Reminder of important clinical lesson

Unusual association of eclamptic encephalopathy and Japanese encephalitis

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Summary

Eclampsia is one of the hypertensive disorders of pregnancy causing considerable maternal morbidity and mortality. It is more common in developing nations, where access to adequate obstetric care is limited to a small population. Eclampsia is clinically characterised by encephalopathy, seizures, headache and cortical blindness and occurs in the setting of pre-eclampsia that comprises hypertension, proteinuria and peripheral oedema. Japanese encephalitis has emerged as a serious neurological disorder in South East Asian countries. In this case report, we highlight the unusual association of eclamptic encephalopathy and Japanese encephalitis in a patient belonging to northern India. This combination of illnesses caused diagnostic difficulty and delayed hospitalisation. A high index of suspicion is required in the endemic zone of Japanese encephalitis if the expected results are lacking in patients with eclampsia.

BACKGROUND

Eclampsia is one of the hypertensive disorders of pregnancy, mainly manifesting with central nervous system symptoms. The eclamptic disorder is one of the leading causes of maternal mortality, more so in the developing nations. The neurological symptoms described are moderate to severe headache, nausea, vomiting, altered sensorium, cortical blindness and recurrent seizures.¹ The cerebrovascular involvement is the determining factor of mortality. MRI findings correlate with the presence of vasogenic oedema, manifesting as hyperintense signals on T2-weighted images (figure 1) and restriction on diffusion weighted images (figure 2). The lesions are mainly localised to parieto-occipital cortex and subcortical regions.² Japanese encephalitis is one of the important causes of virus encephalitis, causing widespread morbidity and mortality, particularly in South East Asian countries including India.³ In this case report, we present a patient of eclamptic encephalopathy associated with Japanese encephalitis. This misled the treating obstetrician and caused delayed hospitalisation.

CASE PRESENTATION

A 21-year-old female primigravida in the last trimester of pregnancy developed recurrent convulsions, followed by altered sensorium 7 days prior to admission. The convulsions were generalised tonic clonic in nature, sometimes associated with tongue bite and bladder incontinence. The patient had moderate to high-grade fever with occasional vomiting. In this state the patient was admitted to a primary healthcare hospital and delivered through caesarean section. She did not regain consciousness and was referred to a tertiary hospital for further management. She was detected to be hypertensive with significant proteinuria. The diagnosis of eclampsia was considered and magnesium sulphate was administered. The seizures were still uncontrolled. At this juncture, injection phenytoin (18 mg/kg

body weight) was loaded. The seizures continued to occur although with decreased frequency. Her sensorium worsened. In spite of the best treatment offered for eclampsia, the patient had prolonged comatose state with ongoing seizures. There was no history of arthralgia, rash, diabetes and tuberculosis.

General examination revealed raised temperature, tachycardia with elevated blood pressure 190/110 mm Hg. Pedal oedema was present. The Glasgow coma scale score was E3M2V1. The motor system evaluation depicted hyper-tonia in all limbs, which included both spasticity and rigidity. The plantars were bilaterally extensor.

Figure 1 MRI (T2 fluid-attenuated inversion recovery) demonstrated bilateral hyperintense signals in thalamus (arrows).

Figure 2 Diffusion-weighted imaging depicted symmetrical areas of hyperintense signals in the bilateral periventricular region, corpus callosum and thalamus.

INVESTIGATIONS

The haematological parameters revealed the following findings: Hb—8.7 g%, total leucocyte count—11 450/mm³, platelet count—1.43 lac/mm³, random blood sugar—103 mg%, serum Na⁺/K⁺—133/4.1 mEq/l, blood urea—40 mg%, serum creatinine—1.0 mg%, serum bilirubin—0.6 mg%, serum glutamate pyruvate transaminase—109 mg%. The urine examination revealed proteinuria. The thyroid study including triiodothyronine, tetraiodothyronine, thyroid-stimulating hormone and antibody were unremarkable. The autoantibodies for antinuclear antibody, anti-neutrophilic cytoplasmic antibody, rheumatoid factor and antiphospholipid reactants were non-reactive in sera study. The cerebrospinal fluid routine assessment revealed pleocytosis (51 cells, all lymphocytes) with normal protein and sugar values with nonreactivity to herpes virus, Ebstein bar virus and Dengue virus. The cerebrospinal fluid and serum study for Japanese encephalitis virus antibodies was positive in high titres. The HIV was not reactive. The KF (Kayser–Fleischer) ring was not visible on slit lamp examination. The electroencephalographic study depicted generalised slowing.

MRI of brain on T2 Flair images and diffusion-weighted images demonstrated symmetrical areas of altered signals involving bilateral periventricular, splenium and body of corpus callosum and bilateral thalamic regions (figure 2). Magnetic resonance venography and angiography study did not show any abnormality. The final diagnosis was eclamptic encephalopathy associated with acute virus encephalitis due to Japanese encephalitis (figure 3).

TREATMENT

The patient was initially given an injection of magnesium sulphate at a dosage of 2 g per hour for 2 days. Owing to uncontrolled seizures, the patient was loaded with the injection phenytoin 18 mg/kg body weight. During the further course of illness, she was treated with

Figure 3 Repeat MRI study revealed the resolution of the lesion.

antioedema drugs and antibiotics with supportive management in the intensive care unit.

OUTCOME AND FOLLOW-UP

The patient was shifted to the neurology intensive care unit and was prescribed decongestives, antiepileptics and supportive treatment. She was discharged after a month with partial recovery. The patient regained activities of daily living at 6 month follow-up.

DISCUSSION

Eclampsia is characterised by the development of new onset seizures in peripartum woman against the background of the pre-eclampsia syndrome, which comprises hypertension >140/90 mm Hg, proteinuria and pedal oedema. The incidence of pre-eclampsia is around 4–5% in pregnant women.⁴ Neuroimaging, particularly MRI including diffusion-weighted images, shows characteristic abnormalities in eclampsia. The radiological findings described are hyperintense signals in bilateral parieto-occipital lobes, thalamus and basal ganglia on T2-weighted images with restriction (hyperintensity) on diffusion-weighted imaging and corresponding normal to increased apparent diffusion coefficient.⁵ The clinical and neuroimaging findings could be explained by the development of oedema due to an abrupt rise in blood pressure, resulting in dilatation of vessels, impairment of autoregulation and disturbances in the blood–brain barrier.⁶

Our patient suffered from eclamptic encephalopathy and was treated under the care of an obstetrician. Even with the best management of eclampsia, desired results were not obtained. She was finally diagnosed with Japanese encephalitis.

Japanese encephalitis is a mosquito-borne, zoonotic infection caused by Flavivirus, which is a significant public health problem in South East Asia. The cardinal features of Japanese encephalitis described are fever, headache, vomiting, convulsions, altered consciousness, masked facies,

rigidity and abnormal movements.⁷ In about 50% of patients, CT-scanned head shows bilateral low-density lesions in thalamus and brainstem. MRI being more sensitive, it demonstrates more widespread lesions (hyperintense signals on T2-weighted and fluid-attenuated inversion recovery) in the regions of thalamus, cerebral hemispheres and cerebellum.⁸ Our patient had an unusual combination of eclamptic encephalopathy and Japanese encephalitis, which raised diagnostic concerns.

Learning points

- ▶ Many clinical symptoms and radiological findings overlap between eclamptic encephalopathy and Japanese encephalitis.
- ▶ The association of Japanese encephalitis resulted in delayed hospitalisation and recovery, in spite of the best treatment of eclampsia.
- ▶ A high index of suspicion is required in the endemic zone to rule out Japanese encephalitis in the patient of eclampsia in order to have the best clinical outcome.

Competing interests None.

Patient consent Obtained.

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