Basal Ganglia Herniation Into the Fourth Ventricle

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ABSTRACT: ***Background:*** Transtentorial herniation of large cerebral fragments is a rare phenomenon. ***Method:*** Case Study. ***Results:*** Examination of the brain of a 35-year-old male showed massive intracerebral hemorrhage resulting in displacement of basal ganglia components into the fourth ventricle. ***Conclusion:*** Sufficiently rapid intracerebral bleeding can dissect fragments of cerebrum and displace them long distances across the tentorial opening.

RÉSUMÉ: Hernie des noyaux lenticulaire, caudé, amygdalien et de l'avant-mur dans le quatrième ventricule. ***Introduction:*** La hernie de parties volumineuses du cerveau est un phénomène rare. ***Méthode:*** Observation clinque. ***Résultats:*** L'examen du cerveau d'un homme âgé de 35 ans a montré une hémorragie intracérébrale massive causant un déplacement d'éléments des noyaux lenticulaire, caudé, amygdalien et de l'avant-mur dans le quatrième ventricule. ***Conclusion:*** Un saignement intracérébral suffisamment rapide peut disséquer des fragments du cerveau et les déplacer loin à travers l'orifice tentoriel.

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Uncal herniation is common consequence of an enlarging cerebral hemisphere due to tumor growth, brain swelling, or brain hemorrhage.[1] Much less common, however, is transtentorial herniation of large fragments of brain. We present a patient who suffered basal ganglia and thalamic herniation into fourth ventricle following massive intracerebral hemorrhage.

Case Report

A 35-year-old previously well male had apparently been involved in a fight three or four weeks prior to admission. After that he complained of headaches of increasing frequency and severity but did not see a physician. He awoke from sleep with a severe headache, stumbled around for several moments, and then collapsed. On examination at a rural hospital his pupils were equal and slightly reactive. His speech was garbled; he would open his eyes and move his right leg and arm to painful stimuli (Glasgow Coma Score 8). Blood pressure was 120/60 mmHg. He was given mannitol, dexamethasone, diazepam, and phenytoin. Over next two hours he became more alert. At 04:30 he suddenly lost consciousness and developed a fixed dilated left pupil (Glasgow Coma Score 3). He was transferred to our facility.

Cranial CT scan at 09:30 showed 7x7x4cm hematoma in the left frontotemporal region with extension into the lateral ventricles (Figure 1). There was marked subfalcine herniation, left uncal herniation, and midbrain distortion. The right lateral ventricle was dilated. The size of the tentorial opening was within normal limits. In the region of the fourth ventricle was a 2x3cm ovoid mass, the identity of which was unknown, surrounded by a narrow rim of blood (Figure 2). He died following organ donation, 36 hours after the initial presentation.

Examination of the brain after formalin fixation revealed diffuse brain swelling and bilateral uncal herniation. No berry aneurysms were found. There was no evidence of trauma or blood on the brain surface. Sectioning of the cerebrum revealed a large hematoma in the left frontal horn of the lateral ventricle with some blood in the enlarged right lateral ventricle. The white matter extending from the margin of the hematoma into the left superior temporal gyrus and two lateral parietal gyri was discolored and softened. The basal ganglia and thalamus on the left side could not be identified. Horizontal sections through the brainstem and cerebellum revealed destruction of the left midbrain and distortion of the pons. A 2x3cm piece of thalamic and striatal tissue was found in the fourth ventricle (Figure 3). Extensive microscopic examination of the brain adjacent to the hematoma failed to reveal any vascular anomalies. In the frontal lobe were some examples of arteriolar tortuosity and mild arteriolosclerosis. The herniated tissue, which included the medial ependyma-lined surface of the thalamus, exhibited no histologic evidence of

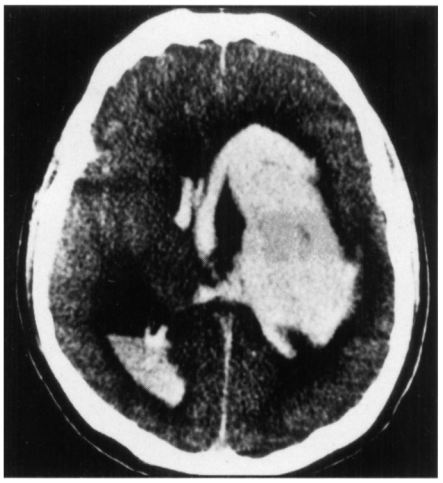

Figure 1: CT scan of the head showing a large hematoma in the left cerebral hemisphere with blood in the posterior horn of the right lateral ventricle.

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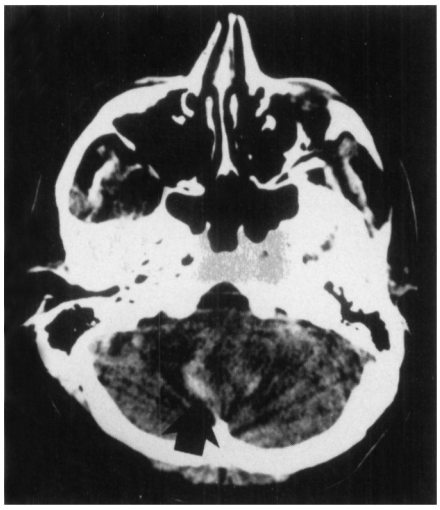

Figure 2: *CT scan of the head showing an ovoid tissue mass in the region of the fourth ventricle (arrow).*

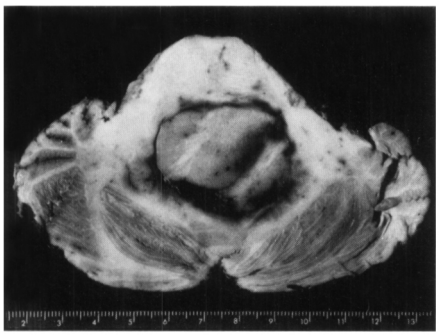

Figure 3: *Photograph of a horizontal slice through pons and cerebellum showing the tissue mass located in the fourth ventricle. The displaced cerebral tissue, which has been sectioned in roughly the horizontal plane, consists of (from left to right) thalamus, internal capsule, striatum, and external capsule surrounded by petechial hemorrhages.*

degeneration and minimal hypoxic-ischemic changes. A small cluster of narrow, thick-walled vessels was found in the herniated putamen, but there was no evidence of old hemorrhage. Despite severe contortion, the internal capsule remained in partial continuity with cerebral peduncle.

Discussion

Of interest in this case is the long distance displacement of a portion of basal ganglia and thalamus. We speculate that the hemorrhage began in the region of the external capsule or insula, dissecting the tissue away from the medial cerebral wall, rather than expanding within the gray matter in a manner more typical of hypertensive basal ganglia hemorrhages. Extremely rapid bleeding then drove the tissue into the posterior third ventricle, and forced it down through the tentorial notch. The CT scan demonstration of an ovoid mass in the fourth ventricle proves that this remarkable herniation event was not simply a consequence of prolonged brain swelling and distortion while the patient was awaiting organ donation.

The cause of the hemorrhage remains a mystery. Arteriolar thickening and tortuosity were identified in the frontal cortex. These changes are more often seen in aged patients and those with hypertension,[2] but there was no history or evidence of hypertension in this patient. The vague history of head injury with subsequent headaches leads to the consideration of "spät apoplexy", late intracerebral hemorrhage which occurs following brain injury.[3,4] Almost all well documented cases of this phenomenon have occurred within a few days of relatively serious head injuries.[5] Nevertheless, a small traumatic intracerebral hematoma may have been present, traces of it being obliterated by the acute hematoma. In approximately 4% of intracerebral hemorrhage cases no cause can be identified; some of these may be due to small vascular malformations which destroy themselves during the hemorrhagic event.[4,6]

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