

Case Report

Vermal cerebellar hemorrhage as the initial manifestation of undiagnosed hypertension

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ABSTRACT

Vermal cerebellar haemorrhage is bleeding affecting the midline region of the cerebellum. It is uncommon, accounting for 5-13% of all intracerebral haemorrhages. It is a grave condition with a high mortality. The diagnosis must be made early, if not, most patients would die within 48 hours. A 70-year-old lady presented to the accident and emergency department with a six hours history of sudden onset generalized tonic-clonic seizures following activity and loss of consciousness. She was unconscious with a Glasgow coma score of 3/15 with neck stiffness, generalized hypotonia and globally diminished reflexes. Her blood pressure was 240/120 mmHg with a mean arterial pressure of 160 mmHg. The brain computerized tomographic scan showed a hyperdense area in the cerebellar vermis with extension to both hemispheres and fourth ventricle. Consciousness was regained after three weeks of conservative management following which she became extremely restless and noticed to have developed titubations with nystagmus of both eyes. She also developed intention tremors, scanning dysarthria, generalized hypotonia, dysmetria, dysidiadochokinesia and intentional bilateral tremors. Her recovery was slow and gradual. Once she was able to stand with support, she was noticed to have florid truncal ataxia, lower limbs ataxia, and titubations.

Keywords: Cerebellar, Haemorrhage, Hypertension, Vermis

INTRODUCTION

Vermal cerebellar haemorrhage is bleeding affecting the midline region of the cerebellum. It is uncommon, accounting for 5-13% of all intracerebral haemorrhages.¹ It is a grave condition with a high mortality. The diagnosis must be made early, if not, most patients would die within 48 hours.²

Uncontrolled hypertension is the commonest etiology of spontaneous cerebellar hemorrhage.^{3,4} There are a few reports on vermal cerebellar bleed in the literature. The cases described include those arising from a ruptured aneurysm of the posterior inferior cerebellar artery, haemophilia A, supratentorial surgery and anticoagulants.⁵⁻⁸ To the best of our knowledge, this rare form of cerebellar haemorrhage has not been reported from our

environment. We describe the clinical presentation of an elderly lady presenting with vermal cerebellar bleed following undiagnosed hypertension. We also highlight our experience with the conservative management of her condition.

CASE REPORT

A 70-year-old lady presented to the accident and emergency department of the University of Port Harcourt Teaching Hospital with a six hours history of sudden onset generalised tonic-clonic seizures following activity (firewood cutting) and loss of consciousness. There was no preceding history of headaches. Her past medical history was significant for an episode of nose bleeding which occurred six months ago but was not given any significance. She was not a previously known

hypertensive or diabetic. She was unconscious with a Glasgow coma score of 3/15 with neck stiffness, generalised hypotonia and globally diminished reflexes. Her blood pressure was 240/120mmHg with a mean arterial pressure of 160mmHg. Her chest revealed bibasal crepitations. Her abdominal examination was normal. Present initial consideration was subarachnoid haemorrhage to exclude intracerebral haemorrhage with intraventricular extension.



Figure 1: Brain computerised tomography image of vermal cerebellar bleed.

The brain computerized tomographic scan (Figure 1) showed a hyperdense area in the cerebellar vermis with extension to both hemispheres and fourth ventricle. Lateral and third ventricles were enlarged. Sulci and gyri were prominent. Electrocardiogram showed left atrial enlargement and left ventricular hypertrophy consistent with long-standing hypertension. Her renal indices and lipid parameters were all within normal limits.

Consciousness was regained after three weeks of conservative management following which she became extremely restless and noticed to have developed titubations with rapid to-and-fro movements of both eyes. She also had intention tremors. Her speech was forceful and mono-syllabic. Examination at this time revealed nystagmus, scanning dysarthria, generalized hypotonia, dysmetria, dysdiadochokinesia and intentional bilateral tremors. Her recovery was slow and gradual. Once she

was able to stand with support, she was noticed to have florid truncal and lower limbs ataxia.

DISCUSSION

An interesting aspect of this case was the evolution of florid cerebellar symptoms and signs once she regained full consciousness. Clinical presentation depends rapidly of enlargement and the size of the bleed. If small, they can present relatively subtle with only signs (e.g. ataxia, nystagmus). Large hemorrhages can impair consciousness and obstruct the fourth ventricle resulting in obstructive hydrocephalus. The clinical presentation of cerebellar haemorrhage has been divided into three groups: those with sudden onset and rapidly progressive signs including coma; those with a headache, vomiting, vertigo, and ataxia who later developed decreased consciousness; and the third group with gradual onset hydrocephalus.^{9,10} Clinical diagnosis is, however, unreliable and some form of radiological investigations is needed. Brain lesions appear as areas of hyperdensities within the cerebellar hemispheres. Extension into the subarachnoid space or fourth ventricle is also common.

The management of acute cerebellar hemorrhage often requires making swift and difficult decisions by the attending neurologist. Certain easily identifiable clinical and neuroimaging findings may assist in appropriate patient triage and timely neurosurgical intervention.¹¹ Treatment of patients with cerebellar haematomas remains a somewhat controversial topic. The major reason for the controversy is the relative lack of ability to predict the outcome. Large hematoma size (>3 cm), decreased the level of alertness especially coma, hydrocephalus, and cisternal obliteration on CT scans predict poor outcomes without surgery.¹² Computer tomographic scan and clinical findings that may be predictors of acute deterioration in cerebellar hemorrhage include: upward herniation of vermal cerebellar tissue through the tentorial notch, intraventricular hemorrhage, brainstem distortion by direct mass effect, haematoma diameter greater than 3 cm, acute hydrocephalus from compression of the fourth ventricle, occlusion of the cerebral aqueduct as a secondary phenomenon, hemorrhage extending into the cerebellar vermis, pinpoint pupils and admission systolic blood pressure greater than 200 mmHg.¹³ Her blood pressure at presentation and the midline location of the bleed were the only predictors of acute deterioration we identified.

Typically, if hemorrhage causes brainstem compression or has a volume greater than 3cm in diameter (20-30 mL) evacuation is beneficial.² If hydrocephalus is the main cause of deterioration, a temporising ventriculostomy is performed. No further surgical therapy may be needed if the patient improves as was with the index case. A decompressive posterior fossa craniotomy should be emergently performed if the patient does not improve immediately.^{14,15}

Our patient was managed conservatively entirely in close liaison with the neurosurgeons. Physiotherapy was instituted and patient discharged in good condition after a six weeks period of hospital stay on anti-hypertensives. A follow-up brain CT scan was not done due to financial constraints. She was followed up in the neurology clinic with progressive neurologic improvements.

CONCLUSION

Though the prognosis and outcome for this condition are very poor, it can be successfully managed if identified early and appropriate treatment instituted promptly. A watch and see approach may be best when patient's condition may be further worsened by neurosurgical intervention.

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