Rupture of an Intracranial Aneurysm within the Subdural Space—In Association with Trauma

A Case Report

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The formation of a subdural hematoma of significant size as a result of the rupture of a cerebral aneurysm is an uncommon occurrence and clinical reports on the subject are sparse. This finding has been reported most frequently in the autopsy studies of bleeding aneurysms. For this reason we are presenting a clinical report with successful recovery following operation in a patient whose course was complicated further by the history of a recent head injury.

The reported incidence of subdural hematoma resulting from ruptured intracerebral aneurysms varies between 1 to 8 per cent. Walton on autopsy findings reported 1.9 per cent in a series of 312 cases. Among clinical reports, Voris found 1 per cent in 100 cases; Laudig et al. 2.9 per cent in 143 cases; Golden et al. 2.1 per cent in 384 cases; McKissock and Walsh 4.9 per cent in 249 cases; Dandy 1.6 per cent in 64 cases; and Strang et al. 0.5 per cent in 420 cases.

Case Report

O.J., a 61-year-old white, right-handed male, was admitted on July 12, 1962 to the New York University Neurosurgical Service at Bellevue Hospital in New York City (Serial #4983-62) as a transfer from another hospital with the following history. The patient was a known alcoholic. While intoxicated on July 3, 1962 he was supposedly injured. Details as to the exact time and place of the incident were uncertain but, immediately after it, he felt pain in both temples and came to find himself on the next morning (July 4, 1962) in a hospital. He was noted to be stuporous with clotted blood in both ears and with a confusion and abrasion of the left forehead. As his level of consciousness improved, he started to complain of bifrontal headache which persisted and became progressively more severe. Pertinent neurological findings were reported as mild confusion and a mild left hemiparesis, including the face. Roentgenograms of the skull revealed a recent linear fracture of the left temporal bone.

Examination on admission to Bellevue Hospital on July 12, 1962 revealed an elderly man. There were signs of healing superficial trauma of the forehead. Blood pressure was 140/80; pulse rate 88; temperature and respiration were normal. The patient was confused, irritable and partially disoriented as regards time, place and persons. Memory and calculation were poor; speech was intact. There was no papilledema. Visual fields and acuity were intact as tested by confrontation. The pupils were round, reacting and equal; extracocular movements were normal. The patient was unable to walk without assistance because of a mild left hemiparesis more pronounced in the lower extremity with an estimated functional loss of 30–40 per cent. Deep tendon reflexes were exaggerated on the left side and bilateral Babinski’s sign was elicited. All sensory modalities were intact. There were no signs of meningeal irritation. Auscultation of the head for bruit was negative.

Course. Results of complete blood studies including hemogram, sugar, electrolytes, and serology were within normal limits; urine was normal; findings of liver-function test and bleeding studies were normal. Roentgenogram of the chest was negative, but films of the skull showed a recent linear fracture of the left temporal area. The pinal body was not visualized. Lumbar puncture yielded xanthochromic cerebrospinal fluid with initial pressure of 300 mm. of water. The chemistries of the cerebrospinal fluid were as follows: protein 59 mg. per cent, sugar 64 mg. per cent, Cl. 119 mEq./l., red blood cells 86/c.mm., lymphocytes 2/c.mm., serology normal.

On July 12, 1962 right carotid arteriography (Figs. 1 and 2) demonstrated an avascular area diagnostic of subdural hematoma and a pea-sized aneurysm adjacent to the inner table of the skull. A feeding vessel was seen crossing the avascular space. Radiologically, this was interpreted as an aneurysm on the periphery of one of the terminal branches (the Rolandic branch) of the right middle cerebral artery.

Operation. On July 13, 1962, right frontotemporal craniotomy revealed an extensive subdural hematoma covering the whole frontal and temporal surface of the brain. Just visible on the surface of the solid hematoma, a greyish-white spot was felt to be firmer than the surface of the surrounding hematoma and was in the position of the angiographic demonstration of the aneurysm. The hematoma was split away from the aneurysm and removed carefully until the rounded, firm, pea-sized structure was reached. A silk ligature was applied to its neck and the aneurysm was excised. Following this, the subdural hematoma was removed totally with its well formed membranes. The hematoma was solid, about 3.5 cm. in its thickest part. Two silver clips were applied, one over the cortex just at the site of the ligated feeding artery, and the other at a corresponding spot in the dura mater. The cortex was xanthochromic in color only at the site of the aneurysm.

Pathological Report (Figs. 3 and 4). The specimen consisted of a round mass adherent to a flat membrane.

Received for publication December 14, 1962.

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Fig. 1 (left). Lateral projection. Right carotid arteriogram demonstrating aneurysm on one of the terminal branches (arrow) of the right middle cerebral artery.

Fig. 2 (right). Anteroposterior projection. Right carotid arteriogram demonstrating the subdural hematoma within which the aneurysm (arrow) could be clearly seen.

Fig. 3. Clotted blood is evident in the upper left field, and a branch of an artery is visible in the lower right. Between these is a fibrinous and necrotic membrane, the origin of which is indicated by the segment to the left which has the histologic character of an arterial wall. Hematoxylin and eosin, X70.
The membrane was 3 cm. in diameter and 0.3 cm. in thickness. One aspect was pale, smooth and glistening. The other was rough, irregular and hemorrhagic, and to this aspect a roughly spherical mass, 0.8 cm. in diameter, was attached. This mass was dark red in color, and semifirm in consistency. On section, it gave the appearance of a laminated clot. Eight irregular fragments of clotted blood accompanied the specimen.

Microscopically, the round structure consisted largely of an anemortem thrombus. In areas, it was undergoing organization and some blood pigment was present. At the point of attachment to the membrane, which proved to be leptomeningeal in character, remnants of a clearly defined arterial wall could be recognized. The leptomeningeal tissues were markedly hemorrhagic and were undergoing organization in areas. A diagnosis of ruptured congenital aneurysm was made, although the wall of the aneurysm itself was not identified.

Postoperative course was smooth and the patient's left hemiparesis cleared rapidly. Marked improvement in his organic mental changes was noted.

Roentgenogram of the skull showed the clips in contact. An electroencephalogram on July 28, 1962 was reported as normal. Psychiatric evaluation was that of a mild organic mental syndrome secondary to chronic alcoholism and head trauma. He was discharged from the hospital on Aug. 8, 1962, ambulatory, and was fully rational with no focal neurological deficit.

Discussion

Clark and Gooddy classified ruptured intracranial aneurysm as (1) rupture resulting in extensive subarachnoid, intracerebral and subdural hemorrhage, (2) rupture resulting in considerable subdural hematoma which caused compression of the brain, and (3) rupture resulting in very small and insignificant hemorrhage. On the other hand, Strang et al. classified the lesion as rupture only when there resulted (1) subdural hematoma with massive subarachnoid and intracerebral hemorrhages, and (2) without significant intracerebral bleeding. It is of interest to note that the relative frequency of the subdural hematoma varies with the location of the aneurysm—thus it is reported as occurring most commonly with bleeding from lesions of the internal carotid artery, next in frequency is the middle cerebral artery, then the anterior communicating artery, and least is the basilar artery. This is attributed to the anatomical relationship of these vessels to the subarachnoid space.

There are many theories postulated as to the mechanism of aneurysmal blood reaching the subdural space:

1. A tear in the arachnoid may be produced by rapid accumulation of blood in the subarachnoid space at the time of the first bleeding.

2. The aneurysm may lie within the subarachnoid space, with its dome being adherent to the inner surface of the arachnoid membrane as a result of previous leakage since the adhesions
come to form part of the wall of the sac; a subsequent rupture may occur directly through into the subdural space.

(3) Massive intracerebral bleeding may rupture the pia-arachnoid.

(4) Bleeding may occur from a carotid aneurysm which arises from the artery as it traverses the subdural space. This last explanation was doubted by Clarke and Walton.\textsuperscript{3}

Clinically, cases may present two distinct syndromes as outlined by Bassett and Lemmen:\textsuperscript{4} one may simulate classical subdural hematoma, the other a leaking aneurysm with subarachnoid hemorrhage. In the present case, the history of chronic alcoholism and signs of recent head injury (bruises over the orbit and a linear fracture in the left temporal area) as well as the neurological deficit (confusion and left hemiparesis) all pointed to correct diagnosis of subdural hematoma on the right side.

However, arteriography demonstrated clearly the presence of aneurysm on the peripheral branch of the middle cerebral artery lying just under the dura mater and the feeding artery bridging the avascular area. It is difficult, of course, to assign the exact role that trauma played in the development of the clinical situation in our patient. The staining of the brain about the base of the aneurysm plus the pathological studies which demonstrated rupture with a loss of all layers of the aneurysmal wall established the fact that the aneurysm had indeed bled.

If the aneurysm had been previously adherent to the overlying arachnoid and dura mater, then conceivably the head injury might have been an important factor in the production of the bleeding.

This case again stresses the emphasis that without the arteriogram it is practically impossible to diagnose a ruptured aneurysm as an underlying cause of subdural hematoma, even to suspect it especially when there is no clinical history of previous episodes suggestive of subarachnoid hemorrhage.

Summary

A case of a subdural hematoma caused by rupture of an intracerebral aneurysm with successful surgical removal has been presented. A brief review of the literature indicates the relative rarity of such a condition.

References