Lesson of the Week

Subdural haematoma in two patients with chronic neurological disorders

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The clinical diagnosis of chronic subdural haematoma is often difficult and as a result delayed; it was made on admission in only 28% of a recent large series of cases.¹ In two patients with chronic neurological disorders (multiple sclerosis and Parkinson's disease) the presentation of subdural haematoma mimicked deterioration of their pre-existing disease. Neurological disability probably predisposes patients to the development of subdural haematoma because of an increased incidence of falls and minor head trauma.

Case reports

CASE 1

A 52 year old woman first developed intermittent vertigo, blurring of vision, and numbness of the left arm in 1975. On examination then there was mild ataxia in the left arm and leg. The tendon reflexes were depressed or absent and the plantar responses extensor. The abdominal reflexes were absent. Proprioception and two point discrimination were impaired on the left, and vibration sense was absent below the ankles. Examination of cerebrospinal fluid, including measurement of the IgG concentration, gave normal results. Computed tomography showed mild cerebral atrophy. The visual evoked response from the left eye was delayed. A diagnosis of probable multiple sclerosis was made.

Over the next seven years the patient was aware of residual clumsiness of her left arm but had no other persistent symptoms. On examination in 1977 she had mild ataxia of the legs, but the physical signs were otherwise unchanged. A few months later she had an episode of numbness of both legs that lasted for one week and was treated with corticotrophin. In February 1982 her gait ataxia increased and as a result she fell and hit her head but did not lose consciousness. At the end of May she noticed more pronounced unsteadiness, weakness of the right leg, transient slurring of speech, and deterioration in her handwriting. These symptoms fluctuated. Two weeks later she was unable to walk independently. She then had severe weakness of her right arm and blurred vision in the right eye for a few days, both of which improved spontaneously. She had had occasional right temporal headaches after her fall, which were relieved by aspirin.

On examination in June 1982 she was vague and disinhibited and recent memory was impaired. She had a cerebellar dysarthria. Colour vision was defective on the right with a visual acuity of 6/9 compared with 6/6 on the left. There was bilateral optic atrophy, a right afferent pupillary defect, nystagmus on upgaze, pyramidal weakness of the right arm and both legs, and cerebellar ataxia in all four limbs, more on the left than the right. The plantar responses were extensor. Sensory testing was unreliable. Her gait was considerably ataxic.

The clinical features of the patient's recent deterioration were

Subdural haematoma may be difficult to diagnose in patients with chronic neurological disorders, particularly if neurological signs can be attributed to longstanding disease

thought to be compatible with the previous diagnosis of multiple sclerosis. A repeat examination of the cerebrospinal fluid showed that the opening pressure was 12 cm H₂O, and IgG electrophoresis showed an oligoclonal pattern. Skull radiographs showed no abnormality. The day after lumbar puncture she became drowsy and had increased weakness of the right arm. Computed tomography showed a large almost isodense subdural haematoma on the left with pronounced mass effect (figure). This was evacuated and the patient's clinical condition subsequently improved. In October 1982 her mental state was normal. She had an ataxic gait but no focal neurological signs and was able to walk with a stick.

Enhanced computed tomogram of case 1, showing a slightly hypodense left subdural haematoma producing mass effect.

CASE 2

A 78 year old woman first presented with tremor in 1967, and Parkinson's disease was diagnosed. She was initially treated with anticholinergic drugs and later with levodopa. She was not appreciably disabled until 1978 when she started to fall occasionally and had typical "on off" effects from her medication.

She was admitted to the Middlesex Hospital in May 1981. She had fallen more often in the few months before admission, and three days previously she had been found lying rigidly on her back on the floor. She had subsequently been confused, immobile, and incontinent of urine. Her treatment consisted of levodopa 500 mg four times a day, orphenadrine 50 mg three times a day, and diazepam 2 mg twice a day. On examination she was disorientated and bradykinetic. There was obvious cogwheel rigidity but normal power in the limbs. The tendon

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reflexes were symmetrically brisk, and the right plantar response was equivocally extensor. Sensory testing was unreliable.

The patient clearly had severe Parkinson's disease and, in addition, a confusional state without any definite focal neurological signs. Skull radiographs showed no abnormality. The day after admission she became more confused and slightly drowsy but was otherwise unchanged. Computed tomography showed a left subdural effusion with considerable mass effect. The patient initially refused an operation and the effusion was not drained until two weeks after admission. She made slow progress after operation but was eventually discharged five months later, independent in personal care but moderately disabled by her Parkinson's disease.

Comment

The neurological deterioration that precipitated admission in each of these patients was initially thought to be related to their chronic diseases. A history of mild head injury was elicited in both, which was not particularly surprising in view of their disturbances of gait. Increasing ataxia preceded head trauma in case 1. Her subsequent course was compatible with the diagnosis of multiple sclerosis; dysarthria and ataxia are common in this disease but are rare features of subdural haematoma.¹ The patient's headache was inconspicuous and did not suggest raised intracranial pressure.

The clinical problem in case 2, comprising increased immobility, falls, and a confusional state, is common in elderly patients with advanced Parkinson's disease. Confusion may be caused by infection or dopaminergic and anticholinergic drugs.

Intercurrent illness is often associated with an increase in extrapyramidal rigidity.

Alcoholism, epilepsy, and cerebral atrophy of any cause are known predisposing factors in the development of subdural haematoma.^{1,2} A review of the case notes of 25 other patients with proved chronic subdural haematoma admitted to the National Hospital during the period 1978-82 showed that five had one or more pre-existing conditions that probably rendered them more liable to head trauma. These included epilepsy (three cases), cardiac arrhythmia (one), a degenerative cerebellar syndrome (one), and motor neurone disease (one).

The diagnosis of subdural haematoma in patients who have longstanding neurological disability requires a high index of clinical suspicion; subacute generalised deterioration may not be associated with conspicuous headache, papilloedema, or definite signs of unilateral cerebral hemisphere compression in this context.

I thank Dr C J Earl and Professor John Marshall for permission to publish details of their patients.

References

- ¹ Luxon LM, Harrison MJG. Chronic subdural haematoma. *Q J Med* 1979;48:43-54.
- ² McKissock W, Richardson A, Bloom WH. Subdural haematoma. A review of 389 cases. *Lancet* 1960;i:1365-9.

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MATERIA NON MEDICA

Tick tock or bleep

Perhaps my upbringing was unusual but the acquiring of a wristwatch was a childhood milestone comparable to, say, the change from school flannel shorts to trousers or sitting the 11 plus examination. Around that time I received a clockwork (British) watch which survived the turbulence of a suburban grammar school but not the oriental horological onslaught fashionable during my late teens. Its successor, a 21 jewelled self winder, ticks on despite many immersions and tumbles, and fends off the quartz challenge—for the moment.

I now meet quite young children wearing wristwatches; often they cannot tell me the time and I wonder what it is they value—presumably the status or decorative effect. Two recent incidents interested me further: first, I was offered a wristwatch with the compliments of a garage where I bought a modest carton of oil; and secondly, my daughter was given one as a Christmas present by a school friend. I see that the acquisition of a wristwatch no longer carries the significance that it did in the 1950s (I suspect the same is true for television). Time, through digital watches, threatens to rule life more nastily: I have heard the hourly alarms bleeping at the theatre, during mass, and one third of the way through an LRCP medicine viva. Although my coexaminer became apoplectic there was no discernible effect on the lacklustre candidate. Are our children and students now so harassed that they need to regulate their lives to the second?—T L CHAMBERS, consultant paediatrician and clinical dean, Bristol.

Irish fare

I had not been to Dublin nor the Abbey Theatre—and you should not visit the one without the other—for 36 years. My 1948 programme is autographed by John McCallum and Googie Withers, not because they were performing that evening (which would have been a remarkable piece of casting) but because they happened to be in the audience and the intimacy of the tiny theatre made it easy for any person out front to approach any other. That theatre, ex public mortuary, was destroyed by fire in 1951; the new Abbey, if lacking some of the charm of the old is a more comfortable and worthy home for the Republic's national theatre. To see a Brendan Behan

play at the Abbey promised to be an experience indeed, but perhaps we expected too much. You do not routinely see the finest Shakespearian acting at Stratford and this production of *The Quare Fellow* for all its competence somehow failed to generate the almost unbearable tension that held us when we first saw the play in Liverpool. Had it dated? I think not, although as we filed out we overheard one member of the audience suggest so and go on to say that maybe the Abbey company was not of the quality that he recalled.

But Snaffles restaurant was no disappointment. In a city where the detritus of the very many junk food establishments litters the streets, where some restaurants offer little that is not grilled or fried (what is wrong with Irish stew?), and where others charge prices that cannot possibly be justified this small, unpretentious, quiet, and, above all, friendly establishment was a highlight of our visit. Not exactly cheap, but worth every penny: you cannot help warming to gentlemen who will sell you Ch Lafite-Rothschild for £150 a bottle and recommend the house wine. "Arguably the best restaurant in Ireland," someone said. I am not arguing.—W K COWAN, consultant histopathologist, Gateshead.

Clinical controversy: early finger clubbing

In medical examinations patients are often said to show "early finger clubbing." Usually they simply have nails that have not been properly manicured for some time. An adjective is required that describes the degree of clubbing present, not its duration and state of progression as these cannot be deduced from a single examination; increased nail curvature may later be lost for various reasons.¹ A three grade scale of moderate, mild, and severe has been proposed.¹ Definitions of these adjectives might be hard to achieve and would probably vary among subspecialties but they would at least convey more information than the meaningless term early finger clubbing.—JOHN F FIELDING, Dublin.

- ¹ Fielding JF, Cooke WT. Finger clubbing in regional enteritis. *GUT* 1971; 12:442-4.