



Wernicke Encephalopathy: Nursing Implications in Diagnosis and Neurological Recovery-An Updated Review

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Abstract:

Background: Wernicke Encephalopathy (WE) is an acute neurological disorder primarily caused by thiamine deficiency. It commonly manifests in patients with chronic alcohol use but can also occur in other contexts such as malnutrition, hyperemesis gravidarum, and prolonged parenteral nutrition. WE is life-threatening, affecting both the central and peripheral nervous systems, and is often misdiagnosed or underdiagnosed due to its nonspecific presentation. Timely diagnosis and intervention are essential to preventing irreversible neurological damage.

Aim: This updated review aims to explore the nursing implications for the diagnosis and neurological recovery of patients with Wernicke Encephalopathy. It highlights the clinical features, underlying causes, and treatment strategies for effective management.

Methods: A comprehensive review of current literature was conducted, focusing on the clinical presentation, pathophysiology, risk factors, assessment methods, and treatment options for WE. Studies related to thiamine deficiency, diagnostic criteria, nursing management, and medical interventions were analyzed.

Results: The review confirms that thiamine deficiency remains the primary cause of WE, most commonly linked to chronic alcohol use. Diagnosis relies on the clinical triad of confusion, ataxia, and ophthalmoplegia, although these symptoms may not always present together. Thiamine supplementation, particularly

through parenteral administration, is crucial for the management of WE. However, persistent neurological deficits may develop, transitioning to chronic Korsakoff syndrome if not addressed promptly.

Conclusion: Early identification and intervention are crucial in managing Wernicke Encephalopathy. Nurses play a pivotal role in monitoring symptoms, administering treatments, and providing education to patients and caregivers. Timely thiamine therapy, along with management of associated deficiencies, is essential to optimize recovery and prevent long-term neurological damage.

Keywords: Wernicke Encephalopathy, thiamine deficiency, nursing management, neurological recovery, Korsakoff syndrome, alcohol use disorder, malnutrition, ataxia, ophthalmoplegia.

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Introduction:

Wernicke Encephalopathy (WE) is an acute neurological disorder that manifests through a clinical triad comprising ophthalmoparesis with nystagmus, ataxia, and confusion. This condition is life-threatening and results from thiamine deficiency, predominantly affecting both the peripheral and central nervous systems. It is essential to differentiate Wernicke encephalopathy from Korsakoff syndrome, a preventable neuropsychiatric disorder commonly suspected as a consequence of at least one episode of Wernicke's encephalopathy. Korsakoff syndrome is characterized by significant memory impairments, particularly deficits in both anterograde and retrograde memory, although immediate memory typically remains intact. In this disorder, patients exhibit preserved sensorium, but short-term memory is diminished, and they often fabricate stories while being fully conscious. Confabulations can be either spontaneous, which is typically observed in acute Wernicke's encephalopathy, or provoked, which is a common feature of chronic Korsakoff syndrome [1][2].

Nursing Diagnosis

Key nursing diagnoses for patients with Wernicke encephalopathy include deficient knowledge, confusion, deficits in mental status, nutritional deficiencies, impaired healing, and a heightened risk for injury and death. These diagnoses highlight the multifaceted nature of the condition and its profound impact on various aspects of the patient's health.

Causes

Thiamine deficiency is the primary cause of Wernicke encephalopathy, most commonly associated with chronic alcohol use disorder. Although alcohol-related thiamine deficiency is the leading cause, other contributing factors include severe malnutrition, hyperemesis gravidarum, prolonged parenteral nutrition, malignancies, immunodeficiency syndromes, liver diseases, hyperthyroidism, and severe anorexia nervosa. Chronic alcohol consumption impairs the absorption of thiamine from the intestine and may also involve genetic predispositions, inadequate dietary intake, reduced liver storage of thiamine, and concomitant nutritional deficiencies [3][2][4].

Wernicke encephalopathy (WE) is primarily caused by thiamine (vitamin B1) deficiency, which disrupts the normal functioning of the central and peripheral nervous systems. Thiamine is essential for glucose metabolism, and its deficiency impairs several enzymatic processes that are critical for neuronal function. The most common etiology of thiamine deficiency is chronic alcohol use, as alcohol interferes with thiamine absorption in the gastrointestinal tract, reduces its storage in the liver, and enhances its renal excretion. Alcoholism also frequently coexists with poor nutrition, further exacerbating thiamine deficiency. Other causes of WE include severe malnutrition, hyperemesis gravidarum, prolonged intravenous feeding without adequate thiamine supplementation, malabsorption syndromes, and conditions such as liver disease, hyperthyroidism, and anorexia nervosa. In some instances, WE can develop in patients with chronic illnesses or those undergoing intensive treatments, such as cancer patients on prolonged parenteral nutrition. Furthermore, genetic predisposition may also play a role in the development of WE, as some individuals may have impaired thiamine transport or metabolism.

Additionally, conditions associated with impaired thiamine absorption, such as Crohn's disease or bariatric surgery, increase the risk of WE. The pathogenesis of WE is multifactorial, with nutritional deficiencies, impaired thiamine absorption, and metabolic disturbances working synergistically to cause neuronal damage, particularly in the brain regions involved in memory, coordination, and eye movement regulation.

Epidemiology:

The prevalence of Wernicke encephalopathy (WE) is difficult to accurately determine due to its clinical presentation, which is often subtle and may be easily overlooked. Autopsy studies suggest a prevalence rate of 1% to 3%, though clinical diagnoses are frequently underestimated, and the true rate may be higher. The incidence of WE is most commonly associated with chronic alcoholism, which remains the leading risk factor. It is estimated that 10-20% of chronic alcoholics may develop WE, though this varies with the severity of alcohol dependence and nutritional status. The condition is also seen in individuals with other risk factors such as severe malnutrition, hyperemesis gravidarum, and gastrointestinal surgeries like bariatric procedures. WE occurs more frequently in developed countries due to the prevalence of alcohol use disorder and related malnutrition. However, in low- and middle-income countries, the incidence of WE is likely higher due to vitamin deficiencies, particularly in populations with limited access to balanced nutrition. Gender differences in the incidence of WE suggest a slightly higher prevalence in males, with a female to male ratio of approximately 1:1.7. However, there is no significant evidence linking WE to specific racial predispositions. As a life-threatening condition, WE often presents in acute stages, but the severity can range from mild symptoms to rapidly progressing encephalopathy, which complicates early diagnosis and intervention. Additionally, as the population of individuals with alcohol use disorders continues to rise globally, the incidence of WE is likely to increase, highlighting the need for timely recognition and intervention.

Risk Factors

The prevalence of Wernicke encephalopathy is primarily derived from autopsy studies, which report incidence rates between 1% and 3%. Clinical records-based studies show a lower prevalence, likely due to the condition being frequently overlooked or misdiagnosed. The incidence of WE is notably higher in developing countries due to prevalent vitamin deficiencies and malnutrition. The gender distribution for WE is approximately 1:1.7 (female to male ratio), with no definitive evidence suggesting that any particular racial group is predisposed to the condition [5].

Pathophysiology:

The pathophysiology of Wernicke encephalopathy (WE) is primarily attributed to thiamine deficiency, which leads to impaired cellular metabolism and neuronal dysfunction. Thiamine, a cofactor for several enzymes involved in glucose metabolism, is crucial for the production of ATP, the primary energy source for cells. When thiamine levels are deficient, the cellular machinery that supports energy production becomes impaired, particularly in high-energy-demand organs such as the brain. The most affected areas of the brain in WE are the mammillary bodies, the thalamus, and the brainstem, which play key roles in memory, motor control, and coordination. Damage to these areas results in the characteristic symptoms of WE, including confusion, ataxia, and ophthalmoplegia. The mechanism by which thiamine deficiency causes neuronal injury involves the disruption of enzymatic reactions in the pentose phosphate pathway and the citric acid cycle, leading to cellular energy failure. Furthermore, thiamine deficiency leads to the accumulation of oxidative stress and neuroinflammation, which exacerbates neuronal damage. In some cases, the absence of adequate thiamine causes demyelination and neuronal necrosis in affected regions of the brain. Additionally, the deficiency impairs the synthesis of neurotransmitters, including acetylcholine, further compromising cognitive and motor function. Over time, this results in both acute neurological symptoms and the potential for chronic cognitive deficits, such as those seen in Korsakoff syndrome. Although thiamine supplementation can reverse many of the acute symptoms, the damage to the brain, particularly in the chronic phase, can be permanent if not addressed early.

Assessment

Wernicke encephalopathy should be suspected in patients with a history of chronic alcohol abuse or any form of malnutrition, presenting with acute changes in mental status, ophthalmoplegia, ataxic gait, delirium, and hypotension. The classic triad of Wernicke encephalopathy includes altered mental status, ataxic gait, and ophthalmoplegia; however, up to 90% of patients may not present with all components of the triad. Ocular symptoms, particularly nystagmus, serve as hallmark signs of WE, and other oculomotor symptoms can involve the oculomotor, abducens, and vestibular nuclei, leading to conjugate gaze palsies. Ataxia, particularly broad-based gait, is also prominent in Wernicke encephalopathy, with some patients experiencing further deterioration to the point of being unable to walk. A comprehensive neurological examination, including cerebellar testing, is essential. Disorientation and altered sensorium are key features of encephalopathy, and some patients may exhibit hyperactive delirium, potentially exacerbated by alcohol withdrawal. In rare cases (less than 5%), patients may experience a severely depressed level of consciousness, progressing to coma and death. Additional warning signs include hyperthermia, hypotension, and peripheral neuropathy, often affecting the lower extremities, which manifests as distal sensory loss.

Evaluation

The evaluation of Wernicke encephalopathy requires a thorough patient history, a focused physical examination, and relevant laboratory investigations, including appropriate imaging. Although no specific laboratory tests exist for diagnosing WE, a complete blood count and comprehensive metabolic panel can assist in excluding other central nervous system abnormalities. Brain imaging typically does not provide diagnostic utility in Wernicke encephalopathy. Caine et al. established criteria in 1997 for diagnosing WE, which remain 85% sensitive when two or more of the classic features—ataxia, confusion, and ophthalmoplegia—are present. Furthermore, the patient's risk factors should be considered, as Wernicke encephalopathy was once thought to be exclusive to alcoholism but is now increasingly recognized in patients suffering from chronic malnutrition, post-bariatric surgery, hyperemesis gravidarum, liver disease, hyperthyroidism, and severe anorexia nervosa [1][6][7].

Differential Diagnosis:

The differential diagnosis of Wernicke encephalopathy (WE) is broad, as many neurological conditions can present with similar symptoms, such as ataxia, confusion, and ophthalmoplegia. Acute alcohol intoxication or withdrawal is often considered, especially in individuals with a history of chronic alcohol use. However, the clinical presentation of alcohol withdrawal typically involves additional signs such as tremors, agitation, and seizures, which are not characteristic of WE. Other disorders that may mimic WE include hepatic encephalopathy, which can present with altered mental status and ataxia, but is typically accompanied by a history of liver dysfunction and abnormal liver function tests. Metabolic encephalopathies, such as those caused by electrolyte imbalances (e.g., hyponatremia, hypoglycemia) or uremia in patients with renal failure, can also present confusion and altered mental status. Moreover, other thiamine deficiency-related disorders, such as Korsakoff syndrome and beriberi, should be considered in the differential diagnosis. In Korsakoff syndrome, memory deficits are more prominent and are characterized by anterograde and retrograde amnesia with confabulations, whereas the acute phase of WE involves more generalized neurological dysfunction. Central pontine myelinolysis, often seen in patients with rapid correction of hyponatremia, can cause ataxia and altered mental status but lacks the characteristic ocular findings of WE. Furthermore, infectious causes of encephalopathy, such as viral or bacterial meningitis, should be considered, particularly in the presence of fever and a history of infection. Neurological conditions such as multiple sclerosis or brain tumors may also present with gait disturbances and cognitive decline, though these are typically associated with a more gradual onset and different imaging findings. The accurate diagnosis of WE requires careful consideration of patient history, clinical symptoms, and laboratory tests, including thiamine levels, to rule out other potential causes.

Medical Management:

The primary objective in the treatment of Wernicke encephalopathy (WE) is the swift and effective correction of thiamine deficiency within the brain. As a medical emergency, WE is considered a reversible condition requiring immediate intervention, although its onset can be either acute or chronic. Parenteral thiamine administration is the most effective means of providing rapid correction. However, some patients may experience persistent neurological deficits, and in certain cases, the acute phase can evolve into chronic Korsakoff syndrome. The recommended dosage for thiamine treatment in WE may reach up to 500 mg, administered one to three times daily via parenteral routes. Malnourished patients often require higher doses. Evidence suggests that thiamine therapy can significantly improve the confusional state, leading to rapid resolution of ataxia, ophthalmoplegia, and nystagmus. Thiamine should generally be administered prior to or alongside glucose solutions, as glucose metabolism can decrease thiamine levels, exacerbating the neurological symptoms of WE. Additionally, it is crucial to address magnesium deficiency, as it may hinder recovery from WE, particularly in individuals with alcoholism [8][9][10].

Nursing Management:

Nursing management of WE requires close monitoring and timely interventions. Key nursing responsibilities include continuous assessment of vital signs and mental status, including the Glasgow Coma Scale (GCS). Intravenous thiamine administration should be closely supervised, ensuring patient safety, and creating a calm environment conducive to recovery. Educating caregivers about the disorder is vital for promoting understanding and compliance. Regular monitoring of fluid intake and output, as well as cognition and memory, is essential. Nurses should also encourage a nutritious diet, administer magnesium, and assess laboratory values, including electrolytes, glucose, and thiamine levels. Ensuring optimal oxygenation is crucial, and two large-bore intravenous lines should be maintained, as hypotension is a common complication [14][15].

When to Seek Help:

Clinical indicators necessitating urgent intervention include hypotension, severe alterations in mental status, and changes in the patient's level of consciousness (LOC) [14].

Outcome Identification:

Wernicke encephalopathy is a serious medical condition with substantial morbidity and mortality. Despite timely thiamine administration, the global confusion typically resolves rapidly, though ataxia and ophthalmoplegia may persist. Patients with minimal neurological impairment generally experience better outcomes with thiamine supplementation. However, individuals who survive WE may develop Korsakoff psychosis, often requiring long-term institutionalization. Fewer than 10% of these patients will recover sufficiently to be discharged from long-term care facilities. A significant proportion of survivors will experience long-term neurological deficits, including ataxia, nystagmus, and Korsakoff syndrome, which profoundly affect quality of life. Regrettably, there are no long-term follow-up studies, and anecdotal evidence suggests that many of these patients experience premature mortality [11][12][13].

Coordination of Care:

Management of WE typically necessitates a multidisciplinary team approach due to the complexity and diverse clinical presentations of the disorder. Neurologists and intensivists are crucial in the management of WE, with additional specialists consulted based on organ involvement. The roles of nurses, dietitians, social workers, and pharmacists are integral to the patient's recovery, particularly since WE patients are often frail, malnourished, and critically ill. A dietary consultation is essential to assess caloric needs and determine appropriate thiamine administration. Pharmacists should counsel patients on the importance of alcohol abstinence, as the cornerstone of therapy is thiamine. Compliance with the prescribed treatment regimen is essential for recovery, and the correction of electrolyte imbalances should be prioritized. Additionally, families should be educated regarding the prognosis and the potential for long-term care if Korsakoff syndrome develops. Ultimately, the goal is to enhance the quality of life and reduce the caregiving burden for families [14][15] [16].

Conclusion:

Wernicke Encephalopathy (WE) is a potentially life-threatening neurological condition caused primarily by thiamine deficiency, with alcohol use disorder being the leading risk factor. It manifests through a classic triad of symptoms, including confusion, ataxia, and ophthalmoplegia, though not all patients will exhibit all three symptoms. The condition's pathophysiology is linked to thiamine's essential role in cellular metabolism, and its deficiency leads to neuronal dysfunction, particularly in the brain regions responsible for memory, coordination, and motor control. Early diagnosis and intervention are critical, as prompt thiamine administration can reverse many of the acute symptoms. Nursing professionals play a vital role in managing WE, from identifying risk factors and symptoms to ensuring the timely administration of thiamine and other supportive treatments. Nursing management also includes monitoring vital signs, cognitive function, and ensuring patient safety during recovery. Moreover, the prevention of WE through appropriate nutritional support and thiamine supplementation in high-risk populations, such as those with chronic alcohol use, malnutrition, and gastrointestinal disorders, is essential. However, delayed or inadequate treatment can lead to irreversible neurological damage, progressing to Korsakoff syndrome, characterized by severe memory deficits. This emphasizes the importance of recognizing WE early, especially in individuals with known risk factors, and implementing appropriate treatment protocols. Additionally, healthcare providers should be educated about the potential for WE in patients with conditions leading to thiamine deficiency, ensuring broader awareness for timely intervention. As the global prevalence of alcohol use disorder and malnutrition increases, the incidence of WE may rise, making it imperative for healthcare systems to prioritize early diagnosis and intervention. Nurses, as frontline caregivers, must be equipped with the knowledge and skills necessary to manage this condition effectively, improving patient outcomes and minimizing the long-term impact of WE.

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تهاب الدماغ فيرنيك: الآثار التمريضية في التشخيص والتعافي العصبي - مراجعة محدثة

الملخص:

الخلفية: التهاب الدماغ فيرنيك (WE) هو اضطراب عصبي حاد ناتج بشكل أساسي عن نقص في الثيامين. يظهر عادة في المرضى الذين يعانون من استخدام الكحول المزمن، ولكن يمكن أن يحدث أيضًا في سياقات أخرى مثل سوء التغذية، التقيؤ المفرط في الحمل، والتغذية الوريدية المطولة. يعد WE مهددًا للحياة، حيث يؤثر على النظام العصبي المركزي والمحيطي، وغالبًا ما يتم تشخيصه بشكل خاطئ أو لا يتم تشخيصه بسبب أعراضه غير المحددة. إن التشخيص المبكر والتدخل العلاجي أمران حاسمان لمنع الأضرار العصبية التي لا يمكن إصلاحها.

الهدف: تهدف هذه المراجعة المحدثة إلى استكشاف الآثار التمريضية في تشخيص وتعافي المرضى من التهاب الدماغ فيرنيك. وتسلط الضوء على السمات السريرية، والأسباب الكامنة، واستراتيجيات العلاج للإدارة الفعالة.

الطرق: تم إجراء مراجعة شاملة للأدبيات الحالية، مع التركيز على العرض السريري، والفسيولوجيا المرضية، وعوامل الخطر، وطرق التقييم، وخيارات العلاج ل WE. تم تحليل الدراسات المتعلقة بنقص الثيامين، ومعايير التشخيص، والإدارة التمريضية، والتدخلات الطبية.

النتائج: تؤكد المراجعة أن نقص الثيامين لا يزال السبب الرئيسي ل WE ، والذي يرتبط في الغالب مع الاستخدام المزمن للكحول. يعتمد التشخيص على مثلث الأعراض السريرية المتمثل في الارتباك، والتهتك العصبي، والشلل العيني، رغم أن هذه الأعراض قد لا تظهر معًا دائمًا. يعد مكمل الثيامين، خاصة من خلال الإغطاء الوريدي، أمرًا بالغ الأهمية في إدارة WE. ومع ذلك، قد تتطور العجز العصبي المستمر، مما ينتقل إلى متلازمة كورسكوف المزمنة إذا لم يتم التعامل معها على الفور.

الخلاصة: يعد التعرف المبكر والتدخل العلاجي أمرًا بالغ الأهمية في إدارة التهاب الدماغ فيرنيك. يلعب الممرضون دورًا محوريًا في مراقبة الأعراض، وإعطاء العلاجات، وتقديم التعليم للمرضى والمقدمين للرعاية. يعد العلاج الفوري بالثيامين، إلى جانب إدارة النواقص المصاحبة، أمرًا أساسيًا لتحسين التعافي ومنع الأضرار العصبية طويلة الأمد.

تهاب الدماغ فيرنيك، نقص الثيامين، الإدارة التمريضية، التعافي العصبي، متلازمة كورسكوف، اضطراب تعاطي الكحول، سوء التغذية، التهتك **:الكلمات المفتاحية**