Lesson of the Week

Hydrocephalus after cerebellar infarction

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An infarction in the cerebellum may be followed by the development of oedema, causing obstructive hydrocephalus and loss of consciousness.

Case report

A 75 year old man was admitted after the abrupt onset of tinnitus, giddiness, nausea, vomiting, and dysarthria. On examination he had a blood pressure of 148/88 mm Hg, was in atrial fibrillation with a ventricular rate of 64/min, and had a soft, short apical systolic murmur. He was alert and orientated and had nystagmus with the fast component to the periphery and maximal on gaze to the left, a partial left sixth nerve palsy, a left lower motor neurone facial weakness, moderate dysarthria, ataxia of the left arm and leg, and inability to sit unsupported. Results of tone, power, and sensory testing were normal, reflexes were slightly brisker on the right, and plantars were flexor.

A CT scan (fig 1) showed a left cerebellar infarction and slight hydrocephalus. An electrocardiogram confirmed atrial fibrillation, and an echocardiogram showed mitral annulus calcification but no mitral valve stenosis.

The nausea, nystagmus, and ataxia gradually improved but 60 hours after

Hydrocephalus may complicate a cerebellar infarction and is readily treated

FIG 1—CT scan on admission showing left cerebellar infarction and displacement of fourth ventricle.

FIG 2—CT scan two days after admission showing pronounced dilatation of lateral ventricles.

the onset of symptoms he began to become obtunded, with no change in the focal signs. A second CT scan (figs 2 and 3) showed oedema around the infarction in the posterior fossa, causing compression of the fourth ventricle and obstructive hydrocephalus. High dose dexamethasone did not halt the worsening impairment of his conscious level. After insertion of a ventricular drain his conscious level rapidly improved, but deteriorated again seven hours after he had removed the drain himself. His conscious level again rapidly improved when cerebrospinal fluid was removed by direct ventricular tap. He subsequently had a ventriculoperitoneal shunt inserted and made a good recovery, and was discharged home, independently mobile, 21 days after admission.

Comment

This patient presented with clear signs of a left cerebellar stroke and his subsequent impairment of conscious level without new focal signs was due to obstructive hydrocephalus caused by oedema around the infarct. After relief of the hydrocephalus he made a good recovery.

The patient's deterioration in hospital may have been mistaken for progression of his initial illness, and without the benefit of a CT

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scan he might have died of a treatable complication of his cerebellar infarction. Cerebellar infarctions are not common disorders, representing less than 5% of strokes, but present an important challenge to the clinician, as accuracy in diagnosis may prove life saving. The patient presenting with an acute onset of dizziness,

FIG 3—CT scan two days after admission showing increased swelling in posterior fossa and further displacement of fourth ventricle to right.

nausea, vomiting, and disturbance of gait should alert the clinician to the possibility of a cerebellar infarction or haemorrhage. Limb ataxia occurs less commonly than truncal ataxia. Nystagmus is a frequent sign, usually more severe towards the affected side, and unlike the nystagmus of labyrinthine disease cerebellar nystagmus tends to change direction with changes of gaze and to be dependent on eye fixation.¹ Dysarthria occurs frequently and the patients may complain of headache.

Distinguishing infarction from haemorrhage in the cerebellum is difficult without the CT scan, and CT is an important aid to diagnosing hydrocephalus, the most important prognostic factor in cerebellar infarction.² Hydrocephalus in these cases occurs from the oedematous infarcted tissue compressing the fourth ventricle and usually may be successfully treated by ventricular drainage or surgical decompression of the posterior fossa. Either procedure may be performed without undue difficulty by a neurosurgeon, and the results are usually dramatic. The deficit after cerebellar infarction, even when extensive, may be minimal once the patient has recovered from the initial event. Patients deteriorating after vascular events in the posterior fossa should have prompt CT scans performed so that the development of hydrocephalus may be assessed and life saving treatment initiated.

We are grateful to Dr J M Oxbury for permission to report this case.

References

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A patient was recently found to have a blood alcohol concentration higher than would be expected from the amount of alcohol consumed. He was taking clomipramine (Anafranil) and clorazepic acid (Tranxene). Could these drugs affect the blood alcohol concentration?

I am not aware of any direct evidence that either of these drugs affects blood alcohol concentrations. Alcohol, which is absorbed partly from the stomach but mainly from the small intestine, is metabolised mainly by hepatic alcohol dehydrogenase, but a small amount is also metabolised by a hepatic microsomal oxidising system and this pathway becomes more important in regular heavy drinkers.¹ Tricyclic antidepressants have anticholinergic properties and delay gastric emptying. This would be expected to slow the rate of alcohol absorption and reduce the peak blood concentration achieved. This effect has been shown in five out of six healthy volunteers.² In the sixth volunteer the opposite effect was seen with the peak blood concentration of alcohol higher after desipramine. No explanation was given for this anomalous result. Neither clomipramine or clorazepate is known to inhibit either pathway of alcohol metabolism, and neither interferes with the methods commonly used to measure blood alcohol concentrations. It seems unlikely that this patient's unexpectedly high blood alcohol concentration can be attributed to his drug therapy.—LINDA BEELEY, consultant clinical pharmacologist, Birmingham.

- 1 Linnoila M, Mattila MJ, Kitchell BS. Drug interactions with alcohol. *Drugs* 1979;18:299-311.
- 2 Hall RC, Brown D, Carter R, Kendall MJ. The effect of desmethylimipramine on the absorption of alcohol and paracetamol. *Postgrad Med J* 1976;52:139-42.

A man of 30 does not know whether he has ever been immunised. His medical records are sparse and he has no living relatives. Should he have a full course of tetanus and polio immunisation or should his immune status be established by serological tests?

Assays are available for tetanus antitoxin and for the three poliovirus antibodies and levels of these antibodies compatible with immunity are recognised. They are not routinely used to assess susceptibility. A man of 30 probably received a primary course of tetanus in infancy but is less likely to have received inactivated or live poliovaccines, which became available in 1956 and 1962, respectively. There is little risk from giving a course of oral poliomyelitis vaccine (three doses at least four weeks apart), though the incidence of vaccine associated paralysis does rise with age. Often repeated tetanus boosters, however, may lead to a sharp local reaction with

systemic upset. It would be pleasing to be able to detect antibody titres in anyone and remedy any gaps found, but the cost of the procedures cannot justify this. So, pragmatically, I would give this man full courses of both poliomyelitis and tetanus vaccines to ensure protection. The poliomyelitis course would be standard. I would start the tetanus course with adsorbed vaccine, 0.5 ml intramuscularly, and would boost at the longer recommended time intervals of 12 weeks and 12 months with simple—that is, non-adsorbed—tetanus vaccine, 0.1 ml intradermally. This would lessen the likelihood of any adverse reaction should a childhood course have been given. I would also issue an immunisation record card for future reference.—GLYN WILLIAMS, lecturer in infectious diseases, Glasgow.

I possess a wooden shuttle from one of the old Yorkshire weaving mills. At one end of the shuttle are a couple of holes through which the yarn passed from the bobbin; when threading the yarn it would be pushed through one end of the hole and then sucked out through the other end. Is there any evidence for the story that this practice sometimes led to carcinoma of the lip?

This is a fascinating question. There was during the nineteenth century and the first part of the twentieth century a considerable risk of skin cancer, particularly affecting the scrotum in men, and to a lesser extent the vulva in women, working in the cotton mills in Lancashire. The problem arose from contact with the mineral oils used for lubricating and cooling the "mules." In theory, the shuttle and bobbin used in weaving should not come into contact with mineral oil, but if they did so then you could expect there to be a risk of cancer of the lip in operatives who sucked the yarn through the hole of the shuttle. I have, however, been unable to find any published evidence of such risk. Nevertheless, there is another health risk associated with the practice described by the questioner. It is the transfer of disease, in particular tuberculosis, from one operative to another. This risk from "shuttle-kissing," as the practice was called, led in 1952 to the Factories (Cotton Shuttles) Special Regulations (Statutory Instrument, 1495, 1952). This regulation prohibited the use after a certain stated date of "suction shuttles," thereby compelling mill owners to go over to the use of "non-suction shuttles." The introduction of this regulation owed much to Miss Bessie Blackburn, for many years HM District Inspector of Factories in Burnley and herself a former weaver.—FRANCIS ROE, independent consultant in toxicology and cancer research, London, and ROBERT MURRAY, independent consultant in occupational medicine, London.