CAROTID ANGIOGRAPHY AND CEREBRAL ABSCESS

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The clinical diagnosis and localisation of a cerebral abscess is sometimes a simple matter. Unfortunately this is not always so, and in many cases the neurosurgeon will need all the information that can be provided by electroencephalography and various radiological techniques. The possible hazards of ventriculography, and more especially of lumbar encephalography, are well recognised, and the information gained is sometimes limited.

In recent years carotid angiography has largely replaced air studies as the investigation of choice, and we have found this method very valuable. This paper, however, describes 3 patients with proven cerebral abscesses, in whom carotid angiography was seriously misleading.

CASE REPORTS

Case 1. H.P., a boy aged 15 months, was admitted to the Cardiff Royal Infirmary on March 30, 1961. Two weeks previously a left hemiplegia developed which in 24 hours became almost complete.

He was first admitted to another hospital, where lumbar puncture revealed cerebrospinal fluid containing less than 1 cell per c.mm. and 30 mg. of protein per 100 ml. Four days later purulent meningitis developed with a temperature of 103.5°F. The lumbar cerebrospinal fluid at that time contained 26,000 white blood cells, mainly polymorphonuclear, and 280 mg. of protein per 100 ml. Pneumococcus was later cultured from this fluid.

He was treated with systemic antibiotics, Sulphaadiazine, penicillin, streptomycin, and Chloramphenicol, but the cells in the cerebrospinal fluid did not fall below 500 per c/mm. and after an initial improvement his clinical state deteriorated. During this period a systolic murmur was noted in the 3rd left intercostal space and this became steadily louder.

On the day of his admission here, under general anaesthesia, right carotid angiography was performed with 35 per cent Hypaque. The resulting roentgen-ray films showed very satisfactory filling of the cerebral vessels, and there was no abnormality to be seen (Figs. 1 and 2).

The electroencephalogram showed high-voltage slow activity in all leads but no sustained focus.

After 3 more days of treatment with antibiotics, including intrathecal penicillin, there was little change in his condition and therefore on April 2, 1961 exploratory burr holes were made under general anaesthesia. The right lateral ventricle was found to contain frankly purulent fluid with 34,240 white blood cells per c.mm., whereas in the lumbar cerebrospinal fluid the count of cells had been steadily falling, and on that day was only 170 white blood cells per c.mm.

He was then treated with intraventricular penicillin in addition to systemic drugs, and thereafter he steadily improved. The systolic murmur disappeared about 3 weeks after admission.

On April 10, 1961 air ventriculography revealed a mildly dilated ventricular system, with an air-filled "diverticulum" extending laterally from the body of the right lateral ventricle (Figs. 3 and 4). This abnormal cavity measured approximately 2.5 × 1 cm. and presumably corresponded to an abscess which had ruptured into the ventricle.

Four months after the illness, at the age of 19 months, he was well, but had a mild left hemiplegia affecting mainly the hand.

We believe that this was a metastatic cerebral abscess, possibly associated with a bacterial endocarditis.

Case 2. E.D., a man aged 62 years, was admitted on Aug. 16, 1960. His left ear had discharged intermittently since childhood, hearing was impaired on that side, and for 4 days he had suffered from pain in that ear.

Two days after admission lumbar puncture yielded cerebrospinal fluid containing 88 polymorphonuclear cells per c.mm. During the course of a radical mastoidectomy on that side an area of dura mater in the middle fossa was found to be exposed.

On Aug. 22, 1960 right-sided convulsions began and spread to become a generalised status epilep-
ticus. This episode was controlled with anticonvulsant drugs but from that time he was dysphasic. On Aug. 23, 1960 electroencephalography showed a focus of delta waves in the left frontal region, and a spike focus in the left temporal region.

On the same day left carotid angiography was performed, and these films showed no abnormality. He was treated with penicillin and streptomycin systemically and for a time his condition remained unchanged.

On Sept. 1, 1960 the cells in the cerebrospinal fluid had risen to 450 per c.mm., and during the succeeding days his state of consciousness deteriorated.

On Sept. 5, 1960 a left posterior temporal burr hole was made and the underlying brain was explored by needling. A small abscess was discovered at a depth of 3.5 cm. and 7 cc. of pus were aspirated. At about this time a suppurative ventriculitis developed which failed to respond even to vigorous chemotherapy. He died on Sept. 17, 1960.

Case 3. P.B., a girl aged 9 years, was admitted on Aug. 17, 1959. This child had a subdural abscess in July 1958. She had been well previously and no cause for this abscess was found. Later multiple cerebral abscesses developed in the left frontal and left parietal regions. These were needled and drained, and were later drained more completely through a formal craniotomy. The first specimens of pus grew a Staphylococcus aureus.

In May, 1959 osteomyelitis had developed in the bone flap, and some sequestra were removed. She was then mildly dysphasic, there was some weakness of her right arm, and she had a right homonymous hemianopia.

During August 1959 she suffered from headache...
and vomiting. She was readmitted on Aug. 17, 1959. She was then alert, and the neurological abnormalities had not changed, but her small cranial defect was tense. Lumbar cerebrospinal fluid contained 280 mg. of protein per 100 ml. though the cell count was normal. Electroencephalogram showed widespread slow waves, predominantly from the left hemisphere, with spikes in the left parietal, left temporal and right occipital areas. During the succeeding days she became more drowsy, and her temperature rose to 100°F.

On Aug. 22, 1959, under general anaesthesia, left carotid angiography was performed with 35 per cent Hypaque. The roentgen-ray films showed stretching of the pericallosal artery in a manner characteristic of hydrocephalus. The vascular pattern otherwise was normal (Figs. 5 and 6).

Two days later ventriculography was carried out. The ventricular fluid was found to be turbid, and it contained 3,500 white blood cells per c.mm. Roentgenograms showed that the posterior part of the left lateral ventricle had failed to fill with air, and a rounded mass could be seen bulging into the ventricle (Fig. 7).

This mass proved to be an intracerebral abscess containing 35 cc. of pus. This was treated by repeated needling, and antibiotic drugs were introduced into it as well as contrast medium. She also was treated with systemic and intrathecal antibiotics, but she deteriorated further and died on Sept. 4, 1959.

**DISCUSSION**

The detection and localisation of a cerebral abscess by angiography or by ventriculography depend upon the distortion or displacement of anatomical landmarks, be they vascular or ventricular.

The occurrence and extent of the displacement of cerebral vessels depend not only on the size of the lesion, but also on its site within the hemisphere. In many parts of the hemisphere an abscess with its surrounding zone of oedema will distort vessels of the carotid system at an early stage. The more deeply placed lesions, however, and those in the more posterior part of the hemisphere, are in general less easily demonstrated by angiography than those that lie superficially or more anteriorly.
It is clear from our 3 cases that a patient may harbour a localised collection of intracerebral pus with a normal carotid angiogram.

The authors suggest that the abscess most likely to be overlooked on a carotid angiogram is the particularly dangerous one that lies deep in the hemisphere close to the ventricular wall. Such an abscess may rupture into the cavity of a ventricle before it has become large enough to produce angiographic changes (our Case 2). After it has emptied its contents into the ventricular system (our Case 1) it will be even less likely to distort the vascular pattern. In our Case 3 the abscess projected into the cavity of the ventricle (already enlarged), so that it reached quite a large size without further distorting the brain tissue and its vessels.

We feel that these 3 cases and 1 similar to our Case 1 described by Brzezinski illustrate the danger of relying too much on the result of a single investigation in the assessment of these difficult problems.

SUMMARY

Three cases of proven cerebral abscess are described with normal carotid angiograms. Carotid angiography is least likely to demonstrate a cerebral abscess when (1) the lesion lies posteriorly in the hemisphere; (2) the lesion lies in the deeper part of the hemisphere; and (3) the lesion projects into or has ruptured into a cerebral ventricle.

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REFERENCES