NEUROCYSTICERCOSIS AS A RARE CAUSE OF POST PARTUM CONVULSIONS

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PRESENTATION OF CASE

23 year old female came with history of 2 episodes of convulsions on her 5th postpartum day at home. She was apparently alright before this. She had another episode of generalised tonic clonic convulsions in casualty, each episode lasting for 2-3mins associated with frothing of saliva, up rolling of eyes, tongue bite and post-ictal confusion. No history of involuntary urination/defecation. There was no preceding history of blurred vision, headache, epigastric pain, pedal oedema, fever, breathlessness, weakness of limbs or pain in the calf muscles. Following episode of convulsions she had slight slurring of speech. She is P2L2, fifth post-natal day, delivered by full term normal vaginal delivery. She had regular antenatal check-ups and had no h/o pre-eclampsia or HTN in either of the pregnancies or any history of convulsions in the past. Antepartum, intrapartum periods were uneventful. She consumes mixed diet and is a pork eater, sleep and appetite normal, bowel and bladder habits regular. On examination, patient was conscious and was responding to verbal commands. There was no evidence of pallor, icterus, and lymphadenopathy. She was afebrile, normotensive and her vitals stable. Systemic examination was normal including the CNS examination. Fundoscopy was normal. Her complete blood counts, urine albumin, Renal and Liver function tests, coagulation profile, serum electrolytes, ECG, chest X- ray were normal. MRI brain did not have any evidence of thrombosis or PRES. MRI brain showed 10 mm ring lesion with tiny hypointense scolex in the left parietal lobe cortex with perilesional oedema suggestive of cysticercosis. There was no evidence of proglottids or eggs in stool examination. Hence diagnosed as neurocysticercosis. She was treated with injection phenytoin, injection levetiracetam and injection dexamethasone 8mg TID. She was started on oral Levipil 500 mg BD and tablet Albendazole 400 mg BD. Rest of her hospital stay was uneventful and discharged on POD 15 on oral Levipil, albendazole and oral prednisolone, asked to review in outpatient basis for further follow-up. Preventive measures advised.

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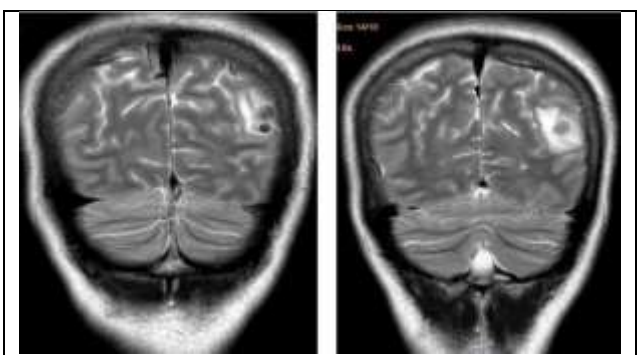

Figure 1

DIFFERENTIAL DIAGNOSIS

- Cerebral Venous Thrombosis
- PRESS
- Eclampsia
- Epilepsy

CLINICAL DIAGNOSIS

Convulsion can be antepartum or post-partum, associated with slurred speech, as there is no history of PIH or Chronic Hypertension, it will be diagnosed by MRI Brain which shows features of neurocysticercosis.

DISCUSSION OF MANAGEMENT

Convulsions in postpartum period should be treated as eclampsia until proven otherwise.[1] But in normotensive and with no proteinuria every effort should be made to identify other causes of convulsions.[2] Neurocysticercosis (NCC) is a parasitic infection with the larvae of *Taenia solium* from contaminated pork. It is a leading cause of seizures in the developing world. Symptoms may be secondary to live or degenerating cysts, or previous infection causing calcification or gliosis.[3] Transient perilesional oedema around calcified foci is common and associated with episodic seizure activity.[4] The prevalence of NCC as a cause of active epilepsy in India was calculated to be one per 1000 population.[5] It is endemic in South America, sub-Saharan

Africa, India, China and regions of South-East Asia which are developing countries.

Cysticercosis is a tissue infection acquired by exposure to eggs of Taenia solium, also known as pork tapeworm. It is a food borne disease which spreads via the faecal-oral route through contaminated food and water. It is associated with lack of sanitation and poor hygiene. The life cycle involves humans as a definite host and pigs as an intermediate host. The eggs after ingestion pass through the lumen of the intestine and migrate into the tissues like brain and muscles. There they form cysts that can persist for years. Symptomatic disease from Taenia solium cysts in the brain is referred to as neurocysticercosis. It most commonly presents as seizures (parenchymal involvement) and less commonly as hydrocephalus.[6]

The diagnosis of neurocysticercosis is mainly based on clinical symptoms, endemicity and imaging studies. MRI is considered superior to CT scan.[7]

The diagnosis in CT scan is depicted as calcified and uncalcified cysts, which represents active and inactive cysts. Cysts in the tissues are seen as ring enhancement and focal enhancing lesions. In CT, the scolex is usually hyperdense, and on T2-weighted MR images, it appears hypointense. Treatment of neurocysticercosis is aimed with antiepileptic drugs and anti cysticercal agents.

Anti cysticercal agents like albendazole (15 mg/kg/day) or praziquantel for 28- 30 days are used. Sometimes corticosteroids are used to ameliorate the inflammatory reaction of host to the dying parasites. Surgery is considered only when there is a need for cyst removal in case of obstruction or for CSF shunts as in case of hydrocephalus. Usually Prognosis is satisfactory with effective anti cysticercal drugs. Anti-epileptics can be tapered after stabilising the patient and effective anti cysticercal agents are started. During pregnancy, treatment of neurocysticercosis consists of anti-convulsant therapy. The pregnancy can be continued till term without any fetal complications. Anti-helminthic drugs should be delayed until post-partum period. Prevention should be aimed at by taking effective measures like proper washing of vegetables and meat, adequate cooking, avoiding indiscriminate defecation and proper disposing of excreta.[8]

CONCLUSION

Neurocysticercosis should be considered as a possible diagnosis in women presenting with seizures which cannot be explained by eclampsia, during her pregnancy or the puerperium. Patients with postpartum seizures who come from endemic areas must be screened for parasitic infestations. Proper history and evaluation imaging techniques helps in the accurate diagnosis and management of the patient. Prognosis is good with effective anti cysticercal drugs. Anti-epileptics can be tapered in most cases. Prevention can be done by deworming in the adolescent age group. Counselling about personal hygiene, proper disposal of excreta and adequate.

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