Amnesia Following Thalamic Hemorrhage

Another Stroke Syndrome

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The clinical manifestations of thalamic hemorrhage frequently comprise hemiparesis, hemianesthesia, and oculomotor abnormalities. Since the advent of computed tomography, an amnestic syndrome following thalamic hemorrhage has been recognized, but the thalamic structures involved and the mechanism of amnesia have remained uncertain. We report a patient with sudden memory dysfunction following hemorrhage into the anterior nucleus of the left thalamus that was shown neuropathologically to disrupt the mamillothalamic fasciculus, one of the principal components of the limbic system. It is considered that the amnestic syndrome following thalamic (anterior nucleus) hemorrhage is due to interruption of the mamillothalamic fasciculus. (Stroke 1988;19:776–778)

Since the introduction of computed tomography (CT), several new clinical syndromes have been identified in association with intracranial hemorrhage.1 The clinical spectrum of subcortical lobar, ganglionic, brainstem, and cerebellar hemorrhages has widened, but the clinical–CT correlation of thalamic hemorrhages has received little attention.2–4 A recent study of 50 patients with thalamic hemorrhages6 reflects previous experience2–5,7 and emphasizes the frequent occurrence of hemiparesis, hemianesthesia, and oculomotor findings (upward gaze palsy with miotic, poorly reactive pupils) as the result of lateral or inferomedial pressure/extension, respectively.

An amnestic syndrome following thalamic hemorrhage has been recently described in four cases.8,9 but only clinical–CT correlation was available. Although amnesia has been associated with thalamic tumor10 and unilateral infarction of the dorsomedial nucleus of the thalamus,11–13 the neuroanatomic explanation for amnesia following thalamic hemorrhage remains uncertain.

We describe a patient with the abrupt onset of memory loss following hemorrhage into the anterior nucleus of the thalamus. Neuropathologic examination revealed disruption of the mamillothalamic fasciculus. It is considered that memory dysfunction in this case resulted from interruption of the mamillothalamic fasciculus in the anterior thalamic nucleus.

Case Report

A 56-year-old man had the sudden onset of memory disturbance. During the preceding 2 weeks he had been attending a physical fitness program, which included a 9-minute treadmill test. His blood pressure before the test was 130/80 mm Hg and his heart rate reached 130 beats/min without symptoms. The day after this workout he developed a vague headache and impaired memory. He smoked 20 cigarettes per day and was in good health.

Examination revealed a mildly febrile (temperature 37.5°C) man whose heart rate was 56 beats/min and regular with blood pressure of 140/90 mm Hg. Routine bedside mental function testing showed errors in orientation, in immediate and 5-minute recall, and in calculation. Results of standard psychometric tests indicated above-average intellectual ability. On the Weschler Adult Intelligence Test his verbal IQ was 111, performance IQ 108, and full-scale IQ 110. No obvious verbal performance discrepancy was present, but administration of the Weschler Memory Scale yielded a memory quotient below normal and impaired ability for new verbal learning. The Benton Visual Retention Test score was also below normal. His neurologic examination was otherwise normal. No neck stiffness was present.

Investigations revealed a normal blood count, urea and electrolytes, plasma glucose, electrocardiogram, electroencephalogram, and skull x-rays. The erythrocyte sedimentation rate was 24 mm/hr.

Unenhanced cranial CT scan revealed an area of high density, consistent with hemorrhage, in the anteromedial aspect of the left thalamus, with extension into the frontal horn of the left lateral ventricle and the third ventricle (Figure 1). CT scan with contrast showed no contrast enhancement. Bilateral selective internal and left vertebral angiograms were normal.

The patient was managed conservatively, and his memory disturbance gradually improved over the subsequent 6 days. A repeat cranial CT scan on Day 7 showed significant resolution of the high-density hemorrhage in the thalamus, third ventricle, and frontal horn of the left lateral ventricle.

On Day 14, he developed a fever (temperature 39.2°C) due to an Escherichia coli urinary tract infection. His conscious state became depressed, and a cranial CT scan revealed acute hydrocephalus. An intraventricular drain was inserted through a right frontoparietal burr hole. Ventricular fluid examination showed changes of florid ventriculitis, and abundant E. coli organisms were cultured. He died the following day without regaining consciousness.
FIGURE 1. Unenhanced cranial computed tomogram (CT scan) showing high-attenuation areas, consistent with hemorrhage, in third ventricle and frontal horn of left lateral ventricle.

Postmortem examination was limited to the intracranial contents. Examination of the brain revealed exudates on the inner surface of the dura mater. The leptomeninges were thickened and covered with a thick layer of purulent exudate. The cortical veins overlying the cerebral hemispheres and the sagittal sinus were thrombosed. The blood vessels at the base of the brain were of normal architectural pattern with minimal atherosclerosis. Uncal grooving was present bilaterally.

One-centimeter-thick coronal slices of the brain showed a markedly edematous cerebral cortex. At the coronal level of the mamillary bodies, a circumscribed hematoma measuring 1 cm in diameter horizontally and vertically and 2 cm in diameter anteroposteriorly was present in the center of the lateral wall of the left lateral ventricle and in the anterior nucleus of the thalamus, at the termination of the mamillothalamic fasciculus (Figures 2 and 3). The ependymal surface overlying the hematoma had been breached, and yellow-brown blood clot was seen within both anterior horns of the lateral ventricles. There was no evidence of a vascular malformation in the vicinity of the hematoma.

Histologic examination of the hemorrhagic area revealed that the ependymal surface overlying the anteromedial thalamus was disrupted, allowing the hemorrhage to communicate with the lateral ventricle. Necrosis with foamy histiocytes and reactive astrocytes was present around the hemorrhage, suggesting that this lesion was at

FIGURE 2. Coronal section of brain, immediately anterior to mamillary bodies, showing circumscribed 1-cm-diameter hematoma (curved arrow) in anteromedial and superior aspect of left thalamus and lateral wall of left lateral ventricle. Site of intraventricular drain inserted through right frontoparietal burr hole is shown (straight arrow).

FIGURE 3. Microscopic appearance of coronal section of left thalamus, mamillary body, internal capsule, caudate nucleus (C), corpus callosum (CC), and third ventricle (V). Hemorrhage (curved arrow) extends from anterior nucleus of left thalamus, at termination of mamillothalamic tract (straight arrow), into left lateral ventricle (curved arrow).
The patient's subsequent clinical course was complicated
by ventricular extension, which can be suspected clinically
and confirmed by unenhanced CT scan. The probable role
of the anterior nucleus of the thalamus and its connections
with the mamillary bodies via the mamillothalamic fasciculus in memory function is also highlighted.

Discussion

Thalamic hemorrhage represents 10–15% of intra-
parenchymal cerebral hemorrhages. The spectrum of
clinical presentation reflects the location, size, and
pattern of extension of the hematoma. Lateral extension
into the internal capsule frequently results in hemi-
paresis and hemianesthesia. If the lateral geniculate
body is involved, a transient homonymous hemianopia
occurs. If the dominant hemisphere is affected,
aphasia may be seen, and mutism, amorphosisynthesis,
and contralateral neglect have been reported following
nondominant thalamic hemorrhage. Inferomedial ex-
tension into the thalamus and dorsal midbrain affects
oculomotor function. The most characteristic
defect is one of upward gaze with miotic, unreacognizable pupils. Other oculomotor signs include convergence
paralysis, nystagmus retractorius on attempted upward
gaze, skew deviation with downward and medial
displacement of the contralateral eye, ipsilateral ptosis
and miosis, forced deviation of the eyes downward, and
transient opsonolus. Medial extension into either the
atrium of the lateral ventricle or the third ventricle
has been recognized on CT to occur in one half to two
thirds of thalamic hemorrhages, resulting in a high
frequency (approximately 25%) of hydrocephalus.

Although a thalamic hemorrhage lying medially with
extension into the thalamus can often be distinguished
clinically from one located laterally, the clinical presenta-
tion of anteromedial thalamic hemorrhage with intra-
ventricular extension is not so clearly documented. The
relatively sudden onset of memory disturbance in this case
after an intensive period of physical exertion and head-
ache was consistent with a vascular event, which was
confirmed by cranial CT scan and autopsy examination.
The patient's subsequent clinical course was complicated
by a urinary tract infection from which he became
septicemic. It is speculated that the intraventricular blood
may have acted as a nidus for the infection to localize to
the ventricular system.

Two thalamic structures have been implicated in
memory functions: the anterior nucleus and mamillotha-
lamic tract and the mediodorsal nucleus and internal
medullary lamina. The mamillothalamic fasciculus (bundle of Vicq D'Azyr) establishes reciprocal connections
between the mammillary bodies and the anterior
thalamic nuclei, which in turn projects into the cingulum.
It is one of the principal fiber bundles of the limbic
system, which has a significant role in memory. The
pathologic data from this case clearly illustrates hemor-
rhage into the anterior nucleus of the thalamus, involving
the mamillothalamic fasciculus, with intraventricular
extension.

The purpose of our report is to increase awareness of

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