Case Report

Varied Ocular Manifestations of Wernicke Encephalopathy: A Case Report and Literature Review

PJO – Official Journal of Ophthalmological Society of Pakistan

Noor Atieqa Inani Shaharuddin[1], Tajunisah Iqbal[2], Sujaya Singh[3]
[1-3]Department of Ophthalmology, Universiti Malaya, Kuala Lumpur, Malaysia

ABSTRACT

Wernicke encephalopathy (WE) is an acute neuropsychiatric syndrome resulting from thiamine deficiency. Though the classical triad of confusion, ataxia, and ophthalmoplegia is well-known, ocular presentations may be subtle or atypical. We report the case of a 49-year-old male admitted to the intensive care unit following an intra-abdominal leak post-Whipple procedure. He received total parenteral nutrition (TPN) for prolonged fasting. After a month, he developed bilateral visual blurring without pain or redness. Examination revealed horizontal gaze-evoked nystagmus, bilateral optic disc swelling, and peripapillary haemorrhages. Brain CT was normal, but neurological signs including ataxia and confusion emerged, prompting a clinical diagnosis of Wernicke encephalopathy. He received high-dose intravenous thiamine with subsequent improvement in vision and resolution of optic disc findings. Blurred vision may be an early ocular manifestation of Wernicke encephalopathy. This case highlights the need for high clinical suspicion and prompt thiamine supplementation in at-risk patients, even without full-blown encephalopathy.

Keywords: Wernicke Encephalopathy, Thiamine Deficiency, Eye Manifestations, Papilledema.

How to Cite this Article: Shaharuddin NAI, Iqbal T, Singh S. Varied Ocular Manifestations of Wernicke Encephalopathy: A Case Report and Literature Review. 2026;42(1):1-5. **Doi: 10.36351/pjo.v42i1.2133**

Correspondence: Noor Atieqa Inani Shaharuddin
Department of Ophthalmology, Universiti Malaya, Kuala Lumpur, Malaysia
Email: atieqashaharuddin@gmail.com

Received: June 27, 2025
Revised: November 4, 2025
Accepted: December 12, 2025

INTRODUCTION

Wernicke encephalopathy (WE) is a neuropsychiatric disorder caused by acute thiamine (vitamin B1) deficiency. While classically associated with chronic alcoholism, it may also occur in malnourished or critically ill patients, especially those on TPN without adequate supplementation. Common neurological features include ophthalmoplegia, gait ataxia, and confusion. Ocular findings such as nystagmus, gaze palsies, and optic disc changes are less frequently reported. This case illustrates a rare presentation of bilateral visual impairment due to papilledema in WE.

Case Presentation

A 49-year-old man underwent a Whipple procedure for mucinous cystadenoma of the pancreatic head. He was initially discharged but returned with abdominal pain and persistent vomiting. Imaging confirmed an anastomotic leak and intra-abdominal collection. He was treated in the intensive care unit with intravenous antibiotics and TPN due to prolonged fasting. After approximately one month on TPN, he reported bilateral blurred vision of four days' duration. There were no ocular pain, redness, or floaters. His vision was 6/24 bilaterally. Pupils were reactive without RAPD. Extraocular movements were full, but horizontal gaze-evoked nystagmus was noted. Anterior segments were unremarkable. Fundoscopy revealed bilateral optic disc swelling with peripapillary haemorrhages. OCT confirmed discs edema (Figure 1), while maculae were normal.

Neurological examination was limited due to lethargy but revealed cerebellar signs. Brain CT excluded mass or haemorrhage. Blood tests revealed

Table 1: Summary of the ocular findings in previously reported Wernicke encephalopathy cases.

No	Author, Year	Age, sex	Cause	Initial VA *	Final VA	Affected eyes	RAPD	Nystagmus	Ophthalmoplegia	Optic disc oedema/OCT RNFL thickening	Retinal haemorrhages	Visual field defect	Investigation (to confirm the diagnosis)	Intravenous thiamine	Ophthalmic sequelae
1	Serlin T et al.[2]	37,F	Malnutrition post gastric sleeve surgery	20/20, 20/20	20/20, 20/20	OU	No	Bilateral horizontal nystagmus	No	Yes	Preretinal hemorrhage OS	N/A	Low serum thiamine level	Yes	None
2	Ikeda et al.[3]	46, F	Malnutrition	N/A	N/A	OU	N/A	Horizontal nystagmus, spontaneous upbeat nystagmus and upgaze-evoked spontaneous upbeat nystagmus	Impaired eye movement	N/A	N/A	N/A	Low serum thiamine level, Brain MRI revealed high intensity in bilateral thalami	N/A	N/A
3	Mumford et al.[4]	24,F	Hyperemesis gravidarum with dietary restriction	N/A	20/17, 20/17	OU	N/A	Vertical and horizontal nystagmus	Impaired abduction OU	Yes	Peripapillary flame haemorrhages	N/A	Reduced red cell transketolase with a thiamine pyrophosphate effect	Yes	None
4	Sia et al.[5]	56,F	Poor diet secondary to oral discomfort after dental extraction, alcoholism	CF, CF	20/15, 20/15	OU	No	Horizontal gaze-evoked nystagmus	None	No disc swelling/pallor. OU superior and inferior RNFL thickening	Flame-shaped haemorrhage inferior to the OS macula	Automated perimetry (Humphrey 30-2): Generalized depression in the right visual field	Low serum thiamine level	Yes	None
5	Sia et al[5]	20,F	Chronic alcoholism	N/A	N/A	OU	N/A	Vertical and horizontal gaze-evoked nystagmus	None	OU RNFL thickening superior and inferior to the disc	Splinter haemorrhages in OD fundus	N/A	N/A	Yes	None
6	Kumar et al.[6]	22,F	Hyperemesis gravidarum	20/32, 20/32	N/A	OU	No	Yes	Sixth nerve palsy OU	Yes	Retinal hemorrhages	Severe visual field constriction OU	MRI brain: hyperintensities in bilateral medial thalami, periaqueductal gray matter, and mamillary bodies	Yes	Centrocecal scotomas OU after a month
7	Wei-Yi et al[7]	29,M	Poor food intake with chronic diarrhoea	NPL, NPL	2/60, 2/60	OU	No	Yes	Sixth nerve palsy OU	Yes	Peripapillary hemorrhages	The mean visual field defect was 23.0 dB in OD and 26.8 dB in OS	MRI brain: hyperintensity over the mammillary body, dorsal medial thalamus, and periaqueductal gray matter	Yes	Optic disc pallor
8	Lawton et al.[9]	37,F	Prolonged vomiting with history of bariatric surgery	20/400, 20/200	20/25, 20/20	OU	Yes	No	No	Yes	Intraretinal hemorrhages	Confrontation testing: Dense central visual field defects OU	Low serum thiamine level	Yes	None

*N/A: Not applicable; CF: Counting fingers; F: Female; M: Male; VA: Visual acuity; RAPD: Relative Afferent Pupillary Defect; OU: Bilateral eye; OD: Right eye; OS: Left eye; OCT RNFL: Optical Computed Tomography Retina Nerve Fibre Layer, dB: Decibels. *All visual acuities converted to Snellen chart.*

anaemia (Hb 97 g/L), thrombocytopenia (platelets 44 × 10^9/L), hypoalbuminemia, elevated ALP, prolonged PT, and elevated CRP. Lumbar puncture was deferred due to thrombocytopenia.

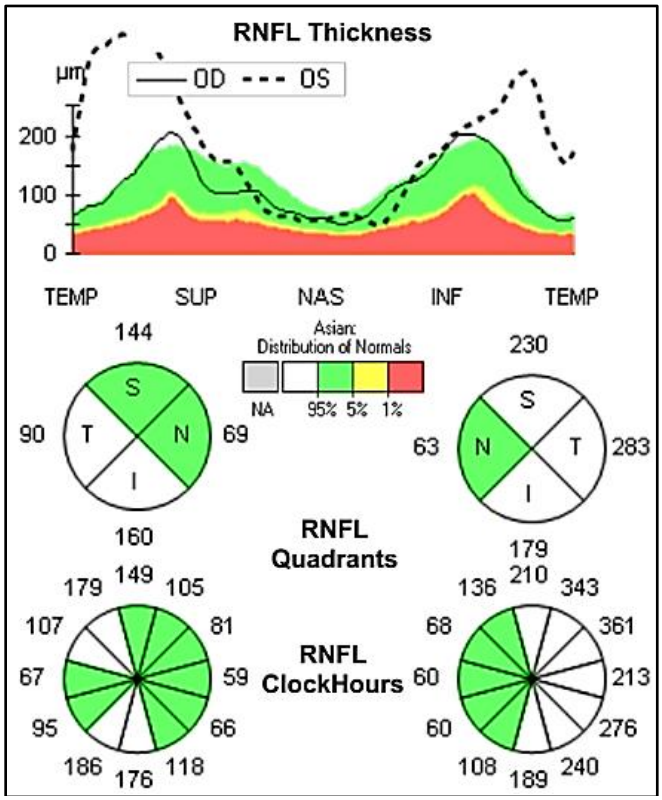

Figure 1: OCT RNFL on presentation shows an increase in the thickness of retina nerve fibre layer which is more marked in the left eye.

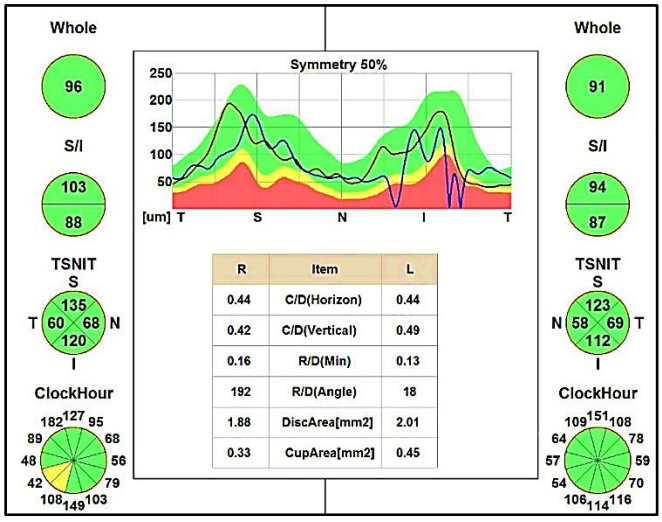

Figure 2: OCT RNFL during 1-month review shows OU resolution of optic disc swelling.

Given the clinical picture of visual symptoms, bilateral papilledema, ataxia, prolonged fasting, and exclusive TPN, WE were strongly suspected. Serum thiamine level could not be tested. Empirical treatment with intravenous thiamine 500 mg three times daily was initiated for three days, followed by 250 mg daily for three more days, and oral thiamine 100 mg thereafter. Parenteral multivitamin (Parentrovite) was also administered.

Vision improved to 6/12 after one week and returned to 6/6 at one-month follow-up. OCT showed resolution of disc swelling (Figure 2). Fundus photography at follow-up revealed residual peripapillary haemorrhages (Figure 3).

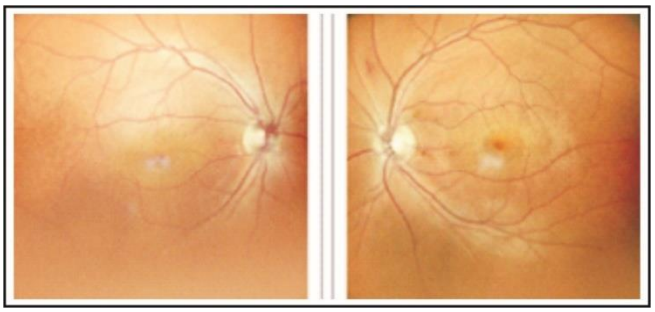

Figure 3: Fundus photo during 1- month review. Both optic discs were no longer swollen with residuals of peripapillary splinter and dot-blot haemorrhages (red arrow) in the left eye.

DISCUSSION

WE remain underdiagnosed, particularly in non-alcoholic patients. Caine's criteria suggest diagnosis when two of the following are present: dietary deficiency, oculomotor signs, cerebellar dysfunction, or mental changes.[1] In our case, risk factors included post-surgical fasting, vomiting, and TPN. Ocular signs preceded systemic manifestations.

In literature (Table 1), ocular manifestations in WE range from nystagmus and ophthalmoplegia to optic disc swelling and retinal haemorrhages. Nystagmus, particularly horizontal gaze-evoked, is among the most frequently described features and is typically attributed to cerebellar or brainstem involvement.[2-5] Several studies have reported spontaneous upbeat nystagmus and cranial nerve VI palsy leading to abduction deficits.[3,4,6,7] These signs may be early and reversible with timely treatment.

Fundus findings, although less common, have been increasingly recognized. Optic disc swelling, peripapillary haemorrhages, and thickening of the

retinal nerve fibre layer (RNFL) are notable presentations. In a review of Japanese prisoners of war, only 2 of 52 WE patients had papilledema, suggesting that this feature is underreported or underrecognized.[8] Serlin et al, described bilateral optic disc swelling in WE patients, sometimes without ophthalmoplegia.[2] Papilledema may reflect increased intracranial pressure or mitochondrial dysfunction affecting retinal ganglion cells and capillaries.

Other notable findings include relative afferent pupillary defect (RAPD), especially in unilateral involvement, and visual field defects such as centrocecal scotomas or peripheral constriction.[5-7,9] Though uncommon, these findings have been documented and may support the diagnosis when present.

Diagnosis of WE remain clinical, especially in resource-limited settings where serum thiamine levels and advanced neuroimaging may not be readily available. Thiamine blood assays are neither timely nor reliable in acute settings. MRI may show characteristic lesions in the thalami, mammillary bodies, and periaqueductal grey matter but should not delay treatment initiation.[10]

Prompt thiamine replacement typically results in rapid resolution of symptoms. Literature supports that most visual outcomes are favourable with early therapy, while delays can lead to optic atrophy and permanent vision loss.[7] In our case, the patient's full recovery of vision and disc appearance after treatment supports the reversibility of ocular manifestations in WE.

This case highlights the importance of including WE in the differential diagnosis of bilateral visual loss with disc swelling in at-risk patients, even in the absence of classic encephalopathy triad. Awareness among ophthalmologists and general physicians can facilitate earlier diagnosis and improved outcomes.

CONCLUSION

Ocular findings in Wernicke encephalopathy are diverse and may include nystagmus, ophthalmoplegia, optic disc swelling, and retinal haemorrhages. Blurred vision may be the first or sole symptom. Early recognition and treatment with thiamine are critical, particularly in malnourished patients or those on prolonged TPN.

Funding: This study was not funded by any organization.

Patient's Consent: Researchers followed the guidelines set forth in the Declaration of Helsinki.

Conflict of Interest: Authors declared no conflict of interest.

REFERENCES

1. **Caine D, Hallsiday GM, Kril JJ, Harper CG.** Operational criteria for the classification of chronic alcoholics: identification of Wernicke's encephalopathy. J Neurol Neurosurg Psychiatry. 1997;**62(1):**51-60. Doi: 10.1136/jnnp.62.1.51.
2. **Serlin T, Moisseiev E.** Fundus Findings in Wernicke Encephalopathy. Case Rep Ophthalmol. 2017;**8(2):**406-409. Doi: 10.1159/000478924.
3. **Ikeda K, Yano H, Higa T, Kinjo M.** Nystagmus in non-alcoholic Wernicke encephalopathy. BMJ Case Rep. 2021;**14(2):**e241130. Doi: 10.1136/bcr-2020-241130.
4. **Mumford CJ.** Papilloedema delaying diagnosis of Wernicke's encephalopathy in a comatose patient. Postgrad Med J. 1989;**65(764):**371-373. Doi: 10.1136/pgmj.65.764.371.
5. **Sia PI, Sia DI, Crompton JL, Casson RJ.** Nerve Fiber Layer Infarcts in Thiamine Deficiency. J Neuroophthalmol. 2015;**35(3):**274-276. Doi: 10.1097/WNO.0000000000000243.
6. **Kumar S, Bolar AR, Shetty R, Govindappa SKG, Pramod MNB, Agadi JB, et al.**Wernicke's encephalopathy: Is visual loss a red herring? Clin Case Rep. 2022;**10(9):**e6372. Doi: 10.1002/ccr3.6372.
7. **Yeh WY, Lian LM, Chang A, Cheng CK.** Thiamine-deficient optic neuropathy associated with Wernicke's encephalopathy in patients with chronic diarrhea. J Formos Med Assoc. 2013;**112(3):**165-170. Doi: 10.1016/j.jfma.2012.10.010.
8. **DeWardener HE, Lennox B.** Cerebral beriberi (Wernicke's encephalopathy); review of 52 cases in a Singapore prisoner-of-war hospital. Lancet. 1947;**1(6436):**11-17. PMID: 20278697.
9. **Lawton AW, 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(1):**112-114. PMID: 28331457; PMCID: PMC5349621.
10. **Zuccoli G, Pipitone N.** Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature. AJR Am J Roentgenol. 2009;**192(2):**501-508. Doi: 10.2214/AJR.07.3959

Authors Designation and Contribution

Noor Atieqa Inani Shaharuddin; Medical Officer: *Concepts, Design, Literature Search, Manuscript*

Preparation, Manuscript Editing, Manuscript Review.

Tajunisah Iqbal; Professor: *Concepts, Design, Literature Search, Manuscript Preparation, Manuscript Editing, Manuscript Review.*

Sujaya Singh; Associate Professor: *Concepts, Manuscript Preparation.*