Sensory Ataxic Hemiparesis in Thalamic Hemorrhage

J.L. Dobato, MD, J.A. Villanueva, MD, and S. Giménez-Roldán, MD

Ataxic hemiparesis with cerebellar-like features has been reported following infarction or hemorrhage of the thalamus. We describe five patients with incoordination and corticospinal tract signs in the limbs opposite a dorsolateral thalamic hemorrhage. In four patients the hemorrhage extended superiorly into the lateral ventricle, the adjacent paraventricular region, and the upper and medial parts of the posterior limb of the internal capsule. Instead of cerebellar dysfunction, these patients exhibited directional errors of movement that improved distinctly when performed under visual guidance. We explain the incoordination on the basis of conscious deep sensory loss in the involved limbs. Perception of light touch, painful pinprick, and temperature were preserved in all patients. We use the term “sensory ataxic hemiparesis” to distinguish these patients from those with “classic” ataxic hemiparesis and cerebellar-like features, a distinction that may be important when planning functional rehabilitation. (Stroke 1990;21:1749-1753)

Thalamic hemorrhage represents approximately 20–30% of all intraparenchymatous hemorrhages of the brain. Early mortality is high although some patients, particularly those in whom the hematoma is small, survive. In such patients residual disability is related to the topographic location of the hemorrhage. Some 76% of surviving patients suffer lesions involving the dorsolateral thalamic area and their functional prognosis is poor in that one third suffer severe associated hemiparesis and hemianesthesia. We describe five patients who survived a hemorrhage located in the dorsolateral thalamus and who exhibited incoordination of the contralateral limbs and severe impairment of proprioceptive sensation while preserving superficial modalities. Corticospinal tract signs were associated ipsilaterally, although only three exhibited rapidly resolving predominantly crural hemiparesis.

Subjects and Methods

Clinical details of the five patients (one woman and four men) are summarized in Table 1. Mean age was 41 (range 24–60) years, and four patients had a history of arterial hypertension. All patients were examined by the authors ≤24 hours after onset. Onset was abrupt, with associated headache in three patients, and included mild confusion and somnolence that subsided rapidly. Three patients initially had a predominantly crural hemiparesis, but there was no motor weakness in the other two despite pyramidal tract signs contralateral to the lesion. Three patients had abnormal ocular disturbances, which included sympathetic palsy in two and upward gaze palsy in another.

We performed a detailed sensory examination in all five patients between 4 and 90 days after the stroke, by which time they were fully conscious and cooperative. The examination included light touch perception, pinprick perception, and warm and cold sensation. The number of errors in perceiving the displacement of proximal and distal joints, two-point discrimination at established levels of the forearm and hand, and vibration sense when a tuning fork was placed on bony prominences at different body sites were compared between the unaffected side and the contralateral limbs and trunk. We asked the patients to identify by touch with both hands a series of different-sized coins, four tokens of varying shape, five small common everyday objects, and a series of three-dimensional geometric shapes.

In patients exhibiting position sense impairment, sensory ataxia was defined as poorly controlled direction of finger-to-nose and heel-to-knee movements or attempts to grasp small objects. Impairment of trajectory or placement was critically compensated for by visual cues. The sensory ataxia thus differed from the incoordination produced by cerebellar disease, in which the short side-to-side corrective movements characteristic of cerebellar dysmetria occur irrespective of whether they are performed under visual guidance.

Unenhanced computed tomography (CT) was carried out in all patients using either a CGR ND-8000 (256x256 matrix, 9-mm slice) or a Philips Tomoscan...
Results

The neurologic status of the patients at short-term follow-up, including the results of sensation testing, is shown in Table 2. Despite full recovery of strength in the affected upper limb, all patients displayed impaired coordination severe enough to interfere with normal everyday activities. Use of the hand was clumsy, objects were frequently dropped, and movements required close visual guidance. During finger-to-nose testing with their eyes closed, the patients typically misdirected their forefingers to their cheek or chin, redirecting movement toward the appropriate target only after the wrong spot had actually been touched. Visual guidance dramatically improved the accuracy of performance, yet movements remained persistently slow and clumsy.

Sustained postures were critically influenced by visual control. Holding the hands out in front with the fingers spread caused a hollow-hand sign, and when performed with the eyes closed, writhing movements of the fingers were sometimes followed by slow outward or upward displacement of the entire limb.

Impaired deep sensation was a consistent finding in all patients, although the severity varied widely. Cases 2, 3, and 4 were unable to reproduce passive movements of the proximal joints of the affected limb, while the other two patients perceived even small displacements of the interphalangeal joints and had intact vibration sense even though they suffered gross astereognosis of this hand. Incoordination was more severe in cases 2, 3, and 4, but even so it was disproportionately mild compared with the intensity of the sensory loss. When reexamined, all patients had recovered thermal sensitivity, touch sense for smooth objects, and painful pinprick sensation, although case 3 inconsistently claimed to perceive painful pinpricks with less intensity hemilateraly on the involved side.

Motor deficits had improved by the time of re-examination in most patients. Gait had returned to normal in case 1, crural paresis persisted in cases 2 and 4 (both of whom had developed spasticity in the affected leg), and case 3 walked with an incoordinated gait caused by hemisensory ataxia. The inability to stand unassisted and a deficit of axial motor control persisted in case 4.

The CT scans performed shortly after onset showed a hemorrhage involving the dorsolateral area of the thalamus in all patients. Mean volume of the hematoma was 7.2 (range 5.0–9.4) cm³. In four patients the hemorrhage extended superiorly to lie close to the body of the lateral ventricle (Figure 1). Ventricular bleeding presumably occurred at this level in three patients since in the more caudal slices a strip of intact tissue seemed to separate the bulk of the hemorrhage from the third ventricle. The hemorrhage extended into the adjacent paraventricular region and the posterolateral portions of the posterior limb of the internal capsule in all except case 3, in whom the hemorrhage extended toward the right upper midbrain.

Discussion

Incoordination of the limbs as a result of lesions involving the thalamus was recognized by Dejerine and Roussy in 1906. The cerebellar or sensory nature of the ataxic movements soon became a matter of controversy, as shown by the opposing views of Vincent in 1911 presented 2 years later at a session of the French Neurological Society. Cerebellar-like incoordination resulting from localized lesions in the thalamus has been widely accepted since the observations reported by Garcin in 1955, although the cerebellar features in his patient became evident only after the coexistent impairment of deep sensation had subsided.

All five of our patients displayed severe incoordination of the limbs contralateral to the thalamic

Table 1. Demographics and Clinical Features at Onset of Patients With Thalamic Hemorrhage

<table>
<thead>
<tr>
<th>Case/sex/age</th>
<th>Risk factors</th>
<th>History</th>
<th>Mode of onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/24 HBP</td>
<td>Pseudoxanthoma elasticum, cesarean delivery</td>
<td>Sudden</td>
<td></td>
</tr>
<tr>
<td>2/M/30 HBP</td>
<td>Insulin-dependent juvenile-onset diabetes mellitus</td>
<td>Sudden-progressive</td>
<td></td>
</tr>
<tr>
<td>3/M/42 None</td>
<td>None</td>
<td>Sudden</td>
<td></td>
</tr>
<tr>
<td>4/M/49 HBP</td>
<td>Recovered (except for minimal residual hemiparesis) from R lobar hemorrhage 2 years earlier</td>
<td>Sudden-progressive</td>
<td></td>
</tr>
<tr>
<td>5/M/60 HBP</td>
<td>None</td>
<td>Sudden</td>
<td></td>
</tr>
</tbody>
</table>

F, female; M, male; HBP, high blood pressure; L, left; B, bilateral; R, right.
hemorrhage. While attempting volitional movements such as finger-to-nose tests with their eyes closed, these patients made gross directional errors and as a consequence the forefingers were guided to portions of the face far from the intended target. The patients usually became aware of such misdirected movement only after the wrong part of the face had been touched, when the movement was corrected. One distinguishing feature was the improved performance of movements executed under visual guidance, which results in only partial compensation in patients with cerebellar dysfunction.\(^6\)\(^7\) Moreover, in our patients the severity of incoordination correlated with the intensity of positional sense impairment, which was severe enough to leave three patients unable to perceive even gross displacements of the entire limb. We therefore suggest that incoordination in these patients was caused by sensory deafferentation, rather than by cerebellar dysfunction.

Because at onset three patients presented with hemiparesis ipsilateral to the limbs subsequently exhibiting incoordination and corticospinal signs, it seems appropriate to consider the relation of our cases to the syndrome of ataxic hemiparesis. Fisher and Cole\(^12\) and Fisher\(^13\) clearly established that incoordination in ataxic hemiparesis displays cerebellar features. These authors' observations and more recent reports indicate that this might be the result of damage inflicted on the dentatorubrothalamicocortical or corticopontocerebellar pathways at several levels\(^14\)-\(^16\) and have included observations of ataxic hemiparesis due to hemorrhage\(^17\) and infarction\(^18\) of the thalamus. The patients described in those reports had to-and-fro oscillations and terminal intention tremors suggestive of cerebellar-like dysfunction, but deep position sense was as a rule preserved. It also cannot be inferred that movements in our patients were clumsy because they were weak\(^19\) since paresis in the upper limb had subsided despite persistent ataxia and since two of our patients displayed no motor weakness at any time.

The term “ataxic hemiparesis” has sometimes been extended to include patients in whom poor coordination was readily explained on the basis of proprioception loss. “Sensory ataxic hemiparesis” may be a better term for such patients, to distinguish them from cases of “classic” ataxic hemiparesis with cerebellar-like features. Hypesthetic ataxic hemiparesis differs from that in our patients not only with respect to the cerebellar-like nature of the incoordination, but also in that the sensory loss may be global,\(^20\) that superficial sensation may be selectively impaired,\(^21\) or that pain may be the sole manifestation of the lesion,\(^22\) but in no cases has the impairment of deep conscious sensation selectively shown by our patients been reported. Features of sensory ataxic hemiparesis can be identified in reports in the literature combining deep sensory loss and pyramidal tract signs in both limbs unilaterally due to vascular lesions located in the pons,\(^23\)-\(^24\) internal capsule and corona radiata,\(^14\),\(^15\) and parietal cortex,\(^25\) but to our knowledge it has not been reported in thalamic hemorrhage.

Even though during the first 48 hours following the stroke two of our patients exhibited global hemisensory deficit, their sensory abnormalities involving light touch, painful pinprick, and temperature quickly subsided, leaving a monomodal loss of

<table>
<thead>
<tr>
<th>Presenting symptoms</th>
<th>Headache</th>
<th>Consciousness</th>
<th>Oculomotor disturbances</th>
<th>Motor</th>
<th>Sensory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild confusional state and headache</td>
<td>Mild</td>
<td>Confusion</td>
<td>None</td>
<td>No weakness; increased L deep reflexes and Babinski's sign</td>
<td>Mild joint position sense abnormality</td>
</tr>
<tr>
<td>Cephalalgia</td>
<td>Severe</td>
<td>Minimal drowsiness</td>
<td>Supranuclear gaze palsy</td>
<td>L hemiparesis, resolved in 4 days; mild residual crural paresis</td>
<td>Deep sense abnormalities in L limbs</td>
</tr>
<tr>
<td>Headache, hemiparesis, hemianesthesia</td>
<td>Severe</td>
<td>Minimal drowsiness</td>
<td>B miosis, R Horner's syndrome, supranuclear gaze palsy, upward nystagmus, pseudoabducens palsy</td>
<td>L hemiparesis, resolved in 7 days</td>
<td>Superficial hemisensory loss and L position sense disturbances</td>
</tr>
<tr>
<td>Unsteadiness</td>
<td>Absent</td>
<td>Drowsiness</td>
<td>None</td>
<td>No weakness; astasia</td>
<td>Rapidly resolving superficial sensory loss; severe position sense abnormalities in R limbs</td>
</tr>
<tr>
<td>Crural paresis</td>
<td>Absent</td>
<td>Minimal drowsiness</td>
<td>B miosis; R Horner's syndrome</td>
<td>Crural paresis progressing to hemiparesis, partially recovering in 7 days</td>
<td>Deep hemisensory loss</td>
</tr>
</tbody>
</table>
TABLE 2. Motor Deficits, Limb Incoordination, and Sensory Disturbances at Short-term Follow-up in Patients With Thalamic Hemorrhage

<table>
<thead>
<tr>
<th>Case</th>
<th>Days after stroke</th>
<th>Corticospinal tract dysfunction</th>
<th>Limb incoordination</th>
<th>Sensory modalities</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Deep</td>
</tr>
<tr>
<td>1</td>
<td>60</td>
<td>Normal strength; hyperactive L deep reflexes and extensor plantar responses</td>
<td>Mild L misdirectional movements and pass-pointing corrected under visual guidance; poor handling on L</td>
<td>L astereognosis</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>Normal upper limb strength, but marked crural paresis associated with hyperactive deep reflexes in L limbs and extensor plantar response</td>
<td>Gross incoordination of movements of L upper limb partially improved under visual control</td>
<td>Severe loss of position sense in both upper and lower limbs; tuning fork perceived up to iliac bone on L; no astereognosis</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>None</td>
<td>Severely incoordinated movements with L limbs improved when performed under visual control</td>
<td>All markedly impaired in L limbs</td>
</tr>
<tr>
<td>4</td>
<td>90</td>
<td>None</td>
<td>Gross directional impairment of R limb movements corrected under visual guidance; marked improvement 14 days later leaving hospital; to-and-fro dysemetrical movements in finger-to-nose test</td>
<td>Position sense of proximal and distal joints absent in R limbs, resolved 2 weeks later</td>
</tr>
<tr>
<td>5</td>
<td>25</td>
<td>Normal limb strength; hyperactive L deep reflexes; L Babinski’s response; mild lower limb spasticity</td>
<td>Gross errors in directional movements partially improved under visual control</td>
<td>Position sense preserved in upper limbs; 30% error rate in joint sense at the L toe; L astereognosis</td>
</tr>
</tbody>
</table>

L, left; R, right.

deep sensation in all cases. Such selective impairment of kinesthetic and proprioceptive sensation does not occur in patients with Dejerine-Roussy syndrome or those with pure sensory stroke due to infarction or hemorrhage involving the territory of the thalamogeniculate artery, in whom sensory impairment is usually global. In the large series of patients with lateral thalamic infarcts reported by Bogousslavsky et al., five patients exhibited selective impairment of superficial sensation and 12 exhibited global loss, but none displayed the selective loss of conscious proprioceptive sensation shown by our patients.

FIGURE 1. Serial computed tomograms in case 4. A: No midline shift is seen, while blood fills third ventricle. B: High-density thalamic mass lesion involving left lateroventral area. Dorsomedial group is spared. C: Hemorrhage arising in upper thalamus may be seen extending into ipsilateral ventricle. Right parietal lobe hypodensity from previous lobar hemorrhage may be observed.
Most patients with thalamic hemorrhage develop hemiparesis due to compression or extension of the hemorrhage to the adjacent capsule.\textsuperscript{1–3} Larger hematoma can cause early depression of the level of alertness,\textsuperscript{4} presumably a reaction to distortion of the brain caused by horizontal displacement.\textsuperscript{5,6} In our patients the hematoma were relatively small, and therefore full consciousness returned soon after a mild initial impairment. The persistent ipsilateral crural paresis in two of our patients may have been caused by involvement of the fibers running to the leg cortex coursing through the posterior limb of the internal capsule near the wall of the ventricle. The ability of the thalamus to stand in case 4 is thought to have been caused by lesions primarily involving the superolateral portion of the thalamus.\textsuperscript{31} In this regard, it is interesting to note that in four of our patients the hemorrhage extended superiorly to the lateral ventricle and involving the upper and medial lateral thalamic hemorrhage extending superiorly to the lateral ventricle as well as into the adjacent periventricular wall.

The lemniscal pathways subserving proprioception, kinesthesia, and vibration sense and ascending in a thalamocortical projection through the posterior limb of the internal capsule presumably were interrupted in four of our patients. The medial lemniscal pathway could have been selectively destroyed in case 3, in whom the hemorrhage extended laterally to the upper midbrain. The dissociated nature of the sensory loss in the other patients, in whom the sensation of light touch, pain, and temperature remained unimpaired, is difficult to explain. Lesions involving the posterior third of the posterior limb of the internal capsule,\textsuperscript{21} or even those lying in a more posterior location\textsuperscript{32} frequently involving the adjacent thalamus, resulted in dysfunction exactly opposite that exhibited by our patients, namely, superficial hemisensory loss with unimpaired position sense. The pathways that convey conscious proprioceptive sense are imperfectly known and have been the subject of review in recent years.\textsuperscript{33} These pathways may be selectively damaged in lacunar stroke\textsuperscript{34} and dorso-lateral thalamic hemorrhage extending superiorly to the lateral ventricle and involving the upper and medial parts of the posterior limb of the internal capsule.

References


KEY WORDS • cerebral hemorrhage • hemiplegia • thalamus
Sensory ataxic hemiparesis in thalamic hemorrhage.
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doi: 10.1161/01.STR.21.12.1749

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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