American Journal of Medical Case Reports, 2024, Vol. 12, No. 3, 38-41
Available online at http://pubs.sciepub.com/ajmcr/12/3/3
Published by Science and Education Publishing
DOI:10.12691/ajmcr-12-3-3

Challenging Discontinuation of Sedation in the Intensive Care Unit May Potentially be Attributed to Thiamine Deficiency. Case Report

Dr.S.AlKaabi, Dr.R.Ebead, Dr.M.Khames*, Dr.M. Amin, Dr.A.Gupta, Dr.E.Marzouk, Dr.S.Ahmed

ICU department Zayed Military Hospital Abu Dhabi, UAE
*Corresponding author: Magdy.aly@msc.mil.ae

Received February 06, 2024; Revised March 09, 2024; Accepted March 15, 2024

Abstract This is a case of 54-years-old critically ill H1N1 ARDS patient with prolonged ventilation and sedation and difficult weaning from sedation and mechanical ventilation. The case gives spotlight on not uncommon cause of delirium and failure of weaning in critically ill patients which is Vitamin B1 deficiency and how it is underestimated and should be put in mind while management those patients. Delirium and the failure to wean patients off sedation and mechanical ventilation in intensive care units (ICUs) are significant challenges that healthcare providers frequently face. This issue became evident in a unique case involving a critically ill H1N1 ARDS patient aged 54, which highlighted the often-ignored cause of these complications - Vitamin B1 deficiency. This critical review aims to delve deeper into the role of Vitamin B1 deficiency in exacerbating the conditions of critically ill patients and argue why it should be a key consideration in patient management.

Keywords: *Acute delirium, acute confusion state, thiamine deficiency, encephalopathy, sedation weaning, ventilator weaning, ICU, critically ill*

Cite This Article: Dr.S.AlKaabi, Dr.R.Ebead, Dr.M.Khames, Dr.M. Amin, Dr.A.Gupta, Dr.E.Marzouk, and Dr.S.Ahmed, "Challenging Discontinuation of Sedation in the Intensive Care Unit May Potentially be Attributed to Thiamine Deficiency.Case Report." American Journal of Medical Case Reports, vol. 12, no. 3 (2024): 38-41. doi: 10.12691/ajmcr-12-3-3.

1. Introduction

Thiamine is a water-soluble vitamin that is absorbed in the jejunum by 2 processes. When the thiamine level in the small intestines is low, an active transport portal is responsible for absorption. When the thiamine concentration is high, a passive mucosal process takes place. Up to 5 mg of thiamine is absorbed through the small intestines. [1] The small intestine is where phosphorylation of thiamine takes place. [1,2]

The body cannot produce thiamine and can only store up to 30 mg of it in tissues. Thiamine is mostly concentrated in the skeletal muscles. Other organs in which it is found are the brain, heart, liver, and kidneys. The half-life of thiamine is 9-18 days. It is excreted by the kidney. [3]

Persons may become deficient in thiamine by not ingesting enough vitamin B-1 through the diet or may become deficient through excess use; the latter may result from hyperthyroidism, pregnancy, lactation, or fever. Prolonged diarrhea may impair the body's ability to absorb vitamin B-1, and severe liver disease impairs its use. [4,5]

Delirium is a common manifestation of acute brain dysfunction in critically ill patients that is associated with poor short-term outcomes and may result in adverse sequelae years after ICU discharge. The strategies described for the prevention, diagnosis, and treatment of ICU delirium is subjects of multiple ongoing investigations. Every ICU clinician should be aware of these strategies, institute routine monitoring for delirium in the ICU, seek to reduce the impact of risk factors for delirium when possible, and use treatment options when necessary. [11,12,13]

Thiamine deficiency maybe the cause in many situations in critically ill patients causing delirium and sedation dependent and difficult weaning from sedation and mechanical ventilation. Other medical conditions such as stroke, drug overdose, other encephalopathy, and infections such as meningitis could mimic WE. These disorders should be thoroughly evaluated, particularly in patients at low risk for WE. [14]

Conversely, WE can be precipitated in high-risk patients such as those with critical illness or sepsis due to increased metabolic demand causing relative thiamine deficiency. In patients with cancer, absolute thiamine deficiency can occur because of malabsorption, malnutrition, or vomiting, triggering WE. [14,15,16,17]

In this case report we can find how thiamine supplementation helped the patient to wean from sedation and MV and improves the delirium state.

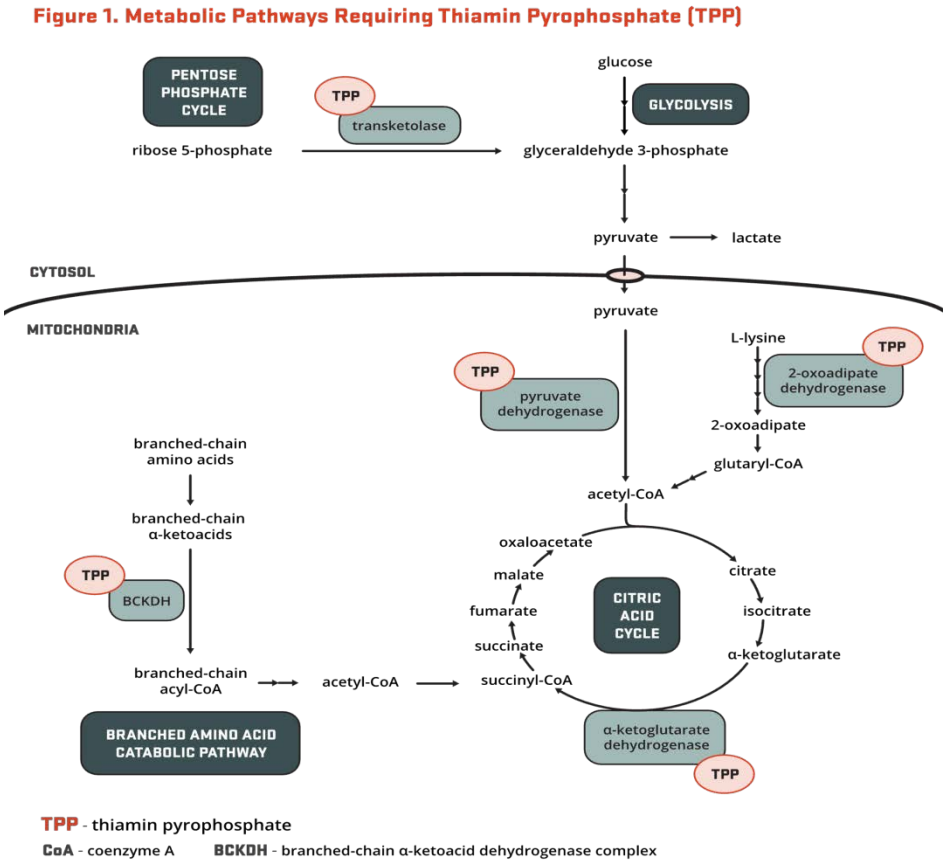

Figure 1. Thiamine metabolism[6]

Table 1. Causes of thiamine deficiency in critically ill patients:[7,8,9,10]

Causes of thiamine deficiency in critically ill patients			
Increased consumption	Increased thiamine depletion	Decreased absorption	Lack of thiamine intake
• Increased metabolic consumption of thiamine can result from the following: • Diets high in carbohydrate or saturated fat intake • Hyperthyroidism • Fever - Severe infection/sepsis • Refeeding syndrome (carbohydrate metabolism is increased)	Increased thiamine depletion can result from the following • Diarrhea • Diuretic therapies • Peritoneal dialysis • Hemodialysis/continuous renal replacement therapy	The following factors can result in decreased thiamine absorption: • Chronic intestinal disease • Alcoholism • Malnutrition • Gastric bypass surgery • Malabsorption syndrome - Celiac and tropical sprue • Folate deficiency - For example, in patients undergoing chemotherapy with high-dose methotrexate • Drug-vitamin interaction can prevent intestinal absorption of thiamine causing deficiency	The following diet-related factors can also cause reduced thiamine intake: • Alcoholic state • Starvation state • Gastric bypass surgery - Due to limited caloric intake during postsurgical repair (approximately 600-900 kcal/day) •Parental nutrition without adequate thiamine supplementation

2. Case Report

A 54y old male patient admitted to the ICU on 21/01/2020 with the history begun over the previous 10 days by fever, nausea, vomiting and dry cough, initially admitted to HDU on 18/01/2020 by SOB, dry cough requiring NC 2L/minute which gave him $SATO_2$ of 95-96% but the patient progressively deteriorating over the following 2 days with increasing respiratory distress, desaturation, increasing O2 requirements upgraded to NR mask with high flow and then NIV, XRChest showed bilateral patchy lung fields with obliteration of cardiopulmonary angle, ABG with mild hypoxia and respiratory alkalosis . However, the patient continued deterioration on 21/01/2020 morning Patient $SATO_2$ decreased to 80% with severe tachypnea so intubation and Mechanical Ventilation started. Bronchoscopy done on 21/01/2020 and BAL sample has been PCR tested confirmed H1N1 ARDS lung pneumonia. Patient has been put on sedation including Midazolam, Fentanyl, Propofol and paralytic agent was add also to control ventilation with high FIO_2 requirement and PEEP, Tazocin, Tamflu, Levofloxacin have been given and Meropenem and Linezolid add later in ICU day 8.

ICU day 12 patient maintaining SATO2 94-95% on SIMV PC 14 PEEP 14 FIO2 50%.

On 31/01/2020 patient requiring more sedation to synchronize with MV so sedation increased to Fentanyl 150mcg, midazolam 5mg, Precedex 1mcg/kg/hr and Propofol 100mg

On 01/02/2020 Fentanyl decreased to 80mcg, midazolam decreased to 4mg, propofol off, Precedex remain the same, Seroquel 25mg TID added From 01/02 to 10/02 patient improved as regard ARDS on PEEP 7, FIO2 40% but still deeply sedated and showed failure of weaning from sedation, patient became distressed, tachycardiaic, desaturation after one hour from just decreasing the sedation or making sedation vacation.

Seroquel dose increased to 50mg TID and Clonidine added to help for weaning from sedation but unfortunately could not wean from sedation at all with hyperactive delirium.

From 11/02 to 29/02 tracheostomy was done but the patient showed failure from weaning from sedation with suffering from hyperactive delirium and agitation

On 02/03/2020 LP was done as recommendation from neurologist to diagnose the cause for agitation with provisional diagnosis of post viral encephalopathy or viral encephalitis but the analysis returned normal.

ICU team decided to give Thiamine 100mg daily for 3 days as a provisional diagnosis of Wernicke's encephalopathy, the patient showed dramatic improvement after 2 doses as regard the weaning from sedation and successfully weaned off from sedation and also from MV completely and became stable on T- piece.

Patient then discharged from the ICU to the medical ward after 10 days from thiamine treatment and also after tracheostomy decannulation on only NC 2L/minute.

3. Discussion

It is obvious from this case that the frequently faced problem of ICU delirium can be caused by thiamine deficiency which in turn frequently underestimated. ICU delirium frequently happened in critically ill patients for many etiologies like sedative-related delirium, Postanesthesia delirium, critical illness delirium caused by hypoxia, sepsis, drug induced.

Assessment of delirium in ICU is very important to decrease the morbidity and mortality and to decrease ICU stay time. Many tools of assessment used to diagnose delirium Confusion Assessment Method for the ICU (CAM-ICU), Intensive Care Delirium Screening Checklist (ICDSC), Richmond Agitation- Sedation Scale (RASS). [18,19]

4. Treatment

The Best Treatment of Delirium is Prevention; Early Mobilization is the Most Important Measure.

The ABCDEF bundle refers to A for Assess, Prevent, and Manage Pain: prevention of agitation and delirium by adjusting the type and dose of analgesic drugs based on the assessment of patients' pain levels; B for Both Spontaneous Awakening Trials and Spontaneous Breathing Trials: daily interruption of sedative and analgesic infusions until the patient is awake and can follow instructions, or until the patient feels uncomfortable or agitated, and it is believed that the sedation therapy should be restarted; C for Choice of Analgesia and Sedation: light sedation does not mean inadequate sedation, deep sedation does not mean excessive sedation, sedation assessment means assessment of agitation and sedation, goal- directed sedation and analgesia based on sedation can reduce the severity and duration of delirium, and prevent delirium; D for Delirium: Assess, Prevent, and Manage: daily delirium assessment and constant monitoring of delirium when RASS scores fluctuate is an important measure for the management of delirium; E for Early Mobility and Exercise; and F for Family Engagement and Empowerment: both E and F can promote the recovery of patients and prevent delirium and shorten its duration. [20,21]

In our case ABCDEF bundle used but the patient persistently delirious, with dependence on sedatives with very difficult weaning from sedations and MV

So we thought about Wernicke's encephalopathy and Thiamine deficiency specially the patient received loop diuretics during his stay in the ICU which is one of the causes of deficiency as mentioned before

5. Conclusion

- Thiamine deficiency in critically ill patient is not uncommon.
- Thiamine deficiency should by considered as one of the causes of ICU delirium
-Thiamine should be given empirically in ICU patient, it can help

References

[1] J. A. Wooley, "Characteristics of thiamin and its relevance to the

management of heart failure.", Nutr Clin Pract, vol. 23, no 5, p. 487–93, 2008.

[2] K. D. Wiley e M. Gupta, "Vitamin B1 (Thiamine) Deficiency", StatPearls, jul. 2023, Acessado: 9 de fevereiro de 2024. [Online]. Disponível em: https://www.ncbi.nlm.nih.gov/books/NBK537204/

[3] B. E. W. JD. Isselbacher KJ, Harrison's Principles of Internal Medicine. 13th ed., NY: McGraw-Hill. 1994.

[4] P. D. Cole e B. A. Kamen, "'Beriberi' Interesting!", J Pediatr Hematol Oncol, vol. 25, no 12, p. 924–926, dez. 2003.

[5] B. R. B. R. eds. Beers MH, The Merck Manual., 17th edition. 1999.

[6] "Thiamin | Linus Pauling Institute | Oregon State University". Acessado: 9 de fevereiro de 2024. [Online]. Disponível em: https://lpi.oregonstate.edu/mic/vitamins/thiamin

[7] M. R. Matrana e W. E. Davis, "Vitamin deficiency after gastric bypass surgery: a review", South Med J, vol. 102, no 10, p. 1025–1031, out. 2009.

[8] F. Francini-Pesenti, F. Brocadello, R. Manara, L. Santelli, A. Laroni, e L. Caregaro, "Wernicke's syndrome during parenteral feeding: not an unusual complication", Nutrition, vol. 25, no 2, p. 142–146, fev. 2009.

[9] P. Wiesen, L. Van Overmeire, P. Delanaye, B. Dubois, e J. C. Preiser, "Nutrition disorders during acute renal failure and renal replacement therapy", JPEN J Parenter Enteral Nutr, vol. 35, no 2, p. 217–222, mar. 2011.

[10] O. S. Al-Attas, N. M. Al-Daghri, A. A. Alfadda, S. H. Abd-Alrahman, e S. Sabico, "Blood thiamine and its phosphate esters as measured by high-performance liquid chromatography: levels and associations in diabetes mellitus patients with varying degrees of microalbuminuria", J Endocrinol Invest, vol. 35, no 11, p. 951–956, dez. 2012.

[11] E. Ely et al., "The impact of delirium in the intensive care unit on hospital length of stay", Intensive Care Med, vol. 27, no 12, p. 1892–1900, 2001.

[12] K. Sriram, W. Manzanares, e K. Joseph, "Thiamine in nutrition therapy", Nutr Clin Pract, vol. 27, no 1, p. 41–50, fev. 2012.

[13] D. A. Sica, "Loop diuretic therapy, thiamine balance, and heart failure", Congest Heart Fail, vol. 13, no 4, p. 244–247, 2007.

[14] M. W. Donnino, J. Vega, J. Miller, e M. Walsh, "Myths and Misconceptions of Wernicke's Encephalopathy: What Every Emergency Physician Should Know", Ann Emerg Med, vol. 50, no 6, p. 715–721, dez. 2007.

[15] E. Isenberg-Grzeda, M. J. Shen, Y. Alici, J. Wills, C. Nelson, e W. Breitbart, "High rate of thiamine deficiency among inpatients with cancer referred for psychiatric consultation: results of a single site prevalence study", Psychooncology, vol. 26, no 9, p. 1384–1389, set. 2017.

[16] C. G. Harper, M. Giles, e R. Finlay-Jones, "Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy", J Neurol Neurosurg Psychiatry, vol. 49, no 4, p. 341–345, 1986.

[17] M. E. Charness e R. L. DeLaPaz, "Mamillary body atrophy in Wernicke's encephalopathy: antemortem identification using magnetic resonance imaging", Ann Neurol, vol. 22, no 5, p. 595–600, 1987.

[18] T. Sharshar et al., "Brain lesions in septic shock: a magnetic resonance imaging study", Intensive Care Med, vol. 33, no 5, p. 798–806, maio 2007.

[19] J. Cerejeira, H. Firmino, A. Vaz-Serra, e E. B. Mukaetova-Ladinska, "The neuroinflammatory hypothesis of delirium", Acta Neuropathol, vol. 119, no 6, p. 737–754, jun. 2010.

[20] M. A. Pisani, T. E. Murphy, K. L. B. Araujo, P. Slattum, P. H. Van Ness, e S. K. Inouye, "Benzodiazepine and opioid use and the duration of intensive care unit delirium in an older population", Crit Care Med, vol. 37, no 1, p. 177–183, 2009.

[21] C. L. Wong, J. Holroyd-Leduc, D. L. Simel, e S. E. Straus, "Does this patient have delirium?: value of bedside instruments", JAMA, vol. 304, no 7, p. 779–786, ago. 2010.