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Case Report

Bilateral facial palsy in an adult with chickenpox

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ABSTRACT

Chickenpox is a result of primary infection with varicella zoster virus. Isolated facial nerve palsy as a complication is rare, and here we report an extraordinary case of bilateral facial nerve palsy following chickenpox in an adult. A 55-year-old male presented to the emergency department with a day's history of facial weakness. He had recently contracted chickenpox with an onset 16 days prior. Physical examination noted crusted vesicles all over the body and a bilateral facial palsy. There were no clinical signs of meningitis or cerebrovascular accident. He was managed with a short course of oral aciclovir and prednisolone and recovered fully after a month. Presentations of facial nerve palsy in chickenpox are rare and should be differentiated from Ramsay Hunt syndrome. Prognosis is relatively good with the majority of known cases seeing complete facial nerve recovery within 6 months.

Keywords: Varicella, Chickenpox, Facial palsy

INTRODUCTION

Chickenpox is a result of primary infection with varicella zoster virus (VZV). It presents classically as a diffuse, pruritic, vesicular rash, accompanied with malaise and fever. It largely affects children, but adults are also susceptible. Possible complications include secondary bacterial skin infections, meningitis, encephalitis, Guillain-Barré syndrome, transverse myelitis, pneumonia, hepatitis, myocarditis, hemorrhagic varicella, and uveitis.[1] Unilateral facial nerve palsy related to primary VZV infection, without other central nervous system (CNS) complications, is few and far between with literature regarding it proving scarce.[2-5]

Here, we report an extremely rare case of bilateral facial nerve palsy following chickenpox in an adult.

CASE REPORT

A 55-year-old male with dyslipidaemia presented to the emergency department with a day's history of facial weakness. This was accompanied by mumbled speech and a difficulty to sip fluids. There was no otalgia, hearing loss, nor vertigo.

He had recently contracted chickenpox with an onset 16 days prior. It started off with lethargy and the appearance of multiple small vesicles all over his body which later ruptured. He was seen at a primary care clinic and was prescribed potassium permanganate ointment for the skin lesions. He did not undergo aciclovir treatment. For the following 2 weeks, there was no fever and the condition had been improving until the occurrence of this new symptom.

On examination, the patient was alert and comfortable without any signs of distress. Dry crusted vesicles were seen all over the body, including the face and bilateral ear conchae. Otoscopy revealed normal ear canals and ear drums on both sides, and they were spared from vesicles. He had bilateral facial lower motor neuron palsy, House-Brackmann grade V. There were no clinical signs of meningitis or cerebrovascular accident.

Complete blood counts, serum electrolytes and glucose tests were normal. Computed tomography of the brain

showed no evidence of acute infarct or infection. Tympanometry was type A with stapedius reflexes absent in both ears. Pure tone audiometry showed no acute hearing issues.

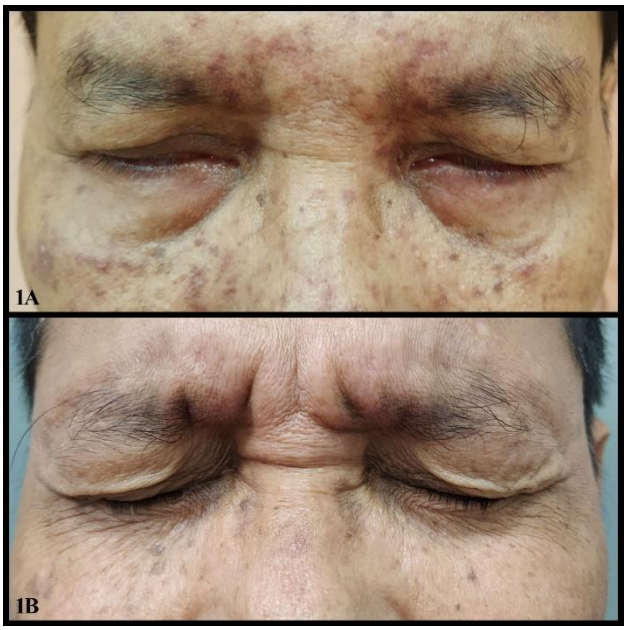

Figure 1: Closure of eyelids (A) incomplete closure at maximal effort at presentation (B) complete closure at 6 weeks

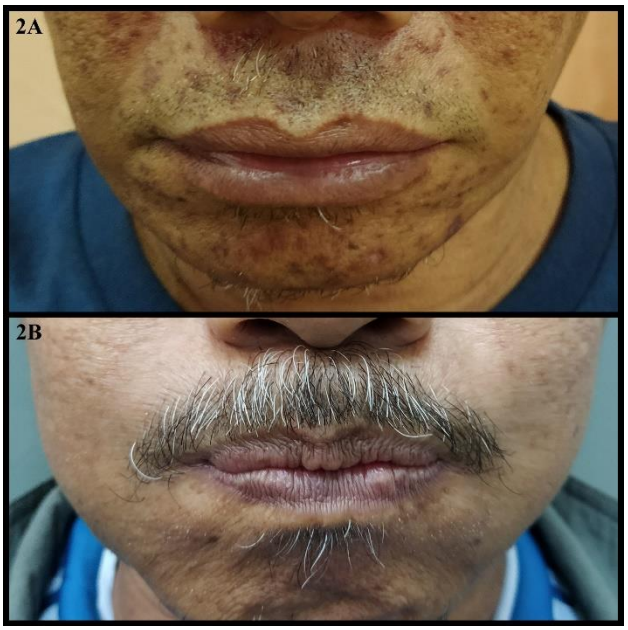

Figure 2: Puffing of cheeks. (A) inability to puff out cheeks at presentation (B) recovered function of lips and cheek muscles at 6 weeks.

After consultation with the internal medicine team, the patient was allowed home with oral aciclovir 800 mg 5 times/day for 7 days, oral prednisolone 60 mg/day for 5 days followed by tapering doses over 2 weeks, and artificial tears. He was followed-up at the ENT clinic after 2 days, then a week, and subsequently a month. He showed positive response to treatment and recovered fully by the last visit with no residual neurological deficits.

Figures 1-3 display comparisons of the patient's reduced facial motor function at first presentation and its recovery at 6 weeks.

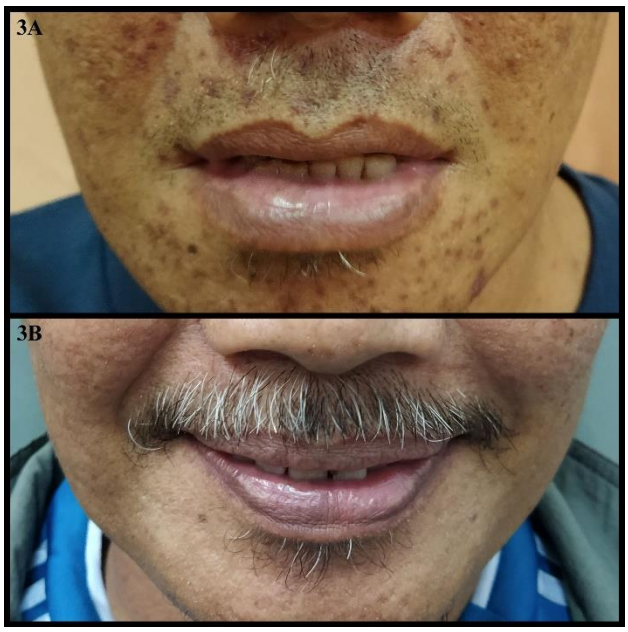

Figure 3: Effort of smile (A) Inability to smile at presentation (B) Decent smile at 6 weeks.

DISCUSSION

Chickenpox is a primary infection occurring in a person exposed to VZV. It is highly contagious, affecting 90% of non-immune exposed individuals, mostly in the paediatric age group. Infections involving adults are less prevalent, but not uncommon. The virus spreads mainly by way of airborne droplets and also via shedding from erupted vesicles. While the incubation period is 10-21 days, infectivity is highest within two days before the onset of rash.[6]

Neurologic complications linked to VZV is estimated at 1-3 every 10,000 cases.[7] These may range from encephalitis, which is more common, to rare complications such as Guillain-Barré syndrome and transverse myelitis. As mentioned earlier, reported cases of isolated facial nerve palsy related to primary VZV infection are extremely scarce. Thorough neurological examination must be performed to rule out the more common complications first.

The pathogenesis of facial palsy in primary VZV infection is not clear, however two mechanisms are theorised. The first is of direct nerve damage by the virus itself, and the second is by immunologically mediated inflammatory response.4 In our case, the patient

developed facial palsy two weeks after the onset of his chickenpox symptoms, and this goes along well with the processes of the second theory mentioned.

Facial nerve palsy, if seen in chickenpox, needs to be made distinct from Ramsay Hunt syndrome. While chickenpox is a result of primary VZV infection, Ramsay Hunt syndrome occurs secondarily by reactivation of VZV at the geniculate ganglion and it commonly presents as a triad of unilateral facial paralysis, ear pain, and herpetic rash over the external auditory canal or pinna.[8,9] Our patient had no ear pain while his rashes were generalised throughout the body and exhibited features of chickenpox in the late eruptive stage.

We would like to highlight the symptoms of mumbled speech and the struggle to sip fluids in this case. The loss of motor function of lip muscles led to the inability to perform certain lip-dependant articulations. Failure to purse the lips and seal the mouth resulted in leaks when drinking. This emphasised the importance of the facial nerve, particularly the buccal and marginal mandibular branches, in speech and swallowing. Patients with bilateral facial palsy experience these symptoms more severely compared to unilateral cases. Our patient was able to adapt to these deficiencies; therefore, he was allowed to be managed as an outpatient with close follow-up.

At the moment, treatment is generally based on clinical judgement as formal practice guidelines are not yet established. Previous reported cases were treated mainly with an antiviral and steroid combination, either oral or intravenous, mostly with positive outcomes.[2-5] Interestingly, one case received only aciclovir and managed to recover fully; while another had aciclovir and dexamethasone, as well as intravenous immunoglobulin, but still had residual facial palsy after 6 months.4 In our scenario, oral aciclovir 800 mg 5 times/day for 7 days and oral prednisolone 60mg/day for 5 days followed by tapering doses over 2 weeks resulted in complete recovery within 6 weeks.

The strength of this report is the capture of an extremely rare complication of chickenpox– isolated facial nerve palsy. Majority of such palsies are unilateral, but here we presented a bilateral case.

However, our limitation was that the patient did not undergo a VZV IgM antibody serology as a confirmatory test for chickenpox. This is because chickenpox is widely considered a clinical diagnosis and there is a perceived lack of cost-benefit in performing such confirmatory tests at our centre.

CONCLUSION

Presentations of facial nerve palsy due to primary VZV infection are rare and should not be confused with ramsay hunt syndrome, due to the need to rule out other possible CNS complications in the former. Extra attention should be given in bilateral cases due to possible feeding disabilities. Once diagnosis is established, the common practice for treatment is with a combination of aciclovir and steroids, either oral or intravenous. Prognosis is relatively good with the majority of known cases seeing complete facial nerve recovery within 6 months.

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