Chronic subdural hematoma simulating transient cerebral ischemic attacks

Case report

ELDAD MELAMED, M.D., SILVAN LAVY, M.D., AVINOAM RECHES, M.D., AND ABRAHAM SAHAR M.D.
Department of Neurology and Department of Neurosurgery, Hadassah University Hospital and Hebrew University-Hadassah Medical School, Jerusalem, Israel

A patient is presented in whom chronic subdural hematoma simulated transient ischemic attacks. The neurological manifestations were those of recurrent, transient episodes of expressive dysphasia preceded by focal sensory deficit. Various pathophysiological mechanisms which could have caused the unusual clinical picture are briefly considered.

KEY WORDS · chronic subdural hematoma · transient cerebral ischemia · dysphasia

As a slowly expanding intracranial mass, chronic subdural hematoma usually has the typical symptomatology of a progressive neurological deficit. In the following report we present an unusual case in which chronic subdural hematoma gave rise to recurrent transient, focal neurological phenomena.

Case Report

A 59-year-old male was transferred as an emergency to the Department of Neurology of the Hadassah University Hospital in Jerusalem. Until September, 1973, the patient had enjoyed good general health, with no history of hypertension, coronary or peripheral vascular disease or diabetes mellitus. On September 14 he developed a dull left frontoparietal headache, which was constant and relieved by analgesics. Two days later he experienced transient, recurrent sensations of numbness in his right hand, involving mainly the thumb and index, but with no weakness. On the following day he developed transient episodes of speech disturbances in which he could not express himself properly, had difficulties in formulating words, confused letters within words, and mixed two languages in the same sentence. On the whole, he had about 15 attacks of dysphasia, each lasting 10 to 15 minutes, often preceded by numbness of the right hand. In between the increasingly frequent attacks, the patient felt quite well except for the headache and, later on, an occasional difficulty in finding the appropriate word while speaking. No clear-cut history of head trauma could be determined.

Examination. On admission the general physical examination was normal. The blood pressure was 120/80 mm Hg. The pulse rate
was 72/min and regular. Good pulses were palpated over both carotid arteries, and there was no bruit. There were no positive neurological findings except for speech disturbances, observed during two attacks, each lasting 10 minutes. Routine laboratory examinations were within normal limits. Skull films were normal. Electroencephalographic (EEG) recordings were repeatedly normal. A left carotid angiogram (Fig. 1) disclosed an avascular, crescent, space-occupying mass over the left hemispheric convexity.

**Operation.** A liquified, chronic subdural hematoma, encapsulated in an old and thick membrane, was removed through two left-sided burr holes. Exploration through a right-sided burr hole was negative. The patient made an uneventful recovery following the operation; no recurrence of his previous symptoms and signs was found in follow-up examinations during the next 6 months.

**Discussion**

Recurrent, transient episodes of expressive dysphasia preceded by focal sensory deficit are often produced by cerebrovascular disease in patients this age. The true nature of the lesion in this case was demonstrated by routine angiographic investigation. The clinical picture is usually that of progressive, though sometimes fluctuating, neurological phenomena. Unlike most patients with subdural hematoma, our patient presented with the unusual symptomatology of short, frequently recurrent and transient episodes of neurological deficit, which had a striking similarity to transient ischemic attacks.

Although rare, intermittent neurological phenomena that mimic transient ischemic attacks have been reported in other intracranial mass lesions such as meningioma, metastatic brain tumor, and brain abscess. The literature is rather scanty concerning chronic subdural hematoma. Groch, et al., describe a case with a sudden appearance of hemiplegia and aphasia which had been preceded by transient hemiparesis. Okihiro, et al., report a case quite similar to ours, with intermittent aphasia and numbness of the right hand.

Several pathophysiological mechanisms by which the chronic subdural hematoma could have produced these unusual manifestations can be offered, although none seems to be certain. The mechanical pressure of the mass on the neighboring cerebral vessels could cause impairment of flow, leading to cerebral ischemia and paralysis of function. The intermittent nature of the attacks could be attributed to a rise and fall in the pressure exerted by the hematoma during changes of head position or performance of the Valsalva maneuver.

Also, hematoma can act as an irritative lesion, producing epileptic discharges. Thus, the intermittent dysphasia could be looked upon as a form of post-ictal paralysis of cortical function and the focal sensory deficit as a kind of an aura. In our patient there was, however, no further spread of symptoms; there were no convulsions nor loss of consciousness, and the EEG failed to show evidence of ictal etiology. Another possible mechanism suggested by Daly, et al., is based on the phenomenon of spreading cortical depression whereby the mechanical stimulation of the cortex by the lesion causes a depression of spontaneous electrical activity that spreads over the cortex; the cortex then becomes inexcitable to electrical stimulation. On the whole, there is obviously no one satisfactory explanation for the appearance of recurrent episodes in this chronic expanding lesion.

**Fig. 1.** Left carotid angiogram, anteroposterior projection, showing an avascular space between the vessels on the left cerebral convexity and the inner table of the skull.
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It is worthwhile to remember that on rare occasions intracranial mass lesions such as chronic subdural hematoma may simulate cerebrovascular insufficiency. Our experience stresses the importance of a complete and detailed workup, including carotid angiography, of every patient with clinical symptoms suggesting transient ischemic attacks.

References

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Address reprint requests to: Eldad Melamed, M.D., Department of Neurology, Hadassah University Hospital, Jerusalem, Israel.