Case Report

Complete Recovery of a Booked Primigravida with Hemorrhagic Stroke Due to Severe Preeclampsia

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ABSTRACT

Stroke is a major contributor to morbidity and mortality during pregnancy and puerperium. Intracerebral hemorrhage (ICH), which is bleeding within the cerebral parenchyma as a result of rupture of a blood vessel, is the second most common type of stroke after ischemic stroke. We report a case of ICH due to severe preeclampsia that was suspected to have occurred shortly before cesarean section in a woman with prior normal prenatal care. She recovered completely without any neurological deficit.

KEY WORDS: Complete recovery, intracerebral hemorrhage, Nigeria, preeclampsia, stroke

INTRODUCTION

Stroke is one of the significant cause of morbidity and mortality worldwide. Stroke is a rare event in young people. However stroke related to pregnancy accounts for about 30% of strokes in the young people. Stroke in pregnancy occurs primarily in late pregnancy and puerperium. Intracerebral haemorrhage (ICH) has been noted to be associated with higher mortality rate than other forms of stroke. The etiology of stroke in pregnancy is generally similar to that of stroke in the general population with the exception of conditions which occur only in pregnancy. Preeclampsia/eclampsia is the most common cause of stroke in pregnancy. It has been noted that preeclampsia is associated with a fourfold increase in stroke during pregnancy. In this case report we present the case of a primigravid woman with sudden onset of pre-eclampsia complicated with ICH. She however recovered completely.

CASE REPORT

A 30-year-old primigravid black woman in her 38th week of gestation presented at the Accident and Emergency Department of Abuja Clinics Limited on February 27, 2013, at 8 pm with complaints of diffuse headache, dizziness, and epigastric pain all of about 12 h duration. She also reported a slight reduction in her urine output. There was no history of visual disturbances, loss of consciousness, or seizure.

The positive findings on physical examination were generalized edema, severely elevated blood pressure of 220/110 mmHg, and epigastric tenderness. She was fully conscious and alert with no neurologic deficit. Obstetric examination revealed a symphysis-fundal height of 36 cm, longitudinal lie, cephalic presentation, and the fetal heart rate of 148 beats/min. Other aspects of the systemic examination were unremarkable. Urine dipstick analysis showed significant proteinuria (4+).

Her pregnancy had been relatively uneventful until 48 h before her presentation at the accident and emergency when she was noticed to have had mildly elevated blood pressure (140/90 mmHg) during a routine prenatal visit, but her urinalysis dipstick test was normal.

She booked for prenatal care at 13 weeks gestation and had made regular prenatal care visits. At booking, her pulse

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Access this article online	
Quick Response Code	Website: www.jbcrs.org
	DOI: ***

How to cite this article: ***

rate was 70 beats/min; blood pressure was 120/80 mmHg, and her weight was 56.3 kg. Obstetric ultrasound scans and urine analysis using dipstick done throughout the prenatal period were normal.

Her clinical history revealed repeated complaints of palpitation that was extensively evaluated on several occasions by different cardiologists, but no cardiovascular abnormality was detected. Laboratory workup at the accident and emergency room showed thrombocytopenia (platelet count of 72,000/μl), hematocrit of 37%, hemoglobin of 12.2 g/dl, clotting time of 5.54 min, and international normalization ratio of 1.19. There was slightly elevated total serum bilirubin (2.2 mg/dl) but normal level of liver enzymes. The serum electrolyte, urea, and creatinine values were essentially normal.

A diagnosis of severe preeclampsia with imminent eclampsia was made. The patient, her husband, and relatives were all counseled extensively on the diagnosis and management options. Stabilization of the patient was immediately commenced.

It was instituted parenteral antihypertensive therapy with hydralazine and seizure prophylaxis with magnesium sulfate using the regimen as proposed by Pritchard. Approximately 1 h after admission, while being stabilized for delivery, she developed sudden alteration in the level of consciousness. There was progressive decrease in the Glasgow Coma Scale (GCS) score. She became disorientated and confused and could recognize neither her husband nor mother.

Emergency lower segment cesarean section was performed under spinal anesthesia leading to the delivery of a healthy 2.5 kg male infant with Apgar scores of 8 and 10 in the 1st and 5th min, respectively.

She was admitted to the intensive care unit. In the immediate postoperative period, she remained drowsy and confused with persistent inappropriate speech and altered consciousness. There was a continuous drop in her GCS score. However, there was no obvious lateralizing sign, visible cranial nerve deficit, or seizure. Fundoscopy showed evidence of papilledema. Computed tomography (CT) scan of the brain was done due to lack of improvement in her level of consciousness within the first few hours after surgery.

The noncontrast brain CT scan done on February 28, 2013, revealed subarachnoid, intraventricular, and intracerebral (left parietal lobe) hemorrhage (ICH) in the left middle cerebral artery arterial distribution. The combined volume of the ICH and intraventricular hemorrhage was 215 ml. There was the deletion of the basal cisterns and minimal perilesional edema. There was no significant midline deviation [Figure 1].

Emergency neurosurgical consultation was sorted. The neurosurgical unit opted for conservative management instead of emergency craniotomy because of worsening thrombocytopenia, uncontrolled hypertension, and the high risk of damage to adjacent normal brain tissue from an anticipated possible reactionary hemorrhage.

She received supplemental oxygen via nasal catheter and carefully titrated intravenous hydralazine. Magnesium sulfate was continued for the next 24 h postoperatively with careful monitoring of her urine output, respiratory rate, and patellar reflex/knee jerk. She was also treated with analgesics, intravenous fluids (1.5–2 L of normal saline per day), and prophylactic antibiotics – single dose ceftriaxone given about 30 min before the cesarean section.

Intravenous mannitol infusion (20% solution infused over 30 min) was given for 24 h to reduce brain edema and conservative management of local mass effect. She also received tablet amlodipine via a nasogastric tube because of worsening blood pressure despite IV hydralazine. She was nursed at 30° head-up position and was later given antioxidants (Vitamins C and E). Her GCS score improved to 14 from GCS score of 8 by the first 24 h after delivery.

Forty-five hours after surgery, the patient became very restless and aggressive with incoherent speech. She was then given parenteral phenobarbitone empirically for 48 h at 60 mg IV 8 hourly.

Serial postoperative hematological profiles revealed a persistent decline in her hematocrit (to 22%) as well as a further drop in her platelet count (to 39,000/μl). She was

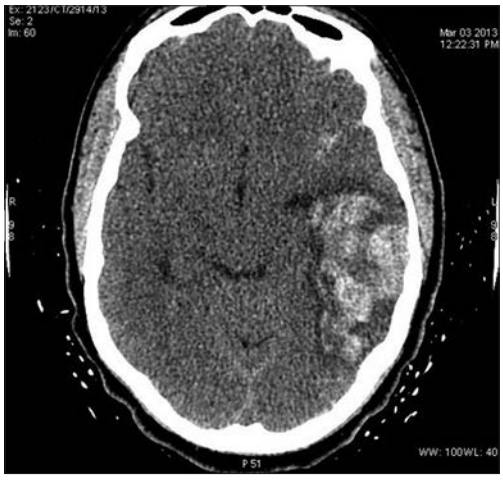

Figure 1: Marked perilesional edema, deletion of basal cistern and effacement of the frontal horn of the left lateral ventricle ipsilateral to the hematoma

subsequently transfused with packed cells which elevated her hematocrit to 31%. Thrombocytopenia was treated by administering platelet concentrates. Repeat CT brain imaging done on the 3rd day showed some resolution of the intraventricular hematoma, organization of the left intracerebral hematoma, effacement of the sulci and gyri, and persistence of the subarachnoid hemorrhage. There was marked perilesional edema, and some basal cisterns were still deleted. There was no significant midline shift [Figure 2]. CT angiography did not suggest any intracranial aneurysm or arteriovenous malformations (AVM) [Figure 3].

At this point, the option of surgical intervention (craniotomy and evacuation of accessible intracerebral hematoma) was suggested by the neurosurgeon but was declined by the patient's relatives.

Four days after the cesarean section, the patient developed high-grade fever and was empirically treated for malaria with a full course of oral artemisinin-based combination therapy and antipyretic. Physiotherapy was done throughout the period. There was persistent papilledema, restlessness, headache, and vomiting until the 8th day.

Repeat hematological profiles done on the 6th postoperative day showed resolution of the anemia and thrombocytopenia with hematocrit and platelet count of 31% and 185,000/µl, respectively. Serum potassium was initially 2.2 mmol/L also normalized at (3.7 mEq/L) by the 7th postoperative day. The dose of oral amlodipine was increased to 10 mg p.o. daily because of persistent high blood pressure spikes.

There was severe constant headache and vomiting. A third brain CT imaging showed resolution of the intraventricular hematoma but the persistence of the intracerebral hematoma with surrounding edema and mass effect. The patient had another 24-h course of mannitol infusion and was commenced on oral phenobarbitone. Surgical intervention was again declined by the patient's relatives. Intermittent bolus doses of diazepam and chlorpromazine were administered to control aggressive behavior. She was continued conservative management until the 22nd day when she was discharged in a stable condition to continue further management as an outpatient.

DISCUSSION

Stroke is not only a leading cause of disability worldwide but also the second leading cause of death in the general population.[1,2] The overall incidence of ischemic stroke during pregnancy is low (3.5–5/100,000 pregnancies) in the developed world with the majority of these events occurring late in pregnancy and the postpartum period.[3] However, when considering stroke in the young people, it has been estimated that stroke related to pregnancy accounts for 12–35% of stroke events in this otherwise low-risk populate.[4] The overall incidence of stroke in pregnancy is low and occurs primarily in late pregnancy and puerperium.[5,6] The incidence of hemorrhagic stroke is similar to ischemic stroke, but ICH has a higher maternal mortality rate and has been estimated to account for 5–12% of the overall maternal mortality.[4]

The etiology of stroke in pregnancy is generally similar to that of stroke in the general population with the exception of conditions which occur only in pregnancy. Stroke risk factors include age above 35 years, cerebral aneurysm, AVM, cerebral amyloid, angiopathy, and vasculopathy.[7] Other predisposing factors are antiplatelet(s), anticoagulant medications, exertion, tobacco use, cocaine use, family history of subarachnoid hemorrhage, female sex, African origin, and

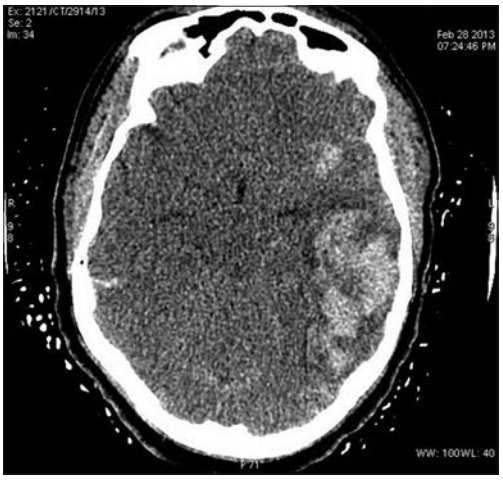

Figure 2: Computed tomography scan showing subarachnoid, intraventricular, and intracerebral hemorrhage, deletion of the basal cisterns

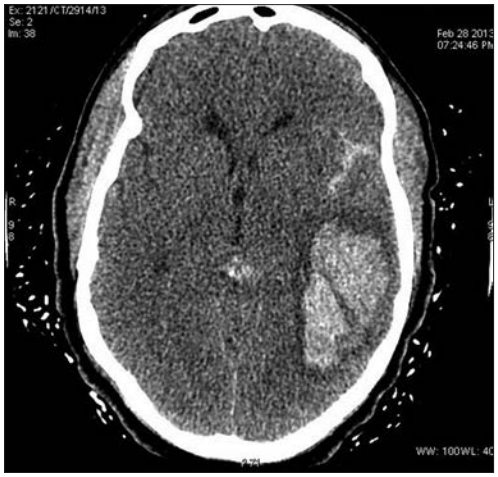

Figure 3: There is reduction of intracerebral hematoma, resolution of intraventricular component, and visible basal cisterns

connective tissue disorder.[8] It can also follow trauma and hypertension. Hypertension, which is the most common risk factor for stroke in the general population, is also a component of preeclampsia/eclampsia that is the most common cause of stroke in pregnancy.[4] It has been noted that preeclampsia is associated with a fourfold increase in stroke during pregnancy.[9] Researchers have noted an increased risk of stroke both in subsequent pregnancy and later in life with a diagnosis of preeclampsia during pregnancy.[10,11] Nonetheless, pregnancy-related stroke is also associated with specific conditions which only occur in pregnancy. These include preeclampsia, eclampsia, amniotic fluid embolism, peripartum cardiomyopathy, and postpartum angiopathy.[12] The risk of ICH has been postulated to be highest in the postpartum period.[11,13,14]

The clinical features in our patient that lead to the suspicion of stroke include persistent headache, altered consciousness, vomiting, incoherent speech, and worsening GCS score. The diagnosis of stroke is usually considered in patients who presented with acute onset of focal neurological changes in the absence of an alternative etiology. Rarely, some patients may present with nonfocal symptoms such as headache and altered consciousness as in our patient. The investigations necessary to establish the diagnosis of ICH include CT scan, CT angiography, and magnetic resonance imaging (MRI). CT scan and CT angiography were used in the diagnosis and assessment of the severity of the neurological injury as well as evaluation for causes and progression of the disease condition. The safety of neuroimaging in pregnancy has been a subject of great controversy among many obstetricians. However, obtaining an early noncontrast computerized tomography (with appropriate shielding) or MRI of the brain will greatly facilitate diagnosis with minimal risk to the fetus during pregnancy.[15,16]

The definitive management of preeclampsia is aimed at the delivery of the fetus and placenta and administration of antihypertensive and seizure prophylaxis.[17,18]

Magnesium sulfate was used for seizure prophylaxis. It has also been confirmed to be effective in the treatment of eclampsia and reduction of the rate of eclampsia and its complications in preeclamptic patients.[19,20]

The cesarean section under spinal anesthesia was our preferred mode of delivered as general anesthesia has been reported to be associated with increased risk of stroke when compared with neuraxial anesthesia in preeclamptic women.[21]

Early emergency neurosurgical consultation is necessary to assess the nature, severity, location of injury, and management options. The need to establish devices to monitor intracranial pressure (ICP), brain temperature and brain oxygen, and patient suitability for decompression will also be assessed. Elevated ICP >20 mmHg may lead to brain herniation and death. Interventions that were done to reduce ICP include sedation to a calm, motionless state, optimizing cerebral perfusion by giving intranasal oxygen, and mannitol infusion. Other interventions that could be done are infusion of hypertonic saline, hyperventilation, hypothermia, paralysis, and induction of coma with medications.[22] Mannitol is also effective in the management of cerebral edema which may result from cerebral ischemic infarction or contusion.

Continuous assessment of the degree of consciousness using the GCS score is essential in the management of stroke. A third brain CT imaging [Figure 4] showed resolution of the intraventricular haematoma.

Fever, as noted in our patient, is a common complication of intracranial hemorrhage. The risk factors for fever include intraventricular blood, blood in the pituitary, damage to the anterior hypothalamus, and ventricular drainage catheter.[23-25] It has been noted that the greater the burden of fever the worse the outcome.[23-25]

Antihypertensive agents are necessary to lower the blood pressure, reduce blood extravasation into the cerebral tissue as sustained severe hypertension is associated with hematoma growth and worsen clinical outcome.[26,27] However, caution must be applied in the use of antihypertensive agents as blood pressure reduction may lead to reduced cerebral perfusion, cerebral ischemia, and infarction.

Where the patient is on anticoagulant, this should be stopped and reversed.[28] She received platelet and red cell

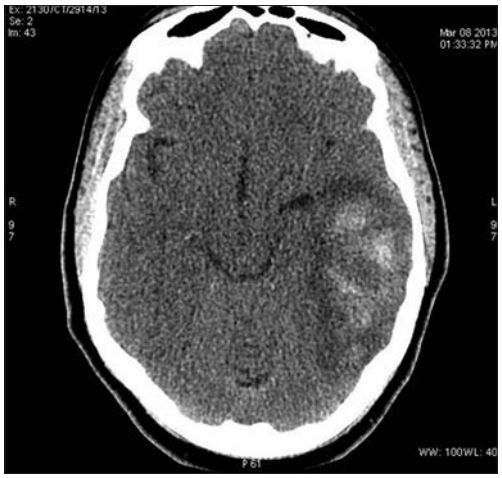

Figure 4: Subarachnoid, intraventricular and Intracerebral haemorrhage, delation of basal cisterns

transfusion. Red cell transfusion is shown to be associated with reduced mortality. Where seizure is present or where there is an unexplained worsening level of consciousness, monitoring with continuous electroencephalogram is necessary.

She made a routine antenatal visit with blood pressure of 140/90 mmHg and urinalysis showing protein of one plus and within 28 h presented in the Accident and Emergency Department with a severe blood pressure of 240/190 mmHg and clinical signs of imminent eclampsia. The sudden, rapid, and dramatic deterioration of the hypertensive state of the patient may suggest the need for obstetrician(s) to consider an early commencement of antihypertensive(s) and a more aggressive treatment of hypertension in women with mild hypertension in pregnancy to prevent/guard against ICH/stroke. This may suggest that any level of rise in blood pressure during pregnancy can lead to hypertensive encephalopathy in a pregnant woman without any previous history of hypertension. Similar assertion has also been reported in another case report.[29]

The prognosis of ICH has been noted to be poor with a case fatality at 1 month being over 40%.[30,31] In the same vein, only a small percentage of those who survive reach independent life after 1 year.[32] However, our patient recovered completely probably due to early diagnosis and immediate institution of critical care. The pregnant woman is vulnerable to delayed diagnosis and possible complications because many diagnostic investigations are avoided during pregnancy due to the concern of causing harm to the fetus.

There is the need for increased vigilance in hypertensive management in preeclamptic patients. ICH occurring in the background of preeclampsia/eclampsia should be managed closely with antihypertensives, antiseizure medications preferably magnesium sulfate.[14,20,33-35] Routine cardiovascular risk reduction counseling should be provided for antenatal women in general but more specifically women with the risk and or history of preeclampsia.

CONCLUSION

Although pregnancy-related stroke is a rare phenomenon, it is an important cause of morbidity and mortality among pregnant women. There should be a high index of suspicion among the obstetricians for early diagnosis. Multidisciplinary management should be the bedrock of care. Adequate follow-up and rehabilitation should be an important component of the care. Obstetricians should increase their use of radiological investigations in the management of neurological conditions in pregnancy.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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