

ORIGINAL ARTICLES

HEMORRHAGE OF THE HEAD OF CAUDATE NUCLEUS

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Abstract

Introduction: Intracerebral hemorrhages involve various parts of the brain. The clinical presentation varies according to location of hemorrhage. The outcome depends on the location and associated complications. This retrospective study aims to study the etiology, clinical presentation and outcomes of caudate hemorrhage.

Methods: We conducted a retrospective study and reviewed 379 patients of intracerebral hemorrhage admitted to Jose Reyes Memorial Medical Centre, Manila, department of Neurology over a 1-year period (July 2011-June 2012). Diagnosis was confirmed by CT scan. Risk factors, clinical findings, neuroimaging features and outcomes were analyzed.

Results: Out of the 379 patients 4 patients had a head of caudate nucleus (CN) hemorrhage and the prevalence was 1.055%. The mean age of the four patients was 41.75 yrs. Three of them were female. The volumes of hemorrhage were 2cc, 4cc, 5cc and 12 cc. Three patients were hypertensive, the fourth patient was an arteriovenous malformation suspect. The most common presentation was headache with vomiting. One patient presented with seizure and decrease in consciousness. Intraventricular extension was present in all patients and three of them had hydrocephalus on CT scan. Two of the patients expired and two were discharged.

Conclusion: Head of caudate hemorrhage is rare. It is a mimicker of subarachnoid hemorrhage (SAH). Hypertension was the foremost factor associated with caudate hemorrhage. Poor outcomes were associated with hydrocephalus.

Keywords: Caudate hemorrhage, Hypertension, Hydrocephalus, Intraventricular Extension (IVE), Subarachnoid hemorrhage (SAH)

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Introduction

The corpus striatum consists of the caudate nucleus, putamen and globus pallidus. Because of their close proximity, the putamen and globus pallidus were once considered as one entity termed the lentiform (lenticular) complex or nucleus. It is now known that the putamen and caudate nucleus (CN) share a common chemocyttoarchitecture and connections, and they are referred to jointly as the neostriatum or simply the striatum¹. The common locations of spontaneous intracerebral hemorrhage are the putamen, thalamus, cerebral hemispheric white matter, pons and cerebellum². In order of frequency, the most common sites of a cerebral

hemorrhage are (1) the putamen and adjacent internal capsule (50 percent); (2) the central white matter of the temporal, parietal, or frontal lobes (lobar hemorrhages, not strictly associated with hypertension); (3) the thalamus; (4) one or the other cerebellar hemisphere; and (5) the pons³. Caudate hemorrhage is seldom reported separately but mostly with putaminal hemorrhage. Caudate hemorrhage accounts from 0.1% to 7% of intracerebral hemorrhages^{4,10}. Vascular supply to the CN relies upon deep perforators from diverse arteries, the two principal ones being the anterior (ACA) and middle cerebral arteries (MCA). ACA supplies a part of the CN head. Heubner's artery is responsible for supplying

the inferior part of the CN head as well as the adjacent anterior limb of the internal capsule and the subfrontal white matter. From Heubner's artery, on average four deep perforators arise, having diameters similar to those of the lenticulostriate branches of MCA. Rupture of this artery leads to hemorrhage into caudate. The medial lenticulostriate arteries originating from the proximal M1 portion of the MCA supply both a small portion of the lateral border of the caudate head and the adjacent internal capsule. The lateral lenticulostriate artery branches, from the mainstream MCA or its superior division branch, supply the largest portion of the CN head, as well as the adjacent internal capsule and the anterior half of the putamen. The CN, due to its paraventricular location, is also perfused by ependymal arteries which flow outward from the ventricular surface into the cerebral parenchyma. The head of CN contributes to formation of the floor of the lateral ventricle⁵. These structures share close anatomical relation and common blood supply. A hemorrhage may involve one or more of above structures. As a result clinical presentation varies based on structures affected by the hemorrhage. Small head of caudate haematomas may readily rupture into the ventricles. When the lesion is large the picture is similar to putaminal haemorrhage but if small, haematomas may mimic subarachnoid haemorrhage with acute headache and meningism with little in the way of focal signs⁶. This retrospective study outlines aetiology, clinical presentation and outcome of caudate hemorrhage.

Methods

We retrospectively reviewed medical records of all patients who had a diagnosis of spontaneous ICH at Jose Reyes Memorial Medical Centre during the period of July 2011 to June 2012. We identified four cases from 379 cases reviewed. We diagnosed caudate hemorrhage when CT scan revealed a high density area mainly at the head of the caudate nucleus marginally contiguous to the anterior horn of the lateral ventricle with or without intraventricular extension. A broader involvement of basal ganglia or internal capsule was not included in the study. Four retrospective cases with head of caudate hemorrhage were included in this study. Risk factors, clinical history, clinical features, laboratory data, ECG, treatment offered and outcomes were obtained and evaluated. The final diagnosis was based on CT scan.

Patients

Patient 1

Miss. A.R. aged 23 years presented on March 21, 2012 with headache. She is a four pack year smoker and alcoholic. The headache started two days prior to admission. The headache was right frontal, throbbing, 8/10 on PAS. The headache persisted and the next day patient vomited. There was no history of illicit drug abuse. On admission her BP was 120/80. The GCS of the patient was 15, there was no cranial nerve or focal neurological deficit, laboratories including bleeding parameters and coagulation profile were normal. E.C.G. and CXR were within normal limits. A plain cranial CT scan revealed acute parenchymal hemorrhage right caudate nucleus (approximate volume 2 cc) with seepage into right ventricular system with resultant beginning obstructive hydrocephalus. She was admitted to neurology ward and was given mannitol, ketorolac and acetazolamide. After nine days of hospital stay the patient was discharged and advised to follow up as outpatient with result of CT angiography. The patient was lost to follow up.

Patient 2

Mrs. D.L. aged 53 years, presented on January 1, 2012 with headache. She is a hypertensive for fourteen years on amlodipine but with poor compliance. She is a non smoker and non alcoholic. On December 27, 2011 the patient started to experience sudden severe frontal headache, throbbing in character, non radiating relieved by analgesics and was associated with vomiting. The next day her BP was 280/100. The patient self medicated with losartan. The headache was persistent and she sought consult at a hospital. She was managed as case of hypertension. The patient went home against medical advice. Due to persistent headache she sought consult at our hospital. On admission her BP was 220/120, GCS 15, there were no cranial nerve or focal neurological deficit. Laboratories including bleeding parameter and coagulation profile were normal. ECG and CXR revealed left ventricular hypertrophy and cardiomegaly respectively. A plain cranial CT scan revealed an acute parenchymal hemorrhage (approx 5 cc), head of right caudate nucleus with ventricular extension into right frontal horn of lateral ventricle. She was admitted and the hospital stay of seven days was uneventful. She received nicardipine, losartan, analgesics, mannitol and acetazolamide. At discharge she was advised to follow up as outpatient. The patient was lost to follow up.

Patient 3

Mrs. L.E. aged 36 years, presented on February 14, 2012 with headache. At 10 am the patient experienced sudden severe frontotemporal headache, throbbing in character, non radiating, measuring 8/10 on PAS and was associated with multiple bouts of vomiting. The patient was a hypertensive for one year and also had a family history of hypertension but the patient was not on any antihypertensive medicine. The blood pressure of the patient was 260/150, GCS 13(E4V3M6), pupils were 2-3 mm isocoric, there was no focal neurological sign, there was nuchal rigidity and Babinski's was elicitable bilaterally. Laboratories including bleeding parameter and coagulation profile were normal. ECG and CXR were unremarkable. A plain cranial CT scan was done and it revealed a hyperdense focus in the left head of CN area consistent with acute hemorrhage with approximate volume of 12 cc with intravenous extension into lateral, third and fourth ventricle. The patient was admitted and was given nicardipine, amlodipine, mannitol, acetazolamide and analgesics. On the fifth hospital day the patient was intubated because of decrease in GCS. Next day the patient expired. The final diagnosis was Brain herniation, Intracerebral hemorrhage, left anterior caudate with intravenous extension and obstructive hydrocephalus, hypertension.

Patient 4

Mr. P.C., aged 55 years, presented on March 19, 2012 with seizure. The patient was a hypertensive and diabetic for five years on losartan, amlodipine and metformin with poor compliance. The patient slept asymptotically on March 18, 2012 at 8 pm. At 12 a.m on March 19, 2012 he was seen having stiffening of extremities with upward rolling of eyeballs. He was immediately brought to our institution. The BP of patient was 200/120, GCS 3, pinpoint pupils, preferential gaze to the right and bilateral Babinski's. The NIHSS score was 36. The patient was intubated and put on mechanical ventilator. Laboratories including bleeding parameters and coagulation profile were normal. ECG revealed sinus tachycardia and borderline left ventricular hypertrophy. Cardiomegaly was noted on CXR. A plain cranial CT scan was done. It showed acute parenchymal hemorrhage involving the head of the right caudate nucleus and anterior limb of the right internal capsule with intraventricular extension resulting into mild communicating hydrocephalus. Right to left subfalcine herniation. subarachnoid hemorrhage, anterior interhemispheric fissure. Patient received nicardipine, mannitol, acetazolamide and analgesics. There were no subsequent episodes of seizure. There was no improvement in the level of consciousness of the patient but the pupils were 2-3 mm isocoric after a few hours. Next day at 12:10 pm the patient expired. The final diagnosis was brain herniation, intracerebral hemorrhage, and right anterior caudate nucleus with massive intraventricular extension. hypertensive cardiovascular disease, diabetes mellitus type 2.

Table 1: Patient characteristics and outcome

Patients	Age	Sex	Risk factors	BP	Aetiology	Symptoms	Signs	Outcome
Patient 1	23	Female	Hypertension Alcoholic	120/80	Undetermined Probably ruptured AVM	Headache vomiting	none	Good
Patient 2	53	Female	Hypertension	220/120	Hypertension	Headache Vomiting	none	Good
Patient 3	36	Female	Hypertension	260/150	Hypertension	Headache Vomiting	GCS 13 Neck stiffness Bilateral Babinski's	Poor
Patient 4	55	Male	Hypertension	200/120	Hypertension	Seizure Decreased consciousness Gaze paresis	GCS 3 Bilateral Babinski's	Poor

Table 2: CT findings and outcome

Patients	Location	Intraventricular extension	Involvement of internal capsule	Volume	ICHS Score	Outcome
Patient 1	Head of right caudate nucleus	Right ventricular system	none	2cc	1	Good
Patient 2	Head of right caudate nucleus	Right frontal horn of lateral ventricle	none	5cc	1	Good
Patient 3	Head of left caudate nucleus	Right ventricular system, mild communicating hydrocephalus	anterior limb of internal capsule	12cc	1	Poor
Patient 4	Head of right caudate nucleus	Right and left ventricular systems, mild communicating hydrocephalus	anterior limb of internal capsule	4cc	3	Poor

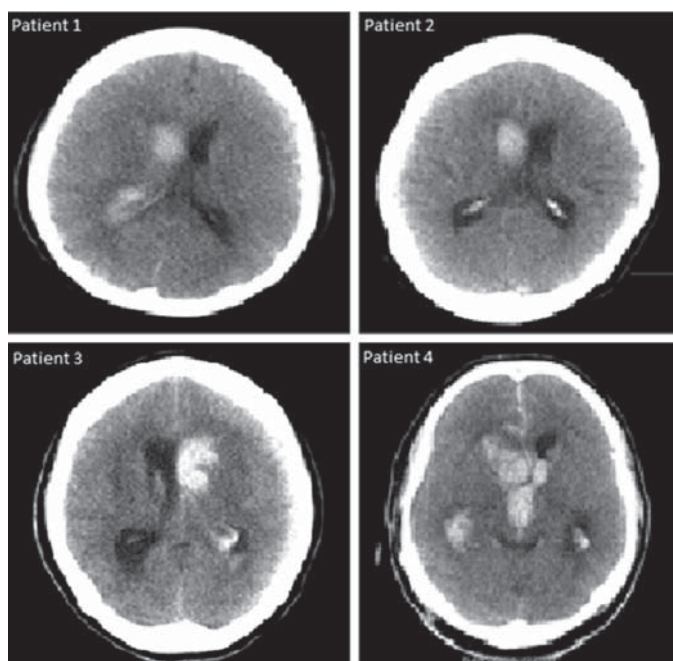


Figure: CT scans

Results

A total of 379 patients were reviewed. Out of them 4 patients were selected. The prevalence rate of the head of caudate nucleus hemorrhage in the patient population was 1.055 %. The mean age of the patients was 41.75 years. The youngest patient was 23 years old and the oldest was 55 years old. Three of the patients were female and there was one male. The male: female ratio in our study was 3:1. Three patients had elevated blood pressures. The systolic pressure was 200 or above they also had elevated diastolic pressure. The mean systolic blood pressure of these three patients was 226.6 mmHg and the mean diastolic pressure was 130 mmHg. The fourth patient was a non hypertensive. She had a smoking history of four pack years and she was an alcoholic. Headache was the predominant chief complaint. In three patients the initial complaint was headache. In all

the three cases it was associated with vomiting. One of the patients presented with seizure and decreased level of consciousness. Two patients who had a GCS of 15 did not present with any neurological findings. One of the patients had a GCS of 13. She also had neck stiffness and bilateral Babinski's. Another patient had a moribund GCS of 3 with bilateral Babinski's. Three out of four caudate haemorrhages involved the right side. All of them had an intraventricular extension. In two of the cases there was marginal involvement of the anterior limb of internal capsule. The volumes of bleed were of the four patients were 2cc, 5cc, 12cc and 4 cc. Two patients were noted to have mild communicating hydrocephalus. The ICHS score of three patients was 1 and one patient had an ICHS of 3. Two patients had a good outcome. They were sent home and advised outpatient follow up. Another two patients succumbed to their conditions. One expired 13hrs and another six days post ictus.

Discussion

Caudate hemorrhage is rare⁷. An intricate study of caudate hemorrhage becomes mandatory because of the following reasons. Firstly, very less has been written about caudate haemorrhages. Secondly, most of the studies on the variant of intracerebral hemorrhage have been on small patient populations. Thirdly, and most importantly clinical presentation of subarachnoid hemorrhage (SAH) and CN hemorrhage are alike. A CN hemorrhage must be entertained especially if there is a characteristic history and clinical signs strongly suggestive of subarachnoid hemorrhage. The initial complaints and neurological findings of caudate hemorrhage mimic subarachnoid hemorrhage, which is characterized by headache and vomiting, accompanied by neck stiffness and alterations in consciousness. Clinical presentations of all the patients were suggestive of subarachnoid hemorrhage. Furthermore a careful clinicotopographical analysis can enable us localize where in CN is the hemorrhage. Hemorrhage of CN head is likely if the patient presents

with headache, vomiting, neck stiffness, decreased consciousness, and behavioural changes. Similarly both head and the anterior limb of internal capsule are involved if the patient presents with gaze abnormalities and hemiparesis, and or hemiasthenia. A more inferior and lateral involvement can be predicted if there is presence of Horner's syndrome⁴. Two patients presented with headache and vomiting. They had a caudate head hemorrhage without involvement of the anterior limb of the internal capsule. One patient had gaze abnormality and the CT revealed involvement of anterior limb of internal capsule.

The independent risk factors of intracerebral hemorrhage are recent heavy and moderate alcohol consumption, hypertension, and likely also anticoagulant treatment in both men and women⁸. Similarly arteriovenous malformation is also a risk factor for CN hemorrhage⁹. Three of the patients presented hypertension the risk was aggravated by poor compliance to antihypertensives. The first patient a non hypertensive and is likely to have an arteriovenous malformation as the patient age tends to be lower than those with hypertension⁵.

Beside headache and vomiting one of our patients presented with a seizure and decreased consciousness. The seizure can be explained by the hyperdensity in the anterior interhemispheric fissure. Despite the prognostic relevance of the ICHS (Intracerebral

Hemorrhage Score), prognosis of caudate haemorrhage does not seem to abide by it. Presence of amount of intraventricular hemorrhage and resultant hydrocephalus seems to govern the outcome. A multivariate analysis done previously by Po-Chou and colleagues in stepwise logistical regression revealed that hydrocephalus was the most important factor associated with a poor outcome in our study. Caudate hemorrhage recovery can be predicted by hydrocephalus¹⁰. Hydrocephalus is the most important factor associated with poor outcome. Two of the patients revealed early signs of hydrocephalus and both had poor outcome. Statistically about 50% cases of caudate hemorrhage survive regardless of hydrocephalus (Michele). The limitation of this study is a small patient population.

Conclusion

Head of caudate hemorrhage is a rare variant of deep cerebral hemorrhage. The clinical presentation mimics SAH, similar presentations range from sudden severe headache to decrease in the level of consciousness. Hypertension was the foremost factor associated with caudate hemorrhage. Hydrocephalus is the most important factor in prognostication and strongly associate with poor outcome.

Disclosure

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