

Neha D. Shah, MPH, RD, CNSC, CHES, Series Editor
Elizabeth Wall, MS, RDN-AP, CNSC, Series Editor

Recognizing Thiamine Deficiency: Keeping Patients Safe and Clinicians Out of Court



Carol Rees Parrish



Neeral Shah



Kate Willcutts

Considerable effort has gone into identifying and diagnosing malnutrition in recent years, and along with that, recognizing those at risk for refeeding syndrome (RS). Less attention has been given to Wernicke's Encephalopathy (WE) despite the rising number of cases, not only reported in the literature, but court cases as well. Court cases have revealed that clinicians are failing to identify malnutrition, and as a result, missing the signs and symptoms of WE with devastating consequences. The purpose of this review is to discuss those at risk for both RS and WE, and, by definition, the malnutrition that overlaps both entities. In addition, frequent findings in court cases will be shared to help clinicians better identify and treat patients at substantial risk for malnutrition, RS, and WE in order to keep patients safe and clinicians out of court.

INTRODUCTION

Refeeding Syndrome (RS) and Wernicke's Encephalopathy (WE) are serious complications that can occur with the initiation and consistent delivery of nutrients in malnourished individuals without adequate guardrails (attention to appropriate practice guidelines). RS is the metabolic response to consistent nutrient provision in a malnourished patient leading to hypokalemia, hypophosphatemia,

and hypomagnesemia and the potentially severe consequences those electrolyte shifts can bring.^{1,2} WE is a severe neurological syndrome due to thiamine deficiency that too often goes unrecognized, underdiagnosed, and undertreated (Table 1).^{3,4}

First, however, one must identify the malnourished patient that should subsequently trigger concern for RS and WE. Court cases

Carol Rees Parrish, MS, RDN, GI Nutrition Support Specialist, Charlottesville, VA; Neeral Shah, MD, Professor of Medicine GI Fellowship, Program Director Director, Academy for Excellence in Education Division of Gastroenterology and Hepatology, UVA Health, Charlottesville, VA; Kate Willcutts, DCN, RDN, CNSC, FASPEN, Clinical Nutrition Manager, VA Western Colorado Health Care System, Grand Junction, CO

have repeatedly found that registered dietitian nutritionists (RDN) in particular have failed to meet the American Society of Parenteral Nutrition (ASPEN)/The Academy of Nutrition and Dietetics (AND)’s consensus standards for nutritional assessment⁵⁻⁷ by not:

- Documenting an adequate nutritional history.
- Documenting an adequate anthropometric evaluation with weight loss history over time and/or physical assessment.
- Reviewing prior admission/emergency department (ED) records that demonstrate persistent, or ongoing decline of nutritional health.
- Identifying the risk for developing micronutrient deficiencies.

Second, malnutrition which generally implies macronutrient “deficiency,” regardless of etiology, rarely appears in isolation from micronutrient deficiency, particularly thiamine (due to its short half-life and limited body stores). Although clinicians are aware of the need for thiamine in the setting of RS, many clinicians have a low index of suspicion for thiamine deficiency in other settings, delaying necessary treatment that can result in the development of WE and its potentially devastating consequences, or worse, Korsakoff’s Syndrome (Table 1). The importance of the clinician’s role

in early identification and treatment of patients at risk for both RS and WE cannot be overstated and is the focus of this article in order to avoid serious complications and subsequent malpractice claims.

About Thiamine

Thiamine (vitamin B1), a renally excreted, inexpensive water-soluble vitamin, has limited body stores of approximately 30 mg and a short half-life of 1-12 hours.⁸ Thiamine will last only 9-18 days in well-nourished individuals,⁹ but will deplete sooner if stores are insufficient or have been drawn upon without replenishment (e.g., 5% dextrose [D5W] infusion or parenteral nutrition [PN] administration without added thiamine or multivitamin infusion [MVI]). As thiamine is primarily absorbed in the upper jejunum, and to some extent the duodenum, consideration of the patient’s anatomy will be important when supplementing via the oral/enteral route.

Thiamine is a critical, rate-limiting cofactor to several enzymes involved in carbohydrate metabolism and can be rapidly depleted when carbohydrate is infused or ingested as the need for thiamine pyrophosphate (TPP) increases (such as in refeeding a malnourished patient). Thiamine deficiency exhausts supply to these enzymes, resulting in decreased adenosine triphosphate (ATP) synthesis, oxidative damage, and cell death; metabolic acidosis can also occur, reflected by an

Table 1. Signs and Symptoms of Wernicke’s Encephalopathy (“Thiamine-Deficient Encephalopathy”)^{32,33,34}

Nervous System Involvement (Dry Beriberi)
<ul style="list-style-type: none"> ◆ Wernicke’s Encephalopathy – (acute phase & reversible): <ul style="list-style-type: none"> • Classic triad: altered mental status (delirium, confusion, drowsiness), gait ataxia, ocular signs (nystagmus) – only 16% - 30% of patients with Wernicke’s present with all three • Also, fatigue, irritability, decreased reflexes, tingling sensation (arms & legs), blurred vision, change in mobility • Consider WE in any patient with unexplained delirium in the ICU ◆ Korsakoff Syndrome – (chronic phase & irreversible) <ul style="list-style-type: none"> • Permanent mental impairment (memory loss, amnesia, tremor, coma, disorientation, and vision problems)
Cardiac involvement (Wet Beriberi)
<ul style="list-style-type: none"> • Acute high-output cardiac failure with peripheral edema
Gastrointestinal involvement (GI Beriberi)
<ul style="list-style-type: none"> • Abdominal pain (potential clue: elevated serum lactate), nausea, emesis
Major barrier to pt diagnosis → a low index of suspicion by clinicians

Table 2. Thiamine Considerations for the Clinician

<p>Absorption</p> <ul style="list-style-type: none"> • Duodenum and proximal jejunum (will your patient be affected based on anatomy?) • Active alcohol consumption may alter oral thiamine absorption²⁶ <p>Excretion</p> <ul style="list-style-type: none"> • Losses are significantly increased up to 2 times baseline by loop diuretics⁹ • Renal replacement therapies also significantly decrease plasma thiamine <p>Treatment considerations</p> <ul style="list-style-type: none"> • Short half-life – 1-12 hours⁸ • Note: Oral gummy multivitamins rarely contain thiamine due to its offensive taste (Table 5) • IV thiamine has a 2-hour half-life, hence, to maintain blood levels to treat Wernicke’s, 2-4 doses per day are needed to achieve rapid correction by steep plasma: CNS gradient³⁵ • Administer IV thiamine slowly (over 30 minutes), diluted in 100mL of normal saline as it is very painful otherwise⁴
--

elevation in serum lactate concentration.^{8,10} For thiamine considerations for clinicians, see Table 2.

Refeeding Syndrome and Wernicke’s Encephalopathy: A Continuum

While several national and international nutrition societies have focused on identifying and preventing RS in recent years, WE has not garnered such attention. Unfortunately, too many clinicians are under the impression that WE is limited to those with alcohol use disorder, yet a myriad of other patient populations are also at risk.¹¹⁻¹⁷ In fact, RS and WE often share the same high-risk groups and may occur concurrently^{4,11-17} (Table 3). Two major warning signs present in almost all reported WE cases are extreme weight loss and vomiting.¹⁶ Many associations have published guidelines recommending thiamine repletion for RS;^{1,18,19} however, clinicians seldom realize that beyond carbohydrate metabolism, if WE is lurking, much higher thiamine doses are required (Table 4).

Wernicke’s Encephalopathy (“Thiamine-Deficient Encephalopathy”)

WE is an acute medical neurological emergency, that if left untreated, can progress to chronic Wernicke–Korsakoff syndrome, leading to impaired memory and cognitive functions, and coma and death in severe cases. It has been estimated that 80% of WE cases go undetected,³ likely due to the fact that the classic signs and symptoms may

not all be present (Table 1). In fact, WE classically presented as a triad of global confusion, gait ataxia, and ophthalmoplegia, yet this triad is only seen in 16%-20% of cases.¹⁴ The current dominant paradigm that WE only appears in those who have alcohol use disorder needs to change in order to prevent the rising cases and consequences of WE. Extensive case reports in patients with conditions other than alcohol use disorder highlight this fact, in particular, the post bariatric surgery patient population.¹⁵ Similar case examples from these and other populations resulting in lawsuits due to inadequate early treatment for WE will be addressed later in this article.

Why Does Thiamine Deficiency (and Wernicke’s) Elude Clinicians:¹⁰

Thiamine deficiency is often missed for several reasons: 1) signs and symptoms of thiamine deficiency can vary and mimic those of unrelated disorders; 2) patients may have micronutrient deficits including a thiamine deficiency in the setting of a secondary medical issue that steers the clinician in a different direction; 3) results of serum thiamine testing take up to 7-10 days, delaying not only the diagnosis, but timely treatment; 4) the best biomarker, as well as normal range cutoffs, have yet to be determined; 5) controversy exists regarding the best treatment regimen for WE as the best universal treatment guidelines have yet to be established.²⁰

Table 3. Patients at Risk for Refeeding Syndrome and/or Wernicke’s Encephalopathy^{2,16,36-38}

Patients at Risk for Refeeding and/or Wernicke’s
<ul style="list-style-type: none"> ◆ Nutrient deficit/significant weight loss: <ul style="list-style-type: none"> • Chronic, poor oral intake for ANY reason • Food insecurity, homelessness, refugees • Eating disorders/malnutrition/ underfeeding • Malabsorption syndromes • Dysphagia/esophageal disorders • Prolonged fasting/NPO post-op for > 7 days • Chronic alcohol or drug use disorder • Morbid obesity with significant weight loss • Oncology patients • Persistent diabetic ketoacidosis, or non-ketotic, hyperosmolar state ◆ Alcohol misuse/withdrawal <ul style="list-style-type: none"> • Head & neck cancer patients (high alcohol misuse) ◆ Anyone with nausea/vomiting x 2 weeks or greater <ul style="list-style-type: none"> • Recurrent visits to emergency departments for dehydration from nausea/vomiting and D5-containing IV solutions started @ high infusion rates without MVI or thiamine added • Hyperemesis gravidarum • Bariatric surgery with excessive diarrhea/vomiting/weight loss • Gastroparesis • Esophageal or pyloric stricture, gastric outlet obstruction, etc. ◆ PN-dependent patients not infusing MVI due to intolerance or supply shortages ◆ Unexplained heart failure or lactic acidosis ◆ Chronic congestive heart failure on diuretics ◆ Psychiatric illness with weight loss

Diagnosing and Preventing Wernicke’s Encephalopathy

Diagnosing WE begins with a high level of suspicion in the right clinical context: i.e., a patient who has lost a significant amount of weight, persistent vomiting for 2 weeks or more, or consumes an excessive amount of alcohol. Significant or severe weight loss has been defined as:^{5,7}

- Significant (1-2%) / severe (>2%) over 1 week
- Significant (5%) / severe (>5%) over 1 month
- Significant (7.5)% / severe (>7.5%) over 3 months
- Significant (10%) / severe (>10%) over 6 months
- Significant (20%) / severe (>20%) over 1 year

However, clinical judgment must also be used and not rely on the numbers alone. For example, if a patient unintentionally loses 4.8% in 3 weeks, it is still considered a significant loss of weight.

Laboratory Testing

Laboratory testing for a serum thiamine level should never delay thiamine treatment – if a thiamine assay is drawn, thiamine dosing should follow immediately after. If laboratory testing is pursued, use whole blood thiamine (direct measurement of thiamine and its phosphate esters). Results can take up to 7 - 10 days, therefore, if Wernicke’s is suspected, it is important to start treatment with thiamine right after the lab draw. Serum or plasma thiamine testing suffer from poor sensitivity and specificity; < 10% of blood thiamine is contained in plasma and is affected by recent oral intake or IV infusion. Therefore, whole blood thiamine should

be ordered instead. Of note, there is no established lab value at which WE is diagnosed.

Imaging

Use of magnetic resonance imaging (MRI) to detect WE has a sensitivity of only 53%, hence, MRI scans can only reliably rule-in suspected cases of WE.²¹ CT imaging is not suitable to use in patients with suspected WE.²² Brain abnormalities are quickly reversed after thiamine treatment, so an MRI should be performed prior to thiamine administration, but only if the MRI can be done right away, or precious time can be lost as treatment is of the highest priority in these cases. It has been suggested that the brain injury is related to focal lactic acidosis, blood–brain barrier disruption, neural cell excitotoxicity, inflammation, or inadequate ATP at the cellular level.³

Due to the alarming lack of recognition of WE, clinicians must change their differential diagnosis to not only include RS, but also note that WE may also be present in order to prevent the negative clinical, and potentially legal consequences.²³ In order to improve identification and subsequent treatment of WE, clinicians must have a high level of suspicion for thiamine deficiency when at risk

patients present for care (see examples below).

What do the following cases have in common?

- ✦ 42-year-old male presents with ongoing nausea and vomiting post gastric sleeve bypass surgery; frequent ED admissions treated with intravenous (IV) antiemetics and D5W at 100mL/hour;
- ✦ 27-year-old pregnant female presents with persistent hyperemesis for the past 5 weeks;
- ✦ 58-year-old male presents with almost daily nausea and vomiting due to a pyloric stenosis;
- ✦ 36-year-old female admitted with ongoing nausea and vomiting over 2 months due to diabetic gastroparesis;
- ✦ 32-year-old female admitted for an endoscopic retrograde cholangiopancreatography (ERCP) to remove gallstone complicated by acute pancreatitis, unable to eat despite attempts over the course of 5 weeks; D5W initiated, then peripheral parenteral nutrition, then central PN, all without thiamine supplementation;
- ✦ 63-year-old male with daily excessive

Table 4. Thiamine Supplementation Guidelines for Wernicke’s Encephalopathy and Refeeding Syndrome^{1,4,25,39,40}

Author/Association	Recommended Supplementation/Dosing Guideline
European (EFNS) Wernicke’s Guidelines (Galvin 2010)	<ul style="list-style-type: none"> • At risk: 200mg IV, TID before any carbohydrate is given • Signs of WE: 500mg IV TID x 3 days, then 250mg daily x 5 days
Mechanick 2019	Repletion dose for thiamine deficiency varies based on route of administration and severity of symptoms: <ul style="list-style-type: none"> • Oral therapy: 100 mg 2-3 times daily until symptoms resolve • IV therapy: 200 mg 3 times daily to 500 mg once or twice daily for 3-5 d, followed by 250 mg/d for 3-5 d or until symptoms resolve, then consider treatment with 100 mg/d orally, indefinitely, or until risk factors have been resolved • IM therapy: 250 mg once daily for 3-5 d or 100-250 mg monthly Magnesium, potassium, and phosphorus should be given simultaneously to patients at risk for refeeding syndrome.
ASPEN Refeeding Consensus (da Silva 2020)	<ul style="list-style-type: none"> • 100mg before feeding or initiating IV dextrose in pts at risk • 100mg/day x > 5–7 days in pts with severe starvation/chronic alcoholism/ other high-risk/signs of deficiency
Dingwall 2022	<ul style="list-style-type: none"> • No clear benefit between 100mg, 300mg, 500mg IV, TID in at risk vs. symptomatic for WE
Wijnia 2022	<ul style="list-style-type: none"> • 250mg IV or IM, daily x 3-5 days

Note: Guidelines are based on expert opinion, not randomized, controlled trials

alcohol intake and 20 lb. weight loss over past month is admitted for mandibular resection due to carcinoma

The unifying presentation in all of the above cases was one or more of the following: ongoing vomiting for > 2 weeks, inability to eat normally over an extended period of time, and significant, unintentional weight loss. Weights were often not obtained or just “self-reported” by patients, thus frequently inaccurate. ED visits were common, and subsequent hospitalizations were just long enough to relieve the symptoms of nausea/vomiting, but not long enough to generate a nutrition consult from an admission nutrition screen (if one was even done, especially in an ED), or one directly from a provider. Nor was empiric thiamine treatment given. If a nutrition consult was completed in these cases, the focus was on getting the patient to consume adequate calories and protein, rather than exploring the patient’s micronutrient adequacy. It is worth noting that rare is the patient who was previously taking a vitamin/mineral supplement, whether as part of standard post bariatric care or for other reasons, who continues vitamin/mineral

supplementation when they stop eating. Further perpetuating the potential for WE, treatments often include a carbohydrate containing hydration fluid such as D5W in response to dehydration from ongoing vomiting. D5W is usually ordered at 100mL/hour, without additional IV micronutrients, thus consuming any serum thiamine remaining along with other micronutrient stores.^{22,24}

Of note, the expected weight loss in the post bariatric surgery patient is often quoted as 2-4 lb/week, or 8-16 pounds per month. The American Society of Metabolic and Bariatric Surgery 2019 guidelines list expected weight loss post-surgery based on the type of bariatric surgery.²⁵ They cite the target weight loss from the original total body weight (not excess weight) as: 20-25% for laparoscopic adjustable gastric banding; 25-30% for sleeve gastrectomy; 30-35% for Roux-en-Y gastric bypass and 35-45% for biliopancreatic diversion with duodenal switch. However, limited literature exists addressing what constitutes an “excessive” loss of weight (too much, too fast) after bariatric surgery that would spark a warning to clinicians to investigate further for possible malnutrition, RS, and WE. One systematic review of bariatric surgical procedures found that those patients admitted with WE had a median weight loss from surgical procedure to admission of 35 kg and a weight loss rate of 0.44 kg/day (~ 1 lb/day).²² Hence, if this amount of weight loss is associated with WE, until further data is available, to prevent WE in the first place, a weight loss less than this amount should prompt further assessment at this time.

Treatment

WE prevention is simple. It is repletion of thiamine, an inexpensive water-soluble vitamin. Advantages of prompt administration of thiamine, in adequate doses (Table 4), are that it leads to improvement in ocular signs within hours to days, vestibular function and gait ataxia during the second week, and confusion subsides over days to weeks.²⁶ However, based on the findings reported in this article, it behooves clinicians to use thiamine judiciously, especially in the IV form, to prevent shortages already experienced at individual facilities as well as nationally vs. treat every patient “just in case.”

Table 5. Thiamine Content of a Few Selected Gummy Vitamins (DRI = 1.2 mg)

- Contains small amount of thiamine**
- Smarty Pants® Women’s Formula (only 0.12 mg in 6 gummies)
- Vitamin or vitamin/mineral gummy combinations that do not have thiamine:**
- Kirkland™ Adult multivitamin Gummies
 - Vitafusion™ MultiVites
 - Vitafusion™ Men’s Multi
 - Vitafusion™ Women’s Multi
 - Kirkland™ Children’s Daily Multivitamin Gummies
 - Smarty Pants® Kids formula Gummies
 - L’ilCritters™ Gummy Vites
 - One A Day® Women’s VitaCraves
 - One A Day® Women’s Prenatals
 - Centrum® Adults Multigummies
 - Centrum® Men 50+ Multigummies
 - Nature’s Promise® Prenatal Multivitamin
 - Nature’s Promise® Men’s Multivitamin
 - Nature’s Promise® Women’s Multivitamin

Other Nutrients Required for Thiamine Utilization

Magnesium, a necessary cofactor in carbohydrate metabolism, if depleted, impedes conversion of

thiamine to TPP, further accelerating thiamine deficiency. Folate, similarly, is required to regenerate TPP. Although it is not consumed in the process, additional folate may be required.²⁷ The examples

Table 6. Findings from Attorneys Trying Wernicke’s that Clinicians Need to Change

<p>1. Nutrition Screening – Is It Working?</p> <ul style="list-style-type: none"> ◆ Ensure that there are proper institutional triggers for timely evaluation and follow up by a registered dietitian nutritionist (RDN) when a pt is admitted with persistent nausea/vomiting, oral nutrition intolerance, and significant weight loss history. <p>2. Failing to Identify Malnutrition:</p> <ul style="list-style-type: none"> ◆ By not meeting ASPEN/AND’s consensus standards⁵⁻⁷ for nutritional assessment including failure to: <ul style="list-style-type: none"> • Document an adequate nutritional history. • Document an adequate anthropometric evaluation including weight loss history and physical assessment. • Look at prior admission/ED records that may show nutritional decline. • Document risk for development of permanent neurological impairments of micronutrient deficiencies. <p>3. Relying on other providers’ review of nutrition progress notes rather than communicating and collaborating directly with other providers when orders for vitamin levels and empiric vitamin repletion are warranted to prevent adverse outcomes.</p> <p>4. NOT Relying on other providers’ progress notes that document patient showing signs of micronutrient deficiencies such as acute neurological symptoms.</p> <p>5. Failing to: Acknowledge Ongoing Problems with Poor Oral Intake</p> <ul style="list-style-type: none"> ◆ Nutrition interventions which repeatedly recommend supplements that the patient either cannot, or will not tolerate, i.e.: <ul style="list-style-type: none"> • Ensure, Boost, or oral multivitamins and minerals without confirming the patient has been able to take or tolerate them when nausea and vomiting persist. ◆ Nutrition notes that continue to recommend: <ul style="list-style-type: none"> • “Advance diet as tolerated,” • “Honor food preferences,” or • “Oral nutrition supplements (Ensure or Boost) as tolerated.” <ul style="list-style-type: none"> ➤ In such cases, the RDN has failed to confer with physicians and recommend the addition of enteral, parenteral, or vitamin/mineral supplementation. <p>6. Failing to:</p> <ul style="list-style-type: none"> ◆ Evaluate and treat micronutrient deficiencies and focus only on macronutrient deficiencies. ◆ Recommend earlier B vitamins, including IV thiamine repletion, for patients with persistent nausea/vomiting and oral nutrition intolerance. ◆ Follow ASPEN Parenteral Nutrition Safety Consensus Recommendations stating^{41,42} <ul style="list-style-type: none"> • The clinician responsible for prescribing and/or charting the PN macronutrient formulation(s) is ultimately responsible for prescribing the IV micronutrients to ensure complete nutrition is provided. <ul style="list-style-type: none"> ➤ The highest risk regarding routine doses is not delivering them with PN. • Multivitamins (MVI) shall be prescribed daily in PN admixtures. • When MVI products are not available, thiamine, ascorbic acid, pyridoxine, and folic acid shall be prescribed daily. ◆ If RDNs are only responsible for recommending macronutrients in PN at a particular facility, because “the pharmacist is responsible for electrolytes, MVI, and trace elements,” make sure this is in writing in hospital policy and procedures and consider a smart phrase for this.

Adapted from the version compiled by Steven R. Davis, J.D., Davis & Davis Law Firm, Houston, TX, and used with permission.

of the role of magnesium and folate demonstrate that in caring for the malnourished patient, it is important that the clinician presume pan-nutrient deficiency and ensure that all vitamins and minerals are adequately provided in the repletion process. This can be achieved by providing a complete vitamin and mineral supplement. Additional thiamine should also be given in the case of RS and WE (Table 4). Of importance for the clinician and patient, popular chewable gummy multivitamin supplements, rarely contain thiamine due to the objectionable taste (Table 5).

Legal Ramifications

As the problem of WE grows, a sub-culture of lawyers who specialize in this diagnosis has emerged. A quick internet search reveals the vast scope of law firms marketing their services to those who have suffered WE due to inadequate medical treatment. To provide optimal patient care and avoid such malpractice lawsuits, it behooves the clinician to become familiar with common errors leading to lawsuits. Informal communications with an attorney specializing in Wernicke’s cases (who has tried over 27 cases so far) resulted in a list of common errors made by clinicians (Table 6). Recognizing these errors provides a road map for change for clinicians to prevent WE and subsequent lawsuits.

Special Issue: Bariatric Vitamin/Mineral Supplementation

Post bariatric surgery patients are one of the more prevalent populations in which WE has arisen including all types of bariatric surgery, even vertical sleeve gastrectomy,^{15,28} prompting numerous court cases. Since post bariatric surgery patients are at high risk of micronutrient deficiency, it is incumbent upon the clinician during the assessment process to question whether they are still taking their prescribed vitamin and mineral supplements,²⁹ which ones, and how much. If a patient has stopped taking the supplements, the clinician should determine when they stopped and for how long. It is not uncommon for some patients to stop these supplements, not recognizing their importance. Some of the documented reasons why patients stop their supplementation are listed below:^{30,31}

- Did not think they still needed them
- Did not like taking them
- Could not find them
- Did not like the taste
- Could not remember to take them
- Could not afford them

To help keep post bariatric surgery patients

Table 7. Sample Contract with Bariatric Patients Regarding Vitamin/Mineral Supplement Adherence²⁹

I understand that I am required to take the following vitamins and minerals (dosing may vary amongst institutions) as specified for the rest of my life (or until my bariatric surgeon or PCP advises me otherwise), example:

- Initials: _____ 50 mg thiamine
- Initials: _____ 500mg B12
- Initials: _____ 3000 units vitamin D3
- Initials: _____ Complete vitamin and mineral supplement

If there comes a time that I am not taking the prescribed vitamins and minerals for any reason, I will contact my PCP or bariatric surgeon to let them know.

Sign & Print name _____ Date _____

safe, at every hospital or clinic encounter, clinicians should:

1. Inquire about and document the following:
 - ◆ What vitamins and minerals is the patient taking - how often, and in what doses?
 - ◆ The patient's history of taking these supplements, including whether the intake is consistent or sporadic. If sporadic, how long do they go without taking them?
 - ◆ For patients not taking vitamins or minerals, inquire why this is the case, and make a new plan with the patient to ensure adherence to the prescribed regimen.
2. Create a smart phrase (in electronic medical record) stating what was specifically reviewed with the patient in terms of

the importance of vitamin and mineral adherence and, for each vitamin and mineral, the prescribed dose and frequency of administration.

3. Finally, it may be prudent to ask every patient choosing to undergo bariatric surgery to sign a contract that is kept in the patient's chart addressing vitamin/mineral supplementation (Table 7).

CONCLUSION

Much attention has been paid to identifying and diagnosing malnutrition; and in addition, recognizing those at risk for RS. WE continues to be thought of, incorrectly, as a complication solely of too much alcohol intake combined with poor nutrition. WE can be found in any patient, not just bariatric patients presenting with prolonged nausea and vomiting or those at risk

Table 8. Actionable Interventions the Clinician Can Do Now

1. Ensure the nutrition screening tool is working, if not, change it to better capture high-risk patients.
 - If it is not being filled out, address that with nursing, or hire diet technicians whose sole job is to screen new admissions.
 - Consider the use of artificial intelligence or dietetic technicians for more focused admission nutrition screening, etc.
2. Consider creating a smart phrase for both refeeding syndrome and Wernicke's such as:
 - 100mg thiamine daily (tab or IV) x 3-5 days (if refeeding).
 - 500mg thiamine IV, TID x 3 days (if Wernicke's), followed by 250mg daily vs. BID, IV (or enteral) x 5 more days.
 - Start a complete vitamin/mineral supplement daily x 5 days, then reevaluate.
 - Check refeeding labs (basic metabolic, magnesium, phosphorus) BID x 3 days, then reevaluate.
3. Obtain accurate weight history, not just the current admission, but from 1, 3, and 6 months prior → ensure accurate weights are done when patient is admitted.
4. If a patient is severely malnourished, assume the patient at risk for both RS and WE (Table 3).
5. Did the patient present with nausea/vomiting for > 2 weeks?
 - Include ED visits, outside hospital admissions, etc.
6. Be specific in your nutrition recommendations:
 - 15-20kcal/kg to start (include ALL calorie sources including D5 drips).
7. Refeeding: start thiamine, 100mg daily (tab or IV) x 3-5 days – including with all D5 IV fluids, PN, enteral feedings.
8. Wernicke's: 500mg thiamine IV, TID x 3 days; then, 250mg daily vs. BID, IV (or enteral) x 5 more days.
9. Ensure adequate magnesium status.
10. Start complete vitamin and mineral supplement daily x 5 days – find out the actual names in the hospital formulary EMR and recommend by name to providers for ease of ordering.
11. Check refeeding labs (basic metabolic, magnesium, phosphorus) BID x 3 days, then reevaluate.
12. If appropriate → good glycemic control to ensure nutrient utilization and protect endogenous stores.

Table 9. Additional Resources

- ◆ Video recording – available free:
 - "Advanced Practice Provider (APP) Lecture Series - Vitamin B1: The Management and Treatment in Clinical Practice - American Society for Metabolic and Bariatric Surgery" (asmbs.org); recorded 4/20/23 presented by Emma J. Patterson, MD, FRCSC FACS FASMBS: asmbs.org/videos/advanced-practice-provider-app-lecture-series-vitamin-b1-the-management-and-treatment-in-clinical-practice/
- ◆ What nystagmus looks like: youtube.com/watch?v=HXOaRGNnijU

for RS and should be treated with thiamine and other micronutrients accordingly. Sadly, litigation of WE cases continues to rise displaying the fact that clinicians are failing to identify malnutrition, RS, and WE with dreadful consequences. It is clear that an evaluation of why nutritional screening practices are failing and patients with severe malnutrition and nutrient deficiencies are not being recognized is warranted. We owe it to our patients to keep them safe, and no clinician wants to find themselves in court for malpractice. See Table 8 for actionable suggestions that can begin to start addressing this pressing issue; Table 9 provides additional resources of interest for clinicians. ■

References

1. da Silva JSV, Seres DS, Sabino K, et al; Parenteral Nutrition Safety and Clinical Practice Committees, American Society for Parenteral and Enteral Nutrition. ASPEN Consensus Recommendations for Refeeding Syndrome. *Nutr Clin Pract.* 2020;35(2):178-195.
2. Krutkyte G, Wenk L, Odermatt J, et al. Refeeding Syndrome: A Critical Reality in Patients with Chronic Disease. *Nutrients.* 2022;14(14):2859.
3. Kohnke S, Meek CL. Don't seek, don't find: The diagnostic challenge of Wernicke's encephalopathy. *Ann Clin Biochem.* 2021;58(1):38-46.
4. Wijnia JW. A Clinician's View of Wernicke-Korsakoff Syndrome. *J Clin Med.* 2022;11(22):6755.
5. Malone A, Hamilton C. The Academy of Nutrition and Dietetics/the American Society for Parenteral and Enteral Nutrition consensus malnutrition characteristics: application in practice. *Nutr Clin Pract.* 2013;28(6):639-50.
6. The Academy of Nutrition and Dietetics New Revised 2024 Scope and Standards of Practice for the Registered Dietitian Nutritionist; pp 42-45.
7. White JV, Guenter P, Jensen G, et al. Academy Malnutrition Work Group; ASPEN Malnutrition Task Force; ASPEN Board of Directors. Consensus statement: Academy of Nutrition and Dietetics and American Society for Parenteral and Enteral Nutrition: characteristics recommended for the identification and documentation of adult malnutrition (undernutrition). *JPEN J Parenter Enteral Nutr.* 2012;36(3):275-283.
8. Marrs C, Lonsdale D. Hiding in Plain Sight: Modern Thiamine Deficiency. *Cells.* 2021;10(10):2595.
9. Frank LL. Thiamin in Clinical Practice. *JPEN J Parenter Enteral Nutr.* 2015;39(5):503-20.
10. Smith TJ, Johnson CR, Koshy R, et al. Thiamine deficiency disorders: a clinical perspective. *Ann N Y Acad Sci.* 2021;1498(1):9-28.
11. Antel K, Singh N, Chisholm B, et al. Encephalopathy after persistent vomiting: Three cases of non-alcohol-related Wernicke's encephalopathy. *S Afr Med J.* 2015;105(6):442-3.
12. Mangione D, Vassiliadis A, Gullo G, et al. Wernicke Syndrome: Case Report and Literature Review of Contributing Factors-Can Malpractice Dynamics Be Identified? *J Clin Med.* 2024;13(3):716.
13. Mifsud F, Messenger D, Jannot AS, et al. Clinical diagnosis, outcomes and treatment of thiamine deficiency in a tertiary hospital. *Clin Nutr.* 2022;41(1):33-39.
14. Okafor C, Nimmagadda M, Soim S, et al. Non-alcoholic Wernicke encephalopathy: great masquerader. *BMJ Case Rep* 2018;11:e227731.
15. Oudman E, Wijnia JW, van Dam M, et al. Preventing Wernicke Encephalopathy After Bariatric Surgery. *Obes Surg.* 2018;28(7):2060-2068.
16. Oudman E, Wijnia JW, Oey MJ, et al. Wernicke-Korsakoff syndrome despite no alcohol abuse: A summary of systematic reports. *J Neurol Sci.* 2021;426:117482.
17. Scalzo SJ, Bowden SC, Ambrose ML, et al. Wernicke-Korsakoff syndrome not related to alcohol use: a systematic review. *J Neurol Neurosurg Psychiatry.* 2015;86(12):1362-8.
18. Friedli N, Stanga Z, Culkin A, et al. Management and prevention of refeeding syndrome in medical inpatients: An evidence-based and consensus-supported algorithm. *Nutrition.* 2018;47:13-20.
19. Mehanna HM, Moledina J, Travis J. Refeeding syndrome: what it is, and how to prevent and treat it. *BMJ.* 2008;336(7659):1495-8.
20. Cantu-Weinstein A, Branning R, Alamir M, et al. Diagnosis and treatment of Wernicke's encephalopathy: A systematic literature review. *Gen Hosp Psychiatry.* 2024;87:48-59.

21. Antunez E, Estruch R, Cardenal C, et al. Usefulness of CT and MR imaging in the diagnosis of acute Wernicke's encephalopathy. *AJR Am J Roentgenol.* 1998;171(4):1131-1137.
22. Aasheim ET. Wernicke encephalopathy after bariatric surgery: a systemic review. *Ann Surg.* 2008;248(5):714-720.
23. Hershkowitz E, Reshef A, Munich O, et al. Thiamine deficiency in self-induced refeeding syndrome, an undetected and potentially lethal condition. *Case Rep Med.* 2014;2014:605707.
24. Li L, Shin J-H, Sharma K, et al. Wernicke encephalopathy after sleeve gastrectomy. *AIM Clinical Cases.* 2024;3:e230116.
25. Mechanick JI, Apovian C, Brethauer S, et al. Clinical Practice Guidelines for the Perioperative Nutrition, Metabolic, and Nonsurgical Support of Patients Undergoing Bariatric Procedures - 2019 Update: Cosponsored by American Association of Clinical Endocrinologists/American College of Endocrinology, The Obesity Society, American Society for Metabolic & Bariatric Surgery, Obesity Medicine Association, And American Society of Anesthesiologists - Executive Summary. *Endocr Pract.* 2019;25(12):1346-1359.
26. Patel S, Topiwala K, Hudson L. Wernicke's Encephalopathy. *Cureus.* 2018;10(8):e3187.
27. McLean J, Manchip S. Wernicke's encephalopathy induced by magnesium depletion. *Lancet.* 1999;353(9166):1768.
28. Tang L, Alsulaim HA, Canner JK, et al. Prevalence and predictors of postoperative thiamine deficiency after vertical sleeve gastrectomy. *Surg Obes Relat Dis.* 2018;14(7):943-950.
29. Parrott J, Frank L, Rabena R, et al. American Society for Metabolic and Bariatric Surgery Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient 2016 Update: Micronutrients. *Surg Obes Relat Dis.* 2017;13:727-741.
30. Ahmad DS, Esmadi M, Hammad H, et al. Malnutrition secondary to non-compliance with vitamin and mineral supplements after gastric bypass surgery: What can we do about it? *Am J Case Rep.* 2012;13:209-213.
31. Smelt HJM, Pouwels S, Smulders JF, et al. Patient adherence to multivitamin supplementation after bariatric surgery: a narrative review. *J Nutr Sci.* 2020;9:e46.
32. Crook MA, Sriram K. Thiamine deficiency: The importance of recognition and prompt management. *Nutrition.* 2014;30(7-8):953-954.
33. Nakamura ZM, Tatreau JR, Rosenstein DL, et al. Clinical Characteristics and Outcomes Associated with High-Dose Intravenous Thiamine Administration in Patients with Encephalopathy. *Psychosomatics.* 2018;59(4):379-387.
34. Peterson BD, Stotts MJ. Beyond the Banana Bag: Treating Nutritional Deficiencies of Alcohol Withdrawal Syndrome. *Practical Gastroenterol.* 2021;June(6):48-58.
35. Donnino MW, Vega J, Miller J, et al. Myths and misconceptions of Wernicke's encephalopathy: what every emergency physician should know. *Ann Emerg Med.* 2007;50(6):715-21.
36. Erick M. Gestational malnutrition, hyperemesis gravidarum, and Wernicke's encephalopathy: What is missing? *Nutr Clin Pract.* 2022;37(6):1273-1290.
37. Marziliano A, Teckie S, Diefenbach MA, et al. Alcohol-related head and neck cancer: Summary of the literature. *Head Neck.* 2020;42(4):732-738.
38. Matz R. Parallels between treated uncontrolled diabetes and the refeeding syndrome with emphasis on fluid and electrolyte abnormalities. *Diabetes Care.* 1994;17(10):1209-13.
39. Galvin R, Bråthen G, Ivashynka A, et al. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. *Eur J Neurol.* 2010;17(12):1408-1418.
40. Dingwall KM, Delima JF, Binks P, et al. What is the optimum thiamine dose to prevent Wernicke's encephalopathy or Wernicke-Korsakoff syndrome? *Alcohol Clin Exp Res.* 2022;46(6):1133-1147.
41. Ayers P, Adams S, Boullata J, et al. ASPEN parenteral nutrition safety consensus recommendations. *JPEN J Parenter Enteral Nutr.* 2014;38(3):296-333.
42. Blaauw R, Osland E, Sriram K, et al. Parenteral Provision of Micronutrients to Adult Patients: An Expert Consensus Paper. *JPEN J Parenter Enteral Nutr.* 2019;43(suppl 1):S16.

