Facial paralysis in cerebral infarction: A case of misdiagnosis and literature review

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Facial paralysis can be classified as central or peripheral facial paralysis based on the location of the underlying lesion, both of which demonstrate facial motor dysfunction. In the currently report, a patient admitted to the department of otology, First People's Hospital of Qinhuangdao, presented with facial asymmetry as the initial symptom of a cerebral infarction and was first misdiagnosed as peripheral facial paralysis. The case is reported as follows.

1. Clinical data

The patient is a 66 years old male, admitted for “facial asymmetry of 2 h”, who also had a history of hypertension. The patient also reported suffering from upper respiratory tract infection. The face and mouth skewed to the right side upon showing teeth. No nausea or vomiting. No ear pus, dizziness or facial numbness. No signs of a tic disorder (Fig. 1).

Considering the history of upper respiratory tract infection and facial asymmetry, he was initially diagnosed with left side facial paralysis, despite the symmetric forehead and eye lid motor functions.

Upon admission, acyclovir antiviral therapy was initiated together with injections of dexamethasone sodium phosphate, fleabane, alprostadil and mecobalamin, as well as mouse nerve growth factor treatment, etc. Four hours after admission, the patient suddenly showed slurred speech and difficulties in using the left limb. Examination confirmed left limb weakness (level III). Eight hours after admission, left limb motion was completely lost with significant left upper limb weakness, facial asymmetry was worse with drooling, and there was intermittent choking upon swallowing. Brain CT results showed signs of infarction and atrophy (Fig. 2). Neurology was consulted on CT, and bilateral basal ganglia and lacunar lesions near the lateral ventricles were diagnosed. Sodium ozagrel (80 mg i.v. bid) and oral aspirin (300 mg) were added to treatment, and dexamethasone was stopped. Examination next day showed that the patient was conscious, still with slurred speech. Blood pressure was at 180/90 mmHg. Left facial weakness remained, ocular movement was normal with no nystagmus. Tongue protrusion deviated to left. Left limb muscle strength remained at level III, with positive Babinski reflex. MRI showed bilateral multiple foci of malacia mostly in the semioloval center near the lateral ventricles, basal ganglia and brainstem. Lab test showed that his glucose was at 22.3 mmol/L. Final diagnoses included acute cerebral infarction, hypertension and type 2 diabetes. The patient recovered after treatment in the neurology department for two weeks.

2. Discussion: causes for initial misdiagnosis

The presentation in this patient is not typical of an infarction, and mainly involved facial weakness without limb abnormalities initially. 2. In the initial assessment of facial paralysis, the signs of central origin, i.e. preservation of upper facial functions, were missed. 3. The patient's history of upper respiratory infection misled the examiner to consideration of Bell palsy as a result of viral infection. 4. Brain CT was not performed before admission.

3. Review of relevant literature

Facial paralysis is classified as central or peripheral based on the location of the underlying lesion, both affecting facial muscle function (Huang et al., 2006; James and Banecke, 2002; Thuy-Anh et al., 2008). Signs of central facial paralysis include: 1) involvement of contralateral side facial expression muscles below the palpebral fissure, while sparing those above the palpebral fissure; so the patient is usually able...
Lesions above the facial nucleus can cause central facial paralysis, including occlusion of the carotid (especially common in the trunk and middle cerebral artery branches), intracranial hemorrhage from aneurysm or other high pressure vascular lesions, and tumor. Facial paralysis in this patient was caused by cerebral infarction, which is ischemic necrosis or softening of the brain caused blood supply disorders. Diseases with relevance to cerebral infarction include diabetes, obesity, hypertension, rheumatic heart disease, arrhythmia, dehydration, arterial inflammations, shock, and rapid and severe drop of blood pressure. Our patient has a history of hypertension, which can lead to cerebral vascular incidents.

CT and MRI are the most commonly used auxiliary tests, with important value in differentiating central from peripheral facial paralysis. Brain MRI in this patient confirmed cerebral infarction near the right lateral ventricle. The main principle in treating cerebral infarction is to improve cerebral circulation by increasing cerebral blood flow and promoting establishment of collateral circulation. Treatments includes use of anticoagulants, neuroprotective and anti-platelet agents, correcting blood pressure and lipids, and thrombolytic therapies.

Peripheral facial paralysis is often caused by inflammation lesions involving the facial nerve, with signs of nerve damage ipsilateral to the lesion side, i.e. weakness of both upper and lower face, but generally not accompanied with limb paralysis. In addition to facial weakness, dysgeusia can also occur in the front part of the tongue. In the bulk of cases reported, Bell's palsy is the most frequent cause of peripheral facial paralysis. In contrast, in central facial paralysis, the lower face weakness is more severe than the upper face; and there are often sings of tongue and limbs weakness on the same side even when facial weakness is not obvious (Han, 2009; Han and Han, 2010; Han et al., 2011).

Jiang et al reported a case of acute cerebral infarction manifested with peripheral facial paralysis and central limb paralysis (Jan, 2013). Despite signs of peripheral facial paralysis, limb motor dysfunction is a clue of central disorders. The facial paralysis did not respond to initial treatment aimed at improving facial nerve function, but improved following treatment for cerebral infarction as shown on brain CT.

From our case, the following can be learnt: 1. The otologist must pay attention to differential diagnosis of facial paralysis in order to avoid misdiagnosing facial paralysis as part of cerebral infarction presentation as otogenic facial paralysis. Paralysis of upper and lower face must be carefully examined to rule out/in central facial paralysis, as the treatment and prognosis of cerebral and otogenic facial paralysis are very different. 2. The otolaryngologist should not focus only on ENT complaints, but also pay particular attention to the patient's history of cardiovascular or metabolic diseases. Thorough history should be collected especially in older patients, as well as careful physical examination of the nervous system. 3. Since limb manifestation may not be present in the early stage of cerebral infarction, the patient should be carefully followed for suspicious signs of central facial paralysis and brain imaging should be considered when in doubt.
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References
