Hemichorea Associated with Ipsilateral Chronic Subdural Hematoma
—Case Report—

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Abstract
Left-sided hemichorea developed suddenly in a 73-year-old male. Computed tomography revealed a left subdural hematoma (SDH) and infarction in the right corona radiata and temporo-occipital region. Hemichorea subsided completely after removal of the SDH. Postoperative single photon emission computed tomography with technetium-99m-hexamethyl-propyleneamine oxime revealed a global low-perfusion area in the right cerebral hemisphere. Right carotid angiography demonstrated severe stenosis of the trunk of the right middle cerebral artery. The cerebral blood flow in the right cerebral hemisphere had probably already decreased to nearly the critical level and was reduced further by the left SDH, inducing the left-sided hemichorea due to dysfunction of the right cerebral hemisphere. This case shows that when hemichorea ipsilateral to a SDH is present, it is important to ascertain whether there is a pre-existing ischemic lesion in the contralateral cerebral hemisphere, particularly in the basal ganglia, thalamus, or corona radiata.

Key words: hemichorea, subdural hematoma, cerebral blood flow, ischemia

Introduction
The etiology of chorea varies widely, but chorea associated with chronic subdural hematoma (SDH) is rare. We report a patient with acute hemichorea associated with ipsilateral chronic SDH.

Case Report
A 73-year-old male complaining of mild headache was admitted in February, 1990. Neurological examination showed no abnormalities. Computed tomographic (CT) scans revealed bilateral subdural effusion and a small infarct in the right corona radiata (Fig. 1). Mild headache was resolved by intravenous administration of glycerol over several days.

He was readmitted in July, 1990, complaining of acute onset of involuntary movements in the left upper and lower extremities. Blood pressure was 140/72 mmHg and a general physical examination was normal. Neurological examination revealed constant, involuntary, irregular, purposeless, and non-rhythmic movements of the left upper and lower extremities. These choreiform movements disappeared during sleep and were aggravated by psychological stress or resting posture. No similar movements were detected in the right upper and lower extremities or in the face. He was fully awake and alert. He denied any history of head trauma, or alcohol or drug abuse. Tactile and pain sensation were slightly impaired on the left body. There was no motor weakness or sensory disturbance in the right upper and lower extremities. Deep tendon reflexes were in-
creased in the left upper and lower extremities. The plantar response was flexor on the right, extensor on the left.

CT scans on readmission disclosed a left chronic SDH and infarcts in the right corona radiata and temporo-occipital region (Fig. 2). The anterior horn of the left lateral ventricle was compressed. The midline structures were slightly shifted to the right. The left-sided hemichorea was probably due to dysfunction of the right cerebral hemisphere caused by the left chronic SDH.

Approximately 20 ml of hematoma was evacuated through a single burr hole. On the 14th postoperative day, single photon emission CT (SPECT) scans with technetium-99m-hexamethyl-propyleneamine oxime (99mTc-HMPAO) demonstrated a global low-perfusion area in the right cerebral hemisphere (Fig. 3). Right carotid angiograms revealed severe stenosis of the trunk of the right middle cerebral artery (Fig. 4). Magnetic resonance (MR) images clearly showed cerebral infarcts in the right corona radiata and temporo-occipital region, and reduced mass effect due to the left chronic SDH, but no lesion in the basal ganglia, thalamus, or brainstem (Fig. 5).

The choreiform movements became markedly less after hematoma evacuation and subsided completely in 1 month without medication. He was discharged ambulatory 5 weeks after the operation.
Fig. 5 Postoperative SE MR images, showing cerebral infarcts in the right corona radiata and temporop-occipital region. No ischemic lesion is found in the basal ganglia, thalamus, or brainstem. upper: 500/25 msec, lower: 2000/90 msec.

Discussion

Involuntary Parkinsonian\(^{16,17}\) or choreiform movements\(^{3,10,15,23}\) in association with SDH is rare. Table 1 summarizes the six reported cases of choreiform movements associated with chronic SDH. There were two bilateral and four unilateral hematoma cases. Generalized choreiform movements were observed in two bilateral and in two unilateral cases. Hemichorea was observed in two cases of unilateral hematoma ipsilateral to the SDH. In all six cases, choreiform movements subsided immediately, or were markedly reduced and resolved completely within 1 month of removal of the hematoma.

Hemichorea is a relatively uncommon movement disorder that usually follows an ischemic\(^{9,11,12,17}\) or hemorrhagic\(^{21}\) lesion in the contralateral caudate nucleus or putamen,\(^{8}\) corona radiata,\(^{2}\) thalamus,\(^{11}\) or subthalamic nucleus.\(^{21}\) Lesions outside these structures may also be associated with hemichorea.\(^{5,11,12}\) However, in Vincent’s\(^{23}\) and our cases, hemichorea ipsilateral to the hematoma was observed. Vincent described a 83-year-old male with a right chronic SDH manifesting as ipsilateral hemichorea. CT scans showed no ischemic or hemorrhagic lesion possibly responsible for the right-sided hemichorea in the left cerebral hemisphere. However, he speculated that the right-sided hemichorea might be due to a pre-existing ischemic lesion in the contralateral thalamic or subthalamic areas. In our case, CT scans detected a pre-existing ischemic lesion in the contralateral cerebral hemisphere.

Our patient was unusual as a small amount of left SDH had probably caused dysfunction of the right cerebral hemisphere without manifesting localizing signs in the left cerebral hemisphere. Also, there were pre-existing ischemic lesions. Usually, chronic SDHs of 40–60 ml cause no symptoms.\(^{14}\) However, some reports\(^{4,7,13,19,20,22}\) on patients with chronic SDH suggested that: 1) the mean hemispheric cerebral blood flow (CBF) decreased on both the side with hematoma and contralaterally; 2) the CBF reduction was always greater in the putamen and thalamus than in the cortex; and 3) reduced CBF occurred even in patients with only headache and minimal or no brain shift on CT scan (hematoma volume 20–70 ml).

We therefore concluded that in our patient the CBF in the right cerebral hemisphere had already decreased to nearly the critical level as suggested by postoperative SPECT with \(^{99m}\)Tc-HMPAO and cerebral angiography. The CBF was reduced further

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age</th>
<th>Sex</th>
<th>Side of hematoma</th>
<th>Choreiform movement</th>
<th>Onset</th>
<th>Hematoma volume (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bean and Ladisch (1977)(^3)</td>
<td>12</td>
<td>M</td>
<td>lt</td>
<td>generalized</td>
<td>gradual</td>
<td>250</td>
</tr>
<tr>
<td>Bae et al. (1980)(^1)</td>
<td>57</td>
<td>F</td>
<td>bil</td>
<td>generalized</td>
<td>acute</td>
<td>40 (rt), 80 (lt)</td>
</tr>
<tr>
<td>Vincent (1980)(^2)</td>
<td>83</td>
<td>M</td>
<td>rt</td>
<td>rt-sided hemichorea</td>
<td>gradual</td>
<td>?</td>
</tr>
<tr>
<td>Kotagal et al. (1981)(^10)</td>
<td>73</td>
<td>M</td>
<td>bil</td>
<td>generalized</td>
<td>?</td>
<td>80 (rt), 80 (lt)</td>
</tr>
<tr>
<td>Saito et al. (1982)(^13)</td>
<td>52</td>
<td>M</td>
<td>rt</td>
<td>lt-sided hemichorea</td>
<td>gradual</td>
<td>100</td>
</tr>
<tr>
<td>Present case</td>
<td>73</td>
<td>M</td>
<td>lt</td>
<td>lt-sided hemichorea</td>
<td>acute</td>
<td>20</td>
</tr>
</tbody>
</table>

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by the left chronic SDH, inducing the left-sided hemichorea due to dysfunction of the right cerebral hemisphere. The CBF in the left cerebral hemisphere might not have decreased sufficiently to cause localizing sign. This case shows that when hemichorea ipsilateral to a SDH is present, it is important to ascertain whether there is a pre-existing ischemic lesion in the contralateral cerebral hemisphere and that a small volume of SDH may cause neurological deficits in a patient with pre-existing ischemic lesion.

References


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