MEDICAL MEMORANDA

Post-traumatic Progressive Cortical Thrombophlebitis

R. S. MAURICE-WILLIAMS

British Medical Journal, 1974, 3, 24

A case is reported in which several days after a relatively minor closed head injury progressive, fatal neurological deterioration set in. Necropsy showed spreading cortical thrombophlebitis originating from a small area of cerebral contusion. Some cases of progressive focal cerebral oedema and necrosis occurring after head injury might have a similar pathological basis, and if this were so the treatment would differ from that generally employed.

Case Report

A previously fit 18-year-old plumber was thrown from his motor-cycle when it collided with a car. He was unconscious for a few seconds but the period of post-traumatic amnesia was not recorded. On arrival at hospital he was fully conscious and had no abnormal neurological signs but there was a small cut on his chin and an extensive abrasion of the left forehead. Next day he was sent home. He remained well until the morning of the sixth day after the accident, when after a bath he collapsed with a generalized epileptic fit. He was immediately readmitted to hospital, where he was found to be alert but completely mute and to have slight weakness of the right limbs. Over the next few hours despite the administration of anticonvulsants and dexamethasone he had numerous epileptic fits. Some were of generalized onset and others began in the right arm and right face. His drowsiness gradually deepened and next day he was transferred to the neurosurgical unit as a case of possible subdural haematoma. During the preceding 24 hours he had had no fluids.

On examination he was found to be drowsy and mute and seemed unable to comprehend commands. His fundi were normal. There was a right lower facial weakness and a moderate right hemiparesis, more pronounced in the arm than in the leg. His temperature was 38°C and his pulse 88/min. The W.B.C. was 21,900/mm³ (81% neutrophils). A left carotid angiogram (Dr. S. H. Shah) showed a slight shift to the right of midline vessels, with stretching of the anterior and middle cerebral arteries, indicating diffuse swelling of the left cerebral hemisphere. There was no evidence of any extracerebral collection but a relatively avascular area was seen in the left frontoparietal region. It was thought that the original injury had caused a left frontal or temporal contusion and that the repeated epileptic fits due to this had led to oedema of the left hemisphere.

He was treated with large doses of anticonvulsants and dexamethasone and a restricted fluid intake. His seizures were difficult to control and when they eventually abated he had a dense right hemiplegia which showed no signs of recovery. A few hours later he developed signs of a fulminating bronchopneumonia and went into status epilepticus. He was intubated, curarized, and hyper-ventilated. His condition on the respirator remained stable but he continued to run a temperature of 39–40°C. Twelve hours later he sustained an irreversible cardiac arrest.

Necropsy (Professor Keith Simpson and Dr. Barbara Smith) showed small contusions over the outer surface of both occipital lobes and both frontal lobes. In relation to the contusion involv-

ing the central part of the left middle frontal gyrus a large cortical vein had thrombosed, and it appeared that a cortical thrombophlebitis had spread backwards from this point over most of the left hemisphere, which was dusky and swollen. The thrombosis had spread to the superior sagittal sinus and to some of the cortical veins emptying into it on the right. There was no extracerebral collection of blood or fluid.

Comment

When a patient sustains a closed head injury, recovers, and then later begins to show progressive neurological deterioration the possible diagnoses are delayed intracranial haemorrhage, cerebral oedema, and meningitis (Rowbotham, 1964). Thrombosis of the cerebral veins or venous sinuses is a rare sequel to head injury but may follow compound injuries of the superior sagittal sinus (Holmes and Sargent, 1915), traumatic osteomyelitis of the skull, or an infected scalp wound (Lewin, 1966). Spreading cortical thrombophlebitis initiated by a trivial cortical contusion in a non-compounded head injury does not appear to have been previously recorded.

More severe cerebral contusions, however, particularly at the temporal pole, often give rise to spreading cerebral swelling and dysfunction. Sometimes extensive debridement of softened and necrotic tissue is necessary to produce decompression and arrest the progression of cerebral oedema. In at least some of these cases the progression of cerebral softening could be due to cortical venous thrombosis with infarction. In such cases the optimum treatment might be adequately to hydrate the patient rather than to keep him relatively dehydrated, as is the widespread practice with fairly severe head injuries. Cerebral oedema could be controlled with large doses of steroids instead of dehydration, as is the practice with non-traumatic cerebral venous thrombosis (Kalbag and Woolf, 1972). In some cases anticoagulants might be considered and the possible benefits weighed against the risks of bleeding from contused areas, especially if certain features, present in this case, indicate a high likelihood of a cortical thrombophlebitis. These features are an unexpectedly high temperature and white cell count, intractable seizures, and worsening neurological deficit despite angiographic exclusion of a mass lesion. Classically angiography in cortical venous occlusion shows a delayed circulation, a failure of filling of affected veins and dilated venous shunts draining into the deep cerebral venous circulation (Krayenbühl, 1954). Oedematous and infarcted areas may appear as a diffuse swelling.

In the treatment of cerebral venous thrombosis there is good evidence that whatever the aetiology antibiotics and anticoagulants substantially improve the prognosis (Kalbag and Woolf, 1972; Fairburn, 1973). The outlook is better if the rate of progression of the disorder is slow, for this permits the opening up of collateral venous channels.

I should like to thank Mr. J. C. M. Currie for permission to report this case, which was treated under his care.

References

- Fairburn, B. (1973). *British Medical Journal*, 2, 647.
- Holmes, G., and Sargent, P. (1915). *British Medical Journal*, 2, 493.
- Kalbag, R. M., and Woolf, A. L. (1972). In *Handbook of Clinical Neurology*, ed. P. J. Vinkeu and G. W. Bruyn, vol. 12, p. 422. Amsterdam, North-Holland.
- Krayenbühl, H. (1954). *Archives of Neurology and Psychiatry*, 74, 261.
- Lewin, W. (1966). *Management of Head Injuries*. London, Baillière, Tindall, and Cassell.
- Rowbotham, G. F. (1964). *Acute Injuries of the Head*, 4th edn. Edinburgh, Livingstone.

St. Bartholomew's Hospital, London E.C.1

R. S. MAURICE-WILLIAMS, F.R.C.S., M.R.C.P., Senior Registrar in Neurosurgery