Chronic Subdural Hematoma Presenting as Transient Neurologic Deficits

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SUMMARY Four patients with symptoms of transient neurological dysfunction were subsequently found to have chronic subdural hematomas (CSDH). The frequency of these episodes diminished significantly after evacuation of the hematoma. The effects of vascular compromise due to the CSDH and to cardiovascular events, more commonly implicated in transient ischemic attacks (TIAs) may be additive. The inclusion of a computerized axial tomographic (CAT) scan in the evaluation of some patients with presumed TIAs is recommended.

ALTHOUGH chronic subdural hematomas (CSDH) present in a protean fashion, they are rarely confused with TIAs.1-4 The fluctuating signs and symptoms of CSDH, seen in a significant percentage of patients,5 are usually superimposed on lesser persistent neurologic deficits. In contrast, the definition of a TIA necessitates complete resolution of symptoms between episodes of vascular insufficiency.7 We have recently encountered 4 patients whose presentation was typical of TIAs, but, who, on further evaluation, were found to have CSDHs. After the CSDHs were evacuated, their symptoms ceased. The advent of aspirin therapy for TIAs, contra-indicated in CSDHs, prompted presentation of the following patients.

Case Reports

Patient 1

Six months prior to admission, a right-handed 53-year-old man experienced abrupt onset of difficulty talking and right arm weakness. These symptoms resolved after 30 min. On the day prior to admission, the patient had 3 similar episodes. The only history of head trauma was a cerebral concussion sustained 16 years before.

Examination disclosed a mild expressive dysphasia and right upper extremity paresis. Carotid pulses were strong and symmetric and there were no bruits. Precordial examination was normal. No retinal emboli were seen. Hemoglobin, white blood cell count, electrolytes, urinalysis, electrocardiogram, and skull radiographs were normal. A technetium brain scan showed increased uptake over the left convexity. Bifrontal and left posterior parietal burr holes were placed. Thick external membranes were encountered and a large left-sided chronic subdural hematoma was drained. The patient was asymptomatic for the next 18 months, but then had a 20 min episode of expressive dysphasia. General physical and neurologic examinations performed shortly after the attack were normal, as was a CAT scan. For the past 12 months he has had no further episodes of transient neurologic dysfunction.

Patient 2

A right-handed 80-year-old woman experienced multiple 5–10 min attacks of difficulty talking and writing over a 4-day period. She was admitted after these symptoms had persisted for 24 h. Two months before, she had transiently lost consciousness during a motor vehicle accident.

During her admission examination, the patient had a 10 min episode of global dysphasia. Carotid pulses were symmetric and without bruits. Examination was otherwise unremarkable except for a right sixth nerve palsy, which had been present since her automobile accident. Directional Doppler showed adequate antegrade flow in both ophthalmic arteries. Lumbar puncture was normal except for a cerebrospinal fluid protein of 64 mg%. Bilateral carotid angiography demonstrated a left fronto-parietal subdural hematoma but no stenotic or ulcerated lesions of the cervical or intracranial portions of the internal carotid arteries (fig. 1a, 1b, 1c). A chronic subdural hematoma was evacuated through left frontal and posterior parietal burr holes. On the seventh postoperative day, the patient became abruptly dysphasic. Immediate re-opening of the burr holes revealed a small reaccumulation of the hematoma. Following evacuation, her symptoms initially improved. During the next 24 h, she had multiple episodes of global dysphasia. A left fronto-parietal craniotomy disclosed a large, gelatinous subdural hematoma. Two days later, the patient again became dysphasic. Another small subdural hematoma was evacuated, the overlying dura was excised, and the bone flap removed. The patient has been asymptomatic for 18 months.

Patient 3:

Three months prior to admission, a 73-year-old right-handed, hypertensive man became dizzy and then lost consciousness. A second episode 2 months later was accompanied by urinary and fecal incontinence. During the week prior to admission, he had 4 episodes of transient difficulty talking.

Physical examination revealed bilateral soft carotid bruits, a grade III/VI mid systolic murmur, and a mild right lower facial paresis. Lumbar puncture showed 20 red blood cells/mm3 and a protein of 118

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mg/dl. A CAT scan demonstrated a subdural hematoma over the left fronto-parietal convexity (fig. 2a, 2b). Thick membranes were found beneath left frontal and posterior parietal burr holes. A small quantity of subdural fluid was evacuated. Postoperatively, loss of vibratory sensation and astereognosis on the right side prompted further studies. A repeat CAT scan was normal. An electroencephalogram showed scattered infrequent slow waves over the left convexity. The signs of parietal lobe dysfunction gradually resolved and the patient has been asymptomatic for 20 months.

Patient 4:

An 81-year-old right-handed male physician was admitted following several 10-30 min episodes of difficulty talking, accompanied by tingling and weakness in the left upper extremity. Similar symptoms had occurred 2 weeks prior to admission. The patient had chronic hypertension and intermittent claudication. Additionally, he had been treated with Coumadin after 2 myocardial infarctions.

Admission examination demonstrated a mild dysarthria and a left hemiparesis and hemihypalgesia. Bilateral carotid bruits and a II/VI systolic ejection murmur were noted. Repeat neurological examination 2 hours after admission was normal. Skull radiographs showed a 4 mm shift of the pineal gland from left to right. Brain scan demonstrated increased uptake over the right fronto-parietal convexity and possibly over the left parietal convexity as well (fig. 3).

The prothrombin time was 24 seconds (control = 11.5 seconds). The patient was given intramuscular vitamin K and also fresh frozen plasma. Bilateral frontal and posterior parietal burr holes revealed a right chronic subdural hematoma and a left subdural hygroma. Percutaneous aspiration of the subdural space was performed postoperatively because of a persistent mild left upper extremity paresis and hypalgesia. No further hematoma was evacuated. Gradual, but
significant, neurologic improvement was noted during his subsequent hospitalization. He was discharged on the 9th postoperative day with only minor weakness in his left arm. He has been asymptomatic over the ensuing 4 years.

Discussion

Unlike most patients with CSDHs who present with impairment of level of consciousness and/or mentation, our patients showed only transient signs of focal cerebral dysfunction. In 1733, Schwenke gave the first description of this phenomenon. Review of our own and 6 other reported cases (table) show that dysphasia is almost always part of the presentation. Loeb found dysphasia in 62% of patients with TIA. However McKissock reported dysphasia in only 18% of his patients with CSDHs.

Transient neurologic dysfunction has been reported with extracerebral masses, although the pathophysiology remains unclear. Melamud suggested 3 possibilities: 1) compression of vessels subjacent to the hematoma, 2) post-ictal cortical suppression, and 3) spreading cortical depression after mechanical stimulation of the cortex. McLaurin postulated that neurologic deterioration in patients with chronic subdural hematomas is sometimes due to increases in regional cerebral edema and not to hematoma enlargement. He also demonstrated that minor changes in vascular displacement are sometimes accompanied by major changes in clinical signs and symptoms. Therefore, a transient increase in regional swelling may cause a critical degree of vascular displacement and consequent ischemia. The focality and transience of the symptoms may be due to local differences in both venous and arterial collateral circulation.

We cannot exclude the possibility that these patients with CSDHs had coincidental TIA. However, the fact that symptoms did not recur in 3 of 4 patients after evacuation of the CSDH suggests a causal relationship.

Regional cerebral blood flow (rCBF) measurements in areas adjacent to CSDHs have not been performed. A diffuse hemispheric decrease in flow has been noted.
by Brodersen, but the finding was thought to be secondary to reduced metabolic demand and not ischemia. CSDHs may cause a noncritical decrease in rCBF that lowers the threshold for regional ischemia due to global decreases in CBF or to regional emboli. The effects of CSDH and cardiovascular phenomena responsible for transient cerebral ischemia may thus be additive, with neither alone causing a critical decrease in rCBF.

Barnett has recently demonstrated that aspirin therapy decreases the incidence of TIAs in some groups of men. However, any type of anticoagulant therapy is contra-indicated in patients with a CSDH because such treatment may encourage hemorrhage from the hematoma capsule. Weisberg reported that the incidence of unsuspected intracranial structural lesions may be as high as 15% in patients who are thought to have TIAs. We therefore recommend that a CAT scan be considered in all patients with suspected TIAs before instituting any type of anticoagulation therapy. SDHs that appear isodense on a CAT scan may suggest an improper diagnosis.

References


### Table: Summary of Published Cases of CSDHs Presenting as Transient Neurologic Deficits

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age</th>
<th>Cause of CSDH</th>
<th>Presenting signs and symptoms</th>
<th>Follow-up: Duration and neurologic status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>53</td>
<td>Unknown</td>
<td>Expressive dysphasia, right upper extremity paresis</td>
<td>3 years: one episode of transient dysphasia</td>
</tr>
<tr>
<td>Case 2</td>
<td>80</td>
<td>Head injury with 6th nerve palsy 2 months PTA</td>
<td>Expressive dysphasia</td>
<td>1 ½ years: Asymptomatic</td>
</tr>
<tr>
<td>Case 3</td>
<td>73</td>
<td>Unknown</td>
<td>Expressive dysphasia, right lower facial paresis, incontinence</td>
<td>2 years: Asymptomatic</td>
</tr>
<tr>
<td>Case 4</td>
<td>81</td>
<td>Coumadin</td>
<td>Dysarthria, left hemiparesis and hemihyphalgia</td>
<td>4 years: persistent mild left upper extremity paresis</td>
</tr>
<tr>
<td>Groch</td>
<td>59</td>
<td>Head injury 2 weeks PTA</td>
<td>Global dysphasia, right hemiparesis</td>
<td>Postoperative recovery</td>
</tr>
<tr>
<td>Melamud</td>
<td>59</td>
<td>Unknown</td>
<td>Expressive dysphasia, right hand paresis and paresthesias</td>
<td>6 months: Asymptomatic</td>
</tr>
<tr>
<td>Okihiro</td>
<td>44</td>
<td>Head injury 6 weeks PTA</td>
<td>Expressive dysphasia, right hand paresis</td>
<td>Postoperative recovery</td>
</tr>
<tr>
<td>Robin</td>
<td>64</td>
<td>Head injury</td>
<td>Post-CSDH evacuation; expressive dysphasia, right upper extremity and facial paresis</td>
<td>Complete recovery after a second evacuation</td>
</tr>
<tr>
<td>Endtz</td>
<td>?</td>
<td>Head injury</td>
<td>Dysphasia, right hemiparesis, amaurosis, drop attacks</td>
<td>Death: autopsy verification of a chronic hygroma</td>
</tr>
</tbody>
</table>
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J E Welsh, G W Tyson, H R Winn and J A Jane

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