Caudate Hemorrhage

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Thirteen patients with caudate hemorrhage are described. All the hemorrhages were in the head of the caudate nucleus, and all the hemorrhages but one ruptured in the anterior horn of the lateral ventricle. In 9 patients, the hemorrhage was related to hypertension and the patients were older. In the other 4 patients, the hemorrhage resulted from rupture of an arteriovenous malformation (AVM) and the patients were under 40 years old. Clinical manifestations were nearly the same in both groups, suggestive of subarachnoid hemorrhage and including hemiparesis in most patients. In patients with hypertension, good recovery was obtained by conservative treatment and in those with ruptured AVMs, resection of AVMs through an anterior transcallosal approach may be safe and recommended. (*Neurosurgery* 18:445–450, 1986)

Key words: Arteriovenous malformation, Caudate hemorrhage, Cerebral hemorrhage, Hypertension, Ventricular hemorrhage

The common sites of hypertensive cerebral hemorrhage are the putamen, thalamus, cerebral hemispheric white matter, pons, and cerebellum (1, 7). The caudate nucleus receives its blood supply from the deep penetrating branches of the lenticulostriate arteries, as does the putamen. Hemorrhage in the caudate nucleus, however, has rarely been described in association with hypertension or with arteriovenous malformations (AVMs) (2, 4–6, 8).

The present report describes 13 patients with caudate hemorrhage, and emphasizes its clinical and computed tomographic (CT) features, and the treatment of choice.

PATIENT POPULATION AND METHODS

From January 1977 to December 1984, we had 313 patients with hypertensive cerebral hemorrhage, 9 of whom had hemorrhage in the head of the caudate nucleus. We also had 70 patients with cerebral AVMs, 4 of them in the head of the caudate nucleus. Thus, caudate hemorrhage represents 3% of hypertensive cerebral hemorrhages and 5.7% of cerebral AVMs. All the hemorrhages were located in the head of the caudate nucleus, and all the patients were diagnosed by CT scan. All 13 patients had contrast-enhanced CT scans, and cerebral angiography was performed in 6 out of 9 patients with hypertensive hemorrhage and in all 4 patients with AVM. All 9 patients classified as hypertensive had had known hypertension, 2 had renal disease, and other 2 suffered diabetes mellitus. The 4 patients with AVM were not hypertensive. We review and analyze the case records of these 13 patients with CT evidence of caudate hemorrhage.

REPRESENTATIVE CASE REPORTS OF HYPERTENSION CAUDATE HEMORRHAGE

Case 5

The sleep of a 51-year-old hypertensive fisherman was disrupted by severe headache and vomiting followed by disorientation. At the time of admission, the patient was drowsy and disoriented, but otherwise neurologically intact, except for nuchal rigidity. CT scan disclosed hemorrhage in the head of the right caudate nucleus with ventricular rupture (Fig. 1). Four-vessel angiography demonstrated no vascular lesions. He rapidly improved in the course of 1 month of conservative treatment and returned to a fisherman's life without deficit.

Case 8

A 61-year-old housewife suddenly vomited while lunching, complained of severe headache, and then collapsed. She was seen at a nearby hospital, where a spinal tap disclosed grossly bloody cerebrospinal fluid (CSF). On admission to our hospital, the patient was alert and cooperative. Neurological examination revealed moderate disorientation and dyscalculia, neck stiffness, a slight right hemiparesis, and bilateral extensor plantar responses. Her history included hypertension. CT scan demonstrated left ventricular hemorrhage (Fig.

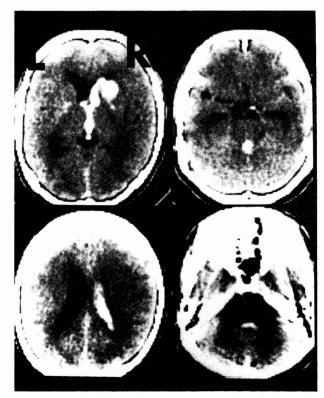


Fig. 1. Case 5. CT scan demonstrates hemorrhage in the head of the right caudate nucleus, with rupture in the anterior horn of the lateral ventricle.

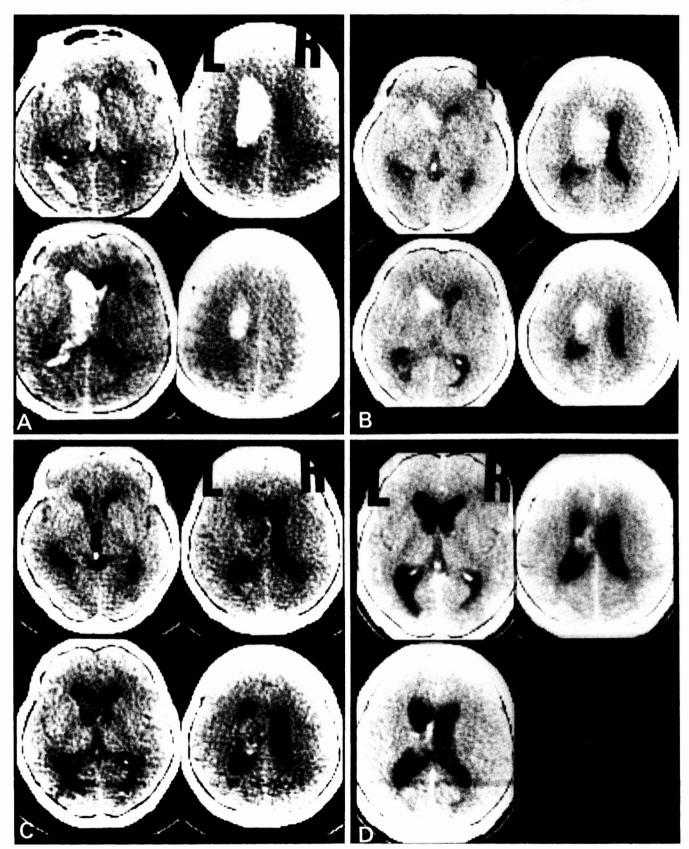


FIG. 2. Case 8. A, CT scan discloses intraventricular hemorrhage (IVH) in the left lateral ventricle. B, three weeks after the hemorrhage, the IVH still persists. C, three months after the hemorrhage, the IVH persists, although the density has decreased. The head of the left caudate nucleus shows marked atrophy. One of reasons we conclude the IVH originates there. D, one year after the hemorrhage, the IVH remains in the body of the lateral ventricle and shows some enhancement after intravenous administration of iodinate contrast material.

2.4). Four-vessel angiography disclosed nothing abnormal. She rapidly improved, and 3 weeks later she was discharged without any sequela. Follow-up CT scan, however, demonstrated interesting findings. At the time of discharge intraventricular hemorrhage still persisted (Fig. 2B). Three months after discharge, the hemorrhage was still seen in the left lateral ventricle, although it decreased in intensity, and the left anterior horn began to dilate, especially at the site of the head of the caudate nucleus (Fig. 2C). Even 1 year after the hemorrhage, although the intraventricular hemorrhage disappeared in the anterior horn, it remained in the body of the lateral ventricle (Fig. 2D), as we had observed before (9).

Case 9

A 54-year-old housewife was found unconscious on her farm. The patient was evaluated at another hospital. She was stuporous, with moderate nuchal rigidity and moderate right hemiparesis. CT scan demonstrated hemorrhage in the head of the left caudate nucleus, with ventricular rupture, and a round mass in the left occipital region (Fig. 3A). On referral to us, she was alert and cooperative. There were right hemiparesis and hemihypesthesia, and right extensor plantar response. Neurological examination revealed moderate loss of recent memory, slight confusion, mild disorientation for time, and moderate loss of attention span. Four-vessel angiography demonstrated some feeding vessels to the left occipital tumor, but did not demonstrate any vascular lesion. The tumor was removed totally through a left occipital craniotomy. The histological diagnosis was meningotheliomataous meningioma. The caudate hemorrhage was not evacuated. Her neurological symptoms and signs gradually decreased over 1 month. She was discharged with only slight loss of recent memory. CT scan demonstrated a moderate low density area in the head of the left caudate nucleus and its adjacent region, with dilatation of the anterior horn, and total removal of the tumor (Fig. 3B).

RESULTS

Clinical and neuroradiological findings, treatment, and results are summarized in Tables 1 and 2.

Hypertensive caudate hemorrhage

There were seven male and two female patients; all nine patients had known hypertension (Table 1). Five took antihypertensive medication regularly and the other four took medicine irregularly. Seven patients developed sudden severe headache and vomiting, which was followed by unconsciousness in four. One was found unconscious on her farm, and another awakened with a right hemiparesis from general anesthesia for clipping of an aneurysm of the left middle cerebral artery. At the time of admission no patient was comatose; four were somnolent or drowsy, and consciousness was nearly complete in the other five, although three of these were moderately disoriented. Eight patients had nuchal rigidity, although the degree was variable. Motor weakness was noted in five: central facial paresis in two, moderate hemiparesis in 2, and moderate paresis of the upper limb in the other. Involuntary blepharospasm-like movement of the contralateral eyelids was transiently observed in one, and papilledema was also noted in one. Hemihypesthesia was present in only one in whom the hemorrhage extended into the anterior limb of the internal capsule. Marked memory loss was present in one, probably due to preceeding multiple cerebral infarcts.

According to the amount of blood within the ventricular system, severity of intraventricular hemorrhage has been graded as mild, moderate, and severe (3). Three had severe hemorrhage, 3 had moderate, 2 had mild, and one did not develop intraventricular hemorrhage. All the hemorrhages occurred in the head of the caudate nucleus, and ventricular rupture almost always took place in the anterior horn, when it occurred. There was no patient with caudate hemorrhage that ruptured in the body of the lateral ventricle. In Case 8, the hemorrhage persisted unabsorbed in the body of the lateral ventricle even 1 year after the hemorrhage, but the site of the

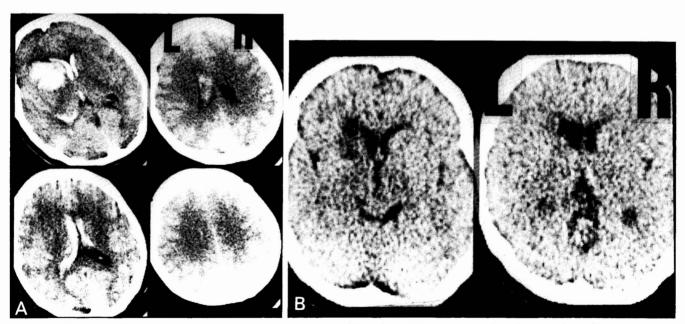


Fig. 3. Case 9. A. CT scan demonstrates hemorrhage in the head of the left caudate nucleus and its adjacent region, with rupture in the lateral ventricle and a round tumor in the left occipital region. B. 4 weeks after the hemorrhage, a moderately sized low density area in the previously hemorrhagic region, with dilatation of the left anterior horn is observed. The occipital tumor is completely removed.

TABLE 1
Summary of Patients with Hypertensive Caudate Hemorrhage^a

Patient	Age	Sex	Hyper- tension	Symptoms	Signs	CT	IVH	Angiography	Treatment	Results	Follow-up Period
I	31	М	+, renal	Headache collapse	Somnolence, nuchal rigid- ity, involun- tary move- ment of L eyelid, L central facial paresis	Hemorrhage in the head of CN, ex- tended to the anterior limb of IC (R)	++	Negative	Removal of hemor- rhage	Excellent	5¾ yr
2	43	M	+, renal	Headache collapse	Somnolence, nuchal rigid- ity, R central facial paresis	Hemorrhage in the head of CN (L)	++	Negative	Supportive	Excellent	5 yr
3	53	M	+, DM	Headache (no LOC)	Nuchal rigidity, disorienta- tion, recent mem- ory loss	Hemorrhage in the head of CN (L) mul- tiple infarcts	+	ND	Supportive	Poor (multi- infarct demen- tia)	5 yr
4	59	М	+	Headache, vomit- ing; gait and speech disturb- ance (no LOC)	Drowsiness, nuchal rigid- ity, papille- dema	Hemorrhage in the head of CN, ex- tended to the anterior limb of IC (L)	++	ND	Supportive	Dead due to DIC, meningi- tis, and broncho- pneu- monia	
5	51	M	+	Headache, vomiting (no LOC)	Drowsiness, di- sorientation	Hemorrhage in the head of CN, ex- tended to the anterior limb of IC (R)	+++	Negative	Supportive	Excellent	4 yr
6	57	M	+, DM	Headache, vomiting (no LOC)	Nuchal rigidity	Hemorrhage in the head of CN (R)	+	ND	Supportive	Excellent	3¾ yr
7	51	M	+	Headache	Nuchal rigidity	SAH		MCA aneu- rysm	Obliteration		
				R weakness	R hemiparesis	Hemorrhage in the head of CN (L)	-	,	Supportive	Excellent	2 yr
8	61	F	+	Headache, vomit- ing, col- lapse	Nuchal rigidity, disorientation, R hemipa- resis	IVH (L)	+++	Negative	Supportive	Excellent	2 yr
9	54	F	+	Collapse, R weakness	Nuchal, rigid- ity, R hemi- paresis, R hemihypes- thesia	Hemorrhage in the head of CN, ex- tended to the anterior and poste- rior limbs of IC (L)	+++	Negative	Supportive		
						L occipital me- ningioma			Resection	Excellent	4 mo

^a Abbreviations: IVH, intraventricular hemorrhage; IC, internal capsule; +, mild; ++, moderate; +++, severe; CN, caudate nucleus; DM, diabetes mellitus; ND, not done; DIC, disseminated intravascular coagulation; SAH, subarachnoid hemorrhage; MCA, middle cerebral artery.

Table 2
Summary of Patients with Caudate Hemorrhage Due to Ruptured AVM^a

Patient	Age	Sex	Hypertension	Symptoms	Signs	IVH	Angiography	Treatment	Results	Follow-up Period (yr)
10	11	М	_	Headache, vomiting, LOC	Drowsy, nuchal ri- gidity, R hemipa- resis	++	AVM	L frontal transcorti- cal	Excellent	7
11	18	M	-	LOC	Drowsy, nuchal ri- gidity, L hemipa- resis	++ ICH	AVM	R frontal transcorti- cal	Excellent	63/4
12	37	M		Headache, vomiting	Neurologically intact	++	AVM	R frontal transcorti- cal	Poor	4
13	35	F	-	Headache, vomiting, LOC	Drowsy, R hemipa- resis	++ ICH	AVM	Anterior transcallosal	Excellent	3

^a Abbreviations: IVH, intraventricular hemorrhage: LOC, loss of consciousness; ICH, intracerebral hemorrhage.

original rupture into the ventricle was in the anterior horn. In 2 patients, the hemorrhage extended to the anterior limb of the internal capsule.

Six patients underwent four-vessel angiography, and neither aneurysm nor AVM was demonstrated. In one patient, the caudate hemorrhage took place during operation for intracranial aneurysm, and in another an asymptomatic occipital meningioma was found on CT scan.

No surgical treatment was given to the caudate hemorrhage, except in one patient with a tentative diagnosis of a cryptic AVM. The treatment given was supportive in 8. The results were excellent in 7; one died of disseminated intravascular coagulopathy complicated by meningitis and bronchopneumonia, and the other was in poor condition because of the preceding multiple cerebral infarcts.

Caudate hemorrhage due to ruptured AVM

Three patients were men and one was a woman. They were all under the age of 40 at onset. None was hypertensive. All the patients had sudden severe headache; three of them then lost consciousness. On admission, three patients were still drowsy and presented with hemiparesis, much worse in the upper limb than in the lower one; two had nuchal rigidity; and only one patient was neurologically intact. CT scan showed that three had a high density area in the head of the caudate nucleus that was contiguous to the anterior horn and accompanied by moderate ventricular dilatation; one had moderate hemorrhage in the head of the caudate nucleus extending to the internal capsule, with very slight ventricular hemorrhage. Intravenous administration of iodinated contrast medium did not disclose the nidus of the AVM, but clearly demonstrated the draining veins in all 4 patients, and angiography clearly showed the AVMs.

The AVMs were resected by a frontal transcortical approach in three and by an anterior transcallosal approach in one. The results were excellent in three, although transient expressive aphasia and weakness were observed in two who were operated on by a transcortical approach. In one patient, marked confusion and disorientation were the significant sequelae after a right frontal transcortical resection in a right-handed young adult. Thereafter, we selected as treatment of choice an anterior transcallosal approach for the AVMs in the head of the caudate nucleus. Details of the AVMs in the lateral ventricles are described in another report (6).

DISCUSSION

Hypertensive cerebral hemorrhage occurs as a result of rupture of such penetrating arteries as the lenticulostriate arteries in the putamen. The rupture is generally considered to be due to necrotic and sclerotic changes in the walls of the arteries. The caudate nucleus is also supplied by the deep penetrating branches of the lenticulostriate arteries, so the hemorrhage would be expected to occur in the caudate nucleus. Nevertheless, reports of caudate hemorrhage have been rare (2, 4, 5, 8). Stein et al have reported 12 patients with hemorrhage in the head of the caudate nucleus (4). Clinical features included acute onset of vomiting, headache, stiff neck, decreased consciousness and behavioral abnormalities, including disorientation and impaired memory. Eight of 12 patients were hypertensive, and the age at onset was older than 40 years in 10. Signs included gaze paresis, Horner's syndrome, and hemiparesis. Seven patients had hemiparesis, slight and transient in 4, and much severer in 3. All 12 patients had hemorrhage in the head of the caudate nucleus with intraventricular rupture. The prognosis for recovery was good in 8; 1 patient was dead, and other 3 left moderate sequelae.

Weisberg reported eight patients with caudate hemorrhage (8). Seven initially suggested clinical symptomatology of subarachnoid hemorrhage and 1 had initial motor weakness. Clinical manifestation included hemiparesis, pupillary abnormalities, and hemihypesthesia. Four patients had internal carotid artery aneurysms, although the exact sites were not described, and the other 4 patients had hypertension. Two of 4 patients with aneurysms died. Two of 4 patients with hypertension also died, and 1 left the hospital with hemiparesis and hemisensory deficit. Weisberg stressed that in caudate hemorrhage due to rupture of an internal carotid artery aneurysm, CT scan showed a high density area contiguous to the anterior horn of the lateral ventricle, and that in caudate hemorrhage related to hypertension, a small hematoma appeared contiguous with the anterior portion of the body of the lateral ventricle with prominent intraventricular extension (8). In contrast to Weisberg's observation, in our series of 13 patients of caudate hemorrhage not one had an aneurysm of the internal carotid artery. Nine patients had hemorrhage related to hypertension, and the other 4 had hemorrhage associated with AVM in the head of the caudate nucleus. In patients with AVM, the age at onset is much younger than in those with hypertension (6). The patients in both groups almost always presented with the same clinical symptoms. The hemorrhage in both groups was contiguous to the anterior horn of the lateral ventricle, and none was closely associated with hemorrhage in the anterior portion of the body of the lateral ventricle in our series.

The prognosis of caudate hemorrhage is also controversial. In our series, the prognosis is not as poor as it is in the Weisberg series. Seven patients with hemorrhage related to hypertension showed good recovery, only one died, and only one was in poor condition, which was not related to the caudate hemorrhage itself but to the preceeding multiple cerebral infarcts. Three patients with AVM showed excellent recovery, and only one was in poor condition.

There have not been sufficient data about the causes, clinical symptomatology, neurological and neuroradiological findings, treatment of choice, and results of caudate hemorrhage. All are still controversial. We must look forward to further investigations.

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COMMENT

In 9 of the 13 patients reported in this series, and the 12 reported by Stein et al. (2), caudate hemorrhage was spontaneous and usually due to hypertension. Most of these 21 patients presented with symptoms and signs of subarachnoid hemorrhage due to rupture of the contiguous hematoma into the anterior horn of the lateral ventricle. The majority also had a hemiparesis.

In these two series, the prognosis for the patient with caudate hemorrhage due to hypertension seems to be good with medical therapy. I also believe these hemorrhages can usually be treated medically following the guidelines we have outlined for the treatment of brain hemorrhage (1).

I disagree with the authors' last paragraph. The cause of caudate hemorrhage has usually been established, the neuroradiological findings characterized, and the treatment is usually a program of medical therapy.

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