Neurocysticercosis Presenting as Postpartum Seizures in A Previously Known Case of Childhood Epilepsy: A Case Report

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Index Terms— Eclampsia, Epilepsy, Neurocysticercosis, Postpartum Seizure, Taenia Solium

1. INTRODUCTION

Post-partum seizures can result from many causes. There may be a considerable overlap in the presentation of these conditions resulting in difficulties in diagnosis and treatment. Convulsions presenting in the puerperium should be treated as eclampsia until proven otherwise. Every effort to identify other causes of convulsions should be made. We present a case of postpartum seizures in a female who is previously known to have epilepsy since childhood.

2. CASE REPORT

A 36-year-old, Gravida 3, Para 2 (G3P2 + 0), Indian female nurse presented to the emergency department. She is a known case of epilepsy since childhood. She had only one episode at the age of eight years. She is not on medication and did not have any subsequent episodes. She presented with generalized tonic-clonic seizure 6 days after normal vaginal delivery (24/10/16). Her antenatal course was uncomplicated and her delivery was without a l**umbar epidural\dural puncture during labour.** Both the immediate and the first 5 days postpartum were uneventful until the 6th day post party when the patient experienced generalized tonic-clonic seizures and tongue bitting. This was witnessed by her husband. There was no history of prodromal symptoms, nausea, vomiting, raised temperature, neck rigidity, headache, dizziness, vision disturbances, focal neural deficit, passing urine or stool, history of head trauma or similar attack antenatally or in previous pregnancies.

The patient's gestational period was uncomplicated. No history of gestational diabetes, hypertension, pre\eclampsia or thrombotic event. Also, her delivery was spontaneous vaginal delivery without regional anesthesia or complications. She had normal lochia with no increase in the blood pressure in the postpartum period. The patient is not alcoholic. She was on calcium and folic acid only with no illegal or over the counter drug use.

At the time of arrival, the patient was conscious, alert but slightly confused, soon after arrival, she became oriented to place, time and person). Her vital signs were within normal limits. Examination revealed no abnormality with normal deep tendon reflexes and no focal deficit. Abdominal examination was unremarkable.

On the day of presentation, the investigations showed a low Hemoglobin level of 11.7 gm/dl (12.0-16), low hematocrit 34.0% (37.0-47.0) and low RBC 3.76 k/ul (4.2-5.5) corresponding to the delivery loss. All other CBC levels were within normal range including the WBCs. She also had normal renal function test, normal urine dipstick, insignificant growth in urine culture and normal urine analysis. The liver function test was normal.

The patient was given Mg sulfate 10 grams Injections in form of two doses 4 g (8ml) and one dose 2 gm

The patient underwent MRI with contrast on the following day (25/10/16) that has revealed a structural lesion; **neurocysticercosis** (Multiple ill-defined rounded foci of hyper-intense T2/FLAIR signal within the left middle frontal gyrus, left post central sulcus, left superior frontal sulcus, anterior aspect of left Sylvian fissure and along the anterior inter-hemisepheric fissure, this demonstrates blooing artifact on gradient image.

The left middle frontal gyrus lesion demonstrates focal edema and smooth ring enhancement in post gadolinium administration).

A spinal MRI with contrast was done 6 days later to rule out any associated pathology with no defensive intramedullary lesions, or pathological enhancement. Vertebral body height, spinal intensity, and alignment are preserved. Para vertebral soft tissues appear unremarkable.

In addition, an Axial un-enhanced CT scan of the brain with coronal and sagittal reformatted was done in the same day that revealed Calcification along the previously seen lesions described in the previous MRI with No mass effect, or midline shift, no hydrocephalus and with normal ventricular system and cisterns. So Neurocysticercosis would be the primary consideration.

- On (26/10/16), The patients was started on Keppra (levetiracetam) 250 mg PO BID for 7 days then 500 mg PO BID until now.
- On (30/10/16), as suggested by Infectious diseases department, she was started on Albendazol 100 mg PO OD, Praziquantel 3600 mg PO OD and Dexamethasone 7mg IV OD with daily liver function test and CBC observation and to continue Keppra (levetiracetam) as was suggested before.
- On (3/11/16), the patient was discharged as planned by both neurology and infectious departments on:
- Albendazol 100 mg PO OD - up to present
- Praziquantel 3600 mg PO OD - up to present
- Dexamethasone 7mg IV OD - up to Jan. 23. 17

3. DISCUSSION

Postpartum seizures contribute for one of the life-threatening conditions that need an urgent intervention. Although that any patient presenting with convulsions in the the puerperium must be treated as a case of postpartum eclampsia, every effort must be done to reach the diagnosis. Putting in mind the chronic diseases that might precipitate the event as hypersensitive encephalipathy and uremia, post-partum seizures could be also the first presentation or a recurrent episode of epilepsy. In addition, it might be a consequence of hypoglycemia, electrolytes imbalance (hypocalcemia, hypomagnesemia, hyponatremia or hypernatremia) or drug/alcohol poisoning or withdrawal. Moreover, head trauma, vascular events as intracranial hemorrhage, cerebral vascular occlusion or ischemia and space occupying lesions such as tumor or abscess are all in the list of the differential diagnosis. Patients with fever, contact with sick people or with recent infection must be also investigated for meningitis, encephalitis, tetanus or HIV infection.

Our patient was investigated vigorously to reach the diagnosis since she was previously a known case of epilepsy in childhood

and had a very smooth uneventful pregnancy. Radiological imaging was started with **MRI with contrast** revealing a structural lesion; **neurocysticercosis** (Multiple ill-defined rounded foci of hyper-intense T2/FLAIR signal within the left middle frontal gyrus, left post central sulcus, left superior frontal sulcus, anterior aspect of left Sylvian fissure and along the anterior inter-hemisepheric fissure, this demonstrates blooing artifact on gradient image. The left middle frontal gyrus lesion demonstrates focal edema and smooth ring enhancement in post gadolinium administration) followed by **MRI of spine with contrast** to rule out spinal pathology which was unremarkable. Moreover, **Axial unenhanced CT scan of the brain with coronal and sagittal reformatted** to confirm the diagnosis was done revealing Calcification noted along the previously seen lesions described in the previous MRI within the left middle front gyrus, left superior frontal sulcus, anterior aspect of the left sylvan fissure and along the anterior r inter-hemisepheric fissure which are again suggestive of infective etiology- (NCC) with normal ventricular system and cisterns and no mass effect, or midline shift, no hydrocephalus. For that, inflammatory markers including **ESR and CRP** which both were high with level of 47 and 1.7 consequently and blood culture which showed no growth were taken.
Our patient was treated as a case of neurocystecrosis (seizures due to Taenia solium (ie, pork tapeworm infestation). She has been on Anti-epilptic drug; Keppra (Levetiracetam) 500 mg PO BiD as a prophylaxis for subsequent seizures. She was also started on Anti-parasitic therapy including Praziquantel 3600 mg PO OD and Albendazol 100 mg PO OD. Since the patient is receiving Anti-parasitic therapy, she was started on Dexamethasone 7mg IV OD to reduce the inflammation associated with the dying organisms. All the therapies are used up to present except Dexamethasone 7mg IV OD that was lastly given on Jan. 23. 17.
WHO recommendations for neurocystecrosis **(NCC)** management include long courses with praziquantel and/or Albendazole, as well as supporting therapy with corticosteroids and/or anti-epileptic drugs, and possibly surgery. The dosage and the duration of treatment can vary greatly and depend mainly on the number, size, location and developmental stage of the cysts, their surrounding inflammatory edema, acuteness and severity of clinical symptoms or signs. Patients who have multiple active and inactive parenchymal cysts; administration of Anti-iparasitic therapy (albendazole 15 mg/kg/d in two daily doses for 8 to 15 days) together with high-dose steroids is the most favorable treatment. Since patients with NCC are at risk of tuberculosis and strongyloidiasis, which can disseminate during treatment with corticosteroids, patients with NCC must be screened for such infection prior to corticosteroid therapy. Surgical intervention is saved for symptomatic patients with hydrocephalus. Endoscopic removal of the cysticerci in the ventricles is recommended for patients without acute symptoms.

4. Learning Points

- One third of patients with pre-eclampsia present with post-partum convulsions.
- Postpartum seizures should be investigated thoroughly.
- Patients with postpartum seizures who come from endemic areas must be screened for parasitic infestations and bacterial infections.

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