

Lenticulocapsular Hemorrhages Presenting as Pure Sensory Stroke

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Key Words

Putamen · Intracerebral hemorrhage · Pure sensory stroke · Sensation

Abstract

Pure sensory stroke (PSS) syndrome is most often produced by a small infarct involving the lateral thalamus. Larger than lacune-sized putaminal hemorrhages have not been considered as a cause of this syndrome. The author describes 3 patients with hypertensive lenticulocapsular hemorrhage presenting with hemisensory symptoms without any other neurological deficits. In these patients, the sensory symptoms were more marked and persistent in the legs than in the other body parts. Neuroradiological data suggested that thalamocortical sensory pathways were exclusively involved. These patients highlight the heterogeneity of the vascular lesion producing PSS syndrome and illustrate that a putaminal hemorrhage should be included in the differential diagnosis of this clinical syndrome.

Pure sensory stroke (PSS) syndrome is usually caused by a minor infarct involving the lateral thalamus [1–4] although small hemorrhages occurring at the thalamus [2–

5] or internal capsule [2, 5, 6] were reported to produce pure or predominantly hemisensory symptoms. To my knowledge, however, relatively large lenticulocapsular hemorrhage has not been reported as a cause of PSS. In this report I describe 3 such patients.

Report of Patients

Patient 1

While driving, a 61-year-old right-handed man with a history of hypertension and diabetes mellitus suddenly felt numbness in his left lower leg which then ascended to the left trunk, arm, neck and the perioral area over the next 2 days. The sensory symptoms persisted, but he did not experience headache, dysarthria, limb weakness or gait difficulty. On admission 20 days later, neurological examination showed that pinprick and temperature senses were decreased to approximately 30% compared to the intact side of the left leg, and 50% compared to the intact side of the left face, forearm and trunk. Vibration, touch and position senses were mildly impaired in the left leg and hand. Cranial nerves, muscle strength and deep tendon reflexes were normal. Cerebellar function tests were normally performed, and Babinski sign was absent. Brain MRI showed a subacute hemorrhage in the right putamen that probably involved the most posterior portion of the posterior limb of the internal capsule and the adjacent thalamus (fig. 1). After his being discharged, the sensory symptoms gradually improved. However, the paresthesia associated with mild sensory deficit remained persistent in his left lower leg (below knee) during the 4-years of follow-up.

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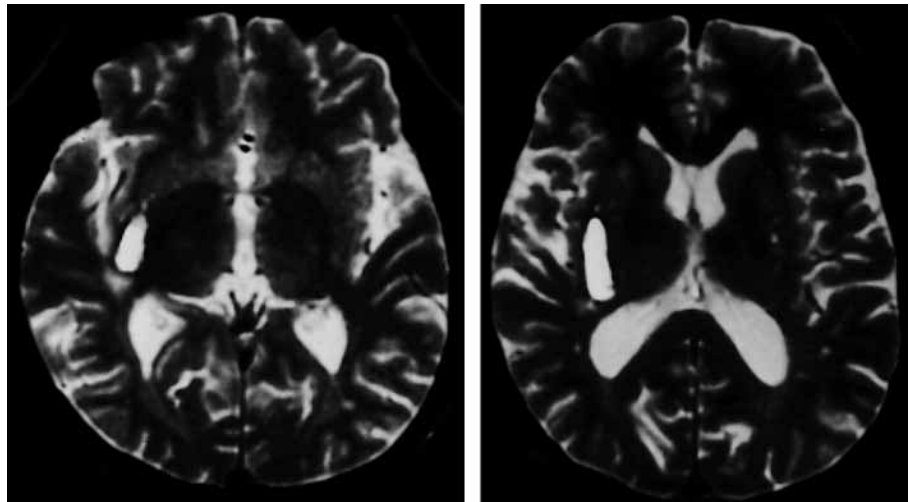


Fig. 1. T₂-weighted MRI showing a right putaminal hemorrhage (patient 1).

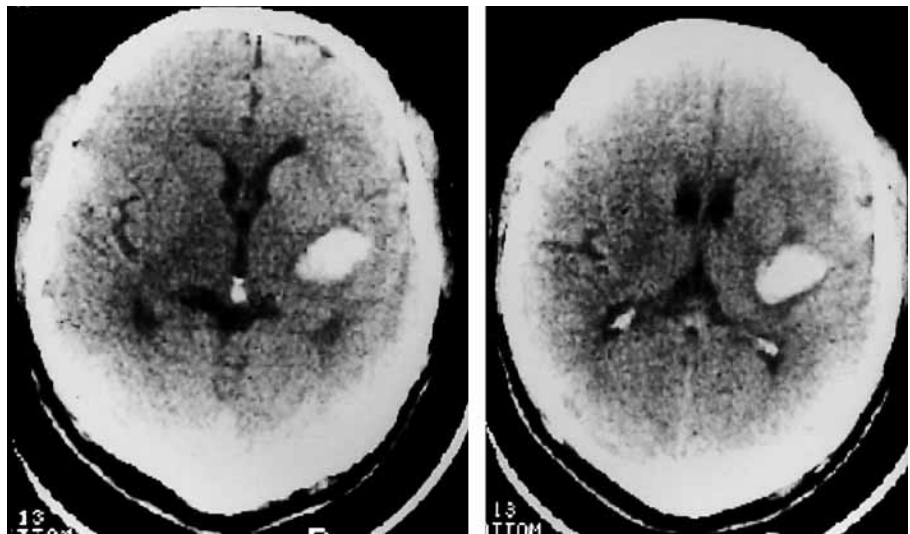


Fig. 2. Brain CT scan showing a hemorrhage in the left putamen (patient 2).

Patient 2

A 41-year-old hypertensive, right-handed man suddenly developed dull, generalized headache associated with numbness on the right side of his body and limbs. On admission 3 days later, neurological examination revealed decreased pinprick, temperature and vibration sensation on the right side, where the deficits were mildest in the face and severest in the leg, especially the right foot. Position sense was also impaired in his right toes. Otherwise, neurological examination was normal. Brain CT showed a left putaminal hemorrhage that probably involved the posterior portion of the posterior limb of the internal capsule and the most dorsolateral portion of the thalamus (fig. 2). Transfemoral angiogram results were normal. His sensory symptom gradually improved, and 1 month later, he had residual paresthesia limited to the medial side of his right lower leg and foot. The paresthesia remained unchanged when he was last examined 4 months after the onset of the stroke.

Patient 3

A 51-year-old hypertensive, left-handed man suddenly developed dull, frontal headache and dizziness associated with a sense of weakness on the right side. On admission the next day, the neurological examination showed decreased senses of pinprick, temperature, vibration and touch in the right half of his body, where the deficits were severer in the leg than in the arm, trunk and the face. Position sense was also impaired in the right toes, but not in the fingers. Otherwise, neurological examination was normal. Brain CT showed a left putaminal hemorrhage that probably impinged on the posterior portion of the posterior limb of the internal capsule and the most dorsolateral portion of the left thalamus (fig. 3). The patient's symptoms gradually improved, but he continued to have the sensory symptoms restricted to the right lower leg, most marked over the anterior part of the sole. One year after the onset of stroke, he still had numbness with occasionally a superimposed distressingly burning sensation in his right foot where the objective senses were mildly decreased. The uncomfortable sensory symptom was partially alleviated by amitriptyline 25 mg/day and carbamazepine 400 mg/day.

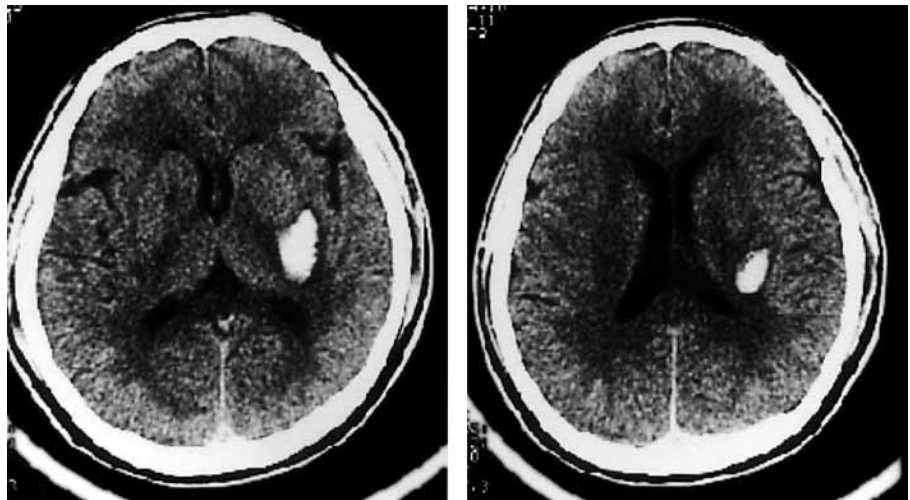


Fig. 3. Brain CT scan showing a hemorrhage in the left putamen (patient 3).

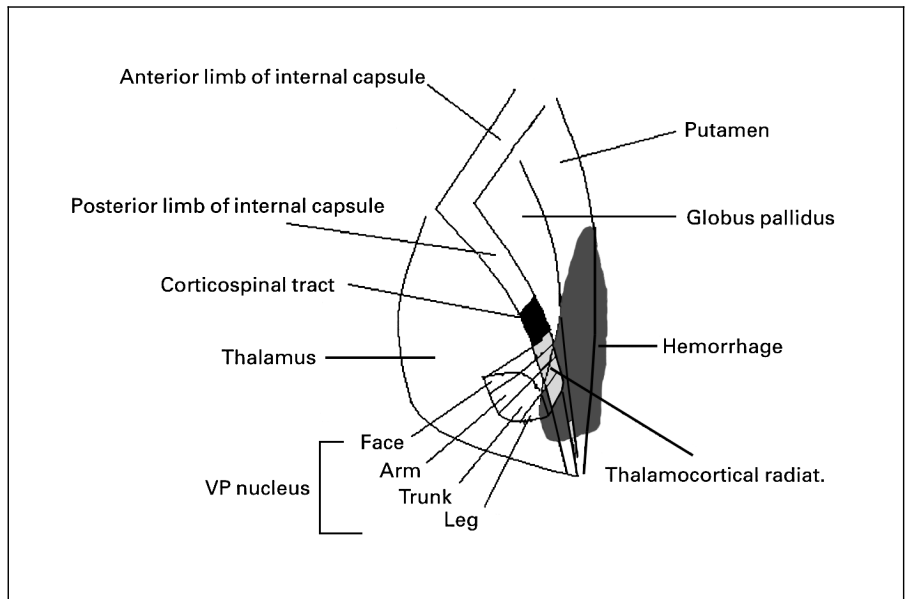


Fig. 4. Schematic drawing of putaminal hemorrhages producing PSS with dominant involvement of the leg. VP = Ventral posterior.

Discussion

The 3 patients described here presented with PSS due to hypertensive putaminal hemorrhages. Although patient 1 was not examined in the acute phase, and the possibility of the presence of other neurological deficits cannot be completely excluded, it is undoubtedly true that his predominant neurological problem was sensory symptoms. A putaminal hemorrhage is the commonest type of spontaneous intracerebral hemorrhage, usually presenting with hemiparesis-hemihypesthesia, aphasia or decreased consciousness [7]. Although small putaminal hemorrhages have been described to produce pure motor hemiparesis [5], those of greater-than-lacune size were not

reported to produce PSS. The neuroimaging results of our patients showed that the hemorrhages may have involved either the most dorsolateral portion of the thalamus and/or the most posterior part of the posterior limb of the internal capsule just adjacent to the thalamus, where ascending sensory tracts are believed to be prominently located [6] (fig. 4). The absence of limb weakness may be explained by the sparing of the descending pyramidal fibers at just posterior to the center of the posterior limb [6]. It seems that a large portion of the hemorrhages confined to the lentiform nucleus did not produce any symptoms.

One interesting observation was that the sensory symptom was distinctly severer in the leg than in the arm/face

in our patients. During follow-up, the patients had persistent paresthesias only in their legs. The mechanisms for the dominant sensory symptoms in the leg remain speculative. It has been shown that the areas of the face, arm and leg are arranged in a medial-lateral direction in the ventral posterior nucleus of the thalamus in primates [7, 8]. Similar topography appears to be preserved in the thalamocortical sensory radiation as well [4, 6]. Considering the neuroradiological data of our patients, the lesions may have involved the most lateral portion of the ventral posterior nucleus of the thalamus and/or the sensory tracts arising from there, which would explain the dominant sensory symptoms in the leg (fig. 4).

Fisher [9] originally stated that PSS is caused by hypertensive lipohyalinotic angiopathy. According to a previous study [10], clinically diagnosed PSS almost always predicts the presence of small vessel occlusive disease. However, small hypertensive hemorrhages [2–5] or cardiogenic embolisms [3] have also been reported to produce PSS. Our patients with larger-than-lacune-sized putaminal hemorrhages further emphasize the heterogeneity

of the vascular lesion producing PSS. A putaminal hemorrhage must be a rare cause of PSS since the above 3 patients were the only ones presenting with PSS out of the 152 patients with putaminal hemorrhage who were admitted to our department during the last 6 years. However, I have occasionally observed patients with putaminal hemorrhage who had predominant, though not pure, hemisensory symptoms. Our patients represent the extreme of these cases. Finally, the presence of headache at onset in patients 2 and 3 is unusual in patients with PSS [2], and could be a clinical clue that these are nonlacunar strokes.

In summary, our patients illustrate that putaminal hemorrhages should be included in the differential diagnosis of PSS. The PSS due to putaminal hemorrhages may be characterized by occasional presence of headache and relatively severe and persistent symptoms in the leg as compared to other body parts. However, more cases should be collected and analyzed to validate this observation.

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