Small Primary Intracerebral Hemorrhage
Clinical Presentation of 28 Cases

Jong S. Kim, MD; Jay H. Lee, MD; Myoung C. Lee, MD

Although there have been sporadic reports of patients with small intracerebral hemorrhages presenting with discrete clinical features, the clinical and distributional characteristics of these hemorrhages have not been adequately investigated.

Case Descriptions We studied 28 patients who had primary intracerebral hemorrhage of a longest diameter ≤1.5 cm as seen in computed tomographic scan and/or magnetic resonance imaging. Small primary intracerebral hemorrhages were found in the basal ganglia in 8 patients (2 with intraventricular hemorrhage), the posterior limb of the internal capsule in 8, the area of the fourth ventricle of the cerebellum in 7 (5 with intraventricular hemorrhage), the pontine tegmentum in 4, and the thalamomesencephalic area in 1. All patients except 3 were hypertensive, suggesting that most of the hemorrhages may have occurred because of rupture of small end arteries secondary to long-standing hypertension. Depending on their location, the hemorrhages clinically manifested as pure motor stroke in 7, pure sensory stroke in 6, vertigo/ataxia in 7, sensorimotor stroke in 4, and ataxic hemiparesis in 2 patients. One patient with thalamomesencephalic hemorrhage showed vertical gaze disturbance, and 1 with basal ganglionic hemorrhage presented with symptoms of acute hydrocephalus secondary to a relatively large amount of intraventricular hemorrhage. The prognosis of small intracerebral hemorrhage was generally excellent except for when patients were very old or when there was a significant amount of intraventricular bleeding.

Conclusions Small primary intracerebral hemorrhage has its predilection sites: basal ganglia, posterior limb of the internal capsule, area of the fourth ventricle of the cerebellum, and pontine tegmentum. Most of the hemorrhages are probably caused by rupturing of the small end arteries in the setting of chronic hypertension. They produce discrete clinical syndromes often mimicking classic lacunar syndrome, of which pure sensory stroke is relatively common. (Stroke. 1994;25:1500-1506.)

Key Words • diagnosis • hypertension • intracerebral hemorrhage • lacunar infarction
Clinical Features of Patients

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex/Age, y</th>
<th>Risk Factors</th>
<th>Initial BP, mm Hg</th>
<th>Site</th>
<th>Clinical Features</th>
<th>ID (LD)</th>
<th>Remarks</th>
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<tr>
<td>1.</td>
<td>F/54</td>
<td>HT, CD</td>
<td>270/140</td>
<td>L, BG</td>
<td>Sensorimotor</td>
<td>CT (1.3)</td>
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<td>2.</td>
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<td>MRI (1.5)</td>
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<tr>
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<td>IVH</td>
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<td>Acute hydrocephalus</td>
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<td>MRI (1.2)</td>
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<td>... M, CB</td>
<td>Vertigo, ataxia</td>
<td>CT, MRI (1.0)</td>
<td>IVH</td>
<td></td>
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</tbody>
</table>

BP indicates blood pressure; ID, identification; LD, longest diameter in centimeters; F, female; M, male; HT, hypertension; CD, cardiac disease; DM, diabetes mellitus; PS, past history of stroke; SM, cigarette smoking; AL, habitual alcohol drinking; L, left; R, right; M, middle; BG, basal ganglia; PIC, posterior limb of internal capsule; TH, thalamus; ME, mesencephalon; PO, pons; CB, cerebellum; FP, facial palsy; CT, computed tomography; MRI, magnetic resonance imaging; IVH, intraventricular hemorrhage; and CO, clinical outcome.

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from the past stroke, except 1 (case 8) who had been dysarthric.

**Basal Ganglionic SICH**

Eight patients showed SICH in the basal ganglia. Seven of them presented with symptoms of lacunar syndrome: pure motor stroke in 5, sensorimotor stroke in 1, and ataxic hemiparesis in 1. In 2 patients (cases 7 and 8), the hemorrhages were extended and ruptured into the lateral ventricle; 1 patient with a minimal amount of IVH presented with ataxic hemiparesis, and another with a relatively large amount of IVH showed stuporous mentality due to acute hydrocephalus secondary to obstruction of the sylvian aqueduct. This patient remained vegetative despite prompt ventriculostomy. Except for this patient, the functional prognosis was good; all patients became ambulatory with minimal dysfunction within several weeks.

A 44-year-old hypertensive woman (case 3) developed right hemiparesis accompanied by mild frontal headache. In the emergency department, her blood pressure was 240/120 mm Hg. She was alert and slightly dysarthric. There was mild right lower facial paresis and right hemiparesis without sensory dysfunction. Babinski's sign was equivocally positive in the right side. Brain MRI performed 3 days after onset showed a small putaminal hemorrhage (Fig 1). The patient's neurological symptoms rapidly improved, and on the seventh hospital day, she complained only of clumsy right hand. A 72-year-old hypertensive man (case 7) developed sudden onset of dysarthria and gait difficulty accompanied by mild frontal headache. In the emergency de-
FIG 1. T₁-weighted magnetic resonance image shows a small, acute hemorrhage in the left putamen (case 3). An old hemorrhage is seen in the right putamen.

Department, his blood pressure was 200/110 mm Hg. He showed dysarthria, lower facial paresis, mild left hemiparesis, and obvious ataxia in his left arm and leg that exceeded that expected with the weakness of the limbs. Sensation was normally perceived, and no neck stiffness was noted. On gait testing, he tended to veer to the left. Brain MRI showed an acute hemorrhage in the area around the right lateral ventricle, probably involving the putamen and corona radiata. The hemorrhage extended into the lateral ventricle, and a small amount of IVH was noted (Fig 2). During admission, he improved rapidly, and on the fourth hospital day, he was able to walk normally.

Capsular SICH

Eight patients showed SICHs in the posterior limb of the internal capsule, which included 1 (case 9) with lenticulocapsular SICH and 6 with thalamocapsular SICH. Four (cases 11, 14, 15, and 16) of the thalamocapsular hemorrhages involved the superior portion of the lateral thalamus. Two patients presented with pure motor stroke, while others presented with predominant sensory dysfunction: 2 with pure sensory stroke, 1 with hypesthetic ataxic hemiparesis, and 3 with sensorimotor stroke of dominant sensory symptoms. The functional prognosis was good in all cases.

A 42-year-old hypertensive man (case 9) felt sudden dizziness and headache associated with loss of sensation in the right side of his body. He had been a cigarette smoker and habitual alcohol drinker. His initial blood pressure was 160/100 mm Hg. On examination, he showed normal muscular strength and decreased sense of pinprick, vibration, and touch in the right side of his body. Brain CT showed a small lenticulocapsular hemorrhage (Fig 3). At 8 months of follow-up, he complained of mild numbness in the right extremities.

Thalamomesencephalic SICH

One patient showed an SICH in the paramedian thalamomesencephalic region and showed vertical gaze disturbance.

A 56-year-old hypertensive woman (case 17) experienced sudden dizziness and vomiting followed by diplopia. In the emergency department, her blood pressure was 190/130 mm Hg. On examination, there were hypotropia of the right eyeball on forward gaze, vertical (upward and downward) gaze paresis of the right eye, and downward gaze paresis of the left eye. Horizontal eye movements were preserved. Pupil size was bigger on the left (4.5 mm) than on the right (2.5 mm), but light
FIG 4. T₂-weighted magnetic resonance images show a small hemorrhage involving the left paramedian thalamus (top) and midbrain (bottom) (case 17).

reflex was normal in both eyes. Otherwise, her neurological examination was essentially normal. Brain CT and MRI showed an SICH in the left medial thalamomesencephalic area (Fig 4).

Pontine SICH

Four patients had SICH in the pontine tegmentum. All presented with pure sensory stroke. Two (cases 18 and 20) showed decreased sense of position and vibration in half of their bodies, and 1 (case 19) showed sensory change (paresthesia and altered vibration sense) limited to the fingertips contralateral to the lesion. One patient (case 21) with a lesion near midline showed paresthesia of the left forearm and bilateral perioral area. The functional prognosis was good in all cases.

A 50-year-old hypertensive man (case 21) developed sudden onset of dizziness followed by facial paresthesia during exercise. In the emergency department, his blood pressure was 190/130 mm Hg. He complained of paresthesia of the anterior face and left forearm. A few days later, the sensory dysfunction was restricted to bilateral perioral and intraoral area and left fingertips. Position sense was mildly decreased in the left fingertips. Otherwise, neurological examination was normal. Brain CT showed a small hemorrhage in the right midpontine tegmentum (Fig 5).

Cerebellar or Brachium Pontis SICH

Seven patients with cerebellar hemorrhage were identified. In all, the lesions were situated in the area of the fourth ventricle: 5 in the right, 1 in the left, and 1 posterior to the fourth ventricle. All presented with vertigo, nystagmus, and disequilibrium. Three patients (cases 25 through 27) showed ipsilateral facial paresis of peripheral type and forced deviation of eyeballs contralaterally. In 5 patients, hemorrhage was also seen inside the fourth ventricle. However, the amount of IVH was small, and no patients suffered symptoms of hydrocephalus. All survived the acute phase of stroke, but 1 (case 22) died from pulmonary embolism 2 weeks after the onset of stroke. Two elderly patients (cases 23 and 25) died from aspiration pneumonia 9 and 3 months after the event, respectively.

A 91-year-old woman (case 25) experienced sudden vertigo and vomiting and became ataxic. In the emergency department, her blood pressure was 120/80 mm Hg. Although oriented, she was lethargic and uncooperative. Both eyes were deviated to the left side and did not move in response to the “doll’s eye” maneuver. Spontaneous left-beating nystagmus was seen in both eyes, and left lower facial palsy was noted. Although the patient was generally weak, there were no focal motor deficits or pathological reflexes. She was unable to stand and showed mild neck stiffness. Brain CT showed an SICH in the right side of the fourth ventricle and a small amount of IVH (Fig 6). During admission, her nystag-
Discussion

Nineteen of our 28 patients showed classic lacunar syndrome: pure motor stroke in 7, pure sensory stroke in 6, sensorimotor stroke in 4, and ataxic hemiparesis in 2. Seven with lesion in the area of the fourth ventricle of the cerebellum presented with vertigo and ataxia. The single most important risk factor for SICH in our series was hypertension. Although we excluded patients under age 40 or with MRI evidence of vascular anomaly, ruptured vascular malformation was not completely ruled out in normotensive patients, and the cause of ICH of the 2 elderly, normotensive patients (cases 6 and 25) remains unknown. Seven patients showed IVH, but only one with a relatively large amount of IVH had symptoms of acute hydrocephalus and showed poor clinical outcome. Three elderly patients died within 1 year after onset because of aspiration or pulmonary embolism. Except for these 4, the prognosis of our patients was generally excellent.

In the basal ganglia, SICHs were seen in rather superior portion, often near the lateral ventricle. These lesions caused lacunar syndromes such as sensorimotor or pure motor stroke that were probably due to involvement of the adjacent corona radiata or internal capsule. Interestingly, 1 patient (case 7) presented with ataxic hemiparesis. Although ataxic hemiparesis has been reported to be caused by capsular or corona radiata infarcts, basal ganglionic hemorrhage has not been previously reported to cause this syndrome. Cerebellothalamocortical or cortico-pontocerebellar pathways may have been involved in the corona radiata in this patient. One patient (case 8) had IVH obstructing the sylvian aqueduct, which produced acute hydrocephalus and subsequent mental deterioration.

Eight patients with capsular SICHs presented with pure sensory stroke, sensorimotor stroke, pure motor stroke, or hyposthetic ataxic hemiparesis. Small capsular hemorrhages producing pure motor stroke, pure sensory stroke, and hyposthetic ataxic hemiparesis have been previously reported. Interestingly, 1 patient (case 17) with SICH in the thalamomesencephalic area presented with bilateral upgaze palsy and ipsilateral monocular downgaze paresis. This so-called “vertical one-and-a-half” syndrome has been described in a few patients with small thalamomesencephalic stroke. Small hemorrhages in the midbrain were reported to produce various signs of oculomotor nuclei involvement or pure sensory stroke secondary to tegmental involvement, suggesting that midbrain may be a common site for SICH. However, these reported patients were frequently young and normotensive, and the cause of the hemorrhage often remained unknown.

In the pons, the SICHs were found exclusively in the tegmentum. All of these patients presented with pure sensory stroke, suggesting that these SICHs occur frequently in the area of the medial lemniscus. The anteriorly located corticospinal tracts and posteriorly located abducens nuclei were spared possibly because of the small size of the lesions. Furthermore, in some patients (cases 19 and 21), sensory abnormality was limited to certain parts of the body. One patient (case 21) was of interest in that a single lesion produced bilateral perioral sensory change. Since sensory fibers from the face run most medially in the medial lemniscus, a single midpontine lesion would involve the sensory fibers from both sides of the face and might cause bilateral perioral paresthesia. Pontine tegmentum may be a common site for SICH, considering occasional case reports of small hemorrhages in this area that have produced clinical syndromes of ataxic hemiparesis, pure motor hemiparesis, sensorimotor stroke, and dysarthria-clumsy hand.

On the other hand, the SICH in the fourth ventricular area of the cerebellum has not been well recognized. Its clinical manifestations were characterized by vertigo, nystagmus, and disequilibrium. Wizer et al previously reported 3 patients with small cerebellar peduncular hemorrhage presenting with a clinical triad of ipsilateral ataxia, facial palsy, and ipsilateral gaze paresis. Three of our patients (cases 25 through 27) showed similar clinical symptoms, suggesting that the facial colliculus and abducens nuclei in the posterior part of the pons were involved. Although 5 patients showed a small amount of intra-fourth ventricular hemorrhage, none suffered from symptoms of acute hydrocephalus.

The SICHs in our series occurred in the basal ganglia, thalamus, pons, and the cerebellum, common sites for ICH and lacunar infarct. However, their distributional characteristics are of interest. In the basal ganglia and posterior limb of the internal capsule, they tended to occur in the relatively superior portion near the lateral ventricle. In the posterior fossa, they were exclusively found near or adjacent to the fourth ventricle. In these areas, the diameter of the centrifugal end arteries should be small and, when ruptured, would cause small hemorrhages. Although SICHs often mimic lacunar infarct, their different predilection sites make overall clinical presentations differ from previous studies of lacunar infarct, in which pure sensory stroke was less common and symptoms of vertigo and/or disequilib-
rrium were negligible. In the study of 19 patients with ICH presenting lacunar syndrome, Mori et al identified 7 patients with sensorimotor stroke, 4 with pure motor stroke, 5 with ataxic hemiparesis, 3 with dysarthria-clumsy hand syndrome, and none with pure sensory stroke. The discrepancy probably was caused by the inclusion of larger ICH occurring in the capsulostriatal area in their study, which would more often cause sensorimotor than pure sensory stroke.

Recently, Challah and Moody reported a patient with pontine SICH who revealed pathological findings consistent with the type II lacune described by Poirier et al. Without pathological confirmation, it remains unknown whether the cases reported here had the pathological characteristics of lacune. Nevertheless, considering the distribution of the lesions, the SICHs of our patients more likely occurred from rupturing of the small end arteries rather than from hemorrhagic transformation after lacunar infarct. This assumption may be supported by an autopsy study of 200 cases where all 13 subjects who showed pathological findings of SICH had concurrent Charcot-Bouchard aneurysms and pre-mortem history of hypertension. Finally, it should also be addressed that our results may not reflect the true distribution of SICH, since we included only symptomatic cases in this study. According to Miyashita et al., old and clinically silent hemorraghes, often small in size, are frequently detected by MRI in the thalamus, putamen, and pons in patients with multiple lacunar infarcts. Therefore, the incidence of SICH may be much higher than previously thought, and further studies are required to elucidate the true incidence and predilection sites of SICH.

In summary, the small “lacune”-sized hemorrhages seem to have predilection sites: the superior portion of the basal ganglia and posterior limb of the internal capsule, the pontine tegmentum, and the area around the fourth ventricle of the cerebellum. They produce discrete clinical syndromes that overlap classic lacunar syndrome, in which pure sensory stroke is relatively common. The distributional characteristics of SICHs suggest that they are most often caused by the rupturing of small end arteries secondary to long-standing hypertension. The prognosis is usually excellent except for when the patients are elderly or the amount of IVH is significant.

References

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