



### Neurocysticercosis in pregnancy

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#### **Abstract :**

Neurocysticercosis is an infection of the central nervous system caused by taenia solium. A 24 yr old P2L2 woman was referred to us on her 7th post operative day as post partum psychosis. She gave history of intermittent headache, vomiting and altered behaviour for 10 days and one episode of seizure. She was drowsy, moving all four limbs with exaggerated deep tendon reflexes. Her vitals were stable. She had elevated lymphocytes and eosinophils her blood indices were normal. CT brain - cerebral edema, MRI brain - starry sky pattern and MRI thigh - multiple hyper intense lesions. She was given tablet albendazole 400mg twice daily for 21 days with anti edema measures and anti convulsants. Patient improved with treatment. Most common presentations of neurocysticercosis include seizure, headache, raised intra cranial tension, stroke, neuropsychiatric manifestations and hydrocephalous. Diagnosis is by CSF analysis that shows mononuclear pleocytosis ,

oligoclonal bands and eosinophilia. Stool examination may or may not show cysticerci. Immunoassay , CT , MRI. Brain biopsy can be used as a last resort. Albendazole and praziquantel are the treatment options available. Though neurocysticercosis is a rare disease, it should be considered as one of the DD in patients presenting with seizures for the first time , even in women during her postpartum period.

**Keyword :** postpartum psychosis, lymphocytosis and eosinophilia, starry sky pattern, tablet. albendazole.

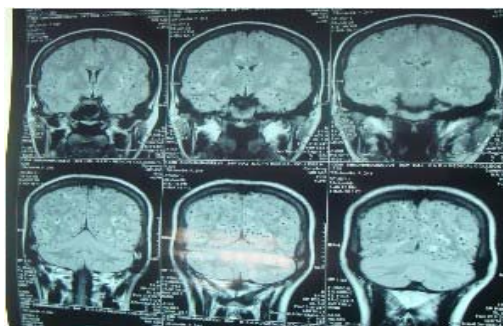
#### **Neurocysticercosis in pregnancy:**

24yr old P2L2 woman with emergency repeat LSCS done one month back, was referred from nearby PHC as ? POSTPARTUM PSYCHOSIS. She gave history of intermittent headache, vomiting for 30 days and altered behaviour for 10 days. There was No h/o convulsions, fever, loss of consciousness, head injury, difficulty in walking/ eating/ breathing/ bowel or bladder habits. No h/o visual disturbance. No h/o foul smelling discharge per vagina. No h/s/o PIH,

PPH. No h/o drug intake. No significant medical or surgical illness in the past.

On examination she was drowsy yet arousable. Afebrile, no pallor / cyanosis / clubbing lymphadenopathy or pedal edema. Vitals - normal. No clinical evidence of DVT. Systemic examination was normal. Per abdominal examination - soft, no organomegaly, no free fluid, caesarean scar was healthy. Per speculum and per vaginal examination was normal. Spontaneous eye movements were present, B/L pupils equally reacting to light. Moving all 4 limbs on command with exaggerated deep tendon reflexes and B/L plantar extensor. Cranial nerve and sensory system were normal. There was no cerebellar signs or signs of meningeal irritation. Differential diagnosis : Post dural puncture headache, Meningitis, Cerebral haemorrhage, Hypo / hyperglycemia, Acid and electrolyte imbalance, CVT, Space occupying lesion, Embolic manifestation, Psychogenic. But for lymphocytosis and eosinophilia all her blood indices were normal.

Motion for ova and cyst were negative. Funduscopy showed early papilledema. CT brain showed effacement of cisterns and sulci, suggestive of diffuse cerebral edema. CVT was suspected, hence MRI was ordered. Meanwhile patient developed blurring of vision, restricted abduction in right eye and had one episode of generalized tonic clonic seizures. CT fundus showed bilateral papilledema. MRI brain showed starry sky pattern suggestive of active lesions of neurocysticercosis and MRI thigh was evident with cysticercal lesions.



She was given Tab. Albendazole 400 mg twice daily for 21 days under steroid coverage with anti edema measures and anti – convulsants. She improved drastically and was discharged.

#### **DISCUSSION :**

Neurocysticercosis is the most common parasitic disease of the nervous system and is the main cause of acquired epilepsy in developing countries. It has also been a problem in industrialized countries because of the immigration of tapeworm carriers from areas of endemic disease. Neurocysticercosis is acquired through consumption of food contaminated with faeces of a *T. solium* tapeworm carrier (i.e., through fecal–oral contract). Eggs of the tapeworm are shed in stool and contaminate food through poor hygiene. When these eggs are ingested and exposed to gastric

acid in the human stomach, they lose their protective capsule and turn into larval cysts, called oncospheres. Oncospheres cross the gastrointestinal tract and migrate via the vascular system to the brain, muscle, eyes, and other structures. Once in the brain, the larval cysts (cysticerci) initially generate a minimal immune response and may remain in the brain as viable cysts for years. Many patients are asymptomatic; others report vague symptoms such as headache or dizziness. The onset of symptoms is usually subacute to chronic, with the exception of seizures, which present in an acute fashion.

**Epilepsy** is the most common presentation (70%) of neurocysticercosis and is also a complication of the disease. Neurocysticercosis is the leading cause of adult-onset epilepsy. Epileptogenesis in patients with neurocysticercosis can be attributed to several factors like inflammation, gliosis, genetics, and predilection for the cysts to travel to the frontal and temporal lobes. The host response to degenerating cysts plays an important role in the associated epileptogenesis. **Intracranial hypertension** : most often, intracranial hypertension is due to obstruction of cerebrospinal fluid (CSF) circulation caused by basal or ventricular cysticercosis. It may also result from large cysts displacing midline structures, granular ependymitis, arachnoiditis, or the so-called cysticercotic encephalitis caused by the inflammatory response to a massive infestation of cerebral parenchyma with cysticerci. Diplopia may result from intracranial hypertension or arachnoiditis producing entrapment or compression of cranial nerves III, IV, or VI. **Stroke** : cerebral infarcts due to occlusion or vascular damage. Strokes may be responsible for paresis or plegias, involuntary movements, gait disturbances, or paresthesias. **Neuropsychiatric disturbances** : Neuropsychiatric dysfunction can range from poor performance on neuropsychologic

tests to severe dementia. **Hydrocephalus** : 10 – 30 %. Presentations of other forms of neurocysticercosis : Spinal neurocysticercosis is rare and may be either intramedullary or extramedullary. The extramedullary form is the most frequent and is responsible of symptoms of spinal dysfunction such as radicular pain, weakness, and paresthesias. Intramedullary presentation may cause paraparesis, sensory deficits with a level, and sphincter disturbances. Systemic cysticercosis is most common in the Asian continent. The parasites may be located in the subcutaneous tissue or muscle. Peripheral nerve involvement as well as involvement of the liver or spleen have been reported.

#### **DIAGNOSIS :**

Neurocysticercosis is commonly diagnosed with the routine use of diagnostic methods such as computed tomography and magnetic resonance imaging of the brain. Peripheral leukocytosis, eosinophilia, and elevated erythrocyte sedimentation rate may be found on routine blood work. A decrease in *N - acetyl aspartate and creatine levels and elevated lactate and metabolites such as alanine and succinate on magnetic resonance spectroscopy*. For the purpose of localization, myelography and cerebral angiography, as well as cisternographies and ventriculographies, may be used. Analysis of the cerebrospinal fluid is indicated in every patient presenting with new-onset seizures or neurologic deficit in whom neuroimaging shows a solitary lesion but does not offer a definitive diagnosis. CSF findings include mononuclear pleocytosis, normal glucose levels, elevated protein levels,

high immunoglobulin G (IgG) index, and in some cases, the presence of oligoclonal bands. Eosinophilia in the CSF suggests neurocysticercosis. Stool examination : may or may not be positive. Enzyme-linked immunosorbent assay (ELISA) is the most widely used test of cerebrospinal fluid (CSF); it has a sensitivity of 50% and a specificity of 65% for neurocysticercosis. Enzyme-linked immunoelectrotransfer blot (EITB) assay in serum is also highly sensitive and specific, initially described as 98% and 100%, respectively. Only in extreme cases of neurocysticercosis is a brain biopsy necessary. A trial of anticysticercal drugs with follow-up imaging shortly thereafter (ie, 2 months) is recommended before considering biopsy.

**TREATMENT** : Two medications are available in the treatment of neurocysticercosis, praziquantel (PZQ) and albendazole. Both agents eliminate the cysticerci or markedly reduce their number. Albendazole appears to be superior to PZQ and seems to be more effective in giant cysts and subarachnoid, intraventricular, or spinal neurocysticercosis. Drugs such as dexamethasone, phenytoin, or carbamazepine may decrease plasma levels of PZQ due to interaction with the cytochrome P-450 microsomal system. This is not seen with albendazole (which is excreted unchanged in the urine). Simultaneous administration of dexamethasone appeared to increase plasma levels of albendazole and decreased its rate of elimination

**During pregnancy** : Albendazole - Category C drug by FDA. It should not be used in pregnant women except in clinical circumstances where no alternative management is appropriate. Patients should not become pregnant for at least 1 month following cessation of albendazole therapy. If a patient becomes

pregnant while taking this drug, albendazole should be discontinued immediately. If pregnancy occurs while taking this drug, the patient should be apprised of the potential hazard to the fetus.

**Albendazole Breastfeeding Warnings** :

There are no data on the excretion of albendazole into human milk. The manufacturer recommends that caution be used when administering albendazole to nursing women.

**Praziquantel in pregnancy** : Category B drug. Can be used both in pregnancy and lactation . Anti epileptics, anti edema drugs and diuretics can be added to the therapy.

**Surgical Intervention** : In the presence of hydrocephalus due to an intraventricular cyst, placement of a ventricular shunt is recommended, followed by surgical extirpation of the cyst and subsequent medical treatment. Long-Term Monitoring : Intracerebral cysticercotic lesions can cause epilepsy in the future. Administration of antiepileptic medication is the same as in any other epileptic syndrome. Follow-up imaging study is recommended after 2-3 months following treatment, especially in cases in which anticysticercal medications are used as a diagnostic tool.

**CONCLUSION** :

Though Neurocysticercosis is a rare disease, it should be considered as one of the DD even in a pregnant patient who presents with seizures for the first time. Neurocysticercosis is becoming a major public health problem in developing countries and is emerging as an increasingly important condition in regions in which the disease is not endemic. Comprehensive programs of long-term intervention involve appropriate legislation, health education, modernization of swine husbandry practices, improvement of efficiency and coverage

of meat inspection, provision of adequate sanitary facilities, and measures to detect and treat human tapeworm carriers.