Ophthalmoplegic tetanus

Local tetanus affecting cranial nerves is uncommon and ocular involvement rare. We describe a patient in whom bilateral internal and external ophthalmoplegia and supranuclear gaze palsy complicated tetanus.

Case history

A 44-year-old man was admitted to Hull Royal Infirmary with severe trismus, tongue biting, left facial weakness, fever, and malaise of sudden onset. Four days later he developed spasms typical of tetanus affecting the legs, with arching of the back and rigidity of the erector spinae. Three weeks earlier he had had a trivial foreign body in the left eye with no signs of local infection. He had been given tetanus antitoxin but not toxoid 20 years before after an injury.

He had a severe and unusual supranuclear ophthalmoplegia (fig 1a) with total paresis of conjugate eye movements to the left. There were slow pursuit movements on vertical gaze up and down, transient bilateral ptosis, and pronounced, very rapid nystagmus on right lateral gaze, particularly in the abducting eye. Pupil reactions to light and accommodation were impaired. Trismus was severe, interdental distance was 1.5 cm. The upper limbs were normal. Tone in the legs was much increased and reflexes were brisk with ankle clonus and flexor plantar responses. Nothing abnormal was found on general examination. The blood pressure was 120/80 mm Hg. The CSF contained 9 lymphocytes, protein 0.35 g/l, and gammaglobulin 20% of total protein. Virus investigations were negative. An electroencephalogram and x-ray examinations of skull and chest were normal.

Large doses of diazepam and chlorpromazine two-hourly were needed to control the spasms, which were readily provoked by noise, startle, and light touch anywhere on the cranium, particularly the mouth. A tracheostomy was done because of one episode of partial asphyxia, but ventilation or curarisation was not necessary. He was given antitetanic human globulin and penicillin at the outset. He slowly recovered over six weeks but slight rigidity of the legs and loss of confidence remained. His eye movements returned to normal (fig 1b). Tetanus toxoid was given.

Supranuclear ophthalmoplegia in patient with tetanus: (a) looking to left—total paralysis of conjugate gaze and mild ptosis; (b) looking up—marked impairment of conjugate upward gaze.

Discussion

Kinnier Wilson¹ stated: "A rare subvariety of cephalic tetanus is distinguished by oculomotor palsy, with or without affection of the seventh cranial nerve." Blepharospasm is the commonest sign, followed by ptosis due to tonic spasm of the lids² or to third-nerve palsy. Pupillary paralysis,³ spastic miosis, and complete ophthalmoplegia⁴ have been reported. In contrast to these signs of local involvement of ocular nerves, our patient presented features of bilateral internal and external ophthalmoplegia with a complete gaze paralysis

and nystagmus. These signs suggest a disorder of nuclear and supra-nuclear pathways in the diencephalon and midbrain. Chromatolysis and neuronophagia have been found in other brain-stem nuclei,⁵ and we think this was the pathological basis of the unusual signs in our patient.

We thank Dr T W G Kinnear for referring the patient.

¹ Wilson, S A K, *Neurology*, 1st edn, p 630. London, Arnold, 1940.

² Walsh, F B, *Clinical Neuro-Ophthalmology*, 2nd edn, p 722. Baltimore, Williams and Wilkins, 1957.

³ Worms, G, *Du Tétanos Céphalique avec Ophthalmoplegia*. Thèse de Lyons, 1905-6.

⁴ Jayme-Goyaz, G G, *American Journal of Ophthalmology*, 1941, 24, 1281.

⁵ Harriman, D G F, *Symposium on Tetanus in Great Britain*. Leeds, National Lending Library, 1967.

Department of Neurology, Hull Royal Infirmary, Yorkshire

S SALTISSE, MB, MRCP, senior house officer (now senior house officer in medicine, University Hospital of South Manchester, Manchester M20 8LR)

R N HAKIN, MRCP, registrar in neurology

J PEARCE, MD, FRCP, consultant neurologist

Lymph node and hepatic granulomata associated with carcinoma

Granulomatous reactions to malignant neoplasms have been described, both locally and in regional lymph nodes. A case is reported here in which a carcinoma appears to have led to both lymph-node granulomata and granulomatous hepatitis.

Case report

A 69-year-old housewife was admitted with a two-month history of diarrhoea and abdominal pain. Four years previously she had had a pelvic clearance for ovarian carcinoma, followed by radiotherapy. Subsequent follow-up had shown no signs of recurrence.

She had a mild fever, her liver was enlarged, but otherwise examination was normal. Investigations showed a non-specific anaemia but otherwise the results of extensive laboratory and routine radiological studies were normal. Barium enema showed an irregular narrowing in the upper sigmoid, probably due to radiotherapy. Toxoplasma dye test suggested old infection. Repeated liver function tests showed an albumin concentration of 34 g/l, falling to 25 g/l; alkaline phosphatase between 14 and 36 KAU/100 ml; alanine aminotransferase between 90 and 200 IU/l; bilirubin and prothrombin time normal. Liver scan showed an enlarged liver without focal defects. A liver biopsy showed multiple granulomata related to the portal tracts and in the liver parenchyma. The architecture was well preserved; stains and culture for fungi and acid-fast bacilli were negative.

At laparotomy, undertaken after an unsuccessful four-week trial of anti-tuberculous therapy, 500 ml of straw-coloured fluid were removed. She had radiation fibrosis involving the rectum and sigmoid, explaining her diarrhoea and changes seen on barium enema. There was no macroscopic evidence of recurrent neoplasm and the small bowel was normal. The liver was enlarged with variably sized scattered nodules throughout both lobes. A trial dissection of the upper rectum and sigmoid colon had to be abandoned and a right transverse colostomy was performed. Biopsy specimens of the liver and a coeliac node were taken. Histologically, the appearances of the liver biopsy were unchanged, but the lymph node contained both multiple non-caseating granulomata and deposits of moderately differentiated adenocarcinoma.

Postoperatively, she developed ascites and complained of weakness, nausea, and vomiting. Treatment with cyclophosphamide and prednisone was started but, since after three weeks she was deteriorating, only symptomatic treatment was given thereafter.

Discussion

Granulomatous hepatitis may result from many causes, but these are usually undetermined in about 20% of cases. Prolonged follow-up may help and antituberculous treatment has been advocated as a