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Wernicke Encephalopathy due to Hyperemesis Gravidarum

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Abstract: *Wernicke encephalopathy is a rare, treatable, reversible yet serious neurological disease due to vitamin B1 deficiency. It is usually associated with heavy alcohol consumption. Other causes are malnutrition due to hyperemesis gravidarum, starvation, anorexia nervosa, cancer chemotherapy, HIV infection, bariatric surgery. It is characterized by classic triad of encephalopathy, ophthalmoparesis/nystagmus and ataxia. We now report two cases of Wernicke's encephalopathy due to hyperemesis gravidarum. Early diagnosis and treatment is necessary to prevent maternal and fetal mortality and permanent amnesic neurological sequelae*

Keywords: *Wernicke encephalopathy, hyperemesis gravidarum, early diagnosis,treatable .*

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I. Introduction :

Wernicke encephalopathy is a rare neurological disorder due to thiamine deficiency. It was described by Carl Wernicke in 1881, in patients presenting with triad of ocular signs , ataxia , confusion [(1)]. But all three components may not be present in all patients. One third of patients have only one component. Some patients present with confusion alone , some begin with ataxia followed by confusion in few days, others with almost simultaneous onset of ataxia, nystagmus, ophthalmoparesis with or without confusion[(1).] It is typically diagnosed among alcoholics. It is relatively rare in hyperemesis gravidarum. Rapid identification and treatment is necessary to prevent maternal and fetal mortality and to prevent amnesic component of disease.

II. Case report 1:

A 25 year old primi with gestational age of 20 weeks was brought with complaints of excessive vomiting for 2 months, difficulty walking for 2 weeks followed by altered sensorium for 1 week. Due to excessive vomiting , she had poor food intake and lost weight of 5 kg . Initially she walked with support but was unsteady, later she confined herself to bed and not responding or obeying commands unless repeated multiple times. At admission she had a GCS of 12/ 15, was inattentive, confused, disoriented. Ocular examination revealed upbeat nystagmus, bilateral abduction restriction (right>left) and she had flaccid paresis of four limbs , absent knee and ankle jerks and flexor plantar response with truncal , gait and mild limb ataxia. She was immediately started on intravenous thiamine 100mg thrice a day in view of severe malnourishment plus classic triad of ocular signs, ataxia and confusion. Her serum biochemistry was normal. (Sodium=142mEq/ lit, Potassium=3.6mEq/lit, Creatinine=0.8mg/dl, Bilirubin=0.7mg/dl, Lactate=4.9mg/dl. She had microcytic hypochromic anemia. MRI brain taken the following day showed symmetrical T2 and FLAIR hyperintensities in both thalami and periaqueductal gray matter. We have also done nerve conduction studies for any coexisting neuropathy due to B1 deficiency and they were normal.

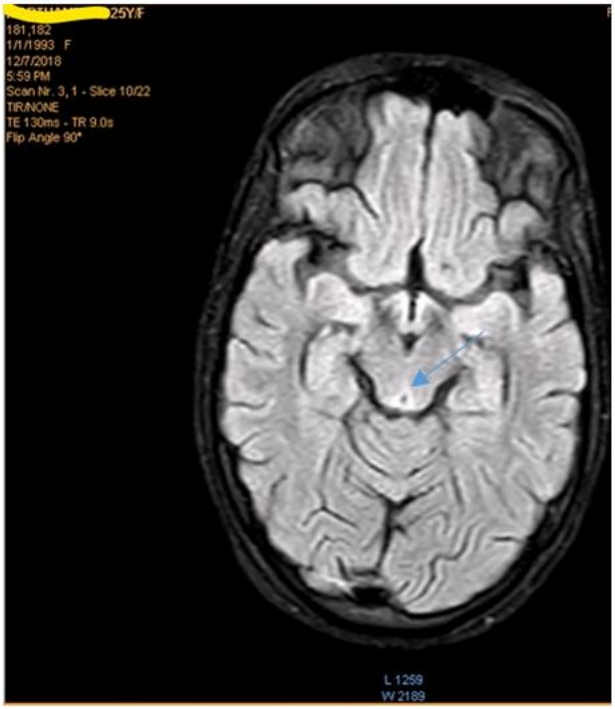

Figure 1*.FLAIR-MRI Brain showing hyperintensities in periaqueductal gray matter*

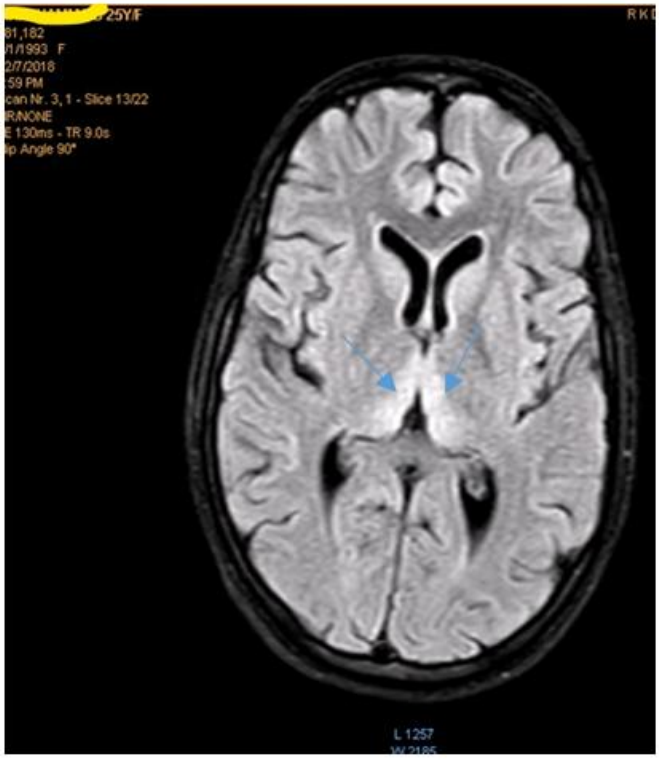

Figure 2. FLAIR-MRI Brain showing symmetrical hyperintensities in both thalami

After a week , patient became conscious , oriented, able to sit , walk without support but gait was widebased and unsteady. Abduction of eye improved but upbeat nystagmus persisted. After 3 weeks ataxia improved further , but gait was widebased and vertical nystagmus persisted. She delivered a healthy male baby at 37 weeks of gestation. She had persistent widebased gait and vertical nystagmus but no features of Korsokoff psychosis.

Case report 2:

A 20 year female was brought with chief complaints of difficulty walking, numb feeling of all limbs, tremulousness of upper limbs for 2 weeks. The complaints were progressive . Initially she walked on her own, later with support then unable to stand and later unable to sit. Her mother complained about her inattentiveness, apathy and she drifts off to sleep easily for the past one week. She conceived 13 weeks back , she had intractable vomiting for 2 months, had intrauterine death at 13 week gestation , underwent extraction, 4 days afterwhich the above complaints started. At admission she had a GCS of 15/15. Vitals were stable. She had both horizontal and vertical gaze evoked nystagmus with paresis of all limbs with stance ataxia, gait ataxia, and limb ataxia with intention tremor. Deep tendon reflexes were hyporeflexic and plantar response was flexor. She was immediately started on intravenous thiamine 300mg/day .Serum electrolytes were normal. MRI showed subtle FLAIR hyperintensities in bilateral thalami

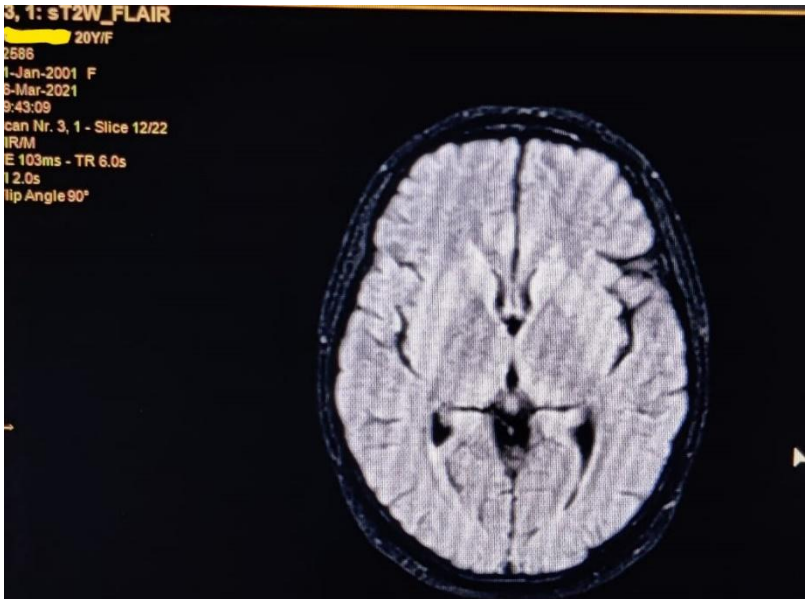

Figure 2.*FLAIR-MRI BRAIN showing subtle hyperintensities in bilateral thalami.*

Her nerve conduction studies were normal at admission and a week later. She improved on thiamine, walked with support in 4 days. But the gait was wide based. Fine horizontal gaze evoked nystagmus persisted. She was discharged a week later with oral thiamine 300mg/ day.

III. Discussion:

Wernicke encephalopathy is mostly seen in alcoholics. But it can occur in any malnourished state. Prevalence of Wernicke encephalopathy in non alcoholic patient varies from 0.04% -0.13%[(2).] Most frequent causes of Wernicke encephalopathy in non alcoholic patient are cancer and gastrointestinal surgery.[(3)]

Thiamine pyrophosphate is biologically active form of vitamin B1. It is essential coenzyme in many biochemical pathways[(4).] Daily requirement of thiamine is 1.1 mg in females and it increases to 1.5 mg /day during pregnancy and even more when absorption is impaired due to hyperemesis[(5).] Wernicke encephalopathy in pregnancy , although reversible, is associated with major complications both for the mother and fetus. Mother can be left with permanent neurological deficits if treatment is delayed and also it can also cause mortality if left untreated. Wernicke encephalopathy can lead to miscarriage, preterm birth, intrauterine growth retardation[(2).]

T2 and FLAIR hyperintensities (representing early reversible cytotoxic edema) in both thalami, mammillary bodies and periqueductal gray matter is the typical radiological lesion[(2).]

Wernicke encephalopathy should be suspected in any patient with severe malnourishment ,particularly when they were given dextrose containing intravenous fluids with out supplementation of thiamine, presenting with triad of ataxia, confusion and ocular signs(nystagmus ,ophthalmoparesis). Thiamine should be started immediately without waiting for imaging because delay in treatment may lead to permanent Korsokoff syndrome. Complete remission of Wernicke encephalopathy is rarely obtained. In a review of 49 cases by Choissi et al, symptom resolution takes months and complete resolution was obtained in only 14 cases.(5)

Both of our patients were primi from low socio economic status with malnutrition and intractable vomiting, presented with classic triad . Both were immediately started on thiamine. But, they were left with persistent wide based ataxic gait and nystagmus.

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