented with a history of a right facial palsy over 2 month. No steroids or other medications were given to the child. There were no complaints of associated hearing loss, tinnitus, headache, dizziness or otalgia. He had a riht-side House-Brackmann (HB) grade V weakness. The patient underwent both magnetic resonance imaging (MRI) and computed tomography (CT) scan of the brain and temporal bones which demonstrated a thickening and contrast enhancement in the right post facial post geniculate segment. This appearance was consistent with venous malformation. Results: Due to the close proximity of the lesion to the facial nerve and high risk for facial nerve injury, serial observation, corticosteroid therapy (1 mg/kg/d oral methylprednisolone, followed by a taper of up to 14 days), propranolol therapy (2x40mg for 30 days) and physiotherapy rehabilitation was recommended instead of surgery to the patient. After 30 days, control EMG revealed the significant improvement in the zygomatic branch of the facial nerve. Decided to contınue propranolol therapy and monthly control has been planned. Conclusion: Facial palsy in children is mostly idiopathic. However, there are many different and identifiable etiologies. Detailed neurologic evaluation is important because of identification of an underlying cause and early treatment.

Objectives

The facial nerve is the most common cranial nerve to have a disorder. It is estimated that children over the age of 10 have an incidence of 10 per 100,000 annually and those under the age of 10 to be less than 3 per 100,000 annually. The most common etiology of facial palsy in children is idiopathic (Bell's Palsy), There are various uncommon secondary causes of peripheral facial palsy, including infectious such as varicella zoster virus (VZV), HIV, Lyme disease, otitis media, benign and malignant tumors, autoimmune/inflammatory processes such as sarcoidosis, amyloidosis, and GuillainBarre syndrome, iatrogenic (trauma or prior intervention), and congenital palsy. The prognosis is favorable in children. However, when a child does not show rapid improvement, one must consider uncommon causes such as malignancies and metabolic diseases.

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Methods

A 8-year-old boy presented with a history of a right facial palsy over 2 month. No steroids or other medications were given to the child. There were no complaints of associated hearing loss, tinnitus, headache, dizziness or otalgia. He had a riht-side House-Brackmann (HB) grade V weakness. The blood routine test, C-reactive protein level were both normal, viral and lyme serology were negative.

The patient underwent both magnetic resonance imaging (MRI) and computed tomography (CT) scan of the brain and temporal bones which demonstrated a thickening and contrast enhancement in the right post facial post geniculate segment. This appearance was consistent with venous malformation.

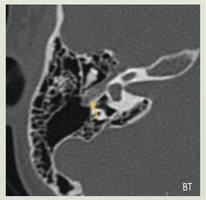
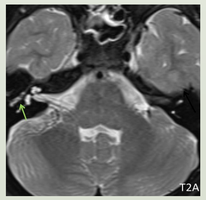


Figure1. Lytic lesion and honeycomb appearance in the right facial nerve post geniculate segment on temporal bone CT (arrows)



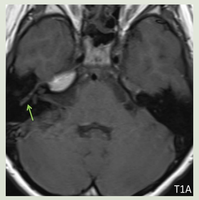
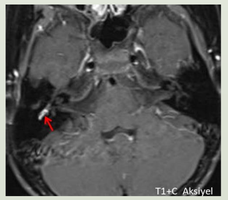


Figure 2-3. T2W hyperintense and T1W isointense lesion in the right facial nerve compared to the brain parenchyma in internal acoustic canal MRI



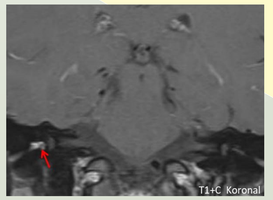


Figure 4-5. Contrast enhancement of the lesion in postcontrast series

Results

Due to the close proximity of the lesion to the facial nerve and high risk for facial nerve injury, serial observation, corticosteroid therapy (1 mg/kg/d oral methylprednisolone, followed by a taper of up to 14 days), propranolol therapy (2x40mg for 30 days) and physiotherapy rehabilitation was recommended instead of surgery to the patient. After 30 days, control EMG revealed the significant improvement in the zygomatic branch of the facial nerve. Decided to contınue propranolol therapy and monthly control has been planned.

Conclusion

Facial nerve venous malformations (FNVMs) are slow-growing, benign vascular lesions that arise from perineural capillary networks. They may involve any segment of the facial nerve and most commonly involve the geniculate ganglion.

FNVMs were previously described as hemangiomas and initially characterized on CT has have irregular ill-defined margins with intralesional bone spicules in a honeycomb pattern. FNVMs account for <1% of temporal bone lesions and 18% of facial nerve tumors. The peak incidence of FNMVs occurs between 30 and 60 years of age slightly more common in females. Symptoms include progressive and sudden facial paralysis and spasm, conductive hearing loss, otalgia, pulsatile tinnitus, aural bleeding, and vertigo. Potential explanations for neural injury have included nerve compression, vascular steal, and invasion.

The most commonly accepted form of the treatment of FNVM is surgical excision. Preservation of facial function is not possible in cases with direct nerve invasion.

Facial palsy in children is mostly idiopathic. However, there are many different and identifiable etiologies. Detailed neurologic evaluation is important because of identification of an underlying cause and early treatment.

We report a case of 8-year-old male who presented with facial paralysis related to a venous malformation involving the GG of the right facial nerve. To the best of our knowledge, this is the first case of facial paralysis reported in literatüre related to a vascular malformation in pediatric patient from Turkey.

References

Rao, D., Fiester, P., Rahmathulla, G., Makary, R., & Tavanaiepour, D. (2020). A case of a facial nerve venous malformation presenting with crocodile tear syndrome. *Surgical Neurology International*, *11*.