



Subdural Hematoma Mimicking a Transient Ischemic Attack Due to Antihypertensive Medication

YEHIA Y. MISHRIKI, MD, Allentown, Pa

ABSTRACT: An elderly man had two completely reversible episodes of aphasia, each occurring after taking his antihypertensive medications. He was subsequently found to have a subdural hematoma. The association between subdural hematomas (and other intracranial mass lesions) and reversible neurologic deficits is discussed, as is the pathophysiology of the phenomenon. In addition, the relationship between the effects of the patient's antihypertensive medications and the neurologic deficits in the setting of a subdural hematoma is explored.

SUBDURAL HEMATOMAS and other intracerebral mass lesions can manifest as intermittent, completely reversible neurologic deficits mimicking transient ischemic attacks (TIAs). The implications for treatment are obvious, since recurring, presumed TIAs are often treated with anticoagulation, a potentially disastrous intervention in patients with subdural hematomas. Herein, I describe a patient who had recurrent transient neurologic deficits induced by the administration of antihypertensive medications and who was found to have a subdural hematoma.

CASE REPORT

An 86-year-old male hypertensive nursing home patient had sudden, nonfluent aphasia accompanied by headache. The episode lasted half an hour before resolving fully. When seen in the hospital, the patient adamantly insisted that it was his new antihypertensive, diltiazem, that had caused his symptoms. It had recently been added to losartan to better control his blood pressure. Shortly after admission, the patient once again had profound, nonfluent aphasia, which was unaccompanied by motor or sensory deficits. The patient indicated that he had a severe headache. On examination, his blood pressure was slightly elevated at 150/86 mm Hg. Auscultation of the lungs disclosed midinspiratory crackles at the bases, and examination of the heart revealed a regular rhythm and a grade 2/6 midsystolic murmur at the base. There were no gallops or clicks. The right carotid artery was of normal

amplitude and contour, whereas the left carotid had a slightly diminished amplitude. There were no carotid bruits. The patient's roommate pointed out that the patient had been conversant only half an hour earlier, and a check with the nurse revealed that the patient had received his antihypertensive medications a short time before the most recent neurologic episode. His blood pressure had been 188/98 mm Hg at that time.

The patient was thought to be having recurrent TIAs, and the headache was attributed to uncontrolled hypertension. A heparin drip was to be started, but fortunately it was withheld until the patient had computed tomography (CT) of the brain. The CT scan revealed a moderate sized acute to subacute subdural hematoma overlying the left frontal lobe. A craniotomy with evacuation of the hematoma was performed, and the patient made a full and uneventful recovery.

DISCUSSION

This case illustrates the well described but little appreciated phenomenon of the occasional intermittence of neurologic symptoms in patients with subdural hematomas. In addition, our patient had the unusual twist of having his symptoms brought on by his antihypertensive medications, presumably by their blood pressure lowering actions.

The association between transitory neurologic signs or symptoms and subdural hematomas was first described by Schwenke in 1733.¹ Since then, many reports have described cases of intermittent neurologic deficits occurring in association with subdural hematomas as well as other "mass" lesions and cerebral aneurysms.²⁻⁵ When transient neurologic deficits have occurred in patients with subdural hema-

From the Department of Medicine/Ambulatory Care, Lehigh Valley Hospital, Allentown, Pa.

Reprint requests to Yehia Y. Mishriki, MD, Lehigh Valley Hospital, Department of Medicine, 1210 S Cedar Crest Blvd, Suite 3600, Allentown, PA 18103.

tomas, they have frequently been aphasias with or without hemimotor or hemisensory deficits and have lasted an average of 30 minutes.⁶ In a significant percentage of these patients, a history of head trauma was conspicuously absent. Various theories have been advanced to explain this counterintuitive phenomenon and have included (1) decreased cerebral blood flow (CBF) due to blood vessel compression or to vascular displacement caused by parenchymal edema; (2) seizure activity caused by disruption of normal cortical electrical activity; (3) fluctuations in intracranial pressure due to changes in head position or Valsalva's maneuver; (4) spreading cortical depression as a result of mechanical irritation of brain tissue; and (5) small, repeated hemorrhages into the subdural space and membrane of the hematoma. Any, or several, of these mechanisms might be operative in a given patient.²

Several studies have investigated CBF in patients with subdural hematomas. Ikeda and colleagues⁷ studied regional CBF in 38 patients with unilateral chronic subdural hematoma. They found that preoperative regional CBF was essentially normal in patients without focal neurologic signs but was in the lower limit of age-matched normal values, in general. It was subnormal in some areas in patients who had hemiparesis associated with the hematoma. Nukui and Aiba⁸ also showed a reduction in CBF in older patients with subdural hematomas and focal neurologic deficits, in contradistinction to younger patients without focal deficits who had normal values. They theorized that CBF was diminished because of an increase in intracranial pressure. Brodersen and Gjerris⁹ had similar results in their study, but they found no significant increase in cerebrospinal fluid pressure at lumbar puncture, which they used as an estimate of intracranial pressure. Furthermore, they could not account for the diminution in CBF by alterations in the patients' PCO₂ levels.

The temporal association between our patient's two episodes of aphasia and his receiving his antihypertensive medications suggests a cause-and-effect relationship. Given

that the patient's hypertension had been poorly controlled, it was possible that he had diminution or loss of cerebrovascular autoregulation. Therefore, cerebral blood pressure and CBF would have had a more linear rather than sinusoidal relationship. Since cerebral blood pressure is the difference between mean arterial pressure and intracranial pressure, a reduction in mean arterial pressure with antihypertensive medications would have resulted in a reduction in cerebral blood pressure and, hence, CBF, in turn causing the neurologic deficit.

This case highlights the fact that subdural hematomas can manifest as intermittent neurologic signs and symptoms mimicking TIAs. It also suggests that a subdural hematoma may cause the sudden development of a neurologic deficit in a patient with hypertension whose blood pressure is normalized with antihypertensive medications. Given that the usual treatment of recurrent TIAs is with anticoagulation (a disastrous intervention in a patient with a subdural hematoma), it is imperative that an imaging study be done in a patient with a transient neurologic deficit to distinguish a true TIA from a subdural hematoma or other intracranial mass lesion.

References

1. Endtz LJ: Post-traumatic hygroma in the eighteenth century. described by Thomas Schwenke. *Surg Neurol* 1978; 10:305-307
2. Melamed E, Lavy S, Reches A, et al: Subdural hematoma simulating transient cerebral ischemic attacks: case report. *J Neurosurg* 1975; 42:101-103
3. Russell NA, Attack DM, Benoit BG: Chronic subdural hematoma, a cause of apparent transient ischemic attacks. *Can Med Assoc J* 1982; 127:1105-1106
4. Moser ML, Johnston DE, Reinmuth OM: Subdural hematoma with transient neurological deficits: a review of 15 cases. *Ann Neurol* 1983; 14:539-542
5. Russell NA, Goumnerova L, Attack EA, et al: Chronic subdural hematoma mimicking transient ischemic attacks. *J Trauma* 1985; 25:1113-1114
6. Traynelis VC: Chronic subdural hematoma in the elderly. *Clin Geriatr Med* 1991; 7:583-598
7. Ikeda K, Ito H, Yamashita J: Relation of regional cerebral blood flow to hemiparesis in chronic subdural hematoma. *Surg Neurol* 1990; 33:87-95
8. Nukui H, Aiba T: Regional cerebral blood flow in patients with chronic subdural hematoma. *No Shinkei Gaisho* 1972; 4:371-382
9. Brodersen P, Gjerris F: Regional cerebral blood flow in patients with chronic subdural hematoma. *Acta Neurol Scand* 1975; 51:233-239