

ance to this treatment but was improved by IVIG. There was a temporal relationship between neurological and renal improvement and IVIG, and this could reflect a common pathogenesis. Recognition of such coexisting disorders provides further evidence of an immune dysfunction in CIDP.

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Acute stage Bell's palsy and narrowing of the palpebral fissure caused by drooping of the eyebrow and the upper eyelid

Widened palpebral fissure on the affected side has been regarded as one of the hallmarks of peripheral facial palsy.^{1,2} The palpebral fissure of the affected eye may, however, be narrower than that of the healthy eye.³⁻⁵

Twenty five patients with Bell's palsy in the acute stage were examined at our outpatient

clinic from January 1990 to May 1991. No other neurological signs were observed in these patients. Ten patients had a narrowed palpebral fissure rather than widened palpebral fissure on the affected side looking straight ahead. Their ages ranged from 32-59 years with a mean of 46.8 years. Seven patients were men and three women, four being diabetic. All 10 patients complained of a drooping of the upper eyelid on the affected side when opening their eyes. All had a furrowless forehead and the corner of the mouth drooped on the affected side, both characteristics of peripheral facial palsy. They were unable to close the affected eye tightly, and five patients had lagophthalmos (incomplete closure). The position of the eyebrow and the upper eyelid on the affected side was lower and the width between their lower margins was shorter than on the non-affected side (fig). The patients experienced difficulty in elevating the eyebrow and the upper eyelid when gazing upwards. The position of the eyes was intact, however, and the extraocular movements were full. The pupils were round, equal in size and reactive to light.

All the patients had CT or MRI brain scans. All were free from intracranial lesions. Neuroelectrophysiological examinations were performed in five patients and in these patients the distal latency of the facial nerve was prolonged and surface electromyogram of the frontal muscle showed decreased activity on the affected side.

The principal muscle involved in opening the upper eyelid and maintaining normal eyelid posture is the superior palpebral levator, which is innervated by the oculomotor nerve. Two accessory muscles are Müller's muscle via the oculosympathetic pathway and the frontal muscle by the facial nerve. The upper eyelid is indirectly elevated by the attachments of the frontal muscle into the eyebrow with the superior orbital portions of the orbicularis oculi muscle.⁶ Thus it is possible that the weakness of the frontal muscle causes drooping of the eyebrow and the upper eyelid, resulting in narrowing of the palpebral fissure. This phenomenon might be more noticeable in Japanese than in

white patients, because Japanese patients are more "heavy lidded".

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Familial recurrent cranial nerve palsy

Familial recurrent cranial nerve palsy is a syndrome of unknown aetiology in which affected family members experience recurrent paresis of the oculomotor, abducens, and facial nerves in an apparent autosomal dominant inheritance pattern.¹ Typically, these individuals experience acute onset of a cranial nerve palsy followed by gradual resolution of the deficit over several months. This presentation and natural history is similar to that of patients with diabetic vasculopathic cranial nerve palsies and prompted us to search for a vascular aetiology in the patient and family members we describe.

A 36 year old man developed binocular, horizontal diplopia in August 1990. He had experienced an idiopathic, peripheral, right facial nerve palsy (Bell's palsy) in October, 1985 and a left Bell's palsy in May 1986. He was an otherwise healthy nurse anesthetist taking no medications. On examination, best corrected visual acuity was 6/6 in each eye and colour vision was normal. Pupils were briskly reactive without anisocoria or a relative afferent pupillary defect. Visual fields were full. Facial sensation was intact, and there was no ptosis or proptosis. There was mild orbicularis weakness bilaterally and evidence of aberrant regeneration of both facial nerves. The patient had an ocular motility disturbance consistent with a left abducens nerve palsy: abduction of the left eye was decreased and there was a 25 prism diopter (PD) esotropia in primary gaze that increased to 45 PD in attempted left gaze. The remainder of the examination was unremarkable.

A complete blood count, thyroid function tests, rheumatoid factor screen, serum protein electrophoresis, assay for acetylcholine esterase antibodies, assay for angiotensin converting enzyme, Lyme titres, edrophonium test, chest radiograph, and magnetic resonance imaging were all normal with the exception of a mildly elevated cholesterol level in the serum (252 ng/dl) and a mildly elevated CSF protein (55 mg/dl). The left



Figure Patient looking straight ahead. Narrowing of the palpebral fissure is seen on the left side.