

CLINICAL REPORTS

Hemifacial spasm in tuberculous meningitis

R. SANDYK
M.D.

Department of Medicine, Johannesburg Hospital, Johannesburg 2001, South Africa

Summary

Hemifacial spasm developed in a 47-year-old man with tuberculous meningitis. The spasms ceased completely following vigorous antituberculous treatment. A selective compression of the facial nerve root along its exit at the brainstem by a localised inflammatory process is the most tenable explanation for the development of the hemifacial spasm.

KEY WORDS: tuberculous meningitis, facial nerve, hemifacial spasm.

Introduction

Hemifacial spasm is a unilateral, involuntary, irregular clonic contraction, frequently beginning in the orbicularis oculi muscle and spreading to the adjacent muscles. It may progress over time to a tonic contraction causing a sustained eye closure and facial grimace. The spasms are aggravated by stress, persist in sleep and may ultimately result in a mild progressive facial weakness (Janneta *et al.*, 1977). Hemifacial spasm may be cryptogenic with no known injury to the seventh cranial nerve, or may be post-paralytic following Bell's palsy or traumatic facial paresis (Ferguson, 1978). Facial spasm has also been described in vascular abnormalities of the posterior fossa, encephalitis, Paget's disease, trauma and arachnoiditis (Eckman, Kramer and Altrocchi, 1971). Recently, a mechanical compression of the facial nerve root exit zone at the brainstem has been emphasised as a major cause of hemifacial spasm (Maroon, 1978).

A patient is reported here in whom hemifacial spasm was a presenting feature of tuberculous meningitis.

Case report

A 47-year-old black man was admitted to hospital with a 3-week history of involuntary, episodic twitching of the right side of his face. The episodes had

lasted up to 2-3 min without interruption and then subsided for minutes. Occasionally they were precipitated by stress or laughter. Past medical and drug history were noncontributory.

Examination revealed constant irregular contractions of the right orbicularis oris, orbicularis oculi and platysma muscles. At the onset of the facial spasms, twitching was noted in the right eyelid, followed by a tonic narrowing of the palpebral fissure and spreading to involve the entire musculature of the right face. Occasionally there was a strong pulling of all muscles from the frontalis down to the platysma muscle, as well as the muscles around the ear.

Examination of the cranial nerves revealed mild hypoaesthesia in the distribution of the right trigeminal nerve, with decrease in the corneal reflex response on that side. Deep tendon jerks were brisk bilaterally with no side difference. Plantar responses were flexor bilaterally. The remainder of the neurological and general examination was normal.

Laboratory analysis revealed an erythrocyte sedimentation rate of 60 mm/hr. Syphilis serology was negative. Roentgenograms of the skull, including basal views were normal. Computerized tomographic (CT) scan of the posterior fossa was normal. Lumbar puncture yielded clear cerebrospinal fluid (CSF) under slightly raised pressure (300 mmH₂O) and analysis showed protein 82 mg/dl, glucose 1.24 mmol/litre (blood glucose 7.3 mmol/litre). Microscopy showed 162 lymphocytes/mm³, 46 neutrophils/mm³ and the presence of acid fast bacilli. *Mycobacterium tuberculosis* was later cultured from the CSF. An electromyogram revealed irregular clusters of spontaneous discharges in the right orbicularis oris, compatible with the diagnosis of facial spasm.

Treatment was initiated with isonicotinic acid hydrazide (400 mg daily), rifampicin (600 mg daily) and ethambutol (1200 mg daily). Following 12 weeks

on this regimen, the severity and frequency of the hemifacial spasms decreased and disappeared completely after 6 months of therapy. The facial numbness persisted however.

Discussion

This patient demonstrated clinical signs of hemifacial spasm and the cause was found to be tuberculous meningitis. This is a most unusual presentation of a basal meningitis.

The precise aetiology of hemifacial spasm remains unknown. Several workers have suggested that the spasms result from an irritation of the facial nerve somewhere along its extramedullary course (Janneta *et al.*, 1977); others have postulated that the spasms originate in the nucleus of the facial nerve itself (Moldaver and Conley, 1980), whereas some postulated a cortical origin (Moldaver and Conley, 1980). Compression of the facial nerve root at its exit zone at the brainstem has recently been suggested to be vital for the development of the hemifacial spasms (Maroon, 1978; Janneta *et al.*, 1977).

The most tenable explanation for the hemifacial spasm in our case would be a selective compression of the facial nerve root along its exit from the brainstem by a localised tuberculous inflammatory process. The presence of trigeminal nerve involve-

ment on that side, and the absence of other cranial nerve pathology would support an extramedullary process. Whatever the precise mechanism may be, it is important that hemifacial spasm was the most obvious neurological sign indicating lesion of the facial nerve in a patient with meningitis on clinical criteria. This case reveals once again that chronic meningitides may present with a wide variety of subtle signs and symptoms. In this case the findings were of right hemifacial spasm in the absence of other signs of meningeal irritation or systemic features and proved highly misleading.

References

- ECKMAN, A.B., KRAMER, R.A. & ALTROCCHI, P.H. (1971) Hemifacial spasm. *Archives of Neurology*, **25**, 81.
- FERGUSON, J.H. (1978) Hemifacial spasms and the facial nucleus. *Annals of Neurology*, **4**, 97.
- JANNETA, P.J., ABBAY, M., MAROON, J.C., RAMOS, F.M. & ALBIN, M.S. (1977) Etiology and definitive microsurgical treatment of hemifacial spasm. Operative techniques and results in 47 patients. *Journal of Neurosurgery*, **47**, 321.
- MAROON, J.C. (1978) Hemifacial spasm, a vascular cause. *Archives of Neurology*, **35**, 481.
- MOLDAVER, J. & CONLEY, J. (1980) *The Facial Palsies. Their physiopathology and therapeutic approaches*, p. 119. Charles C. Thomas publisher, Springfield, U.S.A.

(Accepted 9 December 1982)