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.

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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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