

# Complex Partial Seizures Associated With Unruptured Thrombosed Basilar Artery Apex Aneurysm

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**Complex partial seizures, an unusual presentation of a thrombosed basilar artery apex aneurysm, are described. The possible etiologies causing this presentation are reviewed.**

**KEY WORDS:** Aneurysm; Seizure; Basilar artery, thrombosed; Emboli

Epilepsy is rarely caused by unruptured intracranial aneurysms. In the collaborative aneurysm study only 4% of the patients had seizures antecedent to their subarachnoid hemorrhage [5]. Suggested causative factors are the hamartomalike action of the calcified wall of the aneurysm or subclinical hemorrhage resulting in tissue damage with subsequent development of a discharging focus [8]. The case to be presented is of interest in that the temporal lobe epileptogenic focus was most likely caused by ischemia secondary to embolization from an unruptured thrombosed basilar aneurysm.

## Case Report

A 60-year-old white female was admitted to the University of Nebraska Medical Center after a generalized tonic-clonic seizure earlier in the afternoon of January 19, 1980. The patient related she had been sitting at a table drinking hot chocolate, talking to her son, and had suddenly felt faint. Her son described the ensuing events. His mother became "glassy eyed" and slumped to the floor. She then developed a jerking motion of both upper and lower extremities. She was amnesic for the episode, but she remembered all events up to and immediately after the event. She had no tongue biting or

incontinence. After the event she had residual generalized headache and fatigue.

Approximately 4-6 years before admission she began to experience very brief episodes characterized by the abrupt onset of a "funny" feeling in the pit of her stomach, a fluttering of her chest, and the sensation of blood running up the back of her head. This was followed by a sense of ill being. The attacks lasted only several seconds, and they occurred with variable frequency. Two years before admission an episode of loss of consciousness not associated with tonic-clonic movements was reported. The convulsion before admission was preceded by a "small spell."

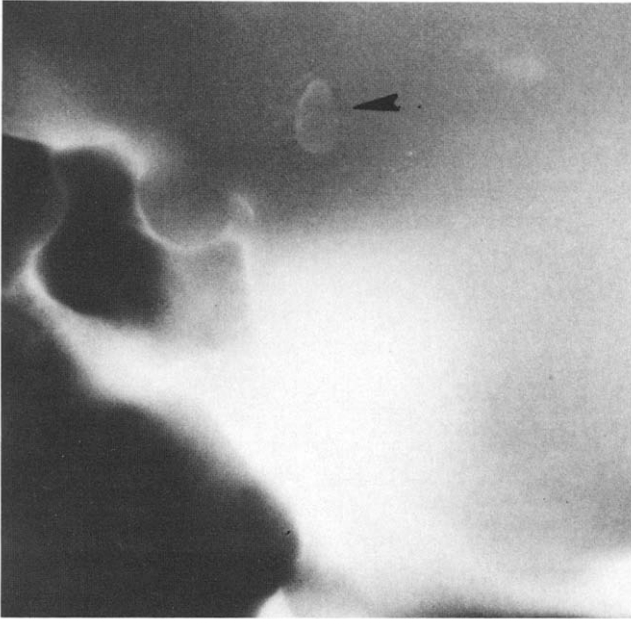
The patient had a long history of hypertension and currently was taking Aldomet, 250 mg twice daily. Her father and sister died of strokes at early ages. Past operations included a cholecystectomy, stapedectomy and tympanoplasty. She specifically denied a previous history of significant head trauma, meningitis, encephalitis, or cerebral vascular accidents. There was no past history suggestive of a prior subarachnoid hemorrhage or transient ischemic events of the anterior or posterior circulation. The remainder of her history was non-contributory.

On admission the patient was alert and oriented. There was no evidence of laceration of her tongue. The blood pressure was 178/94. The remainder of the vital signs were normal. The general physical examination showed normal results. Specifically no murmurs were heard over the precordium or bruits over the carotid or supraclavicular areas. The results of the neurologic examination were normal.

At the time of admission, the laboratory results for hemogram, urinalysis, and electrolyte values were normal. An x-ray examination of her chest showed arteriosclerotic vascular calcification of the aorta. Results of electrocardiogram were within normal limits. A 24-hour Holter monitor recording showed rare supraventricular premature beats.

Tomography of the skull (Figure 1) revealed a calcified density just inferior to the pineal. Computed to-

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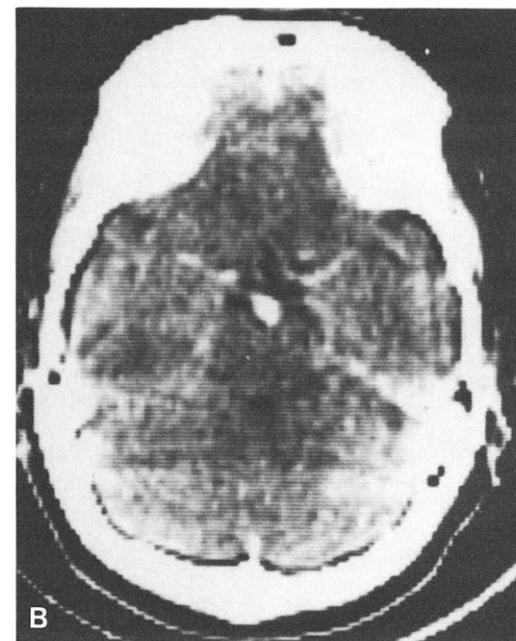
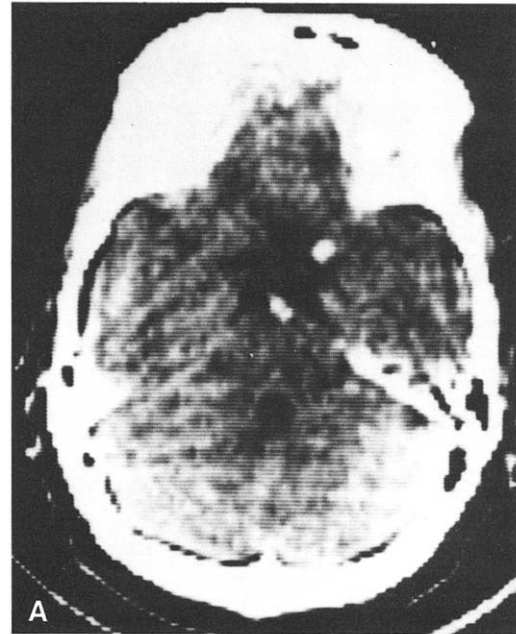
**Figure 1.** Lateral tomogram of the skull through the midline demonstrates an oval collection of calcium with dense margins.

mography scan (Figure 2) showed a calcified mass near the basilar summit. Angiography (Figure 3) revealed a thrombosed basilar artery apex aneurysm. The posterior cerebral arteries were patent and of normal appearance. The initial electroencephalogram (EEG) obtained the day after admission was normal while awake and during drug induced light sleep states (Figure 4A). Small sharp spikes were seen independently over both hemispheres, a finding considered a normal variant. An EEG after sleep-deprivation, obtained 2 months after discharge from the hospital, revealed low-to-moderate bursts of theta activity with mixed sharp waves over the left temporal region (Figure 4B). In-phase cancellations were noted between the F7 and T3 electrodes while instrumental phase reversals occurred between the T3 and T5 electrodes. The small sharp spikes were also seen (Figure 4C). These EEG abnormalities were compatible with an irritative focus within the left temporal lobe.

The patient was placed on a regimen of 300 mg of phenytoin daily; blood levels of 10–20 mg% were maintained. The patient has been followed 2.5 years since the diagnosis. There has been a marked decrease in the frequency of her spells. Over the past 6 months she has only had three brief episodes.

### Discussion

In the older age group, cerebral vascular disease is reported to be a leading cause of epilepsy [27]. The type of vascular disease is often not documented or the as-



**Figure 2.** (A) A computed tomography scan without contrast-medium enhancement. A well-defined area of calcification is intimately related to the distal end of the basilar artery. (B) A computed tomography scan after injection of contrast medium. The film demonstrates a collection of contrast in the proximity of the calcified area seen in the study without contrast medium.

sociation made merely because of the age of the patient. The present case is unusual in that this patient with complex partial and generalized seizures was found to have an unruptured thrombosed aneurysm at the apex of the basilar artery. Although this finding may have been incidental, complete cardiovascular, metabolic, as



**Figure 3.** Lateral vertebral angiogram. The calcified area is located at the distal end of the basilar artery, and probably represents a clot-filled aneurysm of the basilar artery (arrows).

well as four-vessel cerebral angiography failed to uncover other causes. Specifically, the extracranial as well as the intracranial carotid arterial system was normal. Seizures secondary to ruptured aneurysms have been previously reported; however, the aneurysms have been located more commonly on arteries of the anterior circulation [5].

In this case the aneurysm was thrombosed, which is an unusual occurrence [3,7]. Thrombosed aneurysms

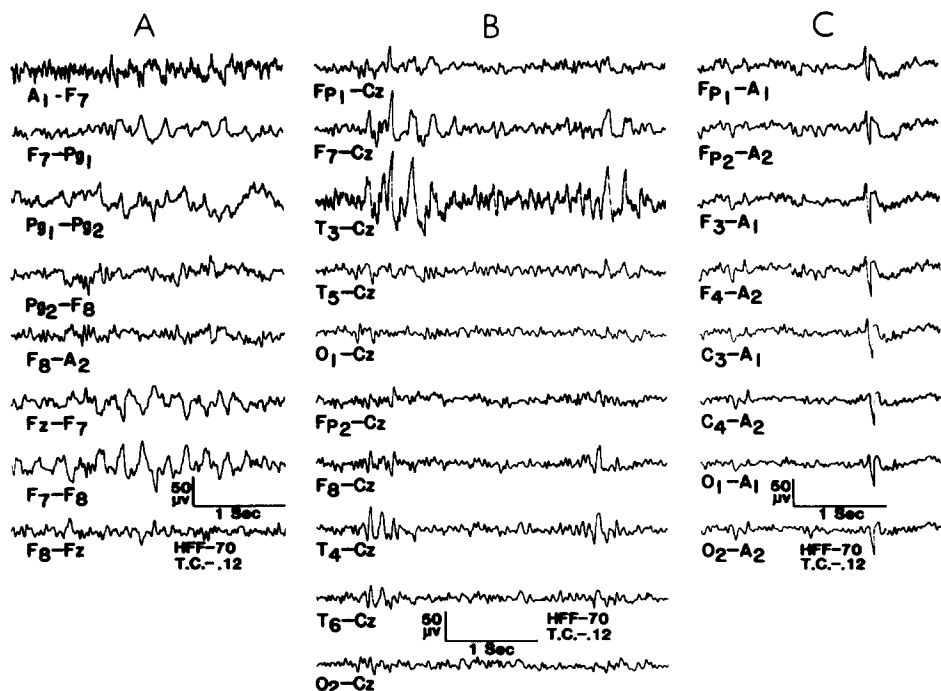
rarely cause subarachnoid hemorrhage or the mass effect of giant aneurysms, but they can embolize. Embolization from aneurysms is considered rare [9], but cerebral infarction related to emboli from intracranial aneurysms has been reported in several cases [1,4,11]. A prior report has described temporal lobe electroencephalographic disturbances associated with major and minor syndromes of vertebral basilar insufficiency [6].

In our patient it seems reasonable to postulate that emboli from this aneurysm traveled through the posterior cerebral artery and its branches to the temporal lobe with resultant microinfarction and the subsequent development of an epileptogenic focus. By history there were no symptoms suggestive of intermittent posterior circulation ischemia; however, destruction of temporal-lobe tissue supplied by distal branches of the posterior cerebral artery may not cause immediate clinical signs. Of interest and support of this mechanism is the report by Stewart et al [10]: his case 4 had symptoms suggestive of complex partial seizures and later suffered a generalized convulsion. Other transient symptoms present in the patient suggested that they were secondary to embolization of a clot from the aneurysm. Seizures occurring acutely with embolization are well documented. Infarction after epilepsy does occur; however, the frequency has not been adequately investigated.

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**Figure 4.** Electroencephalogram samples from tracings performed: A initial, B and C 2 months after discharge. See text for interpretation.



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