Hemifacial spasm: treatment with pizotifen

The surgical treatment of hemifacial spasm has recently been reported in this Journal.1 I report the beneficial effects of pizotifen, a SHT receptor antagonist, in two patients with hemifacial spasm. The original observation was entirely by chance as the drug was introduced to help a patient with frequent migraine attacks.

Patient 1, a 43 year woman pharmacist, had an eight month history of right hemifacial spasm after the removal of a meliobian cyst from the lower eyelid in December 1994. There was also a background history of migraine headache fulfilling the criteria for the diagnosis as defined by The International Headache Society. Pizotifen (1 mg a day), was introduced by her general practitioner in an attempt to reduce the frequency of the migraine attacks. There was a rapid and consistent benefit from the point of view of the hemifacial spasm, although there seemed to be no reduction in the migraine attacks. The patient stopped the pizotifen and within 36 hours, the facial spasms returned. Several more trials of treatment, followed by stopping it, were undertaken, all with the same result. On continued pizotifen treatment, the benefit on her hemifacial spasm has been maintained.

Patient 2, a 52 year old woman clerk presented with a very long history of left hemifacial spasm. She had undergone various non-surgical treatments previously, including botulinum toxin, with variable benefit. There was also a background history of frontal headaches. These were more in keeping with a diagnosis of a tension type headache syndrome. In view of the previous case, pizotifen (1-5 mg at night), was introduced and there was very good improvement in the hemifacial spasm, although it was not completely abolished. The patient was considering a surgical approach for the spasm but decided in view of the considerable improvement that she would await events. The improvement has persisted for more than three months on continued pizotifen.

Considerable caution must obviously apply to the finding of just two patients benefiting from pizotifen treatment in hemifacial spasm. It is interesting, however, that the first patient, being a pharmacist, had undergone several trials of stopping the treatment with the return of hemifacial spasm, only to see consistent improvement on restarting. The mechanism of action is unknown. Antagonising SHT1 receptors with pizotifen might inhibit neurons at the level of the Vth nerve nucleus.

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Orhostatic hypotension caused by a localised dorsal medullary tumour

Orhostatic hypotension is the most dramatic circulatory expression of autonomic failure. To control blood pressure, an intact baroreflex loop is necessary.1 In 1984, Hsu et al reported three cases of brainstem tumours that compromised baroreflex function.2 Here, we describe a patient who presented with severe orthostatic hypotension, respiratory failure, and bulbar palsy associated with a medullary tumour. We examined the autonomic function of the patient. This case is unique, because of its rarity and diagnostic aspects.

A 54 year old man was admitted to Anjio Kosei Hospital on 21 April 1994 because of hoarseness, anorexia, dysphagia, and positional dizziness. These symptoms first appeared in March 1994. He had a history of ankylosing spondylitis and acute myocar-dial infarction diagnosed at the ages of 26 and 51 years, respectively. His general examination was unremarkable except for ankylosis of the spine and a cachectic appearance. Neurological examination disclosed a nasal voice, atrophy of the tongue, and absence of the gag reflex bilaterally. Motor, sensory, and cerebellar functions were normal except for a mild proximal weakness of the limbs, probably from disuse. His gait was normal, and he could use a pair of chopsticks. The deep tendon reflexes were normal. There were no pyramidal signs. His blood pressure decreased from 160/100 to 80/60 mm Hg when he rose from a supine to a seated position, whereas his heart rate remained unchanged. He had no urinary dysfunction.

A plain cranial CT disclosed no apparent lesion. His CSF contained 38 cells per mm,3 and the protein concentration was 90 mg/dl. Repeated cytological analysis of the CSF disclosed no malignant cells. On 26 May, enhanced MRI showed a circumscribed brain tumour located in the dorsal medulla with involvement of the obex. Two additional smaller tumours also were found in the right cerebellum and in the right lateral perriventricle (figure). No lesions in the spinal cord or peripheral nerves were noted. Nerve conduction studies of the limbs were normal. Repeated examinations of the whole body failed to disclose any evidence of a primary tumour. The intracranial tumour was considered to be a malignant lymphoma based on its radiosensitivity and neuroimaging.

After his admission, the patient's postural hypotension gradually worsened, and the frequency of syncopal attacks increased. An episode of respiratory arrest after syncope occurred during defecation on the morning of 7 June. He was unable to breathe spontaneously, and showed clouding of consciousness. However, he could respond voluntarily to some orders. Assisted ventilation was performed for the next two months. Irradiation therapy applied to his whole brain (40 Gy) and to the medullary lesion (14.4 Gy) was performed from 6 June to 19 July. On 20 October, only small enhanced spots were seen on an MRI with contrast. His respiratory status improved and after 17 August, he was able to breathe without aid; however, his episodic orthostatic hypotension and bulbar deficits remained. Both sym pathetic and mineralocorticoid agents given via gastric tube were ineffective in treating his orthostatic hypotension. The use of support stockings to prevent blood pooling in his
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_J Neural Neurosurg Psychiatry_ 1996 61: 118
doi: 10.1136/jnnp.61.1.118

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