Bell Palsy and Preeclampsia Superimposed on Chronic Hypertension

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Bell palsy is an acute idiopathic peripheral facial paralysis of unknown etiology. It is characterized by unilateral weakness of the upper and lower facial muscles. The incidence of Bell palsy is 20–30 cases per 100,000 population per year. A previous report showed that women of reproductive age are affected two to four times more often than men of the same age, and pregnant women are affected 3.3 times more often than non-pregnant women [1]. A high extracellular fluid content, viral inflammation and relative immunosuppression due to pregnancy are thought to be predisposing factors [2]. Some reports noted that hypertension or preeclampsia increases the risk of Bell palsy [3]. It may be associated with hyperacusis on the affected side and loss of taste on the anterior two-thirds of the tongue. The patient cannot close the eye and, on attempting to do so, the eyeball is deviated upward and inward (Bell phenomenon). The corneal reflex is absent. The patient may be unable to produce tears. Rapid recognition is important, because these patients require artificial tears to lubricate the cornea and may need to have the eye taped shut to prevent drying and infection [4].

A 38-year-old female, primigravida at 36.8 weeks' gestation, presented with acute onset of drooling, tearing, and numbness of the face on her left side for 1 day. Pain in her ear and cheek had been noted for 3 days. Upper respiratory infection symptoms had also recently been noted. High blood pressure had been recorded at 11 weeks of pregnancy. At 35.3 weeks of gestation, preeclampsia superimposed on chronic hypertension was found. Ten days later, she developed acute facial paralysis. She denied any other systemic disease or trauma. On an examination, left facial movement impairment was noted. Besides having a drooping face and mouth, the left side of her forehead failed to wrinkle, and she could not close her left eye (Figure 1). There were no left nasolabial fold and only limited retraction of the left angle of the mouth when she tried to smile (Figure 2). A neurologic examination was otherwise normal. Peripheral-type left facial palsy was our impression. Considering the high blood pressure, a stroke

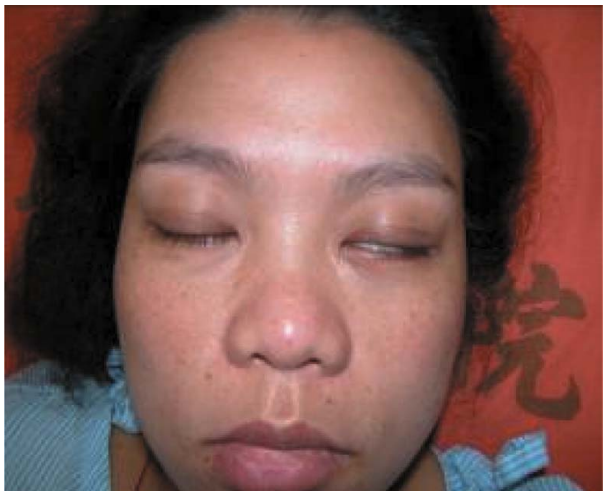

Figure 1. Left eye unable to completely close (with an eyelid fissure of 3–4 mm) and rolling upward.

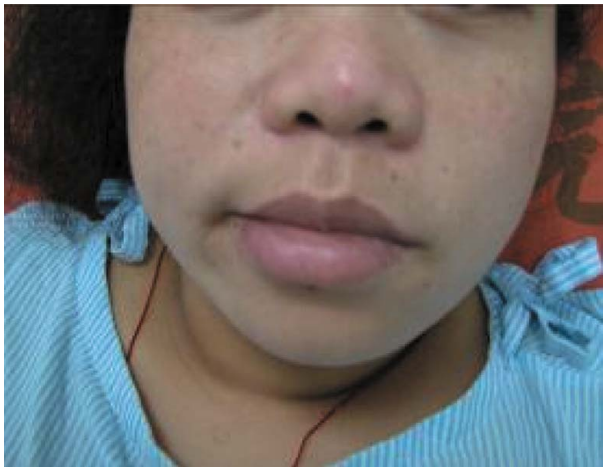

Figure 2. No left nasolabial fold and limited retraction of the left angle of the mouth when attempting to smile.

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Motor nerve conduction studies

Nerve/Sites	Latency (ms)	Amplitude (mV)
Left facial – nasal, ocular, oris		
1. Ear – nasalis	4.80	0.7
Right facial – nasal, ocular, oris		
1. Ear – orbicularis oris	4.25	1.1

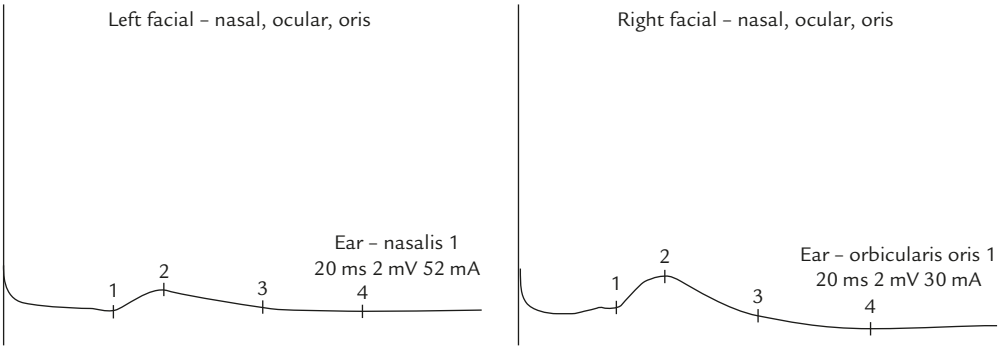

Figure 3. Electroneurogram showing a ratio of the compound motor action potentials between the left and right facial nerves of 0.64 (0.7:1.1).

(central-type facial palsy) could not be ruled out. A neurologist was consulted, and a neurologic examination showed Bell phenomenon with the left eye not completely closing (an eyelid fissure of 3–4 mm) and rolling upward. Thus, peripheral-type facial palsy was diagnosed, and no central lesion was noted. Supportive care with artificial tears and an eye patch were used to protect her eyes. As a result of uncontrolled hypertension, we arrested the pregnancy, and a healthy male baby was delivered by cesarean section at 37.3 weeks' gestation. Three weeks after the episode of facial palsy, electroneurography showed that the ratio of the compound motor action potential between the left and right facial nerves was 0.64 (0.7:1.1) (Figure 3). She experienced good recovery of facial function 1 week later.

In women with preeclampsia, nerve compression because of increased extracellular fluid and thrombosis of the vasa nervorum caused by a hypercoagulable state may account for the higher incidence of Bell palsy [5]. Most cases of Bell palsy occur in the third trimester. Onset is usually acute and painful. Sometimes, hyperacusis resulting from paralysis of the stapedius muscle and loss of taste accompany varying degrees of facial paralysis. Supportive treatment is usually adequate [6]. Complete recovery is anticipated in most women. Poor prognostic markers are recurrence in a subsequent pregnancy and bilateral disease [7].

Bell palsy is often mistaken for a stroke because of its sudden onset, and because it results in numbness and loss of muscular control on the affected side. The patient's history and a neurologic examination can determine whether the facial weakness is central or peripheral [5]. Central weakness (unilateral lower facial area) is always the result of a lesion above the level of the facial nucleus in the pons of the contralateral hemisphere. Cells of the facial nucleus that innervate the lower face receive the corticobulbar fibers primarily from the contralateral cerebral hemisphere. In contrast, cells of the facial nucleus that innervate the upper face receive corticobulbar fibers originating from both cerebral hemispheres. Peripheral-type facial palsy (unilateral, affecting all muscles of facial expression) usually arises from a lesion of the ipsilateral facial nerve, facial nucleus or facial nerve in the pons. It is very important to differentiate whether the facial palsy is the central or peripheral type in a patient with preeclampsia, because the management differs greatly.

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