Hypertensive Putaminal Hemorrhage Presenting as Pure Motor Hemiparesis

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SUMMARY A 44 year old hypertensive man presented with a pure motor hemiparesis, and CT scan showed a putaminal hemorrhage. The clinical course was characterized by rapid resolution of the deficits. This case illustrates a variety of putaminal hemorrhage of good functional and vital prognosis, and stresses the value of CT scanning as a tool for diagnosis and prognosis.

HEMIPARESIS OR HEMIPLEGIA without sensory, visual or speech deficit (Pure motor hemiparesis, PMH) is the classical presentation for lacunar infarction in the internal capsule or basis pontis. Other reported causes of this clinical syndrome include: infarcts or cortical, pyramidal or midbrain location, metastases, multiple sclerosis, nocardial abscess, post-craniotomy hemorrhage, and hemorrhages in the basis pontis or internal capsule. PMH has not been described in the setting of primary hypertensive putaminal hemorrhage. This report documents, by detailed neurological evaluation in the acute stage, an instance of a syndrome of PMH in putaminal hemorrhage.

Case Report

A 44 year old left-handed hypertensive male noticed right arm weakness and slurred speech after awakening on 8/8/82. He had no headache, nausea, vomiting or gait difficulties. Over the following 2 to 3 hours the right arm paresis worsened and a mild weakness of the right leg developed. When examined 4 hours after the onset, he was alert, oriented, and gave an accurate description of the events leading to admission. His speech was dysarthric but free of dysphasia. The blood pressure was 220/130. Motor examination showed a moderate paresis of shoulder abduction and elbow flexion, with minimal weakness of distal movements. The lower extremity had slight paresis of foot dorsiflexion, with intact proximal strength. The deep tendon reflexes were slightly hyperactive in the right arm, and plantar reflexes were flexor. Coordination was intact bilaterally.

Sensation was intact for touch and pin-prick in limbs, trunk and face. The slightest stimulation of individual hairs on the right limbs was felt normally and absence or nystagmus, reactive pupils of 2 mm diameter, normal extraocular movements without gaze preference or nystagmus, reactive pupils of 2 mm diameter, intact facial sensation, preserved palate and tongue movements, and absence of bucco-lingual dyspraxia.

CT scan on admission showed a small area of high attenuation (96 Hounsfield units) at the level of the left
mid-posterior putamen, consistent with fresh hemorrhage (fig. 1). No blood was detected in cuts at the level of the body of the lateral ventricles. Coronal views performed 11 days after admission confirmed the lateral basal ganglionic location of the hematoma (fig. 2), which was impinging upon, but apparently not extending into, the posterior limb of the internal capsule. No ventricular extension of the hemorrhage or post-contrast enhancement were detected. The volume of this hematoma was estimated to be 8.5 cu mm.

The right hemiparesis progressively improved over the 24 to 48 hours following admission, and by day 4 the motor strength of the right limbs was within normal limits. The only residual deficit was a moderate right facial palsy, which persisted until discharge 15 days from the onset.

**Discussion**

Hemorrhage into the internal capsule has accounted for PMH in 6 CT-documented cases. These hemorrhages have been described as lenticulo-capsular, thalamo-capsular, and as primary capsular hemorrhages. The clinical features of 5 of these cases were not provided in detail, as they were part of two large series of PMH cases. Our case represents a documented instance of PMH of an apparently pure putaminal hemorrhage, making exception to the notion that putaminal hemorrhages are always associated with a combination of motor and sensory defects. This observation suggests the clinical spectrum of putaminal hemorrhage needs to encompass cases of PMH, along with the classical forms producing combinations of motor, sensory, visual, oculomotor, language and behavioral deficits.

The rarity of our observation reflects the size and location of the hematoma: its size fell into the lowest portion of the spectrum of putaminal hemorrhage, and, in particular, its lateral location along the mid- and posterior aspects of the putamen was responsible for pressure effects, rather than actual destruction, of the adjacent capsule. In addition, CT sections at a level corresponding to the body of the lateral ventricles showed no extension of the hematoma to that area. This CT scan sign has been correlated with good functional prognosis, as indicative of lack of extension of the bleed across the internal capsule. These anatomical features explain the clinical presentation as PMH, as well as the unusual early regressive course of the deficit, which represents a distinctly uncommon observation in intracerebral hemorrhage in general.

**References**

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J F Tapia, C S Kase, R H Sawyer and J P Mohr

Stroke. 1983;14:505-506
doi: 10.1161/01.STR.14.4.505

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

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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