Hemifacial spasm due to pontine infarction

Currently, hemifacial spasm (HFS) is thought to be due to a compression of the facial nerve at the root exit zone by blood vessels.1 Compression by tumour, aneurysm, or arteriovenous malformation has also been noted. We report a case probably due to a small infarct in the pons.

A 51-year-old man, known to be hypertensive for 15 years, presented with a two-year history of left hemifacial spasm. At the age of forty-six, he had been admitted for a transient ischaemic episode probably in the right internal carotid territory. He recovered, but two years later, represented with a minor right hemiparesis and a transient left facial weakness. Two weeks later, he noted slight fluctuating weakness of the lower eyelid which gradually increased in frequency and severity. The left hemifacial spasm was tonic, involving the orbicularis oculi, orbicularis oris and the platysma and was aggravated by light and emotional stress. The left limb reflexes were brisk. Therapy with botulinum toxin was effective.

The brainstem evoked potentials were impaired centrally on the left. Six months after the onset of the HSF, a blink reflex study4 showed normal R1 (early component) latencies on both sides. The latencies of R2 (the late ipsilateral component) and R2c (the late contralateral component) were shorter on the left (28-70 and 30-1 ms) than on the right (34-35 and 35-1 ms) (control subjects: 32-2+7 and 34-8+2). With the paired stimuli technique, the R2 and R2c responses were obtained when the interstimulus interval was decreased to 100 ms on the left and 200 ms on the right. In the control subjects, the inhibition of the R2 response was complete when the interstimulus interval decreased below 250 ms. The CT scan showed mild sub-cortical atrophy. MRI demonstrated an ecstatic basal artery and decreased signal on the T1-weighted MRI scan and increased signal intensity on the T2-weighted MRI scan in the right centrum ovale and in a small area just above and internal to the left facial nucleus (fig), suggesting a small infarct in the left pons.

To our knowledge, HFS due to a lacunar infarct has never been reported. The onset a few weeks after a regressive hemiparesis and the investigations support this view. The pathogenesis of HFS remains unclear. The ephaptic transmission hypothesis, due to a compression by a blood vessel on the root entry zone, is widely popular. Our case agrees with the claim that the physiological abnormality is situated in the facial nucleus area and that signs of HFS are caused by hyperexcitability of the facial nucleus.4 The results of the blink reflex of our patient, according to the study by Valls-Sole and Tolosa,4 suggest an enhanced excitability of facial motor neurons and of those brainstem interneurons that mediate the blink reflex pathway. In our case, this hyperexcitability might not be due to antidromic impulses from compression of the root entry zone, but to a loss of inhibitory impulses from the brainstem, due to the infarct in the pons.

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Susamethionin is contraindicated in the Guillain–Barré syndrome

Susamethionin induced hyperkalaemia has been described in a variety of disorders.1 Ferguson et al4 described four patients with chronic paraplegia who had developed life-threatening arrhythmias following susamethionin administration. The presumed cause was susamethionin induced hyperkalaemia although this was not documented in their report. We have recently seen a patient with relapsing Guillain–Barré syndrome who developed severe ventilricular arrhythmia secondary to a documented susamethionin induced hyperkalaemia. The potential danger of the use of susamethionin needs to be emphasized in the neurological literature.

A 51-year-old man was admitted with a two-week history of tingling in his hands and feet and progressive weakness in his arms and legs. These symptoms had begun one week after a flu-like illness. Examination revealed a proximal muscle weakness with depressed deep tendon reflexes and normal sensation. Cerebrospinal fluid examination was normal but nerve conduction studies showed evidence of a demyelinating neuropathy. A diagnosis of Guillain–Barré syndrome was made and he was treated with plasmafiltration with significant improvement over the following ten days. He was discharged but was readmitted two months later with an exacerbation of his symptoms.

Examination revealed severe weakness in his arms and legs, absent deep tendon reflexes, bilateral mild facial weakness, mild dysphagia and dysarthria. Forced vital capacity was two litres. He was treated with nasogastric feeding and daily plasmapheresis without any improvement over the following ten days. On day 11 because of deteriorating pulmonary function, it was decided to electively ventilate him. Before ventilation, there was a sinus tachycardia of 126/min but no other evidence of autonomous dysfunction. The arterial partial pressure of oxygen was normal and he was given 100% oxygen for three minutes before the procedure. Anaesthesia was induced with thiopentone and he was then paralysed with susamethionin.

Immediately after the susamethionin was given and before intubation, he developed severe ventricular tachycardia followed by a cardiac arrest. Cardio-pulmonary resuscitation was...