
Short Communication

Hypertensive Putaminal Hemorrhage Presenting With Hemichorea

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SUMMARY A progressive motor deficit primarily manifested by hemichorea developed in a 42-year-old hypertensive man. CT scan demonstrated contralateral putaminal hemorrhage. The patient's course was benign. Previous cases of acute hemichorea familiar to us and documented by CT scans have involved nonhemorrhagic lesions of either the putamen or caudate.

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ACUTE DISORDERS OF MOVEMENT have been seen after nonhemorrhagic infarction of various portions of the basal ganglia. Hemichorea has occurred secondary to infarcts in the caudate nucleus and putamen.¹⁻⁴ Although the putamen is the commonest site of primary intracerebral hemorrhage,⁵ hemichorea was not included in the broad spectrum of clinical presentations seen in one review of 24 cases of putaminal hemorrhage diagnosed by CT scan.⁶ Our report describes a patient in whom hemichorea was the major sign of an acute hypertensive putaminal hemorrhage (HPH).

Case Report

The patient, a 42-year-old man, had a 25-year history of slight hypertension, which had been treated with diuretics. While driving, he noted a slight clumsiness of his left hand, which gradually became more severe overnight and was associated with difficulty in walking.

On examination in our emergency room 18 hours after the onset of symptoms, the predominant finding was an involuntary, continuous arrhythmic purposeless choreiform movement disorder affecting both left extremities. When the patient was asked to extend the left arm, the left hand assumed a dystonic striatal posture. Rapid repetitive movements were diminished greatly in the left extremities. The left-sided deep tendon reflexes were accentuated, and Babinski's sign was extensor. Neurologic examination was otherwise normal.

Slight generalized tremulousness, flushing, sweating, tachycardia, blood pressure of 190/105 mm Hg, and mild AV nicking in the optic fundi were noted. The patient admitted to chronic excessive consumption of alcohol. Treatment with oxazepam (Serax), 30 mg every three to four hours, in addition to 2 oz of whiskey controlled the impending signs of alcohol withdrawal.

CT scan of the brain without contrast enhancement

was carried out on admission. An area of high-density attenuation, consistent with acute hemorrhage, was demonstrated in the right putamen (fig. 1). No extravasation of blood into the ventricles or across the internal capsule was evidenced. EEG was normal.

The choreiform movements gradually cleared over the next 72 hours. The alcoholic tremulousness abated earlier on the left side than on the right side. The patient gradually became able to walk alone after the fifth day. On discharge, the patient had minimal incoordination of the left hand and a subjective diminution in pain and vibratory sensory modalities in the left extremities. Minimal sensory changes were the only residua noted when the patient was seen in follow-up study six weeks later.

Discussion

Acute hemichoreic syndromes are an uncommon manifestation of cerebrovascular disease. Hemichorea as a manifestation of primary intracerebral hemorrhage is unique in our experience. Previous examples of acute hemichorea have included nonhemorrhagic infarcts of either the caudate nucleus or the putamen or both. This case report broadens the clinical spectrum of HPH documented by CT scans.

An extensive review of HPH by Hier et al⁶ did not mention any cases comparable to our experience. Similarly, a recent report of primary caudate hemorrhage by Stein et al⁷ did not include hemichorea as part of the clinical picture, although it has been observed in nonhemorrhagic infarction of the caudate. Hemiballismus, however, has been reported as a manifestation of contralateral focal subthalamic hemorrhage.⁸

The course and prognosis in our patient were similar to those in other patients with benign HPH. The onset was abrupt, followed by gradual evolution of a motor deficit, primarily characterized in our patient by hemichorea with preservation of normal mental status. This combination of clinical factors fits well with the favorable prognostic signs seen on CT scan, namely, no evidence of hemorrhagic extension across the internal capsule or into the ventricles.

It is not clear why the patient's predominant symptoms of HPH were those of a movement disorder rather than the more common combined motor-sensory deficit⁶ or pure motor hemiparesis.⁹ The rapid and total

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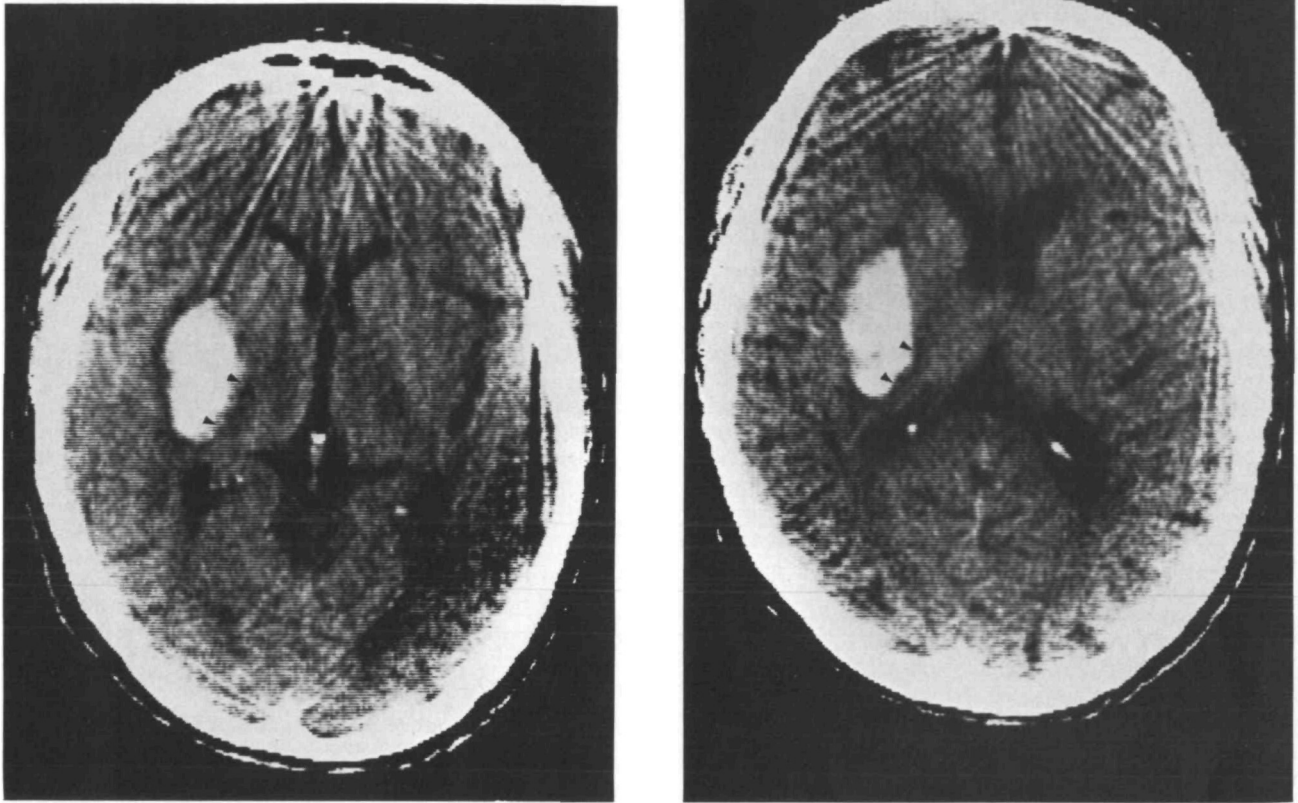


FIGURE 1. Putaminal hemorrhage. (a and b) The area of increased attenuation presumed secondary to acute putaminal hemorrhage extends from the middle and posterior putamen and globus pallidus to the posterior limb of the internal capsule (arrowheads). It extends to or into the external capsule laterally.

resolution of the hemichorea within three days was in contrast to a recently reported case secondary to non-hemorrhagic caudate infarction.⁴ Future reports of hemichorea associated with HPH will be of interest to ascertain whether the movement disorder suggests an acute and irritating, but not destructive, effect of the hemorrhage. If such is the case, hemichorea presenting with HPH may prove to be another indicator of a benign outcome, thus providing further support for a conservative therapeutic approach to these patients.

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