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A Case on Hyperemesis Gravidorum Induced Wernicke's Encephalopathy and Central Pontine Myelinosis

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Introduction

Wernicke's encephalopathy is a neuropsychiatric syndrome due to thiamine deficiency, which is potentially fatal but preventable .In obstetrics , it can complicate hyperemesis gravidarum because of major daily requirement .Now a days there is no consensus on early diagnosis , treatment and prevention of this disorder. We p resent a case report of hyperemesis gravidarm which degenerated into WE .It highlights that the clinical suspicion is necessary to recognize signs and symptoms , to apply the effective preventive measures in situations at risk and to begin urgent treatment in presence of characteristic clinical features.

1959- Victor and Adams first described Central pontine myelinolysis Clinical findings – quadriparesis, pseudobulbar palsy .Pathology – myelin loss confined within the central pons .Was felt as a sequela of alcoholism or malnutrition.

1981- De Masters and Norenberg conclusively demonstrated a link between ODS and rapid correction of hyponatremia

Pathology of central pontine myelinosis

Predominantly - basis pontis sparing the tegmentum, it may extend upto midbrain , very rarely down to medulla. Pathologically, loss of myelin sheath with relative sparing of axons and neurons in sharply demarcated lesion and Absence of inflammatory infiltrate in these lesions.

Reason for localisation within this region-This is a region of maximal admixture of grey and white matter elements. Similarly lesions of EPM are seen in the similar region of grey white apposition.

Duration of Hyponatremia To Cause ODS: Brain damage does not occur when hyponatremia < 1 day duration is rapidly corrected. If persists for > 2 to 3 days same treatment results in ODS. However duration is often not known. Thus, assume that the patient has chronic hyponatremia unless the history suggests acute water intoxication 1.Marathon runner's 2.Psychotic patients 3.Users of ecstasy.

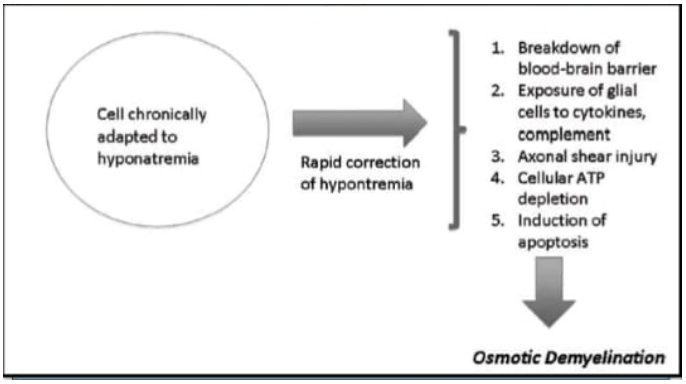

Figure 1

Clinical Course

Biphasic clinical course - initially encephalopathic or seizures from hyponatremia and Then recovering rapidly as normonatremia is restored and thenDeteriorate several days later – manifested by s/o CPM . Dysarthria and dysphagia (secondary to corticobulbar fibre involvement).A flaccid quadriparesis (coticospinal tract) later becomes spastic.Hyperreflexia and bilateral Babinski signs.If tegmentum of pons is involved pupillary , oculomotor abnormalities.Apparent change in conscious level – "locked in syndrome".

Factors which may increase risk of acute hypernatremia encephalopathy

A. **Estrogen:** Inhibits Na-K ATP ase pump and thus ODS is more common in women of child bearing age.
B. **Arginine Vasopressin**: Decreased cerebral perfusion and decrease ATP availability for ion exchange.
C. **Hypoxia:** Limits ATP availability

Figure 2

Diagnosis

Clinical suspicion -Any patient presenting with new onset neurological symptoms with a recent rapid increase in serum sodium. Postliver transplant. Diagnosis clinic radiological. No role for tissue examination

Typical MRI Lesions

Trident shaped / spreading bushfire pattern in central pons. Signal characteristics of affected region include : T1 : mildly to moderately hypointense. T2 : hyperintense , sparing the periphery and corticospinal tracts. FLAIR: hyperintense. DWL: hyperintense. ADC: Signal low or signal loss T1 C + (Gd) : usually there is no enhancement. Radiologic findings don't improve over time: despite complete or nearly complete clinical recovery

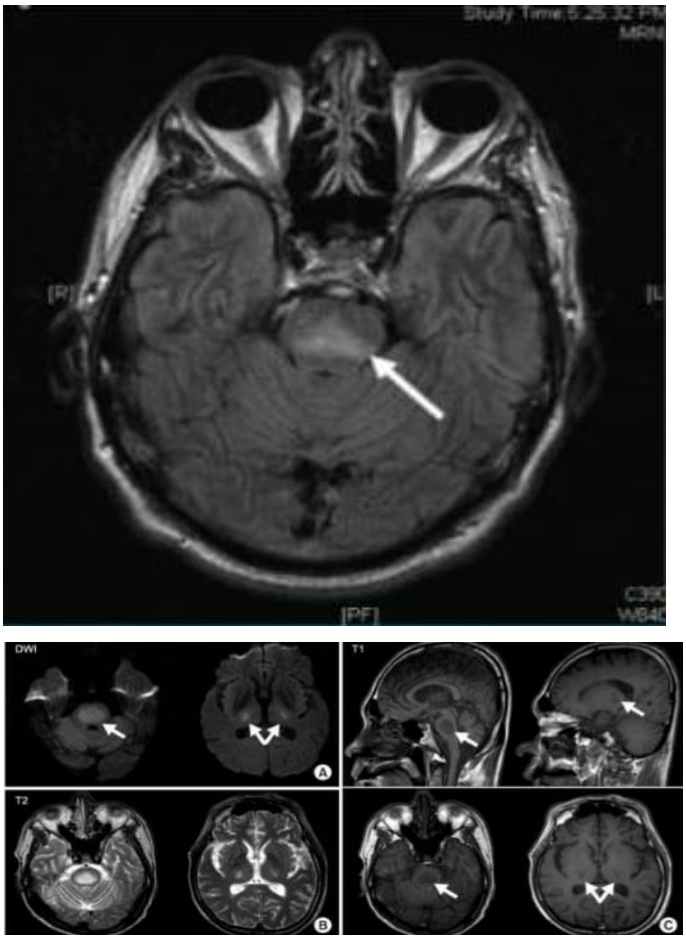

Figure 3

Prevention

Rate of sodium correction to avoid ODS if <10 to 12 mEq/L per 24 hours and if <18 mEq/L in 48 hours **.**Presence of other risk factors require slower rates –max 8 mEq/L per 24 hrs .Some may have risk factors and have urgent symptoms of hyponatremia necessitating immediate correction , such as seizures or obtundation. Generally most life threatening manifestations of hyponatremia will abate with a 5 % rise in serum sodium and Subsequent correction – no more than 8 -12 mEq/L per 24 hours.

Goals of therapy

Rate of lowering sodium is 1 mEq/L per hour with a target a rate of correction of <8 mEq/L in any 24 hour period and <16 mEq/L in any 48 hour period.D 5 % , 6 ml/kg lean body weight infused over 2 hours.It lowers serum sodium by approximately 2 mEq/L. Infusion should be repeated until the therapeutic goals.The d DAVP , 2 mcg , intravenously or subcutaneously q6 H.It can be increased to 4 mcg in those who donot respond .d DAVP is continued , even after D5W infusions have ceased , to prevent serum sodium from rising again d/t excretion of dilute urine.

Case history

A 37 yr old G2P1L1 /Prev LSCS admitted in an unconscious state with complaints of nausea, vomiting since conception, fever for 1 day, breathlessness with BP not recordable, pulse feeble, resp rate-38/min,Temp-100 degree. Due to poor GCS patient was intubated and maintained in mechanical 5ventilation**.**Physician opinion-Inj actrapid 8U IV and Inj Thiamine 200mg iv tds. Neurophysician opinion obtained, as the patient had features of ataxia, ophthalmoplegia and global confusion, a diagnosis of Wernickes encephalopathy was made and the same treated with inj. Thiamine. Bedside ultrasound-normal study of maternal organs. Pregnancy was terminated in view of maternal hemodynamic instability. Patient developed non oliguric AKI. Tracheostomy was done and patient was weaned from ventilator. Patient tolerated oral feeds and was shifted to oral thiamine. Patient was discharged on day 55**.**MRI shows features of central pontine myelinosis

Discussion

Wernicke's encephalopathy is due to the deficiency of thiamine vitamin B1.Essential cofactor in various stages of carbohydrate metabolism.Transketolase is a thiamine dependent enzyme in the pentose pyrose phosphate pathway. Hyperemesis gravidarum is common, Wernicke's encephalopathy is uncommon. Werncike's encephalopathy is a central neurological disorder characterised by classical triad-1.Encephalopathy 2.Ophthalmoplegia\nystagmus 3.ataxia.It is resulting from thiamine deficiency

Etiology

It is most commonly seen in alcoholics. It also occurs in any malnourished state. It should be considered in patient's with1.anorexia nervosa 2.prolonged vomiting associated with chemotherapy 3. GIT disease 4.hemodialysis

Pathophysiology

Thiamine pyrophosphate is the biological active form of vitamin b1.It is an essential coenzyme in many biochemical pathways. Neuronal damage begins once metabolism in brain regions with high metabolic requirements and high thiamine turnover is inhibited. Time to deplete the body stores of thiamine is about three weeks. Daily requirement of thiamine is 1.1mg\day in females and it increases to 1.5mg\day during pregnancy and lactation.

Diagnosis

Diagnosis of wernickes encephalopathy is by clinical manifestations and rapid reversibility of symptoms with thiamine.MRI findings-typical lesions are located in thalamus, mamillary bodies, periaqueductal bodies.Early reversible cytotoxic edema is the most distinctive lesion.

Complications

Wernickes'encephalopathy is reversible. But major complications can arise in pregnant woman. Wernickes encephalopathy can lead to permanent neurological manifestations and Korsakoff syndrome if left untreated which can be fatal. It can lead to miscarriage, preterm birth and intrauterine birth retardation. It can be associated with life threatening complications like central

pontine myelinosis. Our patient presented with classical triad, following vomiting and dextrose administration without thiamine supplements. Clinical and radiological presentation was in favour of acute thiamine deficiency indicating thiamine supplementation. Patient clinically improved after the administration of 200mg of thiamine.

Conclusion

Pregnant women with hyperemesis gravidarum should receive thiamine supplements. If she develops neurological manifestations Wernicke's encephalopathy must be suspected.

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