

RARE CAUSE OF DELIRIUM IN PREGNANCY: CASE REPORT OF WERNICKE’S ENCEPHALOPATHY

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INTRODUCTION:

- Normally a daily intake of 1.1 mg is recommended for adult women, whereas during pregnancy the demands are higher (1.5 mg/day).Thiamine is stored (25-30 mg) in the body, but they can be depleted in 2-3 weeks in certain conditions.¹
- Hyperemesis gravidarum affects about 3% of all pregnancies.
- Complications associated with it include malnutrition, dehydration, vitamin deficiencies, peripheral neuropathy, and more serious neurological complications such as Wernicke’s encephalopathy.
- Hyperemesis gravidarum leading to Wernicke encephalopathy was reported for the first time in 1914, with an incidence of 0.1-0.5%.² It is precipitated by administration of glucose-containing fluids before thiamine, presenting with a triad of ocular signs, ataxia and confusion and may be complicated by life threatening conditions like central pontine myelinosis.

CASE HISTORY:

- ❑ A 19 year old primigravida at 18 weeks of gestation in her first pregnancy presented to the emergency department with history of longstanding hyperemesis.
- ❑ Patient had hyperemesis from the 13th week of gestation, which resolved initially with antiemetics (metoclopramide).
- ❑ The relief was short lived as her hyperemesis recurred again within 10 days and continued despite treatment. She was then hospitalised, but got discharged against medical advice and continued non compliant/limited out patient treatment. By this time she had lost weight -7.7% (4 kg).
- ❑ She further developed the following symptoms, at 17 weeks of gestation and hence was referred to our tertiary care centre
- History of delirium with visual and auditory hallucinations
- Anterograde amnesia
- Mental confusion/altered sensorium
- Inability to articulate
- Marked weakness of upper and lower limbs and became bedridden

PHYSICAL EXAMINATION:

- Not oriented to time and place
- GCS of 12/15
- Afebrile, but with signs of severe dehydration +
- Pulse:108/minute and blood pressure:102/66mmHg
- Respiratory rate:19 breaths/minute

NEUROLOGICAL EXAMINATION

- Bilateral pupils reactive
- Power - 2/5 in bilateral upper limb & 1/5 - bilateral lower limb
- Absent/ delayed deep tendon reflexes

DIFFERENTIAL DIAGNOSIS:

- Wernicke’s encephalopathy
- Guillain Barre syndrome
- Hypokalemic paralysis
- Cerebral venous thrombosis

INVESTIGATIONS:

❖ BLOOD INVESTIGATIONS:

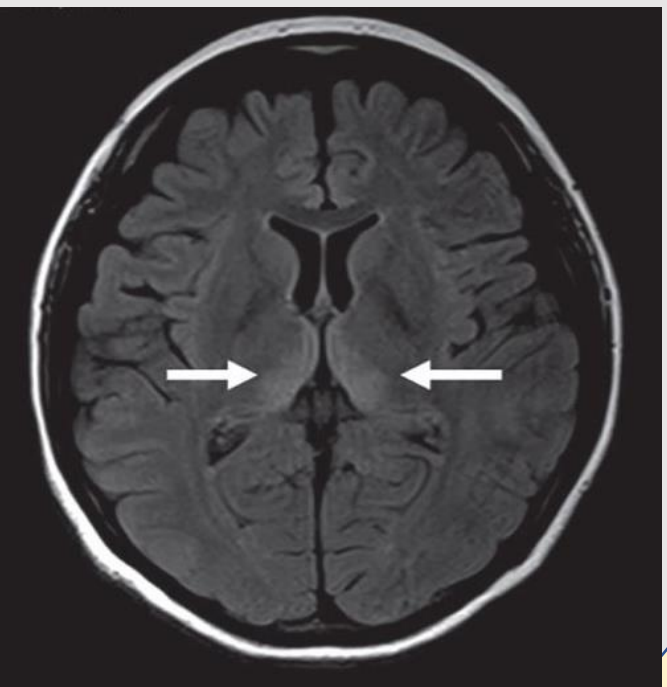
- Potassium (3.1 mmol/l, reference: 3.5-5.4 mmol/l)
- Albumin (2.9 g/dl, reference: 3.5-4.6 g/dl)
- SGOT - 143 IU/L (normal< 40)
- SGPT- 94 IU/L (normal< 40)
- Transient hyperthyroidism-TSH (0.01 mu/L, reference 0.3-4.0 mu/L)

❖ CSF analysis: normal

❖ IMAGING:

MRI done showed features suggestive of wernicke’s encephalopathy in fluid-attenuated inversion recovery(FLAIR) sequence & in diffusion weighted imaging (DWI):

- Bilateral symmetrical hyper signals in medial and posterior thalamic and in periaqueductal area
- Bilateral hypersignal of mammalian bodies in T2 (Cerebral venous thrombosis ruled out by MRI angiography)



MANAGEMENT

- Thiamine replacement was initiated with 500 mg dissolved in 100 ml of saline, and given intravenously every 8 hours for 3 days.
- This was decreased to 250 mg thiamine IV every eight hourly for 3 days, with improvemnt in cognition.
- By day 6 she had a slow but steady improvement in her neurological status.
- By day 7, she tolerated oral feeds, and was shifted to oral thiamine (100 mg orally for another 14 days).
- By day 10 she was able to walk with support
- She also received potassium replacement orally
- By day 13, she was discharged, and she had normal cognitive function and preserved memory
- On discharge, she was continued on physical and occupational therapy as well as thiamine, folate, and multivitamin supplementation.

DISCUSSION

Wernicke encephalopathy (WE) is a disease of the central nervous system caused by thiamine deficiency, with a clinical triad of acute mental confusion, ophthalmoparesis and ataxia³ and can also be diagnosed by Caine's operational criteria, as not all patients present typically. This includes clinical response to thiamine.Neural tissues have the highest thiamine utilization, as it is an important co-factor for enzymes in the pentose phosphate pathway and hence are most coomonly affected.

Glucose supplementation without thiamine can increase the thiamine requirement so care is needed ⁴

As patients with hyperemesis do often present with generalized fatigue and weakness, differentiating this from the pronounced and more specific symptoms seen in Wernicke’s encephalopathy becomes essential at an early stage.⁵

CONCLUSION

Accurate and prompt diagnosis after a detailed history taking and further treatment with high dose thiamine supplementation, especially prior to administering dextrose containing fluids , can easily avoid worsening of patients with this rare cause of delirium in pregnancy.

References

1. Gluckman, S., Hanson, M., Seng, C., & Bardsley, A. (2014-12). Vitamin B1 (thiamine) in pregnancy and breastfeeding. In Nutrition and Lifestyle for Pregnancy and Breastfeeding. Oxford, UK: Oxford University Press. Retrieved 13 May. 2022, from <https://oxfordmedicine.com/view/10.1093/med/9780198722700.001.0001/med-9780198722700-chapter-7>.
2. Treatment of nausea and vomiting in pregnancy. When should it be treated and what can be safely taken? Drug Saf 1998; 19: 155-64
3. Galvin, R, Brathen, G, Ivashynka, A EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. Eur J Neurol 2010; 17: 1408–1418.
4. Chiossi G, Neri I, Cavazzuti M, Basso G, Facchinetti F: Hyperemesis gravidarum complicated by Wernicke encephalopathy: background, case report, and review of the literature. Obstet Gynecol Surv 2006; 61: 255-68
5. Sechi G, Serra A: Wernicke’s encephalopathy: new clinical settings and recent advances in diagnosis and management. Lancet Neurol 2007; 6: 442-55