CEREBRAL ARTERIOVENOUS ANOMALY ASSOCIATED WITH AN IPSILATERAL CAROTID ARTERY OCCLUSION

A CASE REPORT

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We wish to present an interesting case report of a patient with a left-sided cerebrovascular anomaly associated with occlusion of the left internal carotid artery, causing gradual anoxia of the left cerebral hemisphere.

CASE REPORT

History

The patient, a right-handed African Zulu male, aged 33 years, was admitted to our unit on 5 December 1961, with the following history:

He was quite well until one year before when he had his first generalized epileptic attack, and since then he had had 6 further fits. Following the first fit, he noticed that his right arm and leg were weak. This weakness gradually became worse, so much so that about two weeks before admission he had become bedridden, being totally unable to use his right arm or leg.

About six months after the fits started he noticed that he was having difficulty with his speech, and his family stated that for three weeks before admission he had not spoken at all and had been unable to understand them or to make himself understood.

He had never complained of headache, diplopia, dizziness, or noises in the head.

He had not lost weight and his appetite was good. He had never had any previous neurological illnesses, and on looking into his previous medical history, nothing of significance was found.

Neurological Examination (15 December)

Mental state. The patient appeared alert, though at times he tended to fall asleep, but when he had done so he was easily rousable. He had an almost complete receptive and a total expressive aphasia, which made a detailed neurological examination difficult.

Skull. No bruit was heard and no other abnormality was detected.

Spine. No neck stiffness or other abnormality was detected.

Cranial nerves. The fundi appeared normal, but the veins on the left were possibly slightly more distended than those on the right. The visual fields were difficult to test, but appeared to be full. The external ocular movements were normal and there was no obvious squint. The pupils were equal and reacted briskly to both light and accommodation. He had hypoaesthesia of the right side of the face, and an upper motor neurone paralysis of his right facial muscles. The rest of his cranial nerves, as far as could be ascertained, appeared to be normal.

Limbs. The patient had a right spastic hemiplegia with a marked increase of the deep reflexes on that side. The right abdominal reflexes were absent and the right plantar reflex was extensor. He had a right-sided hypoalgesia. He was not cooperative enough to allow the testing of joint position sense.

General Examination

A well-built adult African male; blood pressure 110/60 mm.Hg. Pulse rate 83 per minute, and respirations 21 per minute. His heart, lungs and abdomen appeared to be normal.

Investigations

X-ray. X-rays of heart and lungs were normal, and X-ray of the skull failed to show any abnormality.

Cerebrospinal fluid. Lumbar puncture was performed on 1 December (before admission to this hospital), and a pressure of 300 mm. H_2O was recorded, while examination of the cerebrospinal fluid (CSF) showed 1 lymphocyte per c.mm., a protein level of 46 mg. per 100 ml., sugar 55 mg. per 100 ml. and chlorides 670 mg. per 100 ml. The CSF Wassermann reaction was negative. The lumbar puncture was repeated 9 days later and the pressure as recorded then was only 170 mm. H_2O, while the CSF findings were very similar to those of the previous occasion.

Cerebral angiography. This was performed on 7 December, with the following results:

(*a*) Left carotid injection with 'urografin' revealed a total block of the internal carotid artery at the level of the second bend of the carotid syphon. This blockage was constant on repeated injections (Fig. 1).

(*b*) Right carotid injection showed a perfectly normal right-sided cerebral vascular tree, as well as an excellent cross-circulation to the left hemisphere *via* the anterior communicating artery. The left anterior cerebral artery filled, and the rest of the contrast medium flowed into the left middle cerebral artery and thence, *via* three feeding arteries, into a very large arteriovenous malformation (Figs. 2 and 3).

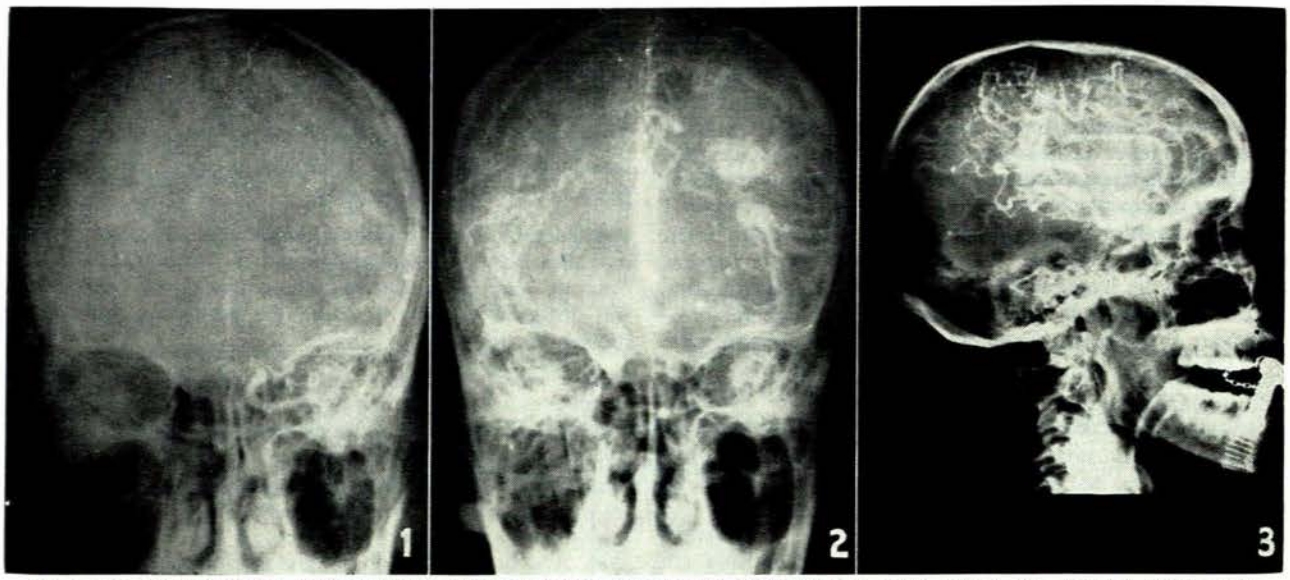

Fig. 1. Left carotid angiogram showing occlusion of the left internal carotid artery in the first part of the syphon. *Figs. 2 and 3.* Right carotid angiogram, showing filling, *via* the anterior communicating artery, of the vessels and the anomaly in the left cerebral hemisphere (Fig. 2 — AP view, Fig. 3 — lateral view).

Operation

This was performed on 12 December. After turning a large left parietal flap, the dura was opened and the three main feeding arteries were clipped just beyond the Rolandic fissure.

Postoperative Course

The patient's postoperative course was smooth and he began to speak within the first 2 weeks after the operation.

An examination in July 1962 showed that, though his speech was still slow and articulation errors were frequently evident, his counting, singing and rhymes were good and he was able to do difficult addition, subtraction and multiplication sums. His writing was still poor, but actual recognition of the written language was quite fair. He perseverated very little, and when this did occur he recognized his error. Nevertheless, he could make himself understood easily and lucidly, not only in his home language (Zulu), but in English as well.

His right leg improved rapidly and on the sixth postoperative day we began to get him out of bed. At the end of the second postoperative week he was walking about, and by the middle of January 1962 he had a barely perceptible limp. At present (July 1962) his facial weakness has disappeared completely, but the fine finger movements of his right hand are very slow and the arm is still spastic.

Joint sense is normal in his toes, but impaired in his right hand. He still has a mild sensory depression to pin-prick on the whole of his right side.

Postoperative Angiogram (1 March 1962)

There is a marked diminution in the circulation through the anomaly, which now fills only partially in the intermediate capillary and venous phases. The left carotid artery still shows an occlusion at exactly the same level.

COMMENT

This patient posed some interesting problems:

Firstly, there was the occlusion of the left internal carotid artery. Was this a thrombosis which occurred recently, i.e. when he had his first fit, or was it an older lesion?

Secondly, the failure of the blood supply from the right side to supply the left hemisphere *via* the anterior communicating artery. This was probably due to a slowly progressive opening up of the circulation throughout the arteriovenous anomaly. As this occurred, more and more blood flowed through the anomaly; consequently the anterior communicating artery (and possibly the posterior communicating artery) was unable to cope with supplying this extra load, and the left hemisphere gradually became anoxic. This was shown by the progressive nature of the signs and symptoms pre-operatively, and the dramatic postoperative recovery.

Finally, from an operative point of view, though we personally believe that on the whole it is better to remove this anomaly than just to clip the feeding vessels, we were loath to remove it in this particular case. Our reasons were that the anomaly was lying right in the middle of the dominant parietal lobe and, as far as we knew and from what we could see at operation, it had never bled.

At any stage in the future, should this patient deteriorate owing to the re-opening of the circulation through the lesion, we shall probably be forced to try to remove the whole arteriovenous anomaly.

SUMMARY

A case of progressive anoxia of the left cerebral hemisphere caused by an arteriovenous anomaly is presented. This anoxia was further increased by an occlusion of the internal carotid artery.

We wish to thank Dr. I. Frack, Superintendent of Baragwanath Hospital, for permission to publish the above case. Our thanks are also due to the photographic section of the Department of Surgery, University of the Witwatersrand.

ADDENDUM

The patient when last seen, on 7 March 1963, was still well and had improved slightly.