

# False Aneurysm with Subdural Hematoma and Symptomatic Vasospasm Following Head Injury

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**A rare case of cortical false aneurysm in a subdural hematoma and symptomatic vasospasm following head injury without skull fracture is reported. Angiography led to the correct preoperative diagnosis and successful surgical treatment, although the patient's symptoms of acalculia, agraphia, and astereognosis, mainly due to the vasospasm of the proximal portion of the middle cerebral artery, remained unchanged. The mechanism of such a cortical false aneurysm and symptomatic vasospasm is discussed briefly.**

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Traumatic acute subdural hematomas usually arise from the laceration of bridging cerebral veins, but those resulting from arterial damage have also been reported [6, 15]. The tearing of intradural cortical arteries usually results from a penetrating injury or skull fracture. A cortical false aneurysm after head injury without skull fracture and producing a subdural hematoma diagnosed preoperatively by angiography is rare. A search of the literature revealed only 14 other similar cases [1, 3, 4, 7, 9-13]. Vasospasm following head injury is well known and its incidence has been reported as 2 to 31% [14]. However, the demonstration of a symptomatic vasospasm following head injury by both angiogram and computerized tomographic (CT) scan has not been described before.

## Case Report

A 36-year-old man sustained a closed-head injury to the left frontal region in a car accident on November 28, 1980, but did not lose consciousness. On admission to a local hospital he was alert but suffered from headache, vomiting,

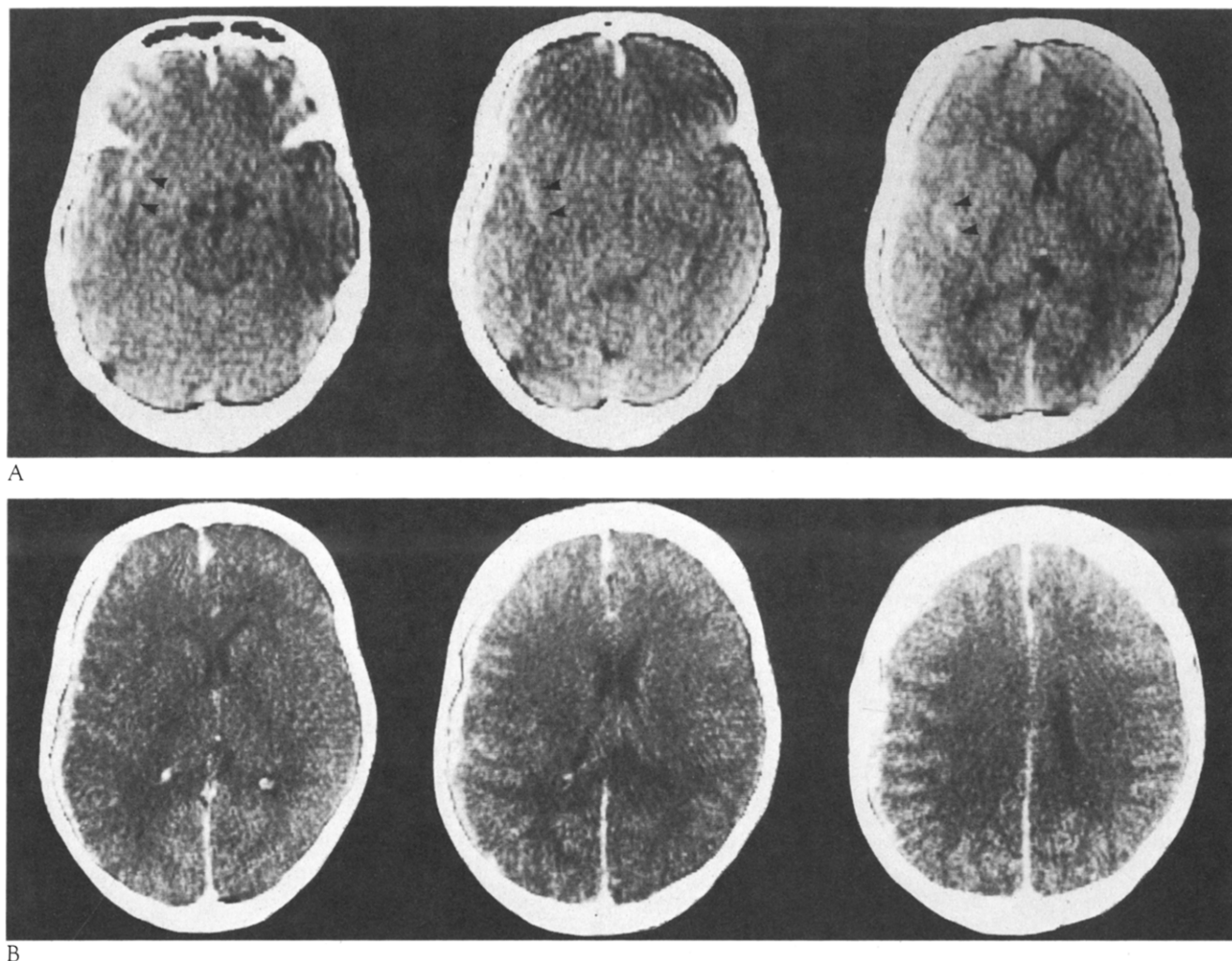
and mild right hemiparesis. CT scan (Fig. 1A) showed a thin, acute subdural hematoma on the left convexity and high density in the left sylvian fissure and anterior interhemisphere. He was treated conservatively, and angiography was not performed at that time. During the next seven days, he gradually improved from the hemiparesis but vomiting and headache persisted. He was transferred to the Hitachi General Hospital on December 5.

On admission, he was alert and oriented. He complained of a severe headache and vomiting. Cranial nerves were all intact but a slight degree of stiffness of the neck was noted. On Barré's testing, the right arm pronated slightly. No other neurological abnormality was noted. Lumbar puncture was not performed. Roentgenograms showed no fracture, but a CT scan revealed a thin, crescent-shaped, high-density area over the left convexity and high density in the anterior interhemisphere. The high density in the left sylvian fissure seen on the earlier CT scan had disappeared. The midline was shifted from left to right by about 1 cm and the left lateral ventricle was compressed. There was no parenchymal abnormal density. A postcontrast CT scan (Fig. 1B) showed bilateral gyral enhancement, which might indicate the presence of chemical meningitis after a subarachnoid hemorrhage. After the diagnosis of acute subdural hematoma was made, the patient was again treated conservatively. On December 8, ten days after the injury, he became disoriented, dysarthric, and incontinent. He also had Gerstmann's syndrome and astereognosis. A CT scan (Fig. 2A) taken on December 12 showed a crescent-shaped low-density and high-density area over the left convexity in place of the previous high density as well as increased midline shift and a low-density area in the left temporoparietal area. A left carotid angiogram (Fig. 3) made on the same day revealed a small avascular area on the left convexity. On the cortical surface, an aneurysm was found as a pea-sized nodule connected with an afferent irregular artery of the peripheral branch of the posterior parietal artery. The aneurysm was filled with contrast medium in the early arterial phase and remained filled in the capillary phase. The pericallosal artery and the internal cerebral vein were shifted 12 mm to the right. There was also a severe vasospasm in the proximal portion of the middle cerebral artery (Fig. 4A). In a contrast-enhanced CT scan (Fig. 2B)

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taken on December 17, an enhanced area with a salt and pepper appearance in the left temporoparietal region was noted.

On December 18, a left temporoparietal craniotomy was performed. No skull fracture was noted and the dura mater was entirely intact. After the dura mater was opened, a large amount of semiliquid clot was evacuated. During removal of the clot, a spot of active arterial bleeding from a pea-sized nodule attached to a cortical artery was found. An old subarachnoid hemorrhage was seen beneath the clot. The vessel was clipped and the aneurysm was resected. The other part of the arachnoid was entirely intact, and no macroscopic ischemic change in the cortex was recognizable.

The patient demonstrated marked clinical improvement after the operation. Headache, vomiting, and dysarthria all disappeared. However, the focal signs such as Gerstmann's syndrome and astereognosis remained unchanged. A post-operative left carotid angiogram (Fig. 4B) showed complete obliteration of the cortical aneurysm, patency of the distal artery, and almost complete disappearance of the avascular

Fig. 1. (A) CT scans performed at another hospital soon after head injury show a thin high-density area over the left convexity, as well as high density in the left sylvian fissure (arrowheads) and anterior interhemispheric fissure. (B) CT scans with contrast enhancement performed at our hospital 7 days after head injury show bilateral gyral enhancement.

area. The vasospasm seen previously in the proximal portion of the middle cerebral artery was reduced. The vessel had regained its normal caliber. A plain CT scan disclosed no area of abnormal density, but the left temporoparietal region was enhanced and had a salt-and-pepper appearance. This infarcted lesion caused by vasospasm might have been responsible for the focal signs. The patient was discharged on December 30 when his neurological deficits were slightly improved. After discharge, he was followed in our outpatient clinics at regular intervals. When he was last examined on May 12, 1981, his parietal lobe signs were still present, although to a very mild degree, and he had returned to his previous job.

A histological section revealed an organizing blood clot

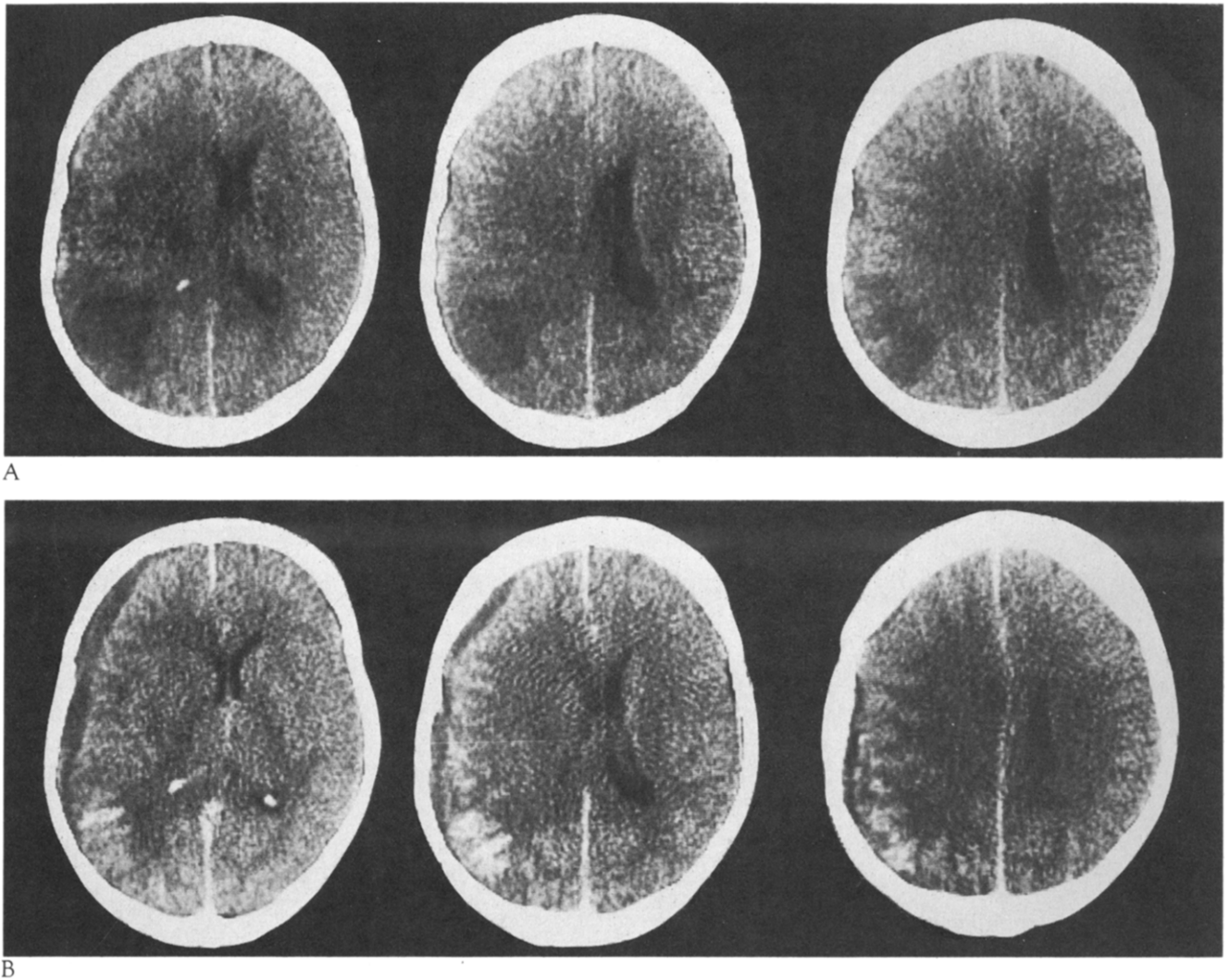


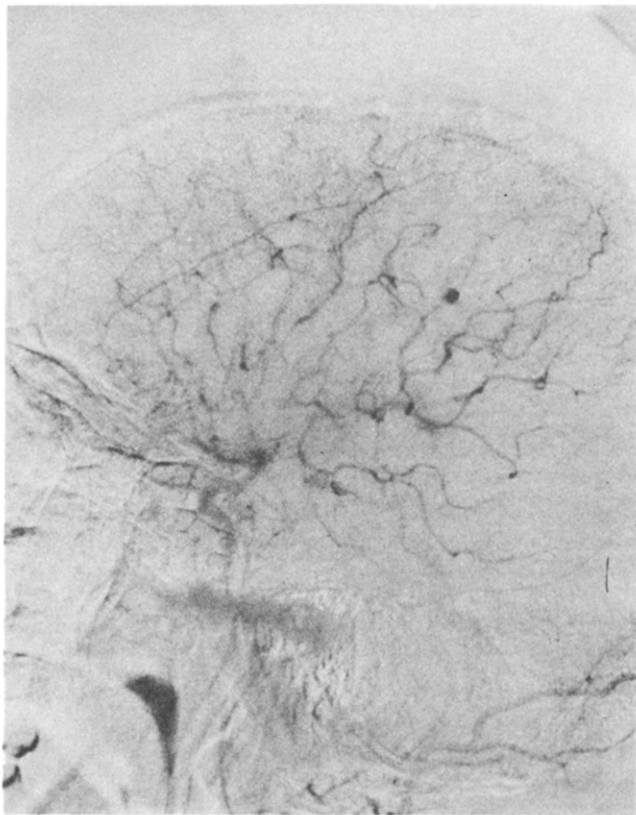
Fig. 2. Consecutive CT scans. (A) Scans taken 14 days after head injury disclose a low-density area in the left temporoparietal area. (B) Scans with contrast enhancement taken 19 days after head injury reveal a left temporoparietal enhanced region with a salt-and-pepper appearance.

and loose connective tissue containing markedly proliferated capillaries. There was no artery or aneurysmal wall in the tissue examined.

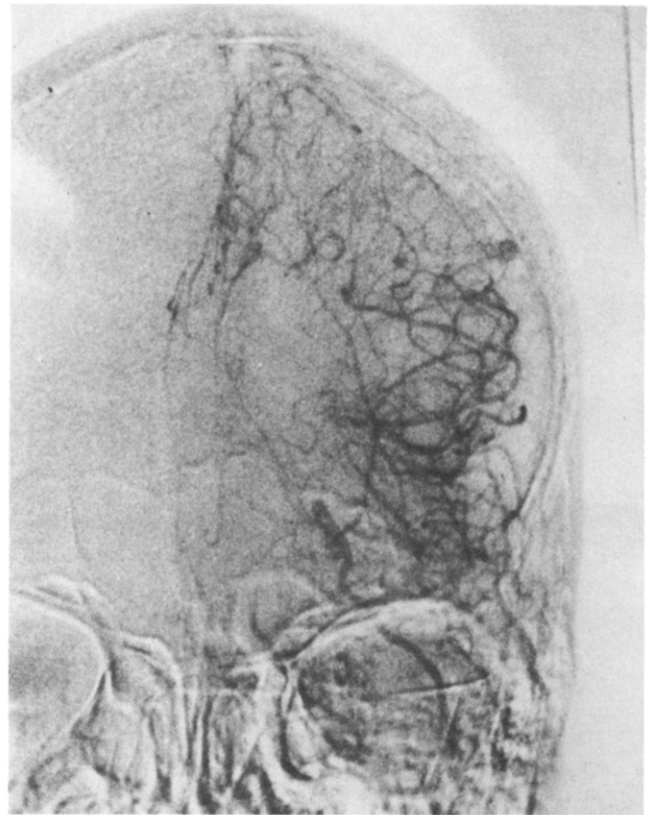
#### Discussion

Cortical false aneurysms in a subdural hematoma following penetrating injury or lacerating skull fracture have been described [2]. This type of pathological condition can be understood as the consequence of direct trauma. However, cortical false aneurysms occurring after trauma without skull fracture (indirect trauma [5]) and associated with a subdural hematoma have rarely been reported and so far we have found only 14 cases [1, 3, 4, 7, 9–13]. In a description

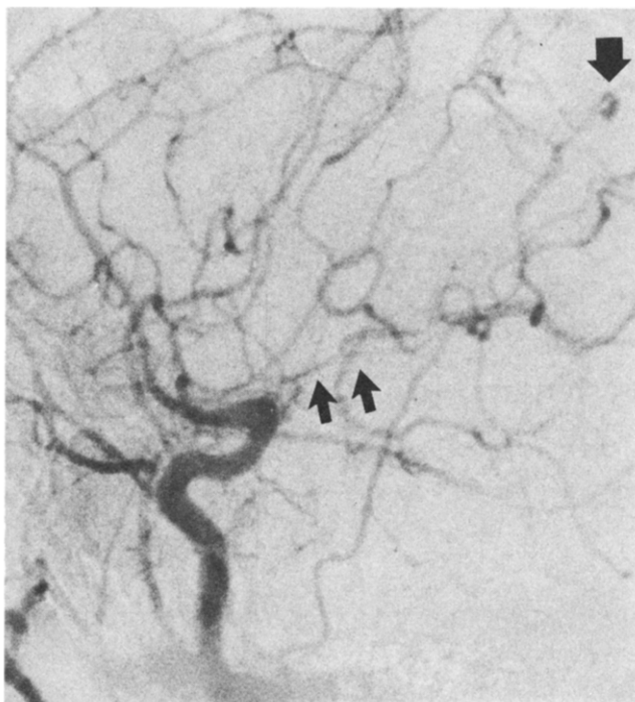
of a subdural hematoma due to arterial bleeding, Drake [6] said that when abrupt brain movement occurs, such arterial tears might appear where surface vessels were adherent to the dura mater or where they supplied small dural branches. Bergström and Hemmingsson [4] also thought that the mechanism responsible for such an occurrence was the existence of a leptomeningeal arterial anastomosis [15, 16]. In a leptomeningeal arterial anastomosis, the cortical artery passes through the arachnoid membrane to the dura mater so that the displacement of the brain beyond a certain extent might lead to the tearing of the arterial wall, forming a subdural hematoma and possibly a false aneurysm. It is easy to suspect that a lesion in the subarachnoid space might lead to subarachnoid hemorrhage at the same time. In the patient described here, a subarachnoid hemorrhage was suspected preoperatively from plain and contrast-enhanced CT scans and was actually observed at operation. In the 15 reported cases, including our case, a subarachnoid hemorrhage was found either on lumbar puncture or during operation.



A  
Fig. 3. Preoperative left carotid angiogram: (A) lateral and (B) anteroposterior views demonstrate an avascular area in the left convexity



B  
and a cortical aneurysm with an afferent irregular artery of the peripheral branch of the posterior parietal artery.



A  
Fig. 4. (A) Preoperative left carotid angiogram: lateral view shows severe vasospasm in the proximal portion of the middle cerebral artery (small arrows) and an aneurysm (large arrow). (B) Postoperative



B  
left carotid angiogram: lateral view shows disappearance of the vasospasm seen previously (small arrows) and obliteration of the aneurysm (large arrow).

ation (4 cases). It seems logical to consider that the spasm of the cerebral artery occurs as a result of subarachnoid hemorrhage associated with the ruptured cortical false aneurysm. Suwanwela and Suwanwela [14] and Wilkins and Odom [17] described symptomatic vasospasm of the anterior circulation after head injury and mentioned its clinical significance. They considered the pathogenesis of the symptomatic spasm of the cerebral artery occurring in association with head injury to be subarachnoid perivascular clot formation and subsequent clot lysis. Jackson and Back [8] also reported a case of posttraumatic hemiplegia caused by delayed arterial spasm and thrombosis: a lumbar puncture revealed grossly bloody cerebrospinal fluid. In conclusion, we consider that the pathological mechanism of symptomatic vasospasms following head injury might be the same as that of spontaneous subarachnoid hemorrhages.

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## Book Review

### Epilepsy—A Window to Brain Mechanism

*Edited by J. S. Lockard and Arthur A. Ward, Jr.*

*New York, Raven Press, 1980*

*296 pp., \$29.50*

*Reviewed by Sidney Goldring, M.D., St. Louis, Missouri*

This book fulfills the promise of its title, but more than that, it is a showcase of the neuroscience program that Arthur A. Ward, Jr., has created in Seattle. In a somewhat sentimental preface, Herbert H. Jasper bridges the gap between Arthur Ward's early training at the Montreal Neurological Institute and the present school of epilepsy research in Seattle. Seventeen chapters follow, all by members of the Department of Neurological Surgery at the University of Washington. Subjects of studies range from

synaptic mechanisms, ultrastructure, and pharmacokinetics to the utility of exploiting a primate clinical model, brain mechanisms for language, neurophysiological underpinnings of surgical treatment, and social aspects of epilepsy. Although the chapters are predominantly reviews of already published work, the incorporation of these multidisciplinary studies under one cover should prove to be an important reference for all who have an interest in epilepsy. For the generalist, an overview by Ward provides insight into his scientific philosophy and succinctly summarizes the chapters that follow. The epilogue closes with the cornerstone of Ward's research strategy, ". . . the loop is closed; research in the laboratory extends to the clinical setting, where observations generate questions that are returned to the laboratory"—a view with which I am in full agreement.