Transient Neurological Symptoms in Patients With Intracerebral Hemorrhage

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Intracerebral hemorrhage (ICH) is a particularly ominous stroke subtype that carries high rates of mortality and morbidity. Prompt recognition and appropriate management are necessary to affect outcomes in these patients. Usually, ICH presents with an abrupt onset of neurological deficits that often progress over time; nausea, vomiting, impairment of consciousness, and significant hypertension at presentation are common features. However, in a significant number of patients, recovery from neurological symptoms starts within a few hours after onset. Most hemorrhages in this cohort were small, with a mean (SD) hematoma volume of 17 (9.9) mL, and were subcortical in location. One patient died of hemorrhage recurrence.

Conclusions and Relevance
Patients with ICH can present with rapidly resolving deficits resembling transient ischemic attacks. Recognition of these instances is important to avoid delays in investigations and to manage these cases appropriately.

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Intracerebral hemorrhage (ICH) is a particularly ominous stroke subtype that carries high rates of mortality and morbidity. Prompt recognition and appropriate management are necessary to affect outcomes in these patients. Usually, ICH presents with an abrupt onset of neurological deficits that often progress over time; nausea, vomiting, impairment of consciousness, and significant hypertension at presentation are common features. However, in a significant number of patients, the deficits are less pronounced and brain imaging is needed to differentiate ICH from an ischemic stroke. Whereas temporary symptoms have been observed in patients with subdural hematomas and subarachnoid hemorrhages due to amyloid angiopathy, transient deficits resembling transient ischemic attacks (TIAs) have not been reported, to our knowledge, in patients with ICH.

We encountered a patient in our practice who had developed a transient language impairment from a left temporal hemorrhage mimicking a TIA. This prompted us to systematically search the medical records at our institution to assess the frequency of TIA-like presentations of ICH, analyze their presenting symptoms and signs as well as imaging patterns, and assess prognosis.

Methods
In this clinical case series, we identified all patients with ICH using International Classification of Diseases, Ninth Revision code 431 from our hospital database from June 1, 2000, to August 31, 2014. Discharge summaries of all records were individually reviewed to exclude patients with traumatic ICH, subarachnoid and subdural hemorrhages, secondary ICH from an underlying neoplasm, and ICH from hemorrhagic transformation of an ischemic infarct. We collected and analyzed demographic information as well as clinical and imaging findings on all remaining patients with a spontaneous ICH. Patients who had transient deficits (symptoms, signs, or both) from an ICH that had resolved on a repeated examination within 24 hours were eligible. The study was approved by the institutional review board at the Beth Israel Deaconess Medical Center. A waiver of
informed consent was granted by the institutional review board owing to the low-risk, retrospective nature of this study.

**Results**

We identified 3207 patients with ICH using *International Classification of Diseases, Ninth Revision* code 431. Among these, 2137 patients had a spontaneous ICH without any coexisting subarachnoid or subdural hemorrhage, neoplasm, or secondary hemorrhagic transformation from a cerebral infarct. Of these, 34 patients had transient deficits, which were defined as clinical symptoms and signs that had resolved within 24 hours. These records were further reviewed to ascertain details of history and imaging findings and to discard records in which significant clinical information and details about the duration of symptoms were lacking (12 cases) or in which additional conditions may have confounded the clinical presentation (5 cases). A cohort of 17 patients was assembled (eAppendix in the Supplement). The baseline characteristics of this cohort are summarized in the Table.

**Table. Baseline Characteristics of the Patient Cohort**

<table>
<thead>
<tr>
<th>Sex/Age, y</th>
<th>Symptom</th>
<th>Initial Examination Findings</th>
<th>Duration</th>
<th>Hemorrhage Volume, mL</th>
<th>Site</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>M/mid-20s</td>
<td>Right-sided paresthesias</td>
<td>BP 120/90 mm Hg; sensory loss; NIHSS score 2</td>
<td>&lt;24 h</td>
<td>11.7</td>
<td>Left subinsular region</td>
<td>Cavernoma</td>
</tr>
<tr>
<td>M/mid-70s</td>
<td>Left foot numbness, arm cluminess, slurred speech</td>
<td>BP 198/92 mm Hg; left pronator drift, extinction; NIHSS score 2</td>
<td>4 h</td>
<td>14.9</td>
<td>Right internal capsule</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>F/mid-60s</td>
<td>Headache, left tongue and lip numbness</td>
<td>BP 200/100 mm Hg; normal NIHSS score 0</td>
<td>30 min</td>
<td>7</td>
<td>Right basal ganglia</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>M/early 60s</td>
<td>Severe word-finding difficulties</td>
<td>BP 220/98 mm Hg; normal NIHSS score 0</td>
<td>30 min</td>
<td>29</td>
<td>Left temporal</td>
<td>Probable amyloid angiopathy</td>
</tr>
<tr>
<td>F/late 50s</td>
<td>Nausea, vomiting, left-sided weakness, dysarthria</td>
<td>BP 120/80 mm Hg; mild left facial droop, left pronator drift; NIHSS score 2</td>
<td>30 min</td>
<td>32.6</td>
<td>Right internal capsule</td>
<td>Possibly hypertensive</td>
</tr>
<tr>
<td>M/early 70s</td>
<td>Speech arrest</td>
<td>BP 201/89 mm Hg; mild anemia, right facial droop; NIHSS score 3</td>
<td>15 min</td>
<td>14.8</td>
<td>Left temporal</td>
<td>Hypertensive or amyloid angiopathy</td>
</tr>
<tr>
<td>M/early 70s</td>
<td>Dizziness, slurred speech, gait unsteadiness</td>
<td>BP 176/100 mm Hg; mild dysarthria, left pronator drift; NIHSS score 2</td>
<td>30 min</td>
<td>10.7</td>
<td>Left basal ganglia</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>F/early 80s</td>
<td>Dizziness, near syncope</td>
<td>BP 140/97 mm Hg; mild lethargy; NIHSS score 1</td>
<td>5 min</td>
<td>8.5</td>
<td>Left internal capsule</td>
<td>Possible amyloid angiopathy</td>
</tr>
<tr>
<td>M/early 50s</td>
<td>Dizziness, gait unsteadiness, mild confusion</td>
<td>BP 100/58 mm Hg; mild inattention, pronator drift; NIHSS score 1</td>
<td>&lt;1 h</td>
<td>9.8</td>
<td>Left basal ganglia</td>
<td>Unclear</td>
</tr>
<tr>
<td>F/mid-80s</td>
<td>Right leg numbness</td>
<td>BP 210/59 mm Hg; left leg sensory loss; NIHSS score 2</td>
<td>30 min</td>
<td>7.7</td>
<td>Left lateral thalamic</td>
<td>Possibly hypertensive</td>
</tr>
<tr>
<td>M/mid-60s</td>
<td>Right hemiparesis, dysarthria, gait unsteadiness</td>
<td>BP 178/95 mm Hg; right hemiparesis, dysmetria; NIHSS score 4</td>
<td>2-3 h</td>
<td>24</td>
<td>Left basal ganglia</td>
<td>Anticoagulation, possibly hypertensive</td>
</tr>
<tr>
<td>F/early 80s</td>
<td>Left leg weakness</td>
<td>BP 200/90 mm Hg; left leg drift; NIHSS score 1</td>
<td>30 min</td>
<td>18</td>
<td>Right internal capsule</td>
<td>Anticoagulation, possibly hypertensive</td>
</tr>
<tr>
<td>M/late 60s</td>
<td>Left arm cluminess, facial droop, slurred speech</td>
<td>BP 182/70 mm Hg; mild left ataxia, dysarthria; NIHSS score 3</td>
<td>5 h</td>
<td>21</td>
<td>Right thalamus</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>M/early 60s</td>
<td>Dysarthria, hand cluminess</td>
<td>BP 141/93 mm Hg; mild dysarthria, limb ataxia; NIHSS score 2</td>
<td>Possibly 6-8 h</td>
<td>7</td>
<td>Left lentiform nucleus</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>F/mid-60s</td>
<td>Dysarthria, headache</td>
<td>BP 197/93 mm Hg; left hemiparesis; NIHSS score 5</td>
<td>&lt;12 h</td>
<td>9.4</td>
<td>Right basal ganglia</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>M/late 40s</td>
<td>Left hemiparesis</td>
<td>BP 132/73 mm Hg; left hemiparesis; NIHSS score 4</td>
<td>&lt;6 h</td>
<td>23</td>
<td>Right putamen</td>
<td>Moyamoya disease</td>
</tr>
<tr>
<td>M/mid-40s</td>
<td>Slurred speech, right leg weakness, gait difficulty</td>
<td>BP 220/120 mm Hg; mild right hemiparesis, aphasia, ataxia; NIHSS score 5</td>
<td>&lt;8 h</td>
<td>40.4</td>
<td>Left putamen</td>
<td>Hypertensive</td>
</tr>
</tbody>
</table>

**Key Points**

**Question:** Do transient signs and symptoms occur with intracerebral hemorrhages?  
**Findings:** In this case series involving 17 carefully selected hospitalized patients, rapidly resolving signs and symptoms resembling transient ischemic attacks were documented. All patients had normal results on neurological examination within 24 hours after symptom onset.  
**Meaning:** Patients with rapidly resolving stroke-like symptoms should undergo prompt brain imaging, even in the context of normal findings on neurological examination, to rule out intracerebral hemorrhage, especially prior to initiating any antithrombotic or anticoagulant therapy.
The median age of the group was 65 years (interquartile range, 56-73 years); 11 of the 17 patients were men. All patients underwent a computed tomographic (CT) scan of the brain within 6 hours of symptom onset and had a documented detailed neurological examination at presentation as well as a subsequent progress note detailing the clinical course during the preceding 24 hours including a follow-up neurological examination within 24 hours.

**Clinical Presentation**

The trajectory of clinical improvement began early in all patients, usually within several minutes. In 9 patients, the deficits lasted less than 30 minutes, in 5 patients they lasted 6 hours or less, and only 1 patient had symptoms lingering more than 12 hours but less than 24 hours. The most common deficits were sensorimotor involving varying degrees of limb weakness, numbness, or incoordination. Three patients developed significant dysarthria and 2 others had major language impairment at onset; 3 patients had dizziness with gait unsteadiness. The typical symptoms of ICH including headache, nausea, vomiting, and reduction in levels of alertness were usually absent. Most patients had moderate to severe hypertension at presentation, although their neurological deficits were mild (mean [SD] National Institutes of Health Stroke Scale score, 2.3 [1.5]; range, 0-5) on initial neurological examination in the emergency department (Table). Five patients took antiplatelet medications either on their own accord or per the advice of their physicians prior to obtaining any imaging studies.

**Imaging Findings**

The prototypical imaging findings on CT scans at admission are shown in the Figure. Most of the hemorrhages were subcortical involving the basal ganglia or neighboring white matter tracts. Two involved the temporal lobes. No patients had intraventricular hemorrhage or hydrocephalus. All hemorrhages were small, with a mean (SD) volume of 17 (9.9) mL as measured by the ABC/2 method. In addition to the initial head CT scans, 11 patients underwent brain magnetic resonance imaging and magnetic resonance angiography, 5 underwent CT angiography of the head, and 2 underwent conventional angiography. In 16 patients who underwent vascular imaging, only 1 had an abnormality consistent with a unilateral moyamoya-type pattern. Of the 11 patients who underwent brain magnetic resonance imaging, 7 showed varying degrees of leukoaraiosis on fluid-attenuated inversion recovery sequences; 2 showed scattered microbleeds on gradient echo sequences; 1 displayed characteristic features of a cavernoma; and 1 showed no other significant abnormality apart from the acute ICH.

**Etiology**

Etiology of the hemorrhage was determined based on discharge diagnosis and review of all discharge summaries and
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REFERENCES


Follow-up

One patient died during hospitalization of recurrence of a massive ICH 4 days after admission. This patient had a pacemaker and did not undergo brain magnetic resonance imaging or vascular imaging. The etiology of her hemorrhage was unclear. Detailed follow-up information was available in 9 patients. The follow-up varied between 1 and 8 months. In all these patients there was no recurrence of symptoms suggestive of a stroke or TIA.

Discussion

Whereas clinical deterioration after ICH has been shown in several studies,6,2 to our knowledge no studies have reported rapid improvement in clinical deficits in patients with ICH. We adopted a 24-hour threshold for labeling a deficit as transient as this time cutoff has traditionally been used for defining TIAs.8 In most patients the improvement started early, usually within several minutes, and the symptoms and signs had resolved well before our adopted time threshold. This is likely an unusual presentation of ICH, although rapid improvement of symptoms with relatively minor deficits may have contributed to a referral bias and led to an underascertainment of cases. Only a prospective population-based study with detailed early neurological assessment and close monitoring can estimate the true incidence.

Our report carries significant clinical relevance. First, it shows that transient deficits from a minor ICH can mimic a TIA or minor infarct and thus require brain imaging for differentiation. Some of our patients received antplatelet medications prior to any imaging. While we did not observe any harm in this small group of patients, prescription of these agents can be hazardous in this situation. Second, this study highlights a need to obtain prompt brain imaging in all patients with suspected TIAs. In many settings, patients with TIAs are not routinely admitted for admission or referred to the emergency department.9,10 Previous reports have shown that even in patients presenting to the emergency department with symptoms of a TIA, CT scans are underused and only 50% to 70% of such patients undergo a scan.11 Delays in obtaining head CTs can substantially reduce the sensitivity of detecting a hemorrhage especially beyond a week.12 Third, in resource-limited settings, clinical scores to diagnose ICH have been derived and validated.13,14 It is worth noting that only a small minority of our patients could reliably be suspected of having an ICH based on their presenting features such as diminished consciousness, nausea, vomiting, headache, and severe hypertension. Our findings urge caution regarding the reliability of these clinical tools in identifying minor hemorrhages.

The biological underpinning driving such an early recovery after ICH is intriguing. The rapid resolution of symptoms may suggest a hemodynamic cause with spontaneous reduction in local mass effect from redistribution of blood along the tracts, especially in patients with subcortical bleeds. In 2 of our patients with lobar hemorrhages, focal seizures with postictal deficits may have played a role. Neuroplastic changes with cortical reorganization are also a possibility, although the time course is less typical.

The major limitation of our work lies in its retrospective nature, where documentation of a detailed neurological evaluation at predefined regular intervals was unavailable. Thus, we relied on the admission and progress notes as well as discharge summaries to record timing of recovery. We meticulously excluded cases in which this information was unavailable. The etiology of the hemorrhage was based on clinician impression, although we independently reviewed all records and imaging studies for corroboration. Routine follow-up information was not available in some of our patients and brain imaging was not repeated in many patients. Most patients with available information fared well, although there was 1 death from recurrent ICH.

Conclusions

This study highlights a previously unrecognized presentation of ICH and emphasizes its clinical relevance for patient care. Further studies are needed to better estimate its true incidence and understand the pathophysiological basis of this presentation.

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