



# Ocular Manifestations of Wernicke's Encephalopathy in a Patient with Hyperemesis Gravidarum Complicating Pregnancy

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## Abstract

Wernicke's encephalopathy (WE) is a condition resulting from thiamine deficiency and has been linked to conditions such as hyperemesis gravidarum, anorexia nervosa, hemodialysis, and chronic alcoholism. The aim of the report is to outline the ocular manifestations of WE. We present the case of a 33-year-old woman, who presented in her second trimester of pregnancy with persistent vomiting, nystagmus, ataxia, global confusion as well as hypotonia, and tremors. She was subsequently diagnosed with WE complicating pregnancy. Ophthalmoscopy showed asymmetric superficial retinal hemorrhages at the peripapillary area in both eyes with associated retinal exudates. On treatment with thiamine supplementation, her systemic condition as well as ocular symptoms resolved. This is a rare case outlining the ocular manifestations of WE. This condition may cause serious, lasting neurologic impairments or death if not properly treated. Only 50% of these pregnancies end in the delivery of healthy offspring. Early diagnosis and treatment of these cases can prevent avertable consequences.

**Keywords:** Deficiency, encephalopathy, hyperemesis gravidarum, pregnancy, thiamine, Wernicke

## Introduction

In the central nervous system, thiamine (Vitamin B1) is a water-soluble vitamin that assumes a significant part as a co-enzyme in the metabolism of carbohydrates (1). Being an essential nutrient, it has limited stores and requires constant dietary supplementation to maintain its intracellular reserve (2). Deficiency of this vitamin classically manifests as the triad of Wernicke encephalopathy (WE): Acute confusional state (encephalopathy), ataxia, and ophthalmoplegia and/or nystagmus (1).

Deficiency of thiamine can result from a myriad of causes such as alcohol abuse, gastrointestinal disorders and surger-

ies (i.e., gastric bypass), acquired immune deficiency syndrome, hemodialysis, malignancies, psychiatric conditions such as anorexia nervosa, infections, shock, and prolonged nutritional deficiencies (3). Thiamine deficiency has also been noted in pregnancy, lactation, and in thyrotoxicosis (4,5).

We describe a patient with WE induced by hyperemesis gravidarum due to persistent vomiting.

## Case Report

A 33-year-old woman in the 16<sup>th</sup> week of her pregnancy presented to the hospital with complaints of generalized weakness and blurring of vision for 3 weeks. The patient reports that the symptoms have exacerbated over the last one and a

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half weeks due to which she is unable to stand or even walk without support. She also gives a history of blurring of vision for both distance and near which was insidious in onset and gradually progressive. Patient is a known case of hyperemesis gravidarum since the first trimester of her pregnancy and is on treatment for the same. She has even been admitted previously in view of the aforesaid complaints. She gives a history of rapid loss of weight in the past 3 months, in which period she had lost a total of 13kg's weight. She was a known case of hypothyroidism since 13 years for which she was on oral medication, i.e., 100 mcg of Tab. Thyronorm taken once daily. There were no other pre-existing ocular or systemic co-morbidities.

On examination at arrival, she was conscious and oriented to person, place, and time. Heart rate was 112 beats/min with blood pressure of 120/80 mmHg. General physical examination revealed pallor and a state of mild clinical dehydration. On examination of the nervous system, the patient was found to have nystagmus, ataxia, as well as hypotonia and tremors involving bilateral lower limbs. Her speech was fluent and coherent but spontaneous speech was reduced. Memory was found to be normal. Reflexes and the remain-

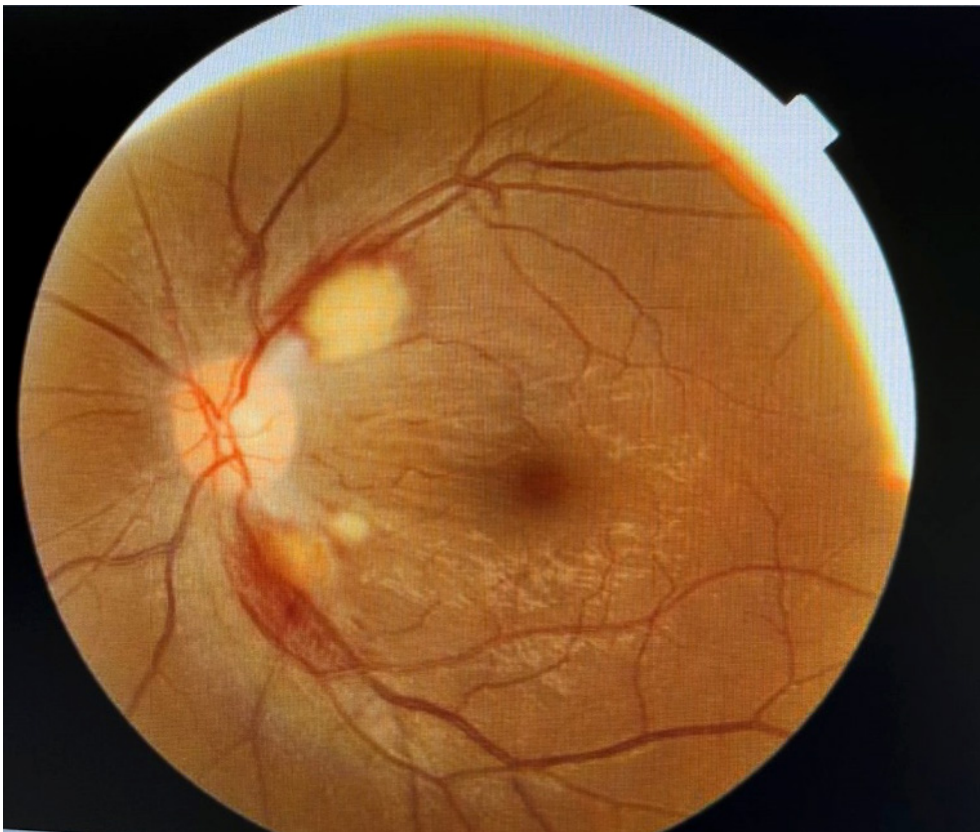
der of sensory and motor system examination were found to be normal. Abdominal examination revealed a uterine size corresponding to 16 weeks of gestational age with good fetal heart rate.

Ophthalmic examination showed a visual acuity of counting fingers at 2 m in both eyes. End gaze nystagmus was noted on dextroversion and levoversion. There was no restriction of extraocular movements. Anterior segment examination was found to be normal with both pupils briskly reactive to light by direct and consensual light reflexes. On dilated fundus examination, disc margins were well defined with a cup to disc ratio of 2:3. A large exudate was present along the superotemporal arcade measuring approximately 1 disc diameter in size, adjacent to which there were a few superficial flame-shaped hemorrhages. Along the inferotemporal arcade, superficial hemorrhages were noted as well. Blood vessels were of normal calibre with A:V ratio being maintained. Foveal reflex was clearly visualized in both eyes (Figs. 1 and 2).

Optical coherence tomography imaging of both eye confirmed the presence of peripapillary subretinal exudates along with hemorrhages in both eyes.



**Figure 1.** Fundus photo of the right eye showing a large exudate measuring 1 disc diameter in size, superotemporal to the disc. Also noted are superficial retinal hemorrhages along the vascular arcades.

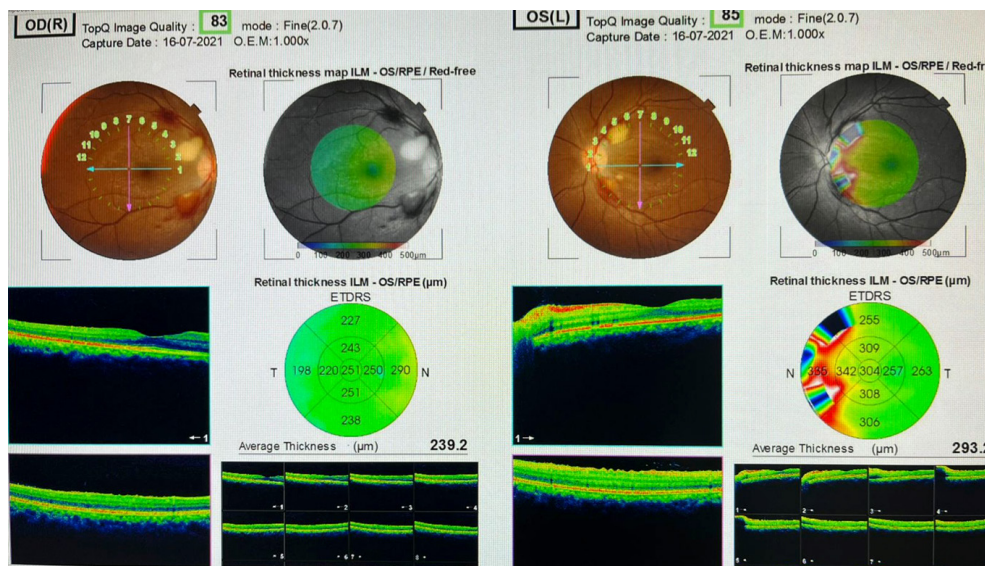


**Figure 2.** Fundus photo of the left eye large exudate measuring 1 disc diameter in size, superotemporal to the disc, and an exudate inferotemporal to disc with overlying hemorrhage.

Laboratory investigations revealed mild anemia, normal total leukocyte count and elevated erythrocyte sedimentation rate (ESR). On urine routine examination, urine albumin was 2+, and pus cells we noted (6-8). T4 levels were raised (14.4), T3 decreased (2.1), and Serum Thyroid-stimulating hormone was 1.48.

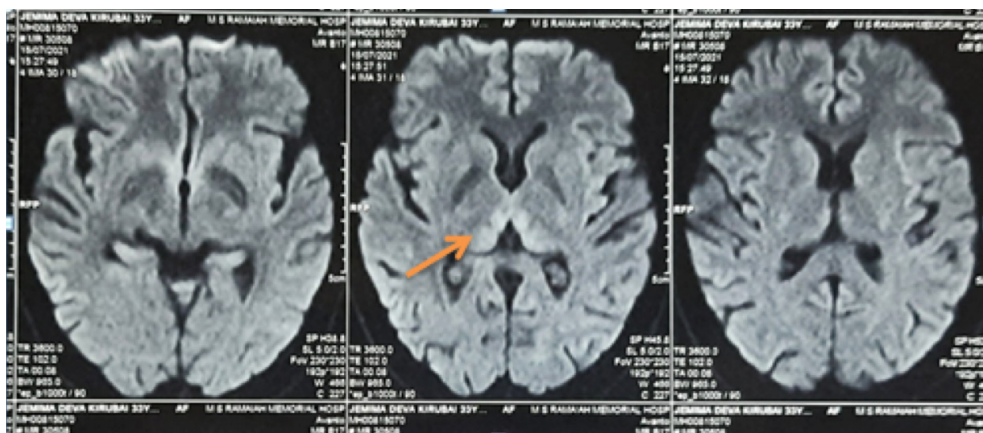
MRI of the brain was reported as bilateral thalamic, periaqueductal hyper intense lesions on fluid-attenuated inversion recovery and T2-weighted images which were symmetrical – suggestive of WE (Fig. 3 and 4).

Patient was diagnosed with WE with ocular manifesta-



**Figure 3.** Optical coherence tomography imaging of both eyes.





**Figure 4.** Bilateral thalamic, periaqueductal hyperintense lesions on fluid-attenuated inversion recovery and T2-weighted images which were symmetrical – suggestive of Wernicke's encephalopathy.

tions secondary to hyperemesis gravidarum with hypothyroidism. She was immediately started on Intravenous thiamine 100 mg TID for 1 week along with IV fluids vitamins and other supportive treatment. She gradually began to improve during her course of stay in the hospital. She was reviewed in the ophthalmology clinic after 2 weeks. Visual acuity in both eyes improved to 6/6 and there was no nystagmus. Fundus examination revealed complete resolution of exudates as well as the hemorrhages.

## Discussion

WE was first described by Dr. Carl Wernicke the 19<sup>th</sup> century as a neuropsychiatric condition. The condition was initially called "polioencephalitis hemorrhagica superioris" due to the characteristic findings on autopsy and was subsequently found to be linked to deficiency of thiamine (6). The it's demand is increased during hyper metabolic states such as pregnancy (1). Its absorption can be impaired even more in conditions such as hyperemesis gravidarum.

Neurologic symptoms of WE induced by hyperemesis gravidarum usually occur between the 14<sup>th</sup> and 20<sup>th</sup> weeks of pregnancy, after at least 3 weeks of persistent vomiting (7). This is in all probability because the time required to deplete the body's nutritional stores if thiamine is about 3 weeks. Delivery of healthy children occurs in only about 50% of these pregnancies (7).

In spite of the fact that WE is reversible with thiamine supplementation, significant difficulties can emerge in the management of the pregnant woman and unborn child. Without active intervention, WE can lead to permanent neurological complications as well as Korsakoff syndrome in the mother, which can be fatal in 10%–20% of cases. On the fetal side, WE can lead to miscarriage, preterm birth, and intrauterine growth retardation (8).

The diagnosis of WE is based on a history, clinical symp-

oms, and laboratory tests. Our patient presents with the classical triad of WE which resulted from severe vomiting associated with hyperemesis gravidarum. MRI showed bilateral and symmetric lesions suggestive of the diagnosis of WE. In addition to this, our patient showed drastic improvement with intravenous thiamine supplementation which further confirmed the diagnosis. Although the classical features and ocular motor abnormalities have been well documented in literature, the fundus findings were only rarely previously documented (9-12). It is postulated that mitochondrial dysfunction, as a result of thiamine deficiency, first gives rise to swelling and haemorrhage of the retinal nerve fiber layer and subsequent optic disc swelling appears only if mitochondrial damage is severe and prolonged (9).

Our patient showed the classical features of WE and was treated promptly for the same. Poor oral intake, physiological demands of pregnancy, and hyperemesis gravidarum precipitated the condition. She continued to receive vitamin supplementation until the end of her pregnancy and ocular features had completely resolved. The rest of her pregnancy was uneventful and she delivered a healthy baby at term.

## Conclusion

In conclusion, WE is a treatable condition which is very much reversible. Prompt recognition and treatment carries a good prognosis. A thorough ophthalmological examination should be a part of the evaluation of patients with risk factors and those diagnosed with WE. Early detection of intraocular changes resulting from thiamine deficiency is vital to early initiation of therapy and prevention of complications. This report adds to the little literature available on the fundus manifestations of WE in the setting of hyperemesis gravidarum.

## Disclosures

**Informed consent:** Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

**Peer-review:** Externally peer-reviewed.

**Conflict of Interest:** None declared.

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## References

1. Gratton SM, Lam BL. Visual loss and optic nerve head swelling in thiamine deficiency without prolonged dietary deficiency. *Clin Ophthalmol* 2014;8:1021–4. [\[CrossRef\]](#)
2. Sechi G, Serra A. Wernicke's encephalopathy: New clinical settings and recent advances in diagnosis and management. *Lancet Neurol* 2007;6:442–55. [\[CrossRef\]](#)
3. Flynn A, Macaluso M, D'Empaire I, Troutman MM. Wernicke's encephalopathy: Increasing clinician awareness of this serious, enigmatic, yet treatable disease. *Prim Care Companion CNS Disord* 2015;17:27008. [\[CrossRef\]](#)
4. Shimomura T, Mori E, Hirono N, Imamura T, Yamashita H. Development of Wernicke-Korsakoff syndrome after long intervals following gastrectomy. *Arch Neural* 1998;55:1242–5.
5. Reuler JB, Girard DE, Cooney TG. Current concepts. Wernicke's encephalopathy. *N Engl J Med* 1985;312:1035–9. [\[CrossRef\]](#)
6. Schabelman E, Kuo D. Glucose before thiamine for Wernicke encephalopathy: A literature review. *J Emerg Med* 2012;42:488–94. [\[CrossRef\]](#)
7. Eliakim R, Abulafia O, Sherer DM. Hyperemesis gravidarum: A current review. *Am J Perinatal* 2000;17:207–18. [\[CrossRef\]](#)
8. Michel ME, Alanio E, Bois E, Gavillon N, Graesslin O. Wernicke encephalopathy complicating hyperemesis gravidarum: A case report. *Eur J Obstet Gynecol Reprod Biol* 2010;149:118–9.
9. Bohnsack BL, Patel SS. Peripapillary nerve fiber layer thickening, telangiectasia, and retinal hemorrhages in Wernicke encephalopathy. *J Neuroophthalmol* 2010;30:54–8. [\[CrossRef\]](#)
10. Lawton AV, Frisard NE. Visual loss, retinal hemorrhages, and optic disc edema resulting from thiamine deficiency following bariatric surgery complicated by prolonged vomiting. *Ochsner J* 2017;17:112–4.
11. Guler A, Alpaydin S, Sirin H, Calli C, Celebisoy N. A non-alcoholic Wernicke's encephalopathy case with atypical MRI findings: Clinic versus radiology. *Neuroradiol J* 2015;28:474–7.
12. Serlin T, Moisseiev E. Fundus findings in Wernicke encephalopathy. *Case Rep Ophthalmol* 2017;8:406–9. [\[CrossRef\]](#)