Fatal cerebral infarction simulating an acute expanding lesion

LOUISE VAN TROTSENBURG AND PIERRE J. VINKEN

From the Neurological and Neurosurgical University Clinics, Wilhelmina-Gasthuis, Amsterdam, the Netherlands

A number of years ago, two papers were published about a few cases of a little known cerebrovascular syndrome (King, 1951; Scarcella, 1956). In these, cerebral vascular occlusion had caused an increase in intracranial pressure which, in the course of four to six weeks, led to clinical and radiological features simulating those of brain tumour. In 1964, we saw two patients in whom cerebrovascular infarction likewise resembled a hemispheric expanding lesion, but unlike the previously published cases with a subacute course, these patients died within a few days. The present paper examines the clinical differentiation of these forms of cerebral infarction.

CASE REPORTS

CASE 1 A woman aged 49 noticed a tingling sensation in the right arm in the evening of 29 December 1964, which soon disappeared; later in bed, she could read the newspaper and hold it with arms extended. During the night her husband awoke because she was making jerking movements. He found her unconscious, with foam on her mouth, which was asymmetrical. The family doctor administered Luminal, whereupon the patient remained quiet. On 30 December, according to the husband, reasonable contact could be established with the patient, but she did not speak. On 31 December it was noticed that the right arm was held in extension and that the left pupil was larger than the right, and therefore a neurologist was consulted in the afternoon of that day.

The neurologist found her unconscious with a right-central hemiparesis and increased reflexes. The left pupil was larger than the right and both failed to respond to light. The cerebrospinal fluid was clear and colourless, containing 23 cells per c.mm. (nearly all polymorphs, no red cells) and 60 mg. protein per 100 ml. The provisional diagnosis of intracerebral haemorrhage was therefore questioned. The serological test for syphilis was negative. The patient was admitted to our clinic because an expanding intracranial lesion was suspected.

On admission she showed Cheyne-Stokes respiration with long periods of apnoea. There was neck rigidity, extension of the right arm and leg, and bilateral Babinski reflexes. The carotid angiograms showed marked displacement to the right of the anterior and middle cerebral arteries. A positive falx sign was seen. The deep

veins were displaced more than a centimetre from the midline. The antero-posterior projection also showed that, along a few centimetres of its curve, the middle cerebral artery was only partly filled with contrast medium. In the lateral projection this artery showed some upward displacement; the same projection showed that its parieto-temporal branch was only partly filled for a few centimetres from its origin, but it showed normal filling more distally. This relative filling defect was not sufficient to warrant the diagnosis of complete vascular occlusion. All signs indicated that in addition to a partial vascular occlusion the patient had a fairly large expanding lesion in the left temporal lobe. On the basis of these findings we decided to operate, in the evening of 31 December.

Operation There was severe cerebral oedema in spite of a 30% urea infusion. The cerebrum was very soft and few vessels, particularly arteries, were visible. There was perivascular discoloration caused by extravasated blood. The ventricular cannula easily sank into the cerebrum under its own weight. No tumour or haemorrhage was found. Decompression was effected by removing the bone flap.

Post-operative course On the day after operation (1 January 1965) the patient was still comatose but the right-sided paresis was less severe. The reaction to pain had improved, the pupils had contracted, and the right one responded to light. On 3 January there was again rapid deterioration, with bilateral extensor spasms and fixity of the pupils. Another urea infusion led to transient improvement but the patient died in the course of that day.

Necropsy disclosed oedema throughout the left hemisphere, and a very large tentorial hernia on that side. The left middle cerebral artery contained a thrombus. The frontal sections revealed haemorrhagic infarction in the region supplied by the middle cerebral artery.

In summary, this report describes the fatal case of a woman aged 49 who initially showed the clinical features of a cerebrovascular accident. In the course of two days, however, there were progressive signs of tentorial herniation apparently caused by an expanding lesion in the left hemisphere. Operation and post-mortem examination disclosed massive infarction in the region supplied by the middle cerebral artery.

CASE 2 A woman aged 44 was unable to speak when she awoke on 11 February; although she could open her eyes she did not respond when addressed and appeared drowsy. She was admitted to our clinic on the same day in a comatose state but still showing reaction to pain. There was a left central hemiparesis with hypertonia, increased reflexes, and bilateral Babinski responses. The midline echo was displaced 3 mm. to the left. The cerebrospinal fluid was clear and colourless and contained 1 lymphocyte per c.mm.

She had an epileptic seizure in the early morning of 12 February; at 5.30 a.m. the right pupil was dilated and neither pupil responded to light. There was Cheyne-Stokes respiration, with extensor spasms. The carotid angiograms showed occlusion of the right middle cerebral artery and a right frontal expanding lesion. Operation was performed immediately and an infusion of 30% urea was given.

Operation The cortex in places was slightly speckled with small haemorrhages. There was oedema and the cortical arteries were thin and pale. No tumour or haematoma was found. The bone flap was removed and the dura closed. The patient died on the same day.

At necropsy a thrombus was found in the right middle cerebral artery; there was severe cerebral oedema with tentorial herniation and softening in the area supplied by the middle cerebral artery.

In summary, this report describes the fatal case of a woman aged 44 who presented the clinical features of an intracranial haemorrhage (*e.g.*, from a vascular anomaly), but the lumbar cerebrospinal fluid was normal. In the course of a single day she developed the syndrome of tentorial herniation due to an expanding lesion on the right. Operation and post-mortem examination disclosed cerebral infarction in the territory of the middle cerebral artery.

DISCUSSION

In both cases, an experienced neurologist initially diagnosed intracerebral haemorrhage of uncertain aetiology. In both cases the preliminary diagnosis was abandoned when lumbar puncture yielded clear and colourless cerebrospinal fluid, containing no red cells even on microscopy. Both patients rapidly developed increased intracranial pressure with the syndrome of tentorial herniation, which was another argument against a cerebral thrombosis and in favour of an expanding lesion of the hemisphere. Echo-encephalography (case 2) and carotid angiography confirmed the presence of an expanding lesion in each case. Operation and post-mortem examination revealed the diagnosis of cerebral infarction with massive, predominantly unilateral, cerebral oedema and severe tentorial herniation.

The literature seems to contain no reports of what may be called 'acute cerebral infarction', as illustrated by the above cases. We found only one

comparable case, with a similar clinical course and similar, well-defined pathological findings. It was described by Nedwich, Haft, Tellem, and Kauffman (1963), and concerned a woman of 30 who died within three days of acute cerebral infarction caused by subintimal dissecting haemorrhage in the right middle cerebral artery. Another case somewhat resembling ours was described by Duman and Stephens (1963). It concerned a man aged 21 who complained of headache and lost consciousness a short time after a fall sustained while water skiing. In hospital he was found to have a hemiplegia. The carotid angiogram disclosed occlusion of the middle cerebral artery at its origin. The patient remained unconscious and showed signs of brain swelling and decerebrate rigidity. His condition deteriorated and he died two weeks after his accident. At post-mortem examination a dissecting aneurysm was found at the origin of the middle cerebral artery.

These two cases resemble ours in that fatal infarction with tentorial herniation occurred after a cerebral vascular occlusion. They differ from our cases in aetiology; in ours we found thrombosis of the middle cerebral artery but no apparent cause. The case of Duman and Stephens (1963) differed from our cases not only in aetiology (trauma) but also in its less acute course.

The occurrence of cerebral oedema following cerebral vascular occlusion is a well known phenomenon, but the exact mechanism remains obscure. In this connexion the work of Shaw, Alvord, and Berry (1959) is of great importance. From extensive pathological material he established that the formation of oedema is part of the process of acute cerebral infarction, and reaches a maximum about the fourth day and recedes about the seventh day. More important is their finding that the patient's clinical condition is determined by the early tentorial herniation rather than by lateral displacement of the hemispheres. This tentorial herniation occurs even before a shift of the midline structures is demonstrable, and reaches a maximum at an early stage. These facts are of great importance in differential diagnosis.

The clinical picture of subacute cerebral infarction as described by King (1951) and later by Scarcella (1956) was not determined by tentorial herniation. The case of Duman and Stephens occupies an intermediate position; while the course was subacute (two weeks), the clinical picture was that of decerebration due to tentorial herniation.

The duration of the clinical course is important in the differential diagnosis. The course of the subacute cases is reminiscent of that of brain tumour. The acute onset of symptoms and the rapid course of acute infarction suggest a cerebrovascular accident, especially a haemorrhage, but the absence of blood

TABLE I

DIFFERENTIAL DIAGNOSIS

	<i>Acute Cerebral Infarction</i>	<i>Subacute Cerebral Infarction</i>	<i>Cerebral Haemorrhage</i>	<i>Brain Tumour</i>
Age (yr.)	30-50	50-70	All ages	All ages
Duration	Days	Weeks	Days-weeks	Weeks-months
Clinical picture	Like that of cerebral haemorrhage	Like that of tumour	—	—
Papilloedema	No	Yes	Sometimes	Sometimes
Cerebrospinal fluid	No erythrocytes, may contain white cells	No erythrocytes, may contain white cells	Erythrocytes or frank blood	No erythrocytes, may contain white cells
Tentorial herniation (mydriasis etc.)	Early	Late	Early or late	Late
Pneumo-encephalography	Lateral shift	Lateral shift	Lateral shift	Lateral shift
Carotid angiography	Marked lateral shift; occlusion of vessel may be visible	Less marked lateral shift; occlusion of vessel may be visible	Lateral shift may be present	Lateral shift; no occlusion of vessel; sometimes abnormal vessels or tumour stain; displacement of vessels
At operation	Pale cortex, deficient filling of cortical arteries; bluish perivascular extravasation; massive oedema; no demarcation; no necrosis	Yellowish cortex; deficient filling of arteries; no oedema; demarcation; necrosis	Haemorrhage	Tumour
At necropsy	Haemorrhagic infarction; marked tentorial herniation; mild or absent pressure conus	Softening and haemorrhage	Tentorial herniation	Tentorial herniation; pressure conus

in the cerebrospinal fluid may suggest other possibilities, such as tumour.

The patients with the subacute condition described by King (1951) and Scarcella (1956) were generally older than our patients and older than the other two patients with acute infarction described in the literature. It might be suggested that the brain in young people more readily develops oedema, a suggestion which is perhaps supported by the more severe oedema after brain injury in children than in adults. But there are cogent arguments against this theory. In post-traumatic disorders of the cerebral circulation in children, signs of increased intracranial pressure are exceedingly rare. Also, statistical data indicate that the prognosis of cerebral thrombosis under the age of 50 is better than over that age. Animal experiments have shown, however, that the prognosis of cerebrovascular occlusion does not depend on age.

Table I, which summarizes the differential diagnostic features from various pertinent disorders, shows that the chief characteristic of acute infarction is the rapidly developing tentorial herniation syndrome in the absence of papilloedema. The table also shows that radiological procedures contribute little to the diagnosis of cerebral infarction. Useful treatment in these cases is limited but it should

probably include infusion of hypertonic solutions in an early stage, followed if necessary by surgical decompression and division of the tentorium.

SUMMARY

A description is given of two cases of what may be called acute cerebral infarction, leading to a fatal issue within a few days. In these cases the clinical picture was characterized by rapidly developing signs of tentorial herniation in the absence of papilloedema. This clinical picture is differentiated from that of subacute cerebral infarction, intracerebral haemorrhage, and brain tumour.

REFERENCES

- Duman, S., and Stephens, J. W. (1963). Post-traumatic middle cerebral artery occlusion. *Neurology (Minneapolis)*, **16**, 613-616.
- King, A. B. (1951). Massive cerebral infarction producing ventriculographic changes suggesting a brain tumor. *J. Neurosurg.*, **8**, 536-539.
- Nedwich, A., Haft, H., Tellem, M., and Kauffman, L. (1963). Dissecting aneurysm of cerebral arteries. *Arch. Neurol. (Chic.)*, **9**, 477-484.
- Scarcella, G. (1956). Encephalomalacia simulating the clinical and radiological aspects of brain tumor. A report of 6 cases. *J. Neurosurg.*, **13**, 366-380.
- Shaw, C., Alvord, E. C., and Berry, R. G. (1959). Swelling of the brain following ischemic infarction with arterial occlusion. *Arch. Neurol. (Chic.)*, **1**, 161-177.