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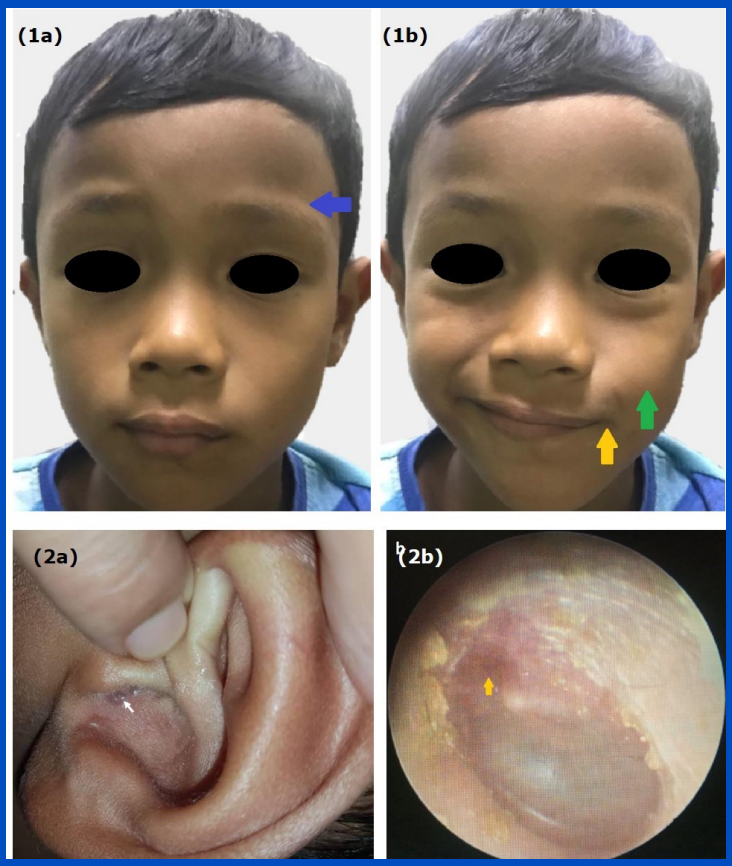

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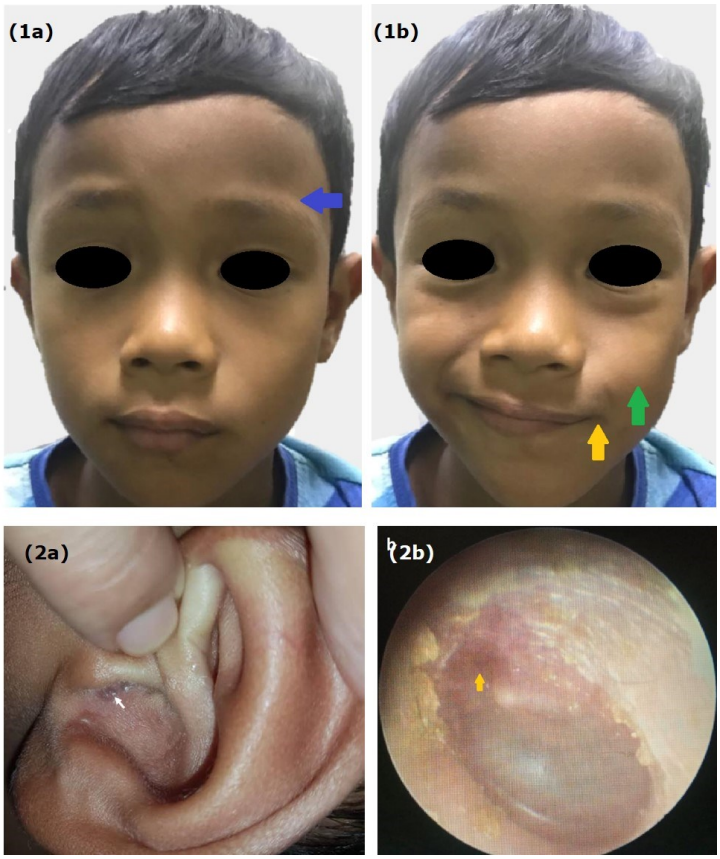

Figure 1 & 2

A seven-year-old boy with coryzal symptoms a week prior presented with a sudden onset of left facial weakness and painful vesicles of his left pinna. He had a low grade pyrexia of 37.6 degrees Celsius. Examination revealed weakness of all the five divisions of the facial nerve motor innervations which corresponded to a left lower motor neuron facial nerve palsy grade 3 on the House Brackman scale (Figure 1). There were several vesicular rash on the pinna (Figure 2). Otoscopy examination showed an intact ear drum with erythema on the posterior superior aspect of the drum. There were no lesions in the ear canal. He had a previous Varicella Zoster infection and is up to date with his vaccinations. Pure tone audiogram revealed normal hearing.

What is the diagnosis?

Answer*: refer to page 89*

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(Refer to page 88)

ANSWER: HERPES ZOSTER OTICUS

In a child presenting with sudden onset unilateral lower motor neuron facial weakness, infective or inflammatory causes should be considered. The presence of typical vesicular rash and coryzal symptoms prior, clinched a clinical diagnosis of Herpes Zoster Oticus.

Herpes Zoster Oticus occurs when there is a reactivation of the dormant virus within the geniculate ganglion of the 7th nerve (CN VII). It is believed to occur when there is an immunocompromised state resulting in diminished cell mediated immunity to the Varicella Zoster virus.[1] The resultant neuritis can potentially affect motor, sensory and autonomic nerves of CN VII as well as other cranial nerves causing a variety of head and neck manifestations, described in Ramsay Hunt Syndrome.

The Varicella Zoster virus may travel down the sensory division of the 7th nerve resulting in vesicular rashes of the ear and subsequently, motor division to cause facial nerve palsy. Vesicular eruptions and ulcerations at the anterior two thirds of the tongue may occur due to neuritis of the auriculotemporal branch of the 7th nerve.[2]

In approximately 50% of the cases, inflammation or compression of the adjacent vestibulocochlear nerve (CN VIII) in the internal auditory meatus is seen resulting in vestibulocochlear symptoms. Patients may present with sensori-neural hearing loss and unilateral vestibulopathy. Vestibulopathy is evident by acute vertigo, tendency to fall to the affected side accompanied by spontaneous nystagmus, and occasionally nausea and vomiting.[2]

It may also cause contralateral CN VII palsy and ipsilateral CN V (trigeminal) palsy, often the ophthalmic division (V1).[3] There have been several reported cases of involvements of lower cranial nerves such as CN IX (glossopharyngeal) and CN X (vagus), albeit rare but possible.[1] CN IX involvement may present with dysphagia and deviated uvula to the contralateral side, while CN X involvement often manifests in hoarseness.[2]

Treatment often involves high dose steroids and antiviral therapy.[1,2,3] High dose intravenous acyclovir (5mg/kg TDS) and dexamethasone (0.1mg/kg TDS) were administered for our patient. His facial nerve palsy improved after 24 hours with near complete resolution after 72 hours. Vesicles dried up after 48 hours. Supportive treatment such as facial physiotherapy can be considered. Prompt administration of eye care with artificial tears, eye lubricant and eye patch is essential to prevent exposure keratitis.

In conclusion, patients presenting with facial palsy should have Ramsay Hunt Syndrome excluded as small vesicles within the ear canal or the pinna may easily be missed. Involvement of other cranial nerve palsies should be ruled out. Early administration of high dose intravenous steroids and acyclovir with low grade facial nerve palsy on presentation offers a better prognosis for a full recovery of the facial nerve function.

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