CASE REPORT

Facial nerve palsy in a child: Bell's palsy? Think again!

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Abstract

Introduction: Half of facial paralysis in children is idiopathic at origin. However, dismissing facial paralysis as being idiopathic without a thorough history and meticulous examination could be disastrous as illustrated by this case.
Case report: We report a case of sphenoid wing meningioma in a 4-year-old girl. She first presented with only facial asymmetry that was noticed by her mother. Examination suggested a left upper motor neuron facial nerve palsy. A sphenoid wing meningioma was found on magnetic resonance imaging (MRI) of her brain. She underwent craniotomy and total tumour excision. Histopathological examination of the tumour showed a grade 1 transitional type meningioma. Meningiomas in children are rare compared to the adult population. Presentations in children may be delayed due to their inability to recognise or communicate abnormalities. Distinguishing between upper and lower motor neuron facial palsy is crucial in decision making for facial paralysis in children.

Introduction

Facial paralysis in children may be caused by infections such as varicella zoster virus and otitis media. Trauma to the head or face is another common cause. Although uncommon, cerebrovascular accidents and tumours must not be overlooked. Only after excluding these, should we classify facial paralysis as being idiopathic. Cerebral meningiomas usually occur in the fourth and fifth decade of life.[1] Meningiomas in children are rare. In the adult population, 32.2% of all intracranial tumours are meningiomas.[1] On the contrary, the incidence of paediatric meningiomas is about 1.5% to 2.9% of all intracranial paediatric tumours.[2] A local study found that only 1 out of 16 paediatric brain tumours was a meningioma.[3] Of all the intracranial meningiomas, only about 1.9% to 2.8% are reported in the paediatric population.[4,5] In a study of 87 paediatric patients with meningiomas, only 7% were aged between 5 months and 5 years and the majority of them were above 5 years.[6] We report a case of a sphenoid wing meningioma in a 4-year-old girl.

Case report

A 4-year-old girl presented with only facial asymmetry for 1 month. She had left upper motor neuron facial nerve palsy as evidenced by loss of nasolabial fold and drooping of the angle of the mouth of her left side. The forehead wrinkling was intact. Her visual fields were taken as grossly normal as the patient was not fully cooperative during the ophthalmologic examination. The visual acuity was 6/18 and 6/24 for her right and left eye, respectively. Fundoscopy was normal with no papilloedema or optic atrophy seen. There was no relative afferent pupillary defect. She did not have any other cranial nerve palsies or cerebellar signs. She had normal tone, power of 5/5 and normal reflexes for her upper and lower limbs. There were no cutaneous signs of neurofibromatosis. A magnetic resonance imaging (MRI) of her brain revealed an enhancing solid mass 32 × 34 × 42 mm^3 arising from the medial sphenoid wing area causing mass effect and compression of the ipsilateral lateral ventricle. There was no evidence of a vestibular schwannoma (**Figure 2**). The patient underwent craniotomy and total tumour excision. Unfortunately, the surgery caused the patient to have left hemiparesis with power of 3/5 as there was an injury to the lenticulostriate arteries supplying the right internal capsule during dissection. Histopathological examination revealed fragments of tumour tissue composed of whirling to storiform architecture of neoplastic cells. There was no obvious mitosis, necrosis or calcification. The cells were focally positive for epithelial membrane antigen – a marker for meningiomas. A final diagnosis of transitional type meningioma WHO grade I was made. Another MRI of the brain was done three months after the operation and did not show

any evidence of recurrence. Her facial nerve palsy and visual acuity remained the same but her hemiparesis was improved to a power of 4/5.

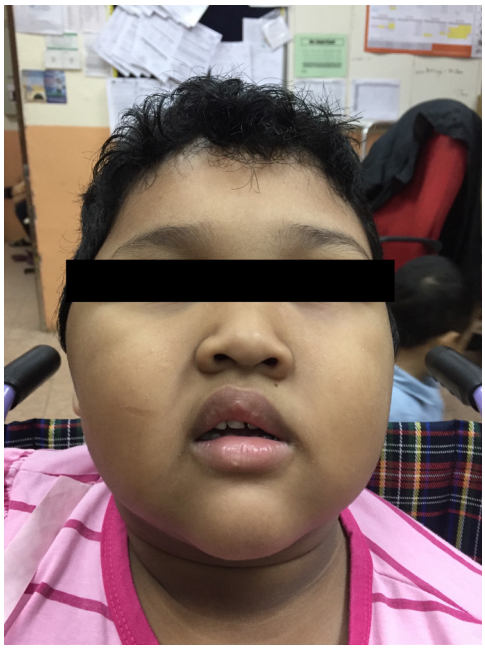

Figure 1. Left upper motor neuron facial nerve palsy

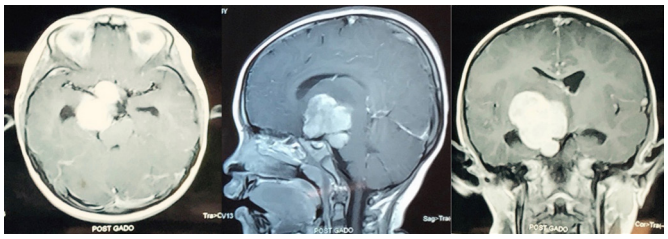

Figure 2. Brain MRI showing enhancing solid mass arising from the medial sphenoid region causing mass effect and compression of ipsilateral lateral ventricle

Discussion

Fifty percent of acquired facial nerve paralysis in children is usually idiopathic and classified as Bell's palsy.[7] The other common causes include complicated otitis media, Ramsay Hunt syndrome, trauma or cerebellopontine angle tumours. It is important to note that these aetiologies cause a lower motor neuron lesion of the facial nerve. Lesions that destroy corticobulbar fibres cause an upper motor neuron lesion of the facial nerve. In upper motor neuron lesions, intracranial tumours and cerebrovascular accidents need to be considered as illustrated in this case.

Neuroimaging is definitely required if the child has upper motor neuron lesion in order to diagnose the pathology in the brain and therefore a good clinical examination is required in order to decide upon the need for neuroimaging. It is important that primary care physicians recognise this and appropriate referral to tertiary centres is made as soon as possible. Intracranial tumours are potentially curable and delay in referral will translate into worse neurological dysfunction, which may be permanent.

To enhance the understanding of differentiating an upper from lower motor neuron facial nerve palsy, an outline of the facial nerve motor innervation and course is explained below.

Corticobulbar fibres project from the cerebral cortex passing through the genu of the internal capsule before reaching the facial nerve nucleus in the pons. From the pons, the facial nerve exits the brainstem at the cerebellopontine angle and enters the internal auditory canal together with the vestibulocochlear nerve. It then runs through the tympanic cavity before exiting the skull base through the stylomastoid foramen. After exiting, the facial nerve passes through the parotid gland and splits into five branches innervating the muscles of facial expression (temporal, zygomatic, buccal, marginal mandibular and cervical).

The facial nerve nucleus located in the pons is unique as it can be divided into two portions. The upper portion is innervated by bilateral corticobulbar fibres from both cerebral hemispheres. This upper portion in turn innervates the frontalis muscles through the temporal branch of the facial nerve. The lower portion in only innervated by the contralateral cerebral hemisphere. This lower portion then innervates the other muscles of facial expression. Thus, any lesion that only affects the corticobulbar fibres (an upper motor neuron lesion) would result in facial paralysis on the contralateral side with sparing of the frontalis muscle as there is still innervation from the corticobulbar fibres to the upper portion of the facial nerve nucleus from the other cerebral hemisphere.

For this case, loss of nasolabial fold and drooping of the left angle of mouth are indicative of left facial weakness. The left facial weakness reported in this case was due to the mass effect/compression to the right internal capsule where the corticobulbar fibres pass.

Meningiomas arise from arachnoid cap cells and are most commonly located along the falx, cerebral convexity or sphenoid bone. Meningiomas of the sphenoid bone are classified according to their location as lateral, middle and medial. In a series of 18 paediatric patients, only one meningioma was located at the medial sphenoid region.[5] Medial sphenoid wing meningiomas may invade the cavernous sinus, superior orbital fissure and surround the carotid, anterior cerebral and middle cerebral arteries; therefore, it is difficult to excise fully without complications. Known risk factors for developing meningiomas are radiation, neurofibromatosis, oestrogen and progesterone. Compared to the female predominance in the adult population, the reported male-to-female ratio in children is 1.2:1.[8]

Paediatric meningiomas are different from adult meningiomas in terms of location of tumour, greater percentage of higher-grade tumours and higher recurrence rates.[3,5] The most common clinical presentations for medial sphenoid wing meningiomas in children include seizures, headaches, ataxia and hemiparesis.[5,6,9] For adults, visual disturbance is the most common presenting complaint.[10] This difference in presentation may be due to children being unable to recognise or communicate visual disturbances and thus allowing the tumour to grow undetected till further compression of adjacent brain occurs. Our patient presented with left upper motor neuron seventh nerve palsy, which is not one of the common presentations of a sphenoid wing meningioma.

Conclusion

Paediatric meningiomas are uncommon but need to be considered when a child presents with just isolated facial nerve palsy. Although the cause of most facial nerve palsies is idiopathic, careful examination can give hint of other potentially lethal diseases.

How does this paper make a difference to general practice?

- It highlights an uncommon but possible cause of facial nerve palsy.
- This case highlights the importance of a good examination to distinguish between upper and lower motor neuron facial palsy.
- This is an example where common symptoms in an adult may not be present in a child as they might not recognise the abnormality and thus not be able to complain about it.
- Meningiomas are curable with complete excision, which is easier with early detection.
- Early referral and detection is required to reduce morbidity from intracranial tumours.

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