CASE REPORT

SIMULTANEOUS OCCURRENCE OF INTRACRANIAL ANEURYSM AND ANGIOMA

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Although both cerebral aneurysms and arteriovenous malformations may have a common congenital basis, their simultaneous occurrence is a rarity.1 Vieten2 illustrated such a case in 1955, in which a 46-year-old male, with two episodes of subarachnoid hemorrhage, was found to have a large saccular aneurysm of the left middle cerebral artery and an arteriovenous malformation in the left occipital area, both verified at surgery. Paterson and McKissock3 described 4 patients who had intracranial arterial aneurysms in addition to an arteriovenous malformation (angioma); one had a right parieto-occipital angioma and a right carotid-cavernous sinus fistula; another, a left Sylvian-point angioma and multiple small aneurysms on the left anterior and middle cerebral arteries; a third patient had a right parietal angioma and two aneurysms on a dilated right internal carotid artery; and the fourth patient had a left cerebellar angioma and a ruptured aneurysm on the right posterior inferior cerebellar artery. None of these patients was operated upon.

Recently, a 26-year-old white male was found to have an aneurysm of the anterior communicating artery and an arteriovenous malformation of the left anterior temporal and inferior frontal area; no surgery was attempted. Because of the paucity of such cases in the literature, this case is being reported.

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V.A. #54175. J.Q.J., a 26-year-old white male, was admitted to the Veterans Administration Hospital on April 14, 1955 with headache, nausea and vomiting of 8 days' duration.

He had been in good health until 1944, 11 years prior to admission, at which time, while in the Navy, there suddenly developed inability to speak. He recovered speech following several Pentothal interviews and was given a medical discharge.

Starting soon thereafter, he experienced dizzy spells, during which he felt light-headed, had difficulty in keeping his balance, but did not fall. These episodes occurred twice a week, and were followed by a severe throbbing headache and, in the last 3 years, by thickened speech.

Seizures were first noted 3 years prior to admission and persisted despite Dilantin and Phenobarbital. A typical seizure might occur while he was eating or drinking, when he would suddenly stare into space, make smacking movements of his lips, salivate, but never vocalize. Occasionally his mouth would tend to draw to the left and his right arm would be held firmly flexed. These episodes would last approximately 1–2 minutes, with the patient having no memory for the event. His activity would continue normally thereafter.

He was hospitalized briefly in June 1953, where work-up, including roentgenograms of the skull and lumbar puncture, was normal, except for electroencephalographic findings (to be discussed subsequently).
For the year prior to the present admission, he noted increased irritability, with temper tantrums, as well as impairment of memory; he was forced to stop work. During the last 3 months, he had occasionally heard voices talking to him.

Eight days prior to admission, he suddenly experienced headache, nausea and vomiting. The headaches were described as sudden in onset, frontal and suboccipital, and quite severe. He was followed at home for several days with sedation and analgesics without relief of pain. Three days before admission, marked nuchal rigidity was noted and a lumbar puncture revealed bloody cerebrospinal fluid. Six generalized seizures occurred in the last 2 days.

Examination. He was a well developed, right-handed white male, extremely hostile, demanding attention, and having attacks of rage. Blood pressure was 110/60, pulse rate 72, and temperature 98.8°F. There was definite nuchal rigidity, with positive Kernig and Brudzinski signs. The myotatic reflexes were hyperactive, but no pathological reflexes were elicited. Bilateral papilledema was noted.

Laboratory data were within normal limits, except for findings on lumbar puncture. The cerebrospinal fluid was bloody, with an initial pressure of 600 mm. water.

Course. During the subsequent 10 days, the patient gradually improved, with subsidence of headache, nuchal rigidity, and papilledema. Repeated lumbar punctures showed improvement in the character of the fluid and reduction in pressure.

On April 25, 1955 bilateral carotid angiography was performed under local and Surital anesthesia. This revealed an angioma (Fig. 1) of the left temporal lobe, surrounding the first portion of the middle cerebral artery, and extending into the inferior frontal region; there was a large vessel draining medially. In addition, an aneurysm of the anterior communicating artery, filling primarily from the right side, was noted (Fig. 2). Surgery of the temporal malformation was thought to be contraindicated because of the extension into the frontal area, as well as its occurrence in the dominant hemisphere, with little likelihood of total excision. Attack on the aneurysm of the anterior communicating artery, without attacking the other malformation, particularly since the immediate source of the bleeding was in doubt, also was considered unwise.

Serial electroencephalograms (May 8, 1953, May 19, 1953, April 15, 1955 and May 3, 1955) all revealed an irregular, slow-wave focus of about 3–6 per sec., with some low-voltage base-line shifting and sharp-wave activity, in the left temporal region. In the most recent record, in addition to the sharp-wave discharges in the left temporal lead, there were independent discharges of similar nature in the right temporal lobe. The electroencephalograms were thought to provide definite evidence for both psychomotor and grand mal seizures.

Fig. 1. Left anteroposterior and lateral angiogram. Angioma in left temporal and inferior frontal areas; aneurysm of anterior communicating artery shows in anteroposterior view only.
During the remainder of hospitalization there was continued clinical improvement on anticonvulsive medication, with complete control of seizures and of attacks of rage, as well as marked improvement in personality. He was discharged on May 20, 1955.

**DISCUSSION**

It is interesting to speculate upon the possible increased frequency with which multiple arterial lesions will be observed, particularly if complete angiography is performed in every case of subarachnoid bleeding. Certainly, this has been true of multiple intracranial aneurysms and undoubtedly there are many unreported cases of aneurysms combined with arteriovenous malformations. The surgeon may well be circumspect in his approach to such lesions, perhaps attacking the aneurysm of the aneurysm-arteriovenous malformation combination, in view of the increased morbidity of the former, and the potentiality for prolonged life with the latter.

**SUMMARY**

A patient is presented with both an aneurysm of the anterior communicating artery and an angioma of the left temporal lobe; the latter lesion was probably responsible for the psychomotor seizures.

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**REFERENCES**