Review Article

Wernicke Encephalopathy: An Updated Narrative Review

Elmukhtar Habas[1], Kalifa Farfar[1], Nada Errayes[2], Amnna Rayani[3], Abdel-Naser Elzouki[1,4,5]

[1]Department of Medicine, Hamad Medical Corporation, [4]Department of Medicine, Weill Cornell Medical College, [5]Department of Medicine, College of Medicine, Qatar University, Doha, Qatar, [2]Department of Epidemiology, University of Lincoln, Lincoln, UK, [3]Tripoli Medical Center, Tripoli, Libya

Abstract

Wernicke's encephalopathy (WE) and Korsakoff Syndrome (KS) are distinct neurological disorders that may have overlapping clinical features. Due to the overlap, they are collectively known as Wernicke–Korsakoff syndrome. WE is related to diencephalic and mesencephalic dysfunction due to thiamine. WE typically manifests as confusion, ophthalmoplegia, nystagmus, and gait ataxia (Wernicke's triad), although they may not consistently occur together. Although WE mostly occurs in alcoholics, other etiologies, such as post-bariatric surgery, must be considered. Early diagnosis and therapy by intravenous thiamine are essential to prevent WE complications and to reduce morbidity and mortality. Therefore, physicians' and patients' awareness of WE is essential for early diagnosis and therapy. Accordingly, this narrative review aimed to provide an update on WE by reviewing articles published between April 2015 to April 2022 about the etiology, pathophysiology, diagnosis, and WE management updates. EMBASE, PubMed, Google Scholar, Google, and Scopus search engines were used to conduct the literature search.

Keywords: Alcohol use disorder, bariatric surgery, ophthalmoplegia, thiamine, vitamin B1, Wernicke's encephalopathy, Wernicke's triad

Address for correspondence: Prof. Abdel-Naser Elzouki, Department of Medicine, Hamad General Hospital, Hamad Medical Corporation, P.O. Box: 3050, Doha, Qatar.
E-mail: nelzouki_1999@yahoo.com

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INTRODUCTION

Thiamine is a water-soluble vitamin that is essential in the metabolic pathways involved in glucose metabolism.[1] Wernicke's encephalopathy (WE) is a neurological disorder that was first described by Carl Wernicke in 1881.[2] While WE is mostly induced by malnutrition due to alcohol dependence (ICD-11), other nonalcoholic conditions can also cause WE.[3] In cases with vitamin B1 deficiency, WE is diagnosed by the occurrence of a minimum of two of the typical WE clinical triad (ophthalmoplegia, gait ataxia, and confusion). These classic WE symptoms are noted in 16%–33% of patients at presentation.[4–6] WE is usually caused after 4–6 weeks of thiamine deficiency.[7] Typical WE brain lesions are detected at autopsy in 0.4%–2.8% of the general population in developed countries, and it most commonly affects moderate and severe alcoholics.[8] Thiamine deficiency affects both the central and peripheral nervous systems. Permanent brain damage can occur when the deficiency is detected late or early but not managed promptly and efficiently.

Korsakoff syndrome (KS) is another neurological disorder that severely alters the working memory. Injury

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of the diencephalon–hippocampal circuit prevents KS patients from combining short- and long-term memories.[9] KS is associated with irreversible memory loss in addition to cognitive and behavioral abnormalities.[10] In addition, KS manifests with confabulation, substantial anterograde, and retrograde deficits of memory. WE and KS have overlapping clinical features. Due to the overlap, they are collectively known as Wernicke–Korsakoff syndrome (WKS). It is not well established whether absence of alcohol consumption reduces the likeliness of WE advancing to KS.[11] Nonetheless, WE must be distinguished from KS because WE is preventable. WE should usually be suspected following an episode of acute/subacute thiamine deficiency. Immediate memory is often retained when the sensorium is intact in KS cases, while short-term memory is usually diminished. Confabulations are often provoked in chronic KS and spontaneous in acute WE.[12] The neurological manifestations of severe acute vitamin B1 deficiency include dry beriberi. Prolonged vitamin B1 deficiency can also result in wet beriberi, which manifests with clinical features of heart failure and other features of KS.[13] The occurrence of both dry and wet beriberi is mainly based on genetic liability.

Awareness of WE among physicians and patients is essential for early diagnosis and therapy, and in turn, to reduce the associated morbidity and mortality. Accordingly, this narrative review aimed to provide an update on WE by primarily reviewing articles published between April 2015 to April 2022 about the etiology, pathophysiology, diagnosis, and WE management updates. EMBASE, PubMed, Google Scholar, Google, and Scopus were used to conduct searches using the following keywords/phrases: Wernicke's encephalopathy, WE pathogenesis, WE, KS, WE-KS therapy, WE diagnosis and therapy updates.

EPIDEMIOLOGY OF WERNICKE'S ENCEPHALOPATHY

Most studies reporting on the prevalence of WE are based on autopsies and have reported rates to be 1%–3%. In the general population, the prevalence of WE has been estimated to be 0.4%–2.8%, with a female-to-male ratio of 1:1.7, with no disposition based on race.[5] Autopsy studies consistently noted a higher rate of Wernicke lesions in the general population than clinical studies, indicating that WE is clinically underrecognized.[8] WE is more common in developing countries mainly due to increase in vitamin deficiencies being induced by moderate to severe alcohol use disorder (DSM-V) and an increase in the prevalence of malnutrition.[5]

ETIOLOGY OF THIAMINE DEFICIENCY

Thiamine deficiency is frequently linked to a severe alcohol use disorder in about 50% of WE patients.[14] Long-term alcohol consumption reduces thiamine absorption in the intestine. Genetic predisposition, a poor diet, and reduced ability of liver to store thiamine are other causes of thiamine deficiency and should also be excluded.[15–17] In addition, prolonged parenteral nutrition, severe malnutrition, liver disease, hyperemesis gravidarum, malignancy, immunodeficiency syndromes, severe anorexia nervosa, and hyperthyroidism are other causes of nonalcohol-related thiamine deficiency that should be excluded as causes of thiamine deficiency. Acute and chronic infection and prolonged carbohydrate or glucose intake are other triggers for WE development in a person with thiamine deficiency. Patients with nonalcoholic WE are more likely to have clinical symptoms and imaging results that differ from those with alcohol-induced WE.[6,18,19]

UNDERLYING CONDITIONS AND DISEASES

WE is broadly classified into alcoholic and nonalcoholic based on the etiologic factor. Unbalanced nutrition, chemotherapy, GI fistula, AIDS, dialysis, severe vomiting, tumor, and prolonged parenteral nutrition are some of the other causes of vitamin B1 deficiency;[20,21] however, they are less frequent than alcohol dependence (ICD-11) worldwide.[14] Oculomotor and, to some extent, cerebellar damage clinical features are the usual presenting features in nonalcoholic-induced thiamine deficiency.[22] In patients with nonalcoholic-induced WE, mental status changes alone without other presenting symptoms and signs may result in delay or misdiagnosis of WE.[23] WE occurs mainly in adults; however, it can also occur in children. WE has been observed in children and infants that were fed isotonic drinks, have excessive food restriction such as in cases of atopic dermatitis, and gluten-sensitive children. Moreover, breast-fed babies of mothers who have vitamin B1 deficiency are also at a higher risk of WE. The common causes and the possible mechanisms of B1 deficiency are presented in Table 1.

PATHOPHYSIOLOGY OF WERNICKE'S ENCEPHALOPATHY

The required vitamin B1 per utilized 1000 Kcal is 0.5 mg, which corresponds to about 1.4 mg daily in a healthy adult. However, these recommended levels are not adequate in cases of regular alcohol consumption, which restricts thiamine uptake. Thiamine is primarily found in foods such as brown rice, yeast, legumes, and cereals made from whole grains.[24] Thiamine is absorbed in the small intestine

Table 1: Causes and mechanisms of thiamine deficiency

Causes	Possible mechanisms
Alcohol dependence (ICD-11) (50% of cases)	Reduces thiamine intake, decreases thiamine absorption by the small intestine, and impairs intracellular thiamine utilization
Hypermetabolic conditions	Hyperthyroidism, infection, and inflammation increase thiamine utilization, decrease absorption, and malnutrition
Gastro-intestinal procedure/starvation	Low thiamine intake and absorption, increase in thiamine utilization as in pregnancy, reduced thiamine storage, and thiamine loss as in short bowel syndrome or bypass surgery lead to Vitamin B1 deficiency
Genetic diseases	Abnormalities of thiamine transporter due to genetic mutation (gene SLC19A3)
Drugs (e.g., 5FU)	Vomiting and loss of appetite reduce thiamine intake
Hypomagnesemia	Associates with alteration of carbohydrate metabolism and cachexia

ICD – International classification of diseases; 5FU – 5-Fluorouracil

through both active transport and passive diffusion, with the jejunum being the major absorption site.[25] The human body's thiamine storage capacity ranges between 30–50 mg, which is usually sufficient on average for 3–4 weeks, corresponding to a consumption of only about 2 mg per day. In the brain, after crossing the blood–brain barrier (BBB), it forms its active metabolite known as thiamine pyrophosphate, which is a coenzyme for multiple enzymes in the Kreb cycle (KC) and the pentose phosphate pathway (PPP). The latter acts in carbohydrate metabolism as a coenzyme.[6]

Brain cellular edema damage in WE are due to both vasogenic and cytotoxic mechanisms. Vasogenic edema is defined as extracellular fluid accumulation due to BBB damage and serum protein leakage from the vessels. On the other hand, cytotoxic edema is characterized by cell swelling because of intracellular fluid accumulation.[26] Vasogenic edema and cytotoxic brain edema, which occur in thiamine deficiency due to KC and PPP dysfunction,[5,10] are thought to cause brain insult in WE. Putamen, caudate, pons, splenium of the corpus callosum, red nucleus, dorsal medulla, substantia nigra of the midbrain, cranial nerve nucleus, dentate nucleus, vermis, paravermian region of the cerebellum, fornix, and pre-and postcentral gyri are the commonly involved areas in the brain, and are detectable by magnetic resonance imaging (MRI) in most cases.[27] The familiarity of WE symptoms and its underlying conditions among clinicians and radiologists is essential for its early detection and prevention of severe permanent neurological damages.

Thiamine is a key vitamin that is essential in maintaining cell membrane integrity and osmotic gradients between the intracellular and extracellular compartments. It is stored in the body tissues, mainly in the liver, as thiamine diphosphate (TDP). TDP is an essential cofactor for multiple KC enzymes and PPP.[5] KC is a vital part of aerobic metabolism, during which ATP is produced. It is the last step in metabolizing carbohydrates, fat, and protein nutrients. KC biochemical reactions occur in cell mitochondria to finalize the nutrients' catabolism to carbon dioxide, water, and energy. On the contrary, in cells with no mitochondria (i.e., red blood cells), PPP is the primary source of pentoses. The pentoses are vital in the synthesis of nucleic acid and the reduction of nicotinamide adenine dinucleotide phosphate (NADPH) formation. NADPH is important for multiple metabolic processes and assists free radical scavenging during oxidative stresses.[5]

Vitamin B1 deficiency decreases intracellular TDP, inhibiting the KC and PPP activity. Inhibiting both cycles reduces ATP production and DNA/RNA and NADPH synthesis, impairing cells' resistance to oxidative stresses. In addition, inhibition of the two cycles causes toxic intermediates such as lactate, alanine, and glutamate accumulation, decreasing intracellular pH and electrolyte disequilibrium and causing cytotoxic edema. In addition, vasogenic edema occurs due to BBB dysfunction. The BBB consists of endfeet that comprises mesenchymal-like cells, pericytes, capillary endothelial cells, and astrocytes terminal processes. The endfeet is crucial for BBB formation and maintenance through synthesizing certain factors and keeping tight and firm junctions.[27,28] In WE, pH reduces to <7.3 due to metabolic acidosis because of lacatic-, hydroxyybutric-, and acetoacetic- acids, but it is compensated by respiratory alkalosis.[29]

MECHANISMS OF THIAMINE DEFICIENCY DUE TO ALCOHOL INTAKE

In cases of alcohol dependence, thiamine utilization by cells is affected in various ways. In cases of acute alcohol intake, thiamine absorption is reduced due to deactivation of the enzyme thiamine diphosphokinase. Magnesium deficiency is a common electrolyte abnormality in chronic moderate to severe alcohol use disorder (DSM-V). Magnesium is necessary for thiamine-dependent enzyme function to form a phosphorylated thiamine complex. Failure of the complex formation causes thiamine deficiency-like features.[30] Drugs such as 5-fluorouracil and fedratinib

affect thiamine function and carbohydrate metabolism enzymes, which cause severe weight loss and systemic muscle wasting, increasing the rate of inflammation and thiamine utilization.[31–33] Vitamin B1 insufficiency due to an increase in thiamine consumption in hypercatabolic status is not uncommon in systemic illness, inflammatory disorders, and hyperthyroidism, especially in individuals who have already depleted their thiamine stores.[29,33,34] It has been reported that thiamine-transporter genetic mutation, such as thiamine metabolism dysfunction syndrome-2 (gene SLC19A3 mutation), affects biotin and other vitamins' intestinal absorption.[35,36]

CLINICAL PRESENTATION OF WERNICKE'S ENCEPHALOPATHY

The European Federation of Neurological Societies (EFNS) guidelines for WE diagnosis, management, and prevention recommend the existence of at least 2 of 4 of the following features: (a) dietary deficiencies, (b) ophthalmoplegia, (c) cerebellar dysfunction, and (d) either an altered mental status or mild memory loss.[8,37] The classical triad (eye signs [ophthalmoplegia], cerebellar dysfunction, and altered mental status) of WE occurs in only 16% of patients.[38] Although confusion, oculomotor abnormalities, and ataxia are the clinical triad of WE, mental status changes are the most common symptom reported in 34%–82% of postmortem-confirmed WE patients.[2] The mental status changes include confusion, inability to concentrate, dizziness, spatial disorientation, memory disturbances, apathy, drowsiness, and cognitive impairment. Complete or partial eye movement restrictions because of thiamine deficiency, are induced mostly due to third cranial nerve palsy; however, complete ophthalmoplegia rarely occurs, but horizontal nystagmus is the most joint ocular abnormality at presentation.[2] Pontine tegmentum lesions manifest as diplopia, decreased visual acuity, bilateral 6th nerve palsy and abnormal eye gaze movement are not uncommon.[27] Vestibular dysfunction and cerebellar vermis damage in WE cause gait ataxia, ranging from inability to stand to minimal gait abnormalities. Hypothermia or hyperthermia (due to the posterior hypothalamus lesions), deafness, and epileptic seizures are infrequent features.[27]

Although thorough clinical and social history is essential, a detailed physical examination must be conducted. Whenever WE is considered, the first essential step is to prescribe thiamine rather than waiting for the results of other laboratory tests. There is no specific laboratory test diagnostic for WE; however, basic laboratory workup and brain imaging are required to support the provisional clinical diagnosis of WE. MRI is more sensitive than CT in detecting acute encephalopathy. Periventricular lesions are common findings;[39,40] however, they are not consistently present. MRI of the brain is a valuable method for confirming a diagnosis of WE or KS.[41] A complete metabolic panel and blood count are essential to rule out other causes of central nervous system abnormalities. The level of erythrocyte transketolase enzyme is sometimes carried out, detecting vitamin B1 deficiency. In addition, measurement of plasma lactate and pyruvate levels is important because thiamine acts as a pyruvate dehydrogenase cofactor, and its inhibition causes an increase in their levels.

It was reported that Caine's criteria have an 85% sensitivity in diagnosing WE [Table 2]. However, Caine's criteria are not entirely validated in nonalcoholic WKS; its application improves the accuracy of WKS diagnosis in alcoholics or other conditions predisposing them to vitamin B1 deficiency.[42]

DIFFERENTIAL DIAGNOSIS

Before diagnosing WE, it is necessary to rule out other conditions that may present with similar neuropsychiatric symptoms, including hepatic encephalopathy, normal pressure hydrocephalus, delirium tremens, alcohol withdrawal syndrome, stroke, and chronic hypoxia. In addition, a brain MRI may be beneficial in confirming the clinical suspicion of WE and ruling out stroke; however, both investigations should not be used to delay initiating thiamine replacement therapy if WE is suspected. While delirium is considered to be common and may be associated with WE,[43] in a large series, the association between these two conditions were reported to be weak.[44] WE should be considered in the differential diagnosis of delirious patients, and common causes of delirium should be ruled out [Table 3].

THIAMINE DEFICIENCY AFTER GASTRECTOMY

Morbid obesity, defined as having a body mass is ≥40 kg/cm^2, has been recognized by the World Health Organization as a

Table 2: Criteria for the clinical diagnosis of Werneck's encephalopathy

Symptoms or signs	Clinical assessment
Thiamine dietary deficiency	History of poor diet intake, low body mass index, low serum B1 level
Changed mental status or memory impairment	Confusion, coma, disorientation, cognitive status abnormality assessed by digit span test, failing the memory test, Impairment on more elaborate neuropsychological tests of memory function
Cerebellar malfunction	Abnormal figure-nose test, heel–shin test, gait abnormalities, ataxia, dysdiadokokinesia
Eye abnormalities	Ophthalmoplegia, ocular gaze movement disturbance, nystagmus (mainly horizontal)

Table 3: Common causes of delirium

Cause	Examples
Drugs	Opioids, sedative-hypnotics, antipsychotics, lithium, skeletal muscle relaxants, antihistamines, heroin, and benzodiazepine
Poisons	Ethylene glycol, methanol, carbon monoxide, cyanide, and hydrogen sulfide
Metabolic abnormalities	Hypercapnia; hypoxemia; hypo- or hyper- function of parathyroid, thyroid, pituitary, and suprarenal gland; porphyria' Wilson disease
Infections	Sepsis, fever-related delirium, and systemic infections
Nutritional	Werneck's encephalopathy, Vitamin B12 deficiency, possibly folate and niacin deficiencies
Organ failure	Liver, heart, polycythemia, hypereosinophilia, leukemic blastic crisis, thrombocytosis, renal failure
Brain disorders	Encephalitis, meningitis, brain or epidural abscess, epilepsy, head injury, hypertension, some psychiatric illness
Others	Hypothermia, hyperthermia, burns, trauma

systemic disease that may lead to disabling comorbidities.[45] Morbid obesity and its complications negatively affect patients' life quality by increasing the rate of stroke, hypertension, osteoarthritis, and diabetes mellitus.[12,46] Bariatric procedures have achieved outstanding results for weight loss and improvement in obesity-related comorbidities; however, complications such as anastomotic leakage, bleeding, stenosis, and nutritional deficiency have been reported. Bariatric surgical procedures as a therapy for morbid obesity include bypass surgery, gastric banding, and sleeve gastrectomy. Worldwide, laparoscopic sleeve gastrectomy is the most common bariatric surgery for weight loss in morbidly obese patients.[47] The rates of this procedure are increasing, with about half a million bariatric surgeries have been conducted yearly worldwide.[17,48] However, most data about WE after bariatric procedures are case reports. In 2018, Zafar *et al.* stated that only 119 cases, including theirs, have reported thiamine deficiency following bariatric surgeries.[49] Thiamine deficiency appears more after Billroth II anastomosis or gastro-jejunostomy because the food passes directly to the second part of the small intestine.[50] It is estimated that the prevalence of thiamine deficiency after bariatric procedures is about 18%.[51]

Generally, obese subjects do not have healthy nutrition and are malnourished, which is more complicated after bariatric surgery. It was reported that excessively prolonged vomiting triggers vitamin depletion, such as thiamine, in a short period because of its low storage.[52] Moreover, quick bodyweight reduction (>30 kg) during the first three months post-bariatric surgery is a warning sign of a higher risk of WE.[53,54] Malnutrition after bariatric surgery is mainly due to vomiting, changes in feeding pattern and style, and the alternation in the lifestyle increases a refeeding syndrome risk.[52]

Neurological complications following bariatric surgeries are rare (in about 1.3% of patients).[53,54] Besides the usual presentations, WE can sometimes present with atypical symptoms such as hypothermia, papilloedema, seizures, and hearing loss.[6,55] Most of the hearing loss improves after thiamine therapy;[6] one case report noted a minimal persistent sensorineural hearing impairment.[55]

Thiamine deficiency is reported in up to 49% of patients undergoing gastric bypass owing to its malabsorptive nature. Nonetheless, there have been a number of reported cases in the literature after sleeve gastrectomy, mainly due to protracted vomiting.[56] Current guidelines for bariatric surgery suggest preventive thiamin supplements for all patients undergoing surgery and even in higher doses for patients suspected of preoperative thiamine deficiency.[46] Thiamin deficiency typically occurs around 6 weeks to 3 months after surgery; however, cases were reported as early as 2 weeks.[57]

MANAGEMENT

WE is a reversible medical emergency that has either acute or chronic forms. As a result, the primary goal of WE therapy is to avoid brain injury by correcting the reduced vitamins quickly and effectively. The WE management is parenteral thiamine replacement once the WE clinical diagnosis is made, especially in a person suspected of dietary thiamine deficiency. Plasma thiamine level must be checked before starting intravenous thiamine; however, the thiamine replacement should begin without waiting for the results of the level of thiamine. In cases with bariatric surgery, 6 months of thiamine plasma levels follow-up and sufficient thiamine supplement must be ensured. Neurological symptoms correction is usually achieved in most patients by parenteral vitamin B1 provided by instant parenteral B1 administration, especially in highly malnourished patients. Oral thiamine is not advisable as the first line of treatment for acute and subacute WE because thiamine absorption is usually limited in chronic alcoholics and undernourished patients.[58]

In the United States, the recommended treatment is 100 mg of parenteral (intravenous or intramuscular) thiamine for 3–7 days (treatment phase) followed by oral thiamine for throughout the duration the patient consumes alcohol. However, case reports have indicated that higher doses (up to 1 g of parenteral thiamine) are required for symptom relief. The Royal College of Physicians experts proposed the administration of 500 mg parenteral thiamine (intravenously) three times a day for 2 to 3 days.[59] If there is no response, the recommendation

is to cease supplementing and evaluate the need for supportive care (unless the patient is comatose). Patients with neuropsychiatric symptoms should be maintained on parenteral administration of 250 mg thiamine for 5 days or if improvement persists after a partial response.[6] "The current practice to avoid thiamine hypersensitivity reactions is recommended to start the infusion slowly, mostly over 30 minutes.

While prescribing low doses of vitamin B1 can result in clinical improvements, some physicians also prescribe high-dose parenteral thiamine based on older studies and anecdotal evidence. However, findings from a recent randomized clinical trial (RCT) found that high dosing did not confer any significant benefit compared with intermediate or lower doses of thiamine. The authors of this RCT also suggested that physicians should alter the treatment strategy to be patient-specific and also to determine and rectify magnesium and other vitamin B deficiencies.[60] In fact, neurological abnormalities may continue, and acute WE may proceed to chronic KS, despite the use of high doses of parenteral vitamin B1.[8] If the Korsakoff amnestic state or the other mental abnormalities are not stabilized after high-dose parenteral thiamine and WE and acute symptoms do not improve after 7 days, then secondary harm prevention approaches must be considered. The EFNS guideline recommends administration of 200 mg intravenous thiamine every 8 hours until the patient is free from the symptoms of KS.[8] The extra considerations include long-term residential care, supported housing, rehabilitation, the treatment of comorbid deficits and medical disorders, and maintenance with oral thiamine to stop future injury.[61]

In WE-comatose patients, intravenous thiamine must be prolonged; however, no recommended duration is yet established. Once the patient has a stable response to thiamine, oral vitamin B1 (50–100 mg) must be continued, especially in those with alcohol dependence (ICD-11) and who have undergone bariatric surgery.[62] To provide more robust recommendations for the management of WE–KS syndrome, additional studies are required.

In a vitamin B1-deficit status, it is recommended to first establish vitamin B1 replacement before starting a high carbohydrate diet, which increases thiamine metabolism enzyme activity and worsens neurological features.[63]

Magnesium deficiency is frequent, particularly in chronic WE; therefore, magnesium supplementation must be initiated without waiting for the laboratory results of magnesium levels in the serum, as it is critical for WE recovery, particularly among patients with alcohol dependence.[12,64] In persistent cognitive impairment (e.g., Korsakoff dementia), cognitive promoters such as acetylcholinesterase inhibitors, and memantine drugs have demonstrated some benefits.[65] However, a case report where the use of rivastigmine in five patients was compared with controls who received conventional treatment found no significant differences in improvements between both groups.[66]

WE management is complex and frequently necessitates a multidisciplinary collaborative effort. Medical intensive care and neurology teams are primarily involved in the care. Severe cases of WE are, in general, treated in intensive care units. Other specialties (particularly psychiatric teams) are essential and must be appropriately involved. Social workers, nurses, dieticians, and pharmacists are also essential in the treatment, given the support requirements of patients with WE.

To reduce the rate of WE recurrence, encouraging alcohol abstinence and ensuring patient compliance are critical. Vitamin supplementation must be given in sufficient amounts to WE patients, especially to those who have undergone weight reduction interventions such as a gastrectomy.[62] Furthermore, patient education about the prognosis and outcomes should be discussed intensively with these patients and their families. If long-term care for WE is required, it should be carefully and thoroughly established. All teams involved in the treatment of patients with WE must maintain close communication to provide standard treatment, improve the quality of life, and reduce the burden on patients' families.[61,67]

OUTCOMES

WE is a serious medical condition associated with a high morbidity and mortality rates. The presence of KS may complicate the cases in about 85% of patients with WE, and may result in death in approximately 20% of these patients.[27] In general, global confusion state rapidly resolves with adequate thiamine replacement, but ophthalmoplegia and ataxia may continue. Patients with few neurological symptoms benefit the most from thiamine supplementation. WE survivors have a chance to proceed to KS psychosis, necessitating long-term hospitalization and care. Fewer than 10% of these patients recover after being discharged from long-term care. Many of these patients are likely to develop long-term neurological abnormalities such as ataxia, nystagmus, and KS, significantly affecting their quality of life. Despite the intensive parenteral thiamine therapy, some WE patients may proceed to KS and need care in specialized centers

to improve the likeliness of recovery. Unfortunately, no long-term follow-up studies have been conducted, and anecdotal accounts indicate that a significant proportion of these patients die prematurely.[68,69]

CONCLUSION

WE is not a rare disease, and can cause severe morbidities and mortality. It is commonly associated with alcohol intake; however, other causes must be considered. Bariatric surgery, which is increasingly being performed, is associated with WE, and thus awareness of WE among surgeons and emergency physicians is necessary. Adequate minerals and vitamin replacement, gradual weight reduction, and vomiting reduction significantly decrease the risk of WE after bariatric surgeries. Prompt treatment initiation is needed in patients suspected to have WE. After restrictive surgical procedures, nutritional deficiencies are easily preventable by early replacement. In fact, the levels of vitamins, especially B1, B6, and B12, should be determined before surgery, and replacement should be immediately initiated. When response to thiamine plateaus, oral supplementation should be provided until the patient can successfully abstain from alcohol consumption.

Data availability statement

Data sharing is not applicable for this article, as no new data were created or analyzed.

Author contributions

Conceptualization: E.H.; methodology/literature review: E.H., K.F., N.E., and A.R.; writing – original draft preparation: N.E. and A.R.; writing – review & editing: E.H. and A-N.E.

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