

LETTERS TO THE EDITOR

An unusual complication of mourning

The emotional trauma of bereavement can, on occasion, be translated into physical trauma. Phrases such as "grinding of teeth", "breast-beating" and "tearing one's hair out" have a traditional association with mourning and lamentation.¹ We report a case where bereavement led directly to an uncommon neurological complication of blunt trauma.

A forty year old housewife from the United Arab Emirates presented with a three week history of nausea, headache, pain in the right eye and a persistent throbbing in the head. Her nineteen year old son had been killed in a road traffic accident one month before presentation, and her relatives reported that in the few days between his death and the onset of the symptoms she had been seen repeatedly striking her head against a wall in her grief.

Physical examination revealed pulsating exophthalmos of the right eye accompanied by a supraorbital bruit. A diagnosis of carotid cavernous fistula was made, and carotid angiography under general anaesthesia confirmed the diagnosis (fig). As part of the same procedure she underwent balloon occlusion of the fistula with preservation of carotid blood flow. Her subsequent recovery was complete and uneventful.

Carotid cavernous fistula is an uncommon condition in which an abnormal communication exists between the carotid artery and the cavernous sinus at the base of the skull. This produces a characteristic syndrome of severe unilateral headache accompanied by ipsilateral carotid and supraorbital bruits, progressive pulsatile exophthalmos, usually ipsilateral, but not infrequently bilateral or even contralateral only, with chemosis, retinal haemorrhages, and variable degrees of ophthalmoplegia and loss of visual acuity which may progress to blindness. Approximately 75% of such fistulae are thought to be of traumatic origin, often found in association with a basal skull fracture, while the remainder are spontaneous, presumably arising from

the rupture of a previously diseased carotid arterial wall. The majority of the post traumatic group are due to blunt trauma, usually following a head injury violent enough to produce unconsciousness, although some follow surgical procedures to the head and neck.^{2,3} Rarely, penetrating trauma is responsible, caused by such diverse items as knives,⁴ air rifle pellets⁵ and fish.⁶

The aetiology of the carotid cavernous fistula in the patient we describe was almost certainly blunt trauma from the repeated striking of her head against a wall while in the acute stage of mourning for the death of her son. It is not uncommon for followers of the Muslim faith, particularly Shi'ites, to traumatise themselves in times of great sorrow. Beating the head against the floor (in the position of prayer) or against a wall or door post, or striking the forehead with the palm of the hand are all recognised. Though such behaviour is presumably often associated with localised trauma of a minor kind, we are not aware of a report of its occurrence as a cause of carotid cavernous fistula.

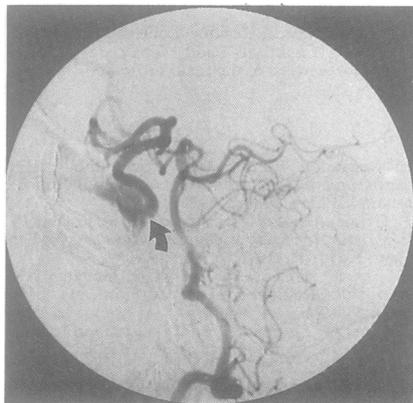
We are grateful to Mr R Jobbins of the World Service of the British Broadcasting Corporation for advice concerning the mourning habits of Arab Muslims.

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Vertebral angiogram, lateral projection. There is retrograde filling of the internal carotid artery as far as a fistula, which is outlined (arrow) by contrast medium shunting into the cavernous fistula.

Chronic polyradiculoneuropathy associated with human T-cell lymphotropic virus type I infection

A chronic progressive myelopathy associated with human T-cell lymphotropic virus type I (HTLV-I) infection named HTLV-I associated myelopathy (HAM) has been recently described in Japan.¹ We report the first patient with chronic polyradiculoneuropathy, found to have HTLV-I infection.

A 53 year old woman presented with progressing symmetrical weakness of the lower limbs over one month. Her reflexes of the lower limbs were absent. The worsening and weakness was progressive. Symmetrical

weakness and atrophy of the upper limbs and trunk also appeared a month after onset. Eventually, after five months she showed severe generalised weakness and areflexia. No involvement of the cranial and sensory nerves or the respiratory function was observed.

Nerve conduction studies revealed prolonged distal latencies (median nerve motor latency: 7 ms), disappearance of H-reflex, and slowing of F-wave recorded from the thenar muscles (latency 45 ms; F-ratio 2.6). Electromyographic examination showed developing denervation of the anterior tibial and gastrocnemius muscles. Sural nerve biopsy did not indicate demyelination or perivascular and endoneurial infiltrates of mononuclear cells. Cerebrospinal fluid (CSF) protein level (max 385 mg/dl) and immunoglobulin in G(IgG) (max 47 mg/dl) was elevated with a normal cell count. Anti-HTLV-I antibody titres were positive in both serum (more than 512 titres) and CSF (32 titres) using the particle agglutination method. Western blot (WB) analysis confirmed the presence of antibodies to HTLV-I in the serum and CSF IgG. The CSF myelin basic protein content was normal. There were no atypical lymphocytes in either the CSF or peripheral blood, and the white blood cell count was normal. Bone marrow study was also normal. She was seronegative for HTLV-III.

She had been treated with prednisolone as advocated by Dalakas and Engel² and gradually improved over more than four months.

Our patient had a symmetrical polyradiculoneuropathy characterised by high antibody titres to HTLV-I in both serum and CSF, confirmed by WB analysis, and a predominantly lower motor neuron disorder. As shown by ratio of CSF to serum albumin, the blood-brain barrier (BBB) was not damaged, and intra-BBB IgG synthesis rate³ was increased.

Cornblath *et al*⁴ described nine patients with inflammatory demyelinating polyneuropathy (IDP), found to have HTLV-III infection. In their patients they considered that IDP most probably resulted from an immunopathological mechanism, reflecting altered immune regulation produced by HTLV-III infection. Tsukada *et al*⁵ described a patient with chronic relapsing polyneuropathy associated with hepatitis B virus (HBV) infection. Electron-dense deposits, suggesting immune complexes composed of HBV, were demonstrated in the endoneurium of the patient.

Thus HTLV-I infection may cause IDP. We assume that this patient had an IDP associated with HTLV-I rather than the virus directly attacking the nerve, while coincidental occurrence of CIDP and positive titre for HTLV-I cannot be excluded. Immunological, genetic or other unknown factors may be interrelated in the clinical manifestation of HTLV-I infection.

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