SHORT REPORTS

Ophthalmoplegia, amblyopia, and diffuse encephalomyelitis associated with pelvic abscess

Duke-Elder[1] included focal sepsis as a cause of optic neuritis, though he thought the association was uncommon. We describe here a patient who presented with optic neuritis with no apparent cause but whose neuro-ophthalmological symptoms disappeared after removal of a pelvic abscess.

Case report

An 18-year-old girl was admitted with a two-week history of headaches, backache, neck stiffness, and hyperacusis. One week before admission she developed intermittent diplopia, failing vision, and ataxia. Her last period had occurred three and a half months earlier. There was no history of drug abuse.

On examination she was feverish, drowsy, slow, and inconsistent in her replies. She had mild neck stiffness and a positive Kernig's sign. There were no marks of injection, bruises, abrasions, rash, or lymphadenopathy. There was a retrouterine mass, the size of a four-month pregnancy, which was fixed, non-tender, and firm.

Neurologically the eyes were immobile on all forms of testing, and the pupils were slightly divergent, irregular, and dilated and did not react to light. She could just detect hand movements but suffered no localised field loss. Examination of the fundi showed bilateral, sharply defined, whitish exudates over the disc and distension of surrounding vessels. There were no haemorrhages and the peripheral vessels were normal. Apart from mild deafness the remaining cranial nerves were normal but her arms were ataxic and she could not stand. All tendon jerks were reduced and the plantar responses were flexor.

Investigations showed a leucocytosis of 23×10^9/l with 89% polymorphs and an erythrocyte sedimentation rate of 76 mm in first hour. Abdominal radiographs showed no pelvic calcification and pregnancy tests gave negative results. Chest and skull radiographs, right carotid angiogram, bilateral cavernous venograms, and EMI scan were all normal. Initial lumbar cerebrospinal fluid (CSF) examination showed normal pressure, cells, and biochemical values. An electroencephalogram showed widespread disorganisation with no specific features.

After treatment with ampicillin and ACTH the fever settled but her level of consciousness and peripheral signs fluctuated. The fundal appearances changed, becoming pinker and more oedematous with venous congestion resembling plerocephalic oedema. Repeat lumbar puncture after three days confirmed that the CSF pressure had risen to 300 mm H_2O but otherwise it remained normal. Treatment with metronidazole was started and her level of consciousness and peripheral neurological signs improved. When metronidazole was stopped after a further 10 days she deteriorated with a swinging temperature. At laparotomy a right sterile pyosalpinx was removed together with the left ovary and fallopian tube, which were affected by several small abscesses. Her mental state and eye movements recovered. The papilloedema resolved and visual acuity returned to normal.

Three months later she complained of gradual loss of sight: visual acuity was 6/36 on the right and 6/12 on the left. The optic discs were pale and the physiological cup obliterated. Perimetry showed concentric contraction in both eyes but no scotoma. Pupillary reflexes were present and eye movements were full, and there were no other neurological signs.

Discussion

This patient initially presented with optic neuritis and a normal CSF pressure, though the subsequent rise in CSF pressure contributed to later swelling of the optic disc. Screening tests for toxic substances, viruses, and specific diseases known to be associated with optic neuritis were negative, and the CSF findings together with the intermittent but symmetrical signs of central nervous system disease made embolic abscess or basal meningitis unlikely. Though our theory remains unproved, we suspect that the pelvic abscess may have caused the optic neuritis. The improvement after metronidazole, the deterioration when the drug was stopped, and the subsequent improvement when the abscess was drained support this suggestion.

Though Duke-Elder[1] considered that focal sepsis was only rarely associated with optic neuritis, other workers have found the two conditions associated in as many as 44% of cases.[2] The presence of a pelvic abscess in this patient provides some further evidence of an occasional association between focal sepsis and widespread neuro-ophthalmological disorder.

We thank Professor R Hoffenberg for his help with the clinical management and this report.

[1] Duke-Elder, S, *System of Ophthalmology*, Vol XII, Neuro-ophthalmology, p 69. London, Kimpton, 1972.
[2] Wilmer, W H, *Archives of Ophthalmology*, 1930, **4**, 817.

(*Accepted 2 January 1979*)

Midland Centre for Neurosurgery and Neurology, Smethwick Warley, West Midlands B67 7JX
BERNARD WILLIAMS, CHM, FRCS, consultant neurosurgeon

Norfolk and Norwich Hospital, Norwich, Norfolk NR1 3SR
T N GHOSH, DO, FRCS, senior registrar in ophthalmology

Department of Medicine, Queen Elizabeth Hospital, Edgbaston, Birmingham B15 2TH
R A STOCKLEY, MD, MRCP, lecturer in medicine

Absence of toxicity in cimetidine overdosage

Cimetidine is a histamine H_2-receptor antagonist that is widely used in the treatment of peptic ulceration. We report four cases of self-poisoning with this drug.

Case reports

(1) A 26-year-old man with a duodenal ulcer was admitted 2½ hours after allegedly taking 80 cimetidine tablets (16 g), 30 mg nitrazepam, and 10 tablets of aluminium hydroxide. He was slightly drowsy and complained of a dry mouth, which was confirmed on examination. No other abnormality was found. Some tablet material was recovered by gastric lavage. He developed slight epigastric discomfort which was relieved by milk, but otherwise he remained well and the dryness of his mouth disappeared within 18 hours. The electrocardiogram and plasma urea, creatinine, electrolytes, and glucose concentrations, and the liver function test results were normal 20 hours after the overdose. The plasma cimetidine concentration, measured by a high-pressure liquid chromatography method based on that of Randolph *et al*,[1] was 57 mg/l (226 μmol/l) at 3·2 h, 40 mg/l (159 μmol/l) at 4·5 h, and 3 mg/l (12 μmol/l) at 23·3 h after the overdose.

(2) A 42-year-old man claimed to have taken 98 cimetidine tablets with a lot of Guinness two hours before admission. His only complaint was of a dry mouth. His tongue looked dry and he smelled of alcohol, but examination was otherwise normal. The electrocardiogram on admission was normal. Vomiting was induced by ipecacuanha with return of tablet fragments. He remained well and was discharged after 12 hours. The plasma cimetidine concentration was 36 mg/l (143 μmol/l) at 2 h and 2·3 mg/l (9 μmol/l) at 11·5 h after the overdose.

(3) A 26-year-old man was admitted 1½ hours after taking 26 cimetidine tablets (5·2 g). He was observed for 10 hours but developed no abnormal symptoms or signs. Two hours after the overdose the plasma cimetidine concentration was 37 mg/l (147 μmol/l).

(4) Ten hours before admission a 37-year-old man allegedly took 30 cimetidine tablets (6 g) and two pints of beer. He developed no symptoms or signs of toxicity. The plasma cimetidine concentration was 4·5 mg/l (18 μmol/l) at 10 h and 2·9 mg/l (11 μmol/l) at 12 h.

Comment

A single 200-mg dose of cimetidine by mouth gives a peak plasma concentration of about 1 mg/l (4·0 μmol/l) at one hour.[1] Our patients had much higher concentrations without ill effect except for dryness of the mouth in two patients and drowsiness in one patient who had also taken nitrazepam. Gill[2] described the case of a man who took about 15 cimetidine tablets four times a day for five days without untoward