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Hypertensive encephalopathy in a patient with unconsciousness: A case report

Mouhammad Ekbal Alkalaf

Abstract--Hypertensive encephalopathy (HE) is characterized by acute onset severe arterial hypertension associated with confusion, headache, seizure and even unconsciousness. It is usually reversible but any delay in recognition and treatment can be life threatening. Typical abnormality in MRI is increased intensity in both occipital white matter areas indicative of edema. Differentials can be ischaemic stroke, ICH, Tumor, acute toxic metabolic encephalopathy. We here describe a patient with no history but admitted with hypertension and loss of consciousness and had several seizures. She recovered with appropriate treatment for hypertension.

Keywords---hypertensive encephalopathy, seizure, unconsciousness.

Introduction

A hypertensive emergency is a life-threatening condition where ongoing target-organ damage occurs due to markedly elevated blood pressure. Pulmonary edema, cardiac ischemic events, acute renal failure, aortic dissection, eclampsia, retinopathy, and encephalopathy may present as a result of organ injury due to hypertension.

Hypertensive encephalopathy is a less commonly encountered type of hypertensive emergency. It is characterized by signs of cerebral edema that occur after a severe episode of hypertension. This condition is usually diagnosed retrospectively after symptoms dramatically resolve by lowering the patient's blood pressure, and other causes of the neurologic disease have been ruled out. Symptoms of hypertensive encephalopathy include the gradual onset of headache, nausea, and vomiting, followed by neurologic symptoms such as restlessness, confusion, seizures, unconsciousness and potentially coma. If hypertension is treated promptly, the symptoms of encephalopathy are usually reversible (1).

Elevated blood pressure is considered as a potential predisposing factor as well as a precipitating factor for acute changes in mental status. The pathogenesis of the link between chronic hypertension and altered mental status (AMS) is uncertain, but proposed mechanisms include: 1) associated brain atrophy leading to

cognitive impairment and 2) atherosclerosis resulting in brain hypoperfusion and cellular hypoxia **(1)**. AMS is also a possible complication of hypertensive crisis (i.e. sudden elevation of blood pressure above 180/120) secondary to cerebral edema, which occurs when the autoregulation of cerebral blood flow is overwhelmed and cerebral vasodilation ensues. Hypertensive encephalopathy is the general term for the presence of altered consciousness and other neurologic findings in the context of a hypertensive crisis **(2)**.

Case presentation

A 60 year old Chinese lady was brought by ambulance in unconscious state. No past history was available as she was a tourist. Her GCS on arrival was 4/15; Pupils bilaterally reacting with normal in size. There was left beating nystagmus which precluded proper fundoscopic examination. Her BP was 210/120 mm of Hg (no discrepancy in any arm with normal peripheral pulses). There was no sign of lateralization in any limbs. Inj. Labetalol was given for reduction in BP with target of 15% reduction initially. She had two episodes of generalized tonic clonic seizure upon arrival which were controlled with Diazepam IV. Airway protection measures were taken.

Blood tests were normal. ECG showed only sinus tachycardia. ABG showed mild respiratory acidosis. CT scan brain showed no abnormality. CT angiogram brain revealed normal arteries in all cerebral territories. MRI brain was normal as well. IV Levetiracetam was started subsequently. EEG studies revealed no significant abnormality. Urine toxicology was normal. CSF analysis did not reveal any infective etiology or any albumino-cytological dissociation. Patients' sensorium improved dramatically with control of blood pressure and she was alert, conscious, and cooperative within 48 hours of presentation without any neurological deficit.

Discussion

The initiation of the patient's altered mental status after persistent hypertension in the critical range, resolution following successful management of hypertensive emergency with CT scan brain which showed no abnormality. MRI brain was normal as well. Labetalol was given for reduction in BP with target of 15% reduction initially. IV Levetiracetam was started subsequently. Patients' sensorium improved dramatically with control of blood pressure and she was alert, conscious, and cooperative within 48 hours of presentation without any neurological deficit. This suggests that his AMS was most likely secondary to hypertensive encephalopathy. Posterior reversible encephalopathy syndrome is another presentation of HE which could not be found in our case as MRI did not reveal any cerebral edema.

Normally, the brain sustains blood flow within a narrow perfusion pressure range without being affected by fluctuations in systemic arterial pressure. For healthy individuals, the pressure ranges are 50-150 mm Hg cerebral perfusion pressure (CPP) or 60 to 160 mm Hg mean arterial pressure (MAP). The CPP = MAP – intracranial pressure (ICP) **(2)**.

With increased MAP, cerebral arteriolar vasoconstriction occurs, and conversely, with decreased MAP, arteriolar dilation occurs to keep the CPP constant. This adaptive process maintains brain perfusion at a constant level despite systemic blood pressure changes. However, a sudden and severe increase in arterial pressure can exceed this autoregulatory mechanism because the arterioles are limited in their ability to constrict. The then intracerebral elevated blood pressure causes a breach in the blood-brain barrier, and vascular fluid diffuses across the capillary membranes into the brain parenchyma. This leads to the development of cerebral edema, increased intracranial pressure, and neurologic deficits such as altered mentation, visual deficits, seizures and even unconsciousness **(1)**.

In previously normotensive patients, acute episodes of hypertension may induce hypertensive encephalopathy at diastolic blood pressures as low as 100 mm Hg. This scenario may be seen with patients that develop eclampsia or in patients receiving cytotoxic and immunosuppressive therapies **(3)**.

A thorough physical exam and history are primarily used to diagnose hypertensive encephalopathy in patients presenting with elevated blood pressure in addition to altered mental status, visual abnormalities, headache, or seizures. Eliciting a thorough drug history is essential for identifying any previously used antihypertensive drugs. Typically, patients who develop hypertensive encephalopathy have chronic uncontrolled hypertension and may have recently discontinued their antihypertensive medication. Individuals that have rapidly developing and/or intermittent episodes of hypertension are also more at risk for developing hypertensive encephalopathy **(1)**.

The majority of patients with this diagnosis have blood pressures in excess of 220/120 mm Hg. **(4)**. These patients should be evaluated for signs of organ damage that can be found during a hypertensive emergency. In particular, thoracic auscultation may reveal signs reflective of cardiac dysfunction, such as extra heart sounds or pulmonary edema, with rales heard on lung auscultation. Fundoscopy may show retinal hemorrhages and papilledema, which is a sign of severe hypertensive retinopathy. A complete neurologic exam can identify whether focal or non-focal deficits are present and may warrant other differential diagnoses for conditions that cause similar symptoms to be considered **(5)**.

This case highlights that state of the patients unconsciousness associated with severely elevated BP without any abnormality detected and that the most probable cause of these is cerebral edema secondary to sudden rise of BP as complete resolution after BP control occurred and no abnormality in imaging and other studies, establishes Hypertensive encephalopathy (HE) as the cause of patients unconsciousness and convulsions.

Conclusions

We here report that the state of the patients unconsciousness associated with severely elevated BP, complete resolution after BP control and no abnormality in imaging and other studies, establishes Hypertensive encephalopathy (HE) as the cause of patients unconsciousness and convulsions. Most probable cause of these

is cerebral edema secondary to sudden rise of BP. Posterior reversible encephalopathy syndrome is another presentation of HE which could not be found in our case as MRI did not reveal any cerebral edema.

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