Medical Memoranda

Spontaneous Intracerebellar Haemorrhage

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Bleeding into the cerebellum is a common form of intracranial haemorrhage. The diagnosis is rarely made during life, though published reports indicate that the incidence is far greater than is generally appreciated. Mitchell and Angrist (1942) and Rey-Bellet (1960) reported the incidence in post-mortem studies to be 10% of all cases with intracerebral bleeding. Isolated reports of cases treated surgically have appeared in the literature, but the largest series recorded is that of McKissock *et al.* (1960), who stressed the difficulties of diagnosis and the importance of urgent treatment. The following six cases show the importance of early clinical evaluation, investigation, and urgent surgical treatment.

CASE 1

A 45-year-old woman developed sudden severe headache, vomiting, and giddiness. There was no loss of consciousness. Her symptoms progressed and about two-and-a-half months after the onset medical advice was sought. She was now admitted to the neurosurgical unit. Examination showed no papilloedema, and the pupils were equal and reacted to light. There was a mild right facial weakness and right-sided ataxia. The plantar reflexes were flexor and the blood pressure 150/110 mm. Hg. A pneumoencephalogram suggested a space-occupying lesion in the right cerebellar hemisphere. Suboccipital craniectomy was performed and 20 ml. of old clotted blood was evacuated from the cerebellum. Her postoperative recovery was uneventful and she was well and working when seen in 1960, four years after operation.

CASE 2

A 64-year-old woman developed a severe occipital headache without loss of consciousness. The headache persisted and two weeks after the onset she was admitted to another hospital, where she was noted to have neck rigidity but no papilloedema. The blood pressure was 150/100. Lumbar puncture yielded clear colourless fluid with a protein level of 15 mg./100 ml. At the end of a further week she became confused and disorientated and was transferred to the neurosurgical unit. On examination she was drowsy and restless; papilloedema was not present, but there was a mild right facial weakness and minimal weakness of the right arm and left leg with increased tone. The left plantar response was extensor. A right carotid angiogram was suggestive of mild hydrocephalus. A pneumoencephalogram suggested a high midline space-occupying lesion in the posterior fossa. Through a suboccipital craniectomy a right-sided cerebellar haematoma was evacuated. Inspection of the haematoma showed no abnormality. She made an uneventful recovery from the operation. Nine years later, at the age of 73 (May 1969), she was well and living in an old people's home.

CASE 3

A 63-year-old man collapsed with severe occipital headache about three hours before admission. He recovered rapidly, made contact with his doctor, and was sent direct to the neurosurgical unit. On arrival he was unconscious and did not respond to any stimuli. The pupils were small and did not react to light. The fundi were normal. The tone was increased in all limbs, and bilateral extensor plantar reflexes were present. The blood pressure was 280/120. Lumbar puncture showed clear cerebrospinal fluid under high pressure. A short time after his admission the left pupil increased in size. A left carotid angiogram suggested mild hydrocephalus but there was no midline shift. The right lateral ventricle was tapped through a burr-hole. The ventricular fluid was under pressure and was slightly blood-stained. This measure brought no clinical improvement. Subsequent Myodil (iophendylate) ventriculography showed non-filling of the third ventricle. An immediate suboccipital craniectomy revealed a swollen and contused right cerebellar hemisphere; a large haematoma was evacuated. The patient failed to improve and died the next day. At necropsy a massive haemorrhage was found in the right cerebellar hemisphere; this had ruptured into the fourth ventricle and had involved the brain stem.

CASE 4

A 68-year-old man with a 10-year history of depressive illness and a five-month history of weakness of the legs was admitted to another hospital after the sudden onset of headache and vomiting. On admission he was found to be dysarthric. There was a mild right facial weakness with a minimal right hemiparesis. Lumbar puncture showed clear cerebrospinal fluid at a moderately high pressure containing 70 mg. of protein per 100 ml. Over the next few days he developed papilloedema with defective conjugate deviation of the eyes to the right. The blood pressure was 160/110. A left carotid angiogram showed evidence of hydrocephalus and he was transferred to the neurosurgical unit four weeks after the onset of symptoms. An iophendylate ventriculogram was consistent with a right cerebellar hemisphere mass, which proved at operation to be a large intracerebellar haematoma, measuring 20 ml.; this was evacuated. There was no evidence of any other abnormality. Postoperatively he made a very encouraging recovery, though he still had a mild right-sided ataxia. At the time of writing he was able to look after himself and was under treatment for hypertension.

CASE 5

A 25-year-old woman was admitted to hospital on 10 March 1968. On the previous day she had complained of sudden severe generalized headache. She had been taking the "pill." She had not lost consciousness. A lumbar puncture performed at another hospital showed blood-stained cerebrospinal fluid and she was transferred to the neurosurgical unit. On examination she was restless and responded to verbal stimuli. The neck was stiff and Kernig's sign was present. No papilloedema was seen. The eyes were deviated to the right and the pupils were small and reacted to light. There was a mild left hemiparesis with increased tone in both lower limbs and bilateral extensor plantar reflexes. The blood pressure was 140/80. Bilateral carotid angiograms suggested dilatation of the ventricles. About four hours after arteriography her respirations became irregular, the pupils did not increase in size, but the light reflex became sluggish. There was no change in the level of consciousness. Ventricular catheters were inserted through bifrontal burr-holes. The ventricular fluid was clear and under considerable pressure. There was no improvement in her clinical state after the ventricular drainage. An iophendylate ventriculogram showed obstruction and shift of the aqueduct to the left. A vertebral angiogram showed a bare area in the lowermost part of the right cerebellar hemisphere. The posterior inferior cerebellar artery did not fill. At suboccipital craniectomy 25 ml. of blood clot was removed from the right cerebellar hemisphere. Inspection of the haematoma cavity showed no abnormality. The postoperative course was satisfactory. On repeat vertebral angiography the posterior inferior cerebellar artery was seen to be filling. She has since been working as a shop assistant.

CASE 6

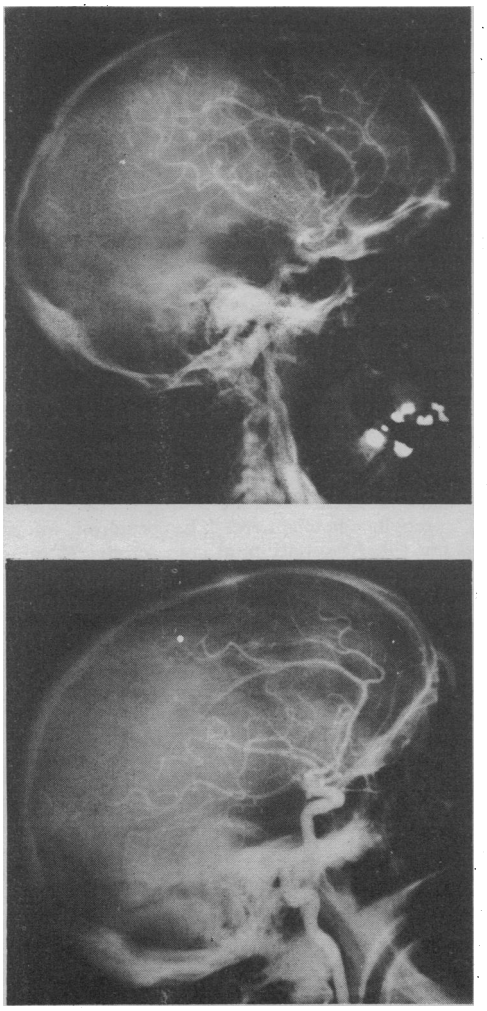
A 49-year-old man was admitted to hospital with a six-month history of bifrontal headaches associated with vomiting. Forty-eight hours before admission his headache increased in intensity and he also developed dizziness. He became drowsy and was admitted to the neurosurgical unit. On admission he was drowsy and had a blood pressure of 180/125. He had papilloedema, with slight impairment of conjugate deviation of the eyes to the left and upwards. The right arm was minimally weak with mild incoordination. Both plantar reflexes were flexor. An air ventriculogram showed slight swelling of the right cerebral hemisphere. A right carotid angiogram suggested a small superficial collection over the right parietal region, but on burr-hole exploration none was encountered. As the fourth ventricle was not seen in the air ventriculogram an iophendylate ventriculogram was

carried out and the appearances indicated a right cerebellar swelling. A vertebral angiogram showed tonsillar herniation but no vascular abnormality. Suboccipital craniectomy revealed a large intracerebellar clot in the right cerebellar hemisphere and this was removed. On inspection of the haematoma cavity no other abnormality was found. The patient made a slow postoperative recovery. At follow-up he complained of dizziness, which was controlled with Avomine (promethazine theoclate); otherwise he had remained well. At the time of writing he was receiving treatment for hypertension.

COMMENT

Vascular hypertension seems to be the most important cause of spontaneous intracerebellar haemorrhage (Hyland and Levy, 1954). According to Bailey (1948), a branch of the anterior superior cerebellar artery supplying the dentate nucleus is often implicated. A pathogenetic correlation of the anatomical sites of microaneurysms with those of spontaneous intracranial haemorrhage in hypertension has been shown (Russell, 1954, Cole and Yates, 1967). About 13% of these microaneurysms occur in the cerebellum and the incidence of spontaneous haemorrhages is around 10%, which is quite a close correlation. The possibility of a haemorrhagic infarct cannot be excluded. Wood and Murphy (1969) recently reported 10 cases of cerebellar infarction in which the clinical picture resembled that of a haemorrhage. A variety of other causes, such as hamartomas, arteriovenous malformations, and aneurysms, may be responsible for the bleeding.

The history is typically that of sudden onset of severe generalized headache, vomiting and confusion, with or without subsequent loss of consciousness. Loss of consciousness occurred in only one of the six cases reported above. There may be other signs of subarachnoid haemorrhage, such as neck rigidity. The cerebrospinal fluid may or may not be blood-stained. The presence of blood in the cerebrospinal fluid would indicate rupture of the haematoma into the subarachnoid space or the fourth ventricle. The fluid was clear in all but Case 5, but Cases 1, 4, and 6 had lumbar punctures several days after the onset, the protein content showing a moderate increase in Case 4. Patients may also show signs of brain-stem involvement such as miosis, impairment of conjugate movements of the eyes, and irregular respiration. This triad has been found with great frequency in intracerebellar haematomas. These signs may develop immediately or may appear only after hours or days (Cases 4 and 5).



FIGS. 1 and 2.—Carotid angiography showing widely swept anterior cerebral artery in lateral views.

The history and ocular signs in most cases will help to distinguish the cerebellar haemorrhage from pontine haemorrhage, brain-stem infarction, and cerebral haemorrhage. About 80% of cases survive the acute episode (McKissock *et al.*, 1960). Some patients may present with symptoms and signs referable to cerebellar dysfunction. A careful history will usually elicit the sudden onset. Michael (1932) and McKissock *et al.* (1960) defined three major groups according to the mode of onset and the clinical course. These divisions represent the various stages in the clinical course of the pathological process.

Carotid angiography is usually the first investigation to be carried out because of the history. The anterior cerebral artery is fairly wide-swept, suggesting early hydrocephalus (Figs. 1 and 2), which may be seen within a few hours of the haemorrhage (Cases 3 and 5). I believe this to be an angiographic sign of great significance, though earlier reports have failed to stress its importance. The early hydrocephalus together with the clinical history is very suggestive of the presence of an intracerebellar haematoma. An iophendylate ventriculogram or pneumoencephalography, depending on the general condition of the patient and presence or absence of papilloedema, is the most helpful investigation. Vertebral angiography will usually exclude an arteriovenous malformation or an aneurysm as the source of the bleeding.

Intracerebellar haemorrhage is a surgical emergency. Prompt action is necessary to diagnose and evacuate the haematoma. Aspiration of the clot through a burr-hole may not be possible, as the haematoma is usually solid. In acute cases ventricular drainage does not seem to help (Cases 3 and 5), as the symptoms and signs are due to brain-stem distortion. In fact, this manoeuvre may prove dangerous because of the tendency to upward herniation of the cerebellum. The reported surgical mortality is fairly high (36% McKissock *et al.*, 1960) but increasing awareness of this condition and prompt surgical intervention will enable the mortality and morbidity rates to be reduced. Probably the most important step to be taken in the diagnosis is to assess the possibility of bleeding from a microaneurysm into the cerebellum.

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