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Post-traumatic bilateral facial palsy

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Abstract
Bilateral facial palsy is an uncommon condition and few clinicians have experience of more than the occasional case. Unlike its unilateral counterpart, an aetiological factor is often demonstrable. A wide variety of recognized diseases may give rise to the condition and thus thorough investigation is required. This paper presents an unusual example of this rare condition.

Key words: Head injuries; Facial paralysis

Introduction

Facial palsy is a common condition in otological practice, with an estimated incidence of 20 to 25 cases per 100 000 population annually (Sherwen and Thong, 1987). Such palsies are usually idiopathic (i.e. Bell's palsy) and unilateral (Ovesen, 1992). However, a rarer bilateral form is occasionally encountered and comprises 0.3 to 2 per cent of cases of facial palsy (Klar *et al.*, 1985). Thus, approximately one case of bilateral facial palsy will occur per five million population per year (George and Pahor, 1991). Whereas unilateral facial palsy recurs in up to seven per cent of individuals, bilateral cases are extremely rare (Stahl and Ferit, 1989). In the diplegic form, the aetiology is apparent in over half, whereas in unilateral cases, the majority are idiopathic (George and Pahor, 1991). The differential diagnosis of bilateral facial palsy includes systemic, infectious, traumatic, neuromuscular, vascular and toxic as well as idiopathic causes (Sherwen and Thong, 1987). We report an unusual presentation of bilateral facial palsy and discuss the possible aetiology.

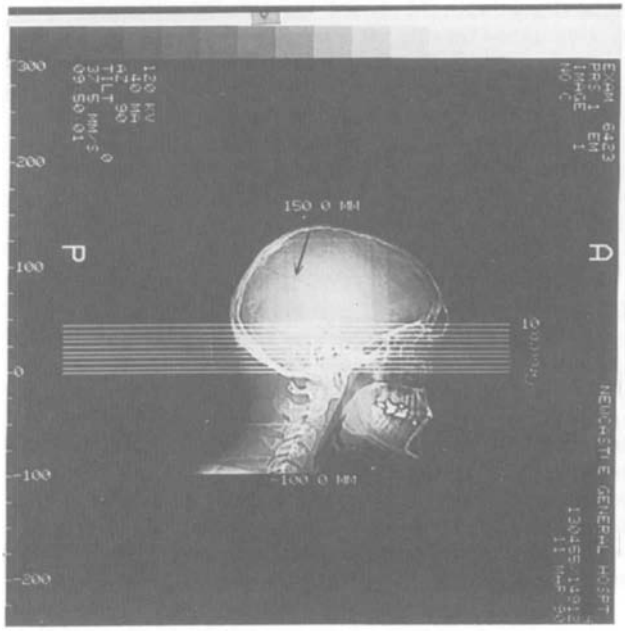

FIG. 1
Scout view from computerized tomography (CT) scan demonstrating left fronto-temporo-parietal skull fracture (arrowed).

Case history

A 34-year-old man underwent vasectomy, as a day case procedure, under general anaesthetic. Upon discharge he collapsed whilst awaiting a taxi and suffered a left fronto-temporo-parietal skull fracture (Figure 1). Although he was initially confused but obeying commands, his condition deteriorated prompting transfer to the regional neurosurgical unit. Upon arrival he was no longer obeying commands, though there were no focal neurological signs.

A computerized tomography (CT) scan revealed a small

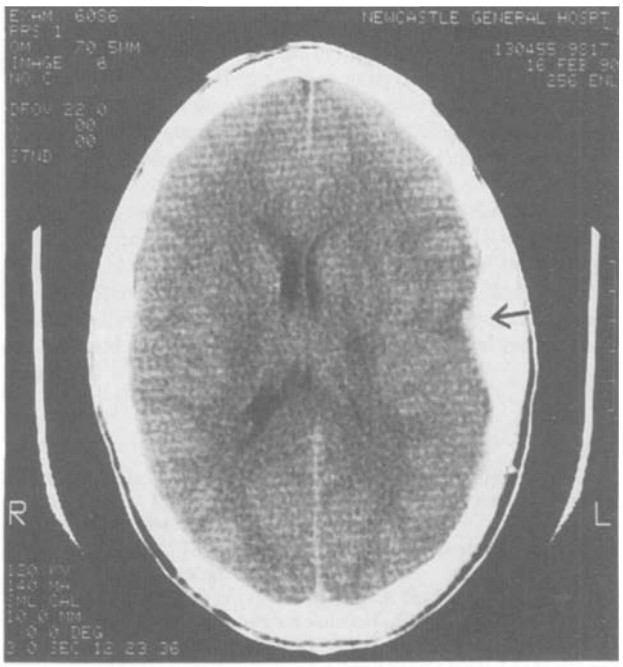

FIG. 2
Contrast-enhanced computerized tomography (CT) scan demonstrating a left extradural haematoma (arrowed) with compression of the lateral ventricle and midline shift.

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extradural haematoma overlying the left temporal lobe with some compression of the lateral ventricle and midline shift (Figure 2). His condition improved without surgical intervention and he was discharged 12 days post-injury, having recovered from a transient mixed dysphasia.

One week after discharge, he developed a complete right lower motor neuron facial palsy and by the tenth day, a left subtotal facial palsy (House grade IV). A further CT scan, including high resolution imaging of the petrous temporal bones showed the previously noted resolving left extradural clot. Throughout the clinical course, the possibility of cerebrospinal fluid (CSF) rhinorrhoea was considered and excluded. At lumbar puncture, the CSF protein concentration was 0.7 g/l (normal range 0.1–0.4 g/l), the glucose concentration 4.8 mmol/l (normal range 2.8–4.5 mmol/l) and a white cell count of 64 mm^3, of which 98 per cent were lymphocytes. CSF culture was negative. Although the patient had been anosmic since the head injury and complained of diminished taste, formal taste assessment was normal. ENT examination revealed normal hearing with intact stapedial reflexes. A diagnosis of sarcoidosis was considered, but both chest X-ray and Kveim test were negative, as was serology for Lyme disease. Electromyography (EMG) revealed no insertional or spontaneous activity and bilaterally dense motor unit interference patterns. There was also a slight excess of polyphasic unstable motor units at all sites. Neuropathies may be divided into those affecting the axon and those affecting the myelin sheath, although more commonly a mixed picture is seen with one feature predominating. In this case, there was some axonal loss, but this was not the predominant feature, there being sufficiently good remaining innervation in all facial muscle groups to suggest a favourable prognosis. The neurophysiologist involved felt that this probably represented a post-traumatic demyelinating neuropathy.

The patient was discharged a few days later and at three-month follow-up, the seventh nerve palsies had resolved apart from a slight delay in the right blink reflex.

Discussion

The investigation of bilateral facial palsy demands the consideration of systemic diseases which may manifest in this manner. Often both the history and examination findings will reveal other symptoms and signs in addition to the facial diplegia and thus suggest likely diagnoses. Where such clues are absent, investigations should include baseline haematological indices, glucose and hepatic function, an autoimmune screen, tensilon test, chest X-ray, CT scan of brain stem and petrous bones, lumbar puncture, audiometry and spirochaetal serology (Sherwen and Thong, 1987; Stahl and Ferit, 1989). Some or all of these may be required in each individual case.

Worldwide, bilateral facial paralysis is most often associated with Guillain-Barré syndrome, leprosy, syphilis, leukaemia, sarcoidosis and bacterial meningitis (Sherwen and Thong, 1987). However, Lyme disease in the USA and a closely related European form, Borreliosis, are tick-borne spirochaetal infections which have been increasingly recognized as causes of both unilateral and bilateral cranial nerve palsies (Pachner and Steere, 1985; Olsson *et al.*, 1988), with the facial nerve most commonly affected (Pachner and Steere, 1985). Borreliosis is most commonly encountered in the Scandinavian countries, and is the causative factor in some 20 to 40 per cent of facial palsies (Ovesen, 1992). Up to a quarter of these may be bilateral (Clark *et al.*, 1985; Pachner and Steere, 1985).

In the case described, Lyme disease, sarcoidosis and trauma were considered diagnoses. The finding of an elevated protein and lymphocytosis in the CSF suggested a lymphocytic meningitis, as may be found in Lyme disease. However, no organisms were seen, CSF culture was sterile and more importantly, Lyme disease serology was negative. In retrospect, the CSF findings could have been secondary to the original head injury.

Sarcoidosis usually occurs in the 20 to 40 year-old age group (Sherwen and Thong, 1987) with cranial nerve involvement in 5 to 20 per cent, particularly affecting the facial nerve (George and Pahor, 1991). The exact pathophysiology of such palsies is unknown. In our case, a Kveim test was negative and a chest X-ray did not reveal any hilar lymphadenopathy, making this diagnosis most unlikely.

Both unilateral and bilateral facial palsy may result from skull base fracture (George and Pahor, 1991). Such palsies are usually of acute onset and may be associated with acoustic trauma. In the case we describe, the palsies were of delayed onset relative to the head injury, audiometry was normal and no fracture was seen on petrous bone computerized tomography. Despite this, EMG confirmed the presence of a demyelinating neuropathy affecting both facial nerves and predicted that recovery should occur. This is entirely consistent with the observed course of events and supports our diagnosis of a post-traumatic demyelinating neuropathy. To our knowledge, this is the first recorded description of such a neuropathy affecting both facial nerves.

References

Clark, J. R., Carlson, R. D., Pachner, A. R., Sasaki, C. T., Steere, A. C. (1985) Facial paralysis in Lyme disease. *Laryngoscope* **95:** 1341–1345.

George, M. K., Pahor, A. L. (1991) Sarcoidosis: a cause for bilateral facial palsy. *Ear, Nose and Throat Journal* **70:** 492–493.

Klar, A., Gross-Kielelstein, E., Hurvitz, H., Branski, D. (1985) Bilateral Bell's palsy due to *Mycoplasma pneumoniae* infection. *Israeli Journal of Medical Science* **21:** 692–694.

Olsson, I., Olsonn, I., Engervall, K., Asbrink, E., Carlsson-Nordlander, B., Hovark, A. (1988) Tick-borne Borreliosis and facial palsy. *Acta Otolaryngologica (Stockholm)* **105:** 100–107.

Ovesen, J. (1992) Bilateral facial palsy caused by the Borrelia spirochaete. *Acta Otolaryngologica (Stockholm)* **Supplement 492:** 115–116.

Pachner, A. R., Steere, A. C. (1985) The triad of neurological manifestations of Lyme disease: meningitis, cranial neuritis and radiculoneuritis. *Neurology* **35:** 47–53.

Sherwen, P. J., Thong, N. C. (1987) Bilateral facial nerve palsy: a case report and literature review. *Journal of Otolaryngology* **16:** 28–33.

Stahl, N., Ferit, T. (1989) Recurrent bilateral facial palsy. *Journal of Laryngology and Otology* **103:** 117–119.

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