Cerebrospinal fluid was normal (leucocytes 0·6 μl; protein 0·43 g/l). Electromyography showed decreased compound muscle action potentials in tibial nerves and signs of degeneration of motor axons (fibrillation potentials). Motor nerve conduction velocities, and distal and F wave latencies were normal. Compound muscle action potential amplitudes were reduced in tibial nerves (right 1·5 mV; left 0·2 mV). Sensory nerve conduction velocity was normal in all four limbs but potential amplitudes were slightly reduced in the sural nerves (right: 6 μV left: 8 μV). Blood chemistry and cell count were normal except for a mild macrocytosis (mean corpuscular volume 99 fl; haemoglobin 11·6 g/dl). Serum cobalamin concentration was 240 pg/ml (normal 200–1000 pg/ml), but serum and red cell folate were lowered, respectively 2·4 ng/ml (normal >3 ng/ml) and 146 ng/ml (normal > 150 ng/ml). Serum thyroxine, creatine, phosphokinase, lactic dehydrogenase and aldolase were normal. Tests for heavy metals and coproporphyrins in urine were negative. Amino acid chromatography detected homocystinuria (16 μmol/1 per 24 hours) whereas no homocystinuria was found in controls. Because of the clinical features, homocystinuria, and lowered folates, a disturbance of folate metabolism was suspected. Activity of MTHFR was assayed on skin fibroblast according to the method described by Kutzbach and Stokstad.[4] MTHFR in the patient was 1 nmol/h/mg protein (mean control value in 20 normal subjects 3·8 nmol/h/mg protein with a range of 2·6 to 5). An artefactual decrease of this enzyme activity due to folate deficiency was excluded because cells were grown in a complete RPMI medium containing folic acid.

She was treated with infusion of thiamine, pyridoxine, cyanocobalamin, and intramuscular folinic acid for nine days; these were then given by mouth. Gait and behaviour improved in two weeks. Homocystinuria and delusions disappeared within 15 days, neuroleptic drugs were stopped, and contact improved progressively. Six weeks after discharge, a new paranoid episode occurred, without neurogenous relapse. She was admitted to a hospital psychiatry department and treated with loxapine with success. Folic acid was continued. Five months later, mean corpuscular volume was 93 fl, haemoglobin 13 g/dl. Her gait was normal, tendon reflexes were present except for the Achilles tendon. She refused further examinations. One year later, her state was unchanged, comparable with that before the neurological episode.

Three out of her four asymptomatic children were investigated. Their total serum homocysteine concentrations were normal and no homocystinuria was detected. In one of them, a slight decrease of MTHFR activity was found (2·48 nmol/h/mg protein). Normal values (4·96 and 5·19 nmol/h/mg protein) were found in the two others.

In this case, enzyme deficiency, relatively well tolerated, was diagnosed in adulthood. Her poor feeding in relation to her psychotic symptoms first led us to suspect a folate deficiency. Because of homocystinuria, a disturbance of folate metabolism was suspected, confirmed by a decrease of MTHFR activity to 25% of the mean control value. Freeman and colleagues[2] described the case of a mildly retarded 15 year old girl with a schizophrenia like behaviour and axonal neuropathy responsive to folic acid. This patient also stopped eating, had low serum folate, later related to an MTHFR deficiency. Botez *et al*, who reported many cases of neurological disorders associated with folic acid deficiency responsive to folate treatment, proposed the hypothesis of a vicious circle between neurological disorders and nutritional folate deficiency.[5] One pathogenic hypothesis for the role of folate in the nervous system is that reduced methionine biosynthesis[3] causes reduced biosynthesis of S-adenosyl-L-methionine, a methyl donor involved in the production of phosphatidylcholine, and consequently in myelin synthesis. A relation between homocystinuria and schizophrenia had been noted. An abnormality in folate metabolism may predispose to schizophrenia.[2]

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1 Rosenblatt DS. Inherited disorders of folate transport and metabolism. In: Scriver CR, Beaudet AL, Sly WS, Valle D, eds. *The metabolic basis of inherited disease*. New York: McGraw-Hill, 1989:2049-64.
2 Freeman JM, Finkelstein JD, Mudd SH. Folate-responsive homocystinuria and "schizophrenia". *N Engl J Med* 1975;**292**: 491–6.
3 Visy JM, Le Coz P, Chadefaux B, *et al*. Homocystinuria due to 5,10-methylenetetrahydrofolate reductase deficiency revealed by stroke in adult siblings. *Neurology* 1991; **41**:1313–5.
4 Kutzbach CA, Stokstad ELR. Mammalian methylenetetrahydrofolate reductase: partial purification, properties and inhibition by S adenosylmethionine. *Biochim Biophys Acta* 1971;**250**:459–77.
5 Botez MI, Peyromard JM, Charron L. Polyneuropathies responsive to folic acid therapy. In: Botez MI, Reynolds EH, eds. *Folic acid in neurology, psychiatry, and internal medicine*. New York: Raven Press, 1979: 401–12.

Transcallosal intraventricular tumour excision, alcohol abuse, and amnestic syndrome: a case study

Amnesia is defined as short-term and long-term memory impairment occurring in a normal state of consciousness and due to a specific organic factor (DSM-III-R). The most frequent alteration in mental activity after manipulation of midline basal cerebral structures is thought to be a transient amnesic syndrome that usually resolves within several weeks. Our patient had an intraventricular tumour (oligodendroglioma of the lateral ventricles) surgically removed with a transcallosal approach. Neuropsychological testing included evaluations completed just before the operation and one and two months later.

Case history

A 32-year-old white, right-handed male factory worker was admitted to an urban hospital with a headache, blurred vision, and ataxia. An MRI scan revealed an interventricular mass (6 × 5 cm) larger in size on the left than the right. Prior head injuries or seizures were not reported although the patient had a significant history of alcohol abuse. He had completed 13 years of schooling.

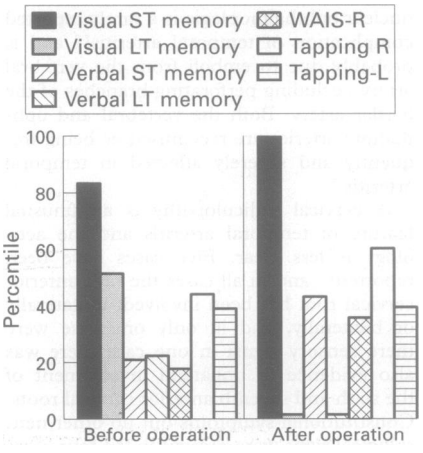
The operation involved a right frontal craniotomy and an interhemispheric, corpus callosal approach. The right frontal lobe was immobilised and a small incision was made in the corpus callosum. The tumour was removed with gentle suction from the right lateral and third ventricles. The left intraventricular tumour was removed with the same technique. The postoperative course was complicated by increased intracranial pressure for which shunts were placed. Over 90% of the tumour was removed and radiation therapy followed surgery.

BEFORE OPERATION

During his first neuropsychological assessment the patient was cooperative and attentive. Initial neuropsychological testing revealed cognitive functioning within the low average range of intelligence with significantly better performance scores than verbal scores. Fine motor skills in both hands were mildly depressed. Verbal memory with a standardised battery was within the 20th percentile (low average) and visual memory skills were at least average (figure). The discrepancy between the verbal and visual skills was consistent with the slightly better visual perceptual skills demonstrated on the intelligence test.

AFTER OPERATION

The patient's behaviour after surgery and transfer to the rehabilitation unit was striking. The patient was not generally orientated to date and was unable to give his correct age. He was found to confabulate and remote memory for significant events was inaccurate. His affect was flat and initiation of conversation limited. The patient was able to learn the location of his room with clues and reminders during his four-week stay but easily became lost in unfamiliar territory. Although the patient's physical



Testing before and after operation showed equal or superior test performance on all measures except long-term (LT) verbal and visual memory. ST = short term.

abilities quickly returned to their previous level, he continued to need cuing to initiate his self care. The patient was discharged home under 24 hour supervision of relatives.

The neuropsychological testing completed after the operation revealed average intellectual skills but slow performance on the timed visual perceptual tasks. Although his intellectual performance was significantly better than the initial testing, intelligence level was slightly below that expected given his educational history and age. The patient's slow processing on the timed visual perceptual tasks probably resulted in a decline in his intelligence score. On a problem solving task his performance was mildly perseverative and, most likely because of his memory disturbance, the patient had difficulty maintaining a strategy over time.

The neuropsychological testing also revealed a long-term memory dysfunction (figure). Although immediate recall of verbal material was more accurate than before the operation, the patient's ability to recall material after a significant delay was severely impaired. When a list of seven words was presented verbally, he was able to repeat six (immediate or working memory). When asked to recall the words after a delay, no words were recalled spontaneously, semantic clues were not helpful, and recognition memory was also found to be severely impaired.

A brief assessment took place two months after the operation. Again, the patient was not able to provide the date, his own age, or recall recent significant events. Formal testing of verbal memory again revealed a severely impaired long-term memory.

Our patient has many symptoms consistent with a diagnosis of an amnestic syndrome. He was unable to acquire new visual or verbal information efficiently and was only able to learn procedures with much repetition and numerous cues. Memory difficulties influenced his ability to perform efficiently on a problem solving task. Neuropsychological testing revealed particularly severe deficits in encoding new information and retrieval after a delay or distraction.

Testing completed before operation showed adequate long-term memory functioning, so it is unlikely that alcohol abuse itself caused pre-existing deficits. The patient's drinking history, however, may have compromised diencephalic structures so that no alternative limbic memory pathways were available after surgical intervention on fornices compressed by the tumour. This would explain the apparent contradiction between our results and Apuzzo's suggestion that the fornix is not required for normal memory functioning.[1]

This is the second published case of surgery in which a transcallosal approach was associated with the development of a non-transient amnestic syndrome as Berti *et al* also presented a patient who demonstrated amnesia after similar surgery.[2] The results from our patient indicate possible interactions between alcohol abuse and outcome from particular neurosurgical approaches.

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1 Apuzzo ML. The diencephalic and paradiencephalic regions, In: Holtzman, RN, Stein BM, eds. *Surgery of the diencephalon.* New York: Plenum, 1989, 119–40.
2 Berti A, Arienta C, Papagno C. A case of amnesia after excision of the septum pellucidum. *J Neurol Neurosurg Psychiatry*, 1990; 53:922–4.

Isolated aneurysm of a spinal radicular artery presenting as spinal subarachnoid haemorrhage

Isolated aneurysms within the vertebral canal are rare, with 11 patients reported in the literature.[1] We present a patient with symptoms and signs of spinal subarachnoid haemorrhage in whom MRI was used to localise the intradural haematoma to restrict selective spinal angiography and in whom an isolated intradural aneurysm of a spinal radicular artery was clipped successfully.

A 59 year old white woman was admitted with acute backache. She was well until the day before admission when she experienced the sudden onset of stabbing frontal headaches that subsided after five minutes and were followed by low back pain radiating up to the midthoracic level. The pain was worse with body movements. On admission she had mild meningism and an incomplete level at T7-T8 with loss of pinprick sensation spanning about three sensible dermatomes. One week later, a girdle-like pain appeared that originated in the back at the thoracolumbar level and radiated into the right upper abdomen. The girdle-like pain and the diffuse backache resolved over the next two weeks together with the meningism and signs of incomplete transection.

CT revealed intracranial subarachnoid haemorrhage but four-vessel angiography failed to show any intracranial bleeding source. A lumbar puncture confirmed the diagnosis of spinal subarachnoid haemorrhage. Spinal MRI showed a subdural haematoma at the lower thoracic levels (figs 1 and 2). Selective spinal angiography at these levels depicted a saccular aneurysm originating from the right radicular artery at T8 (fig 3).

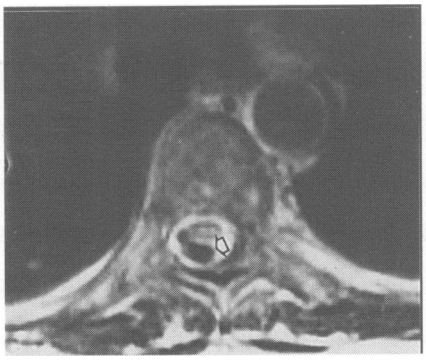

Figure 1 T1 weighted horizontal section at lower thoracic levels showing subdural accumulation of blood (arrow).

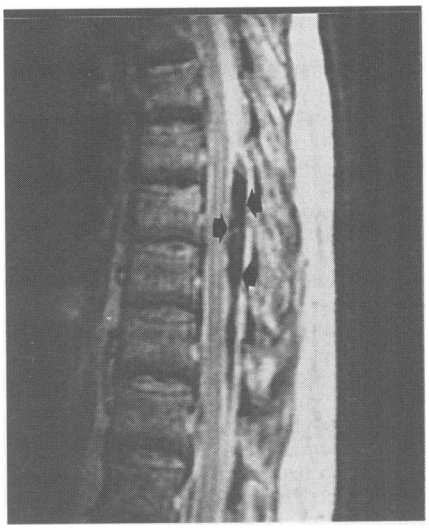

Figure 2 T1 weighted sagittal section through the spine. Note subdural haematoma (arrows).

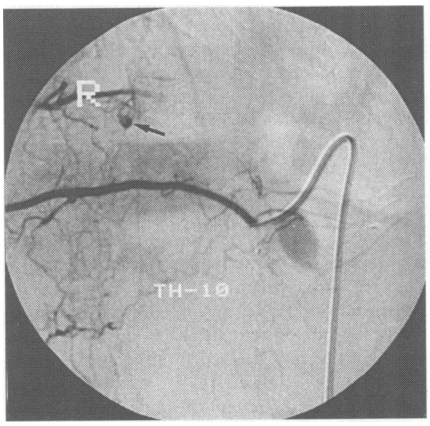

Figure 3 Angiographic demonstration of the aneurysm (arrow).

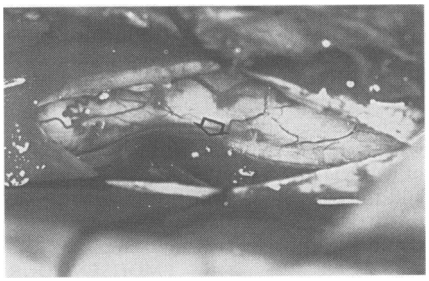

Figure 4 Intraoperative situation before evacuation of haematoma and clipping of the aneurysm (arrow).

On day 32 after spinal subarachnoid haemorrhage (13 April 1992) hemilaminectomy was performed at T8 and T9. After opening the dura, the dorsal radicular fila of nerve root T8 and the denticulate ligament appeared displaced by a saccular aneurysm of the related radicular artery measuring 5 mm in diameter (fig 4). After removal of the surrounding blood clot the spinal cord at the aneurysm site showed a yellowish discolouration. The aneurysmal sac was clipped and resected. Histological examination confirmed the diagnosis of a true saccular aneurysm with a thin fibrotic wall and organised blood adhering to the inner surface.

The patient made an uneventful recovery with complete resolution of neurological