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## TRANSCORTICAL MOTOR APHASIA DUE TO A SUBDURAL HEMATOMA

*To the Editor:*—Emergency physicians are well aware that acute or subacute aphasias typically result either from cerebral infarction or an intracerebral mass such as a tumor or hemorrhage.<sup>1</sup> Well-characterized aphasia syndromes correlate well with distinct cortical lesions in the dominant hemisphere.<sup>2</sup> Wernicke's and other fluent aphasias, characterized by well-articulated speech with abnormal content, usually localize to the parietal or temporal lobes. Nonfluent aphasias such as a Broca's, typified by slow, incorrectly articulated words, are caused by frontal lesions.<sup>1</sup>

Rarely, aphasias can also be presenting symptoms of subdural hematomas (SDHs).<sup>3-13</sup> Here we describe a patient with a transcortical motor aphasia (TCMA) due to a SDH and then discuss its possible pathogenesis and implication in the management of aphasic patients in the emergency department.

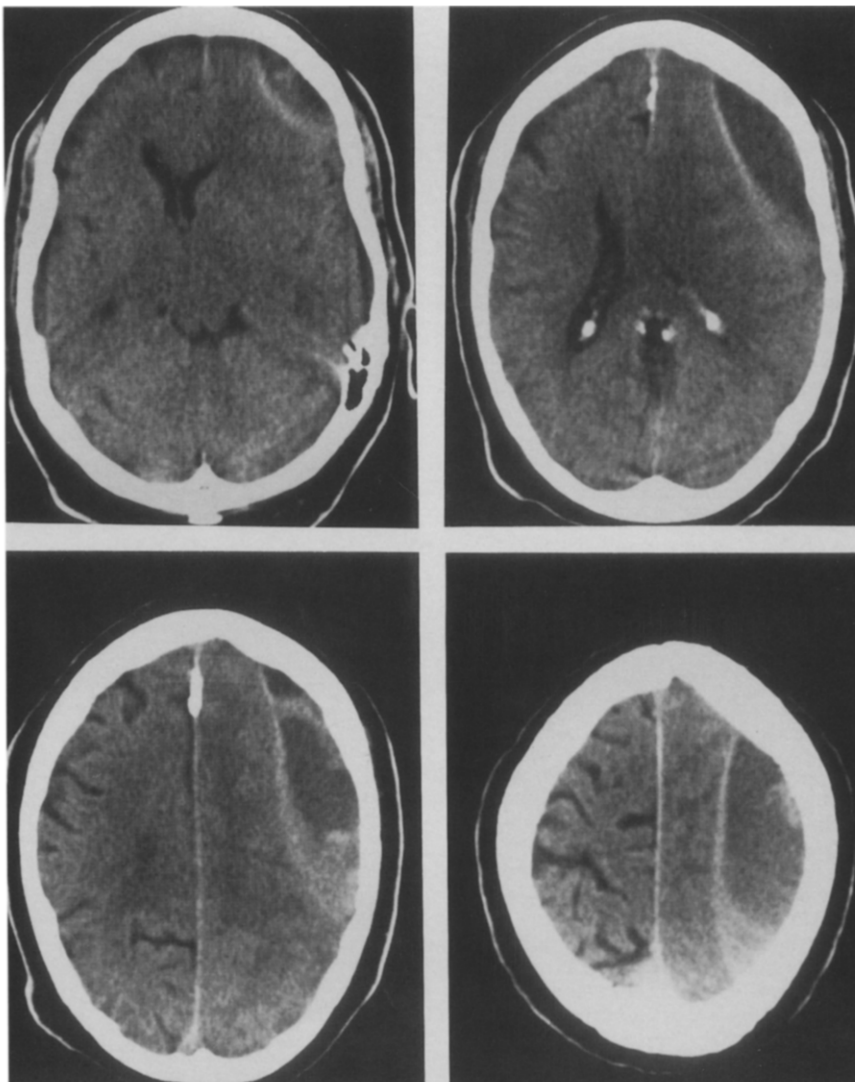
A 53-year-old right-handed man was evaluated in our emergency room for complaints of right-sided weakness and difficulty "getting the words out." The patient stated his symptoms began 3 weeks earlier when he fell and struck his head. His appetite suffered, and his nephew brought the patient for a medical evaluation after he found him disheveled and "confused." He had no headache. His past medical history was remarkable for hypertension, diabetes, gout, and alcoholism.

On physical examination upon admission the patient had a blood pressure of 170/90 mm Hg and a pulse of 70 per minute, and he was afebrile. He was alert and attentive, though he spoke little spontaneously. His speech in response to questions was halting but without dysarthria or paraphasic errors. He was able to understand spoken as well as written words, and he could read aloud. Upon verbal command he was able to whistle, protrude his tongue, and pretend to blow out a match, but he had difficulty waving goodbye with either hand. He named a watch and a pen, but could not name the watch's face, hands, or buckle, or the pen's tip or cover. He was unable to write with either hand. However, he was able to repeat even a phrase as complex as "no ifs ands or buts."

His visual fields were full to confrontation, his optic discs were sharp, and his pupils and eye movements were normal. There were no sensory abnormalities. The right face, arm, and leg were mildly paretic, and the right brachioradialis, biceps, and triceps reflexes were increased compared with the left. The ankle jerks were absent, and the right plantar reflex was extensor while the left was flexor.

Computerized tomography (CT) of the head (Figure 1) revealed a large left frontoparietal, extra-axial hypodense fluid collection containing scattered foci of hyperintensity. The fluid was lined by a convex hyperdense inner rim. Effacement of the left lateral ventricle accompanied left to right midline shift. The findings were thought to be consistent with a chronic SDH with an acute hemorrhagic component and significant mass effect.

The patient underwent a left frontoparietal craniotomy and evacuation of a chronic SDH encapsulated by well-organized inner and outer membranes. The hematoma contained acute as well as old liquefied blood. Ten by eight-centimeter windows in both the inner and outer membranes were removed. Pathologic examination revealed fresh and organizing blood clot as well as hemosiderin depo-



**FIGURE 1.** Computed tomographic scan of the brain shows a large left frontoparietal hypodense fluid collection with scattered areas of hyperdensity consistent with a chronic subdural hematoma with acute components. The subdural hematoma caused a transcortical motor aphasia.

sition and hemosiderin-containing macrophages, indicating old hemorrhage. By 2 days after surgery he had dramatically improved: his speech was fluent, he could write with only minor errors, and his hemiparesis was almost resolved. A follow-up CT on the third postoperative day demonstrated only mild residual fluid and little mass effect.

The diagnosis of TCMA was established based upon his nonfluent spontaneous speech, agraphia, difficulty naming but intact auditory comprehension, reading, and repetition.<sup>2</sup> His mild right hemiparesis, limb apraxia involving even the stronger arm, and normal sensory examination results and visual fields were also consistent with a TCMA.<sup>2</sup>

Patients with TCMA typically have lesions which involve either the supplementary motor area (SMA) or the subcortical white matter anterolateral to the frontal horn of the left lateral ventricle, thereby disconnecting fibers from the SMA to Broca's area.<sup>2,14,15</sup> The SMA, which lies anterior to the primary motor cortex on the medial surface of hemisphere, mediates the initiation and planning of movement and speech.<sup>15</sup> Intact repetition, the hallmark of transcortical aphasias, remains because the lateral inferior suprasylvian pathways are spared.<sup>15</sup>

The resolution of our patient's TCMA after evacuation of the SDH implies a causal relationship. The postoperative CT scan showed no other responsible cerebral infarction or mass lesion. To

our knowledge, this association between a TCMA and a SDH has not been previously reported.

Subdural hematomas, caused by hemorrhage from bridging veins between the dura and arachnoid, are almost always associated with head trauma. The blood typically lies over the convexity atop the frontal and parietal lobes, and in some cases is bilateral. Patients usually complain of a headache, and they may have mild mental status changes and a hemiparesis contralateral to the hematoma; aphasia is uncommon. Surgical evacuation is the treatment of choice.<sup>16</sup>

How could a SDH overlying the lateral aspect of the left frontal and parietal lobes cause a TCMA, which is usually associated with a deep white matter or a parasagittal cortical lesion? Subdural hematomas can cause epileptiform discharges; however, seizures involving language areas usually manifest themselves as speech arrest.<sup>17</sup> Daly et al<sup>18</sup> suggested mass lesions can cause neurologic symptoms by mechanical stimulation of the cortex followed by a spreading depression of cerebral electrical activity,<sup>19</sup> but this effect would be too diffuse to cause a focal symptom. Direct compression or hemispheric pressure differences created by the SDH<sup>20</sup> would also be too widespread. We cannot exclude the possibility that these diffuse processes may have induced the expression of an otherwise subclinical focal lesion.

Vascular insufficiency, due either to compression of adjacent ce-

rebral vessels<sup>8,18</sup> or vascular displacement,<sup>6,11</sup> is a more plausible explanation for a focal deficit in our patient. Branches of the middle cerebral artery supply the white matter adjacent to the frontal horn of the lateral ventricle. The SMA lies in a division of the anterior cerebral artery, which may be compressed against the gyrus rectus proximally or the falx cerebri distally in cases of subfalxian herniation.<sup>6</sup> In three patients similar to ours with convexity SDHs complicated by midline shift, McLaurin angiographically demonstrated loss of flow in the anterior cerebral artery which subsequently returned after surgical drainage.<sup>6</sup>

Although CT scanning is already common in clinical practice, this case reinforces the importance of prompt cranial imaging studies in the emergency department evaluation of patients with aphasia and symptoms otherwise associated with focal lesions. Aphasic patients may be unable to give a history of head trauma, a fall, or a headache. Those with an SDH should receive immediate neurosurgical consultation. Like ours, such patients may respond dramatically to surgical evacuation of the hematoma. Emergency physicians should be keenly aware that in addition to strokes, intracerebral hemorrhages, and tumors, SDHs may lead to classic aphasia syndromes, perhaps as a result of vascular insufficiency.

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## LINGUAL TONSILLITIS: AN UNCOMMON CAUSE OF AIRWAY COMPROMISE RESPONSIVE TO EPINEPHRINE

*To the Editor:*—Lingual tonsillitis is a rare event that harbors a significant potential for airway compromise. The use of epinephrine for treatment of this airway compromise has not been previously reported. We report the case of an adult with lingual tonsillitis who developed respiratory distress responsive to parenteral epinephrine.

A 35-year-old white woman presented to the emergency department with a 2-day history of progressive sore throat associated with increasing malaise, headache, and stiff neck. She noted a temperature of 38.3°C 24 hours prior to presentation and she had been having chills for 12 hours. She reported some nausea and photophobia, but denied vomiting. Over the preceding 12 hours she noted a progression of the sore throat. Swallowing, even her own saliva, and speaking caused severe discomfort. She presented with a towel in her mouth, which she used to collect her saliva to keep it from going to the back of her throat. She denied cough, earache, chest pain, or diarrhea.

Her past medical history was positive for primary hypothyroidism treated with synthroid, and mixed connective tissue disease treated with diflunisal. She had completed a course of steroids 10 days prior to the onset of her symptoms for an exacerbation of her connective tissue disease. She stated she had not taken her usual medications for 24 hours due to her progressive sore throat.

Physical examination revealed a well-nourished white female in obvious distress, with a small towel clenched in her mouth. The patient sat leaning forward and declined to lay back for examination, stating that she felt it was harder to breathe when recumbent. Vital signs included a blood pressure of 108/70 mm Hg, pulse rate of 72 beats per minute, respirations of 20, and a temperature of 36.8°C. Head, eyes, ears, nose, and throat examination was pertinent for markedly dry mucous membranes, and a slightly erythematous pharynx with no obvious exudate. The neck was stiff and had shoddy anterior and posterior cervical adenopathy. The chest was clear to auscultation, and heart examination revealed a regular rate and rhythm without murmur. The abdomen was soft without organomegaly or masses, and was nontender. The skin was warm and dry without rash. The neurologic examination was intact.

The diagnosis of acute epiglottitis was entertained and a soft tissue lateral cervical radiograph was obtained (Figure 1A). As can be seen, the epiglottis is normal in appearance, with some scalloping of the anterior surface of the vallecula. Figure 1B is a line drawing of the radiograph shown in Figure 1A highlighting the epiglottic area.

Due to the abnormality noted on the lateral soft tissue cervical radiograph, an otolaryngology consultant was obtained and an oral endoscopic procedure performed. Only the edge of the epiglottis was viewed and it was normal. One gram of ceftriaxone was given intravenously. A computed tomographic scan of her head without contrast was obtained and was normal. A lumbar puncture revealed an opening pressure of 26 cm of water, and the cerebrospinal fluid was observed to be clear and colorless.

Immediately after the lumbar puncture, the patient sat upright and indicated that she was having trouble breathing. Significant respiratory distress associated with inspiratory and expiratory stridor was