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Unique Presentation of Dry Beriberi Manifesting as Abrupt, Profound, Generalized Weakness in Middle-aged Male with No Comorbidities

Sujay Dayal[1]* and Surbi Dayal[2]

[1]*University of California San Francisco (UCSF) Fresno, USA*
[2]*Western University of Health Sciences, Pomona, CA, USA*

Abstract

Vitamin B1, thiamine, has many pertinent functions relating to the generation of energy and metabolism in the human body. Thiamine can be found in various food products, such as beans, yogurt, seafood and meats. Deficiency can be seen in individuals with poor oral intake or alcoholism and can present as dry beriberi. Dry beriberi involves the Central Nervous System (CNS) and manifests as a spectrum in severity, with symptoms of motor and sensory deficits commonly affecting the extremities.

A 30s year old male with no past medical history presented with acute generalized weakness, so debilitating he could not physically get out of bed. He recently endorsed an alcohol binge three days prior. Thiamine levels revealed<6 nmol/L. Intravenous (IV) infusion of thiamine alongside physical therapy during hospital led to complete resolution of symptoms. Thiamine levels should be checked in patients presenting with neuropathic symptoms, especially in the setting of malnourished states.

Keywords: Dry beriberi • Thiamine • Neuropathic symptoms

Introduction

Thiamine plays many roles in the body. Its active form, Thiamine Pyrophosphate (TPP), serves as a cofactor for many enzymes such as transketolase to function, often involved in decarboxylation reactions [1]. Thiamine is heavily involved in the CNS, especially in propagating nerve impulses and taking part in myelin sheath maintenance [2]. The human body has a limited storage capacity of thiamine, with an average of 25-30 mg stored at one time [3].

Thiamine deficiency can present in two forms, dry and wet. Dry beriberi manifests with more CNS symptoms, whereas wet beriberi presents with more cardiovascular symptoms. Wet beriberi presents as cardiomyopathy, cardiomegaly and heart failure symptoms (i.e. shortness of breath, peripheral edema) [3]. Shonshin beriberi, a fulminant form of wet beriberi, can present as cardiogenic collapse and subsequent multi organ failure if left untreated [3,4].

Dry beriberi on the other hand displays as CNS symptoms, such as decreased body reflexes, symmetrical motor and sensory deficits, etc [1]. One of the forms of dry beriberi is Wernicke's encephalopathy and presents as the triad: altered mental status, ataxia and ocular symptoms such as nystagmus. Another form is Korsakoff syndrome, associated with confabulation.

Case Presentation

A 30s year old male with no past medical history presented after not being able to get out of bed. Information was collected from patient and his wife. Patient and spouse hosted a baby shower three days prior to admission where patient endorsed drinking copious amounts of alcohol in celebration. The next day patient described feeling hung over but was able to function properly throughout the day. The day of admission patient was able to get out of bed at 7am and went to the backyard to shut off some lights. He was able to return back to the bed after which he became increasingly weak and fatigued. Around 8am, patient's wife tried to wake up patient but patient was unable to get up from bed, mentation still intact. He described the sensation as "something is pushing down on me". Patient remained in bed until 2pm due to the propound generalized weakness and lethargy and Emergency Medical Services (EMS) was subsequently called.

When EMS arrived, point of care glucose was 70. IV dextrose was given with repeat blood sugar 241, although no resolution of symptoms. Patient in the hospital was afebrile, hemodynamically stable breathing room air. Pertinent labs: CBC and CMP within Normal Limits (WNL), TSH 0.734, T4 0.77, glucose 209, CK WNL, CRP/ESR WNL, VBG unremarkable. It is worth noting patient's TSH and T4 labs were also both low and similar from labs three years prior to hospital admission. EKG, chest x-ray and urinalysis were unremarkable. Urine drug screen positive for cannabis. CT head without contrast negative. MR spine thoracic was negative. MR spine lumbar showed L5-S1 disc protrusion. Toxicology was consulted for possible environmental causes given patient's brief backyard exposure, felt low suspicion for toxin etiology. Patient was given 1 L IV fluids and fentanyl/morphine for pain.

On initial presentation, patient was in no acute distress, mentation intact, however unable to get out of hospital bed. He denied any pain, previous episodes of such symptoms, recent travel or sick contact exposure, or performing any activities out of the ordinary. The only symptoms patient endorsed were the following: nausea and generalized headache. Patient described the muscle weakness happening all at once and symmetric, not progressive. Patient endorsed chronic alcohol consumption (around three beers a day unknown start time) and marijuana usage, denied the following: allergies, medications, surgeries, contributory family history. He is a construction worker and his fellow colleagues were not exhibiting similar symptoms.

Physical exam showed a mental status of alert and oriented times 3 (able to remember name, location and reason why at hospital but unable to remember date). Patient was somnolent but able to follow instructions. Pupils

***Address for Correspondence:** Sujay Dayal, University of California San Francisco (UCSF) Fresno, USA, E-mail: sujay.dayal@ucsf.edu*

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were equal, round and reactive to light and accommodation. Mouth exam was benign and he was able to perform facial tasks such as lift eyebrows, smile and stick out tongue and move side-to-side. Brudzinski and Babinski signs were negative and thyroid without masses, enlargement, or tenderness to palpation. Cardiovascular, respiratory and gastrointestinal exams were benign. Patient did not have any edema upper or lower extremities. When patients' arms and legs were lifted up, they fell back down immediately. When arms and legs were stabilized, patient had +2/5 muscle strength in wrist, biceps/triceps, feet and quadriceps/hamstrings muscle groups. Pulses were intact at radial and posterior tibial bilaterally (b/l) and extremities were cool to touch. Patient had gross sensation to light touch at dermatomes V1-V3, C6, L4. Deep tendon reflexes were intact at biceps and patella b/l.

Patient was started on IV Synthroid for subclinical hypothyroidism. Thiamine levels later showed<6 nmol/L and IV thiamine was initiated. A lumbar puncture was done, with results unremarkable. Patient had continued recovery following initiation of thiamine, Synthyroid and physical therapy and had complete resolution of symptoms within seven days of hospital. Neurology was consulted, who agreed with medical management and recommended MRI brain with and without contrast pituitary focus and MR spine cervical, which all came back negative. Final recommendations made by them were the following: unclear what etiology of weakness is, but given improvement without compelling definitive evidence of Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and given that Intravenous Immune Globulin (IVIG) is not benign, would favor deferring treatment at this time and one week Electroencephalogram (EEG) outpatient study to evaluate for f waves. Psychiatry was consulted, who recommended further medical workup and causes be ruled out before a diagnosis of functional neurological symptom disorder can be applied.

After seven days of hospital stay, patient was cleared for discharge with oral Synthroid and thiamine. Physical therapy initially recommended acute care rehab, however given complete resolution of symptoms patient was cleared for home without physical therapy needs. Neurology and primary care appointments were set up.

Investigation

Generalized, acute weakness lends itself to a robust set of investigations and differentials (Figure 1). The first decision made was to assess whether the patient was stable or unstable and following the ABCDE of medicine: airway, breathing, circulation, disability and exposure. As this patient was stable hemodynamically, the severity of the situation was significantly reduced. The next step was to rule out the serious conditions such as bleeds and potential spinal cord compression syndromes such as cauda equina that would require urgent surgical intervention and hence imaging was ordered in the emergency department (i.e. CT head and MR brain, cervical, thoracic and spine) all of which were negative. Once the ABCDE and urgent medical conditions were ruled out, more workup could be initiated.

Labs were done to investigate further causes such as anemia or acute hepatic/renal failure, which came negative. Additional labs such as aldolase, ANA, EBV, hepatitis, Jo1, lyme serology, NMDAR, AchR, P/Q-type VGCC antibody, ganglioside gm-antibody IgG serum, urine porphobilinogen and blood cultures were all ordered for rare etiologies, all negative.

Appropriate consultants were brought to the case. Toxicology was consulted first given the unusual timeline of events of symptoms post-backyard exposure, with their analysis of less likely a toxin exposure further narrowing the etiologies. Neurology and psychiatry were later consulted and their recommendations of the etiology being less likely AIDP or functional neurological symptom disorder respectively also helping the case. Physical therapy was efficient in meeting with the patient daily and trying to optimize motor functioning quickly and effectively. Appropriate medications were brought, with IV Synthroid and thiamine later transitioned to oral helping bring resolution to patient's symptoms.

Outcome

Patient had complete resolution of neuropathic symptoms within seven day hospital course. Patient was discharged on oral Synthroid and thiamine and appointments were made for primary care and neurology. Neurology wanted to have the patient undergo an EEG to evaluate for f waves, which can be indicative of AIDP, a form of Guillain Barre Syndrome (GBS). Patient unfortunately never came to primary care and neurology appointments. Fortunately, patient has not come back to hospital since.

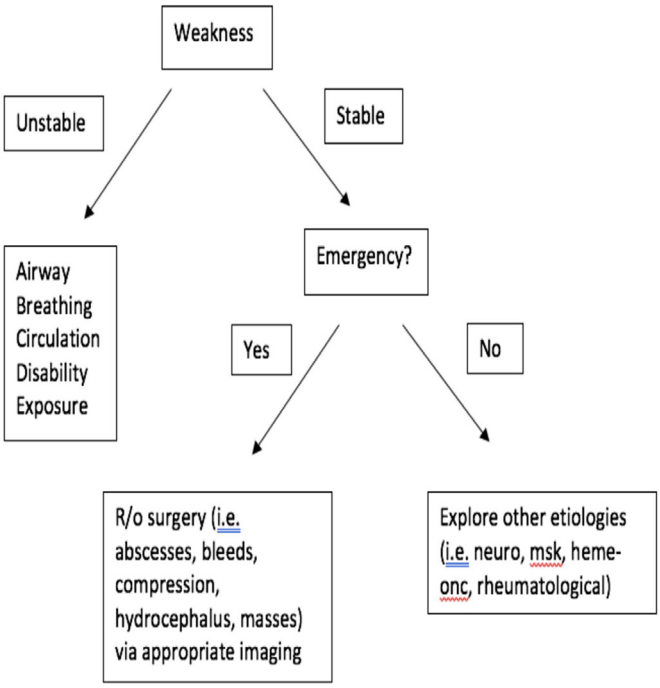

Figure 1. Rough schematic of how to approach generalize weakness.

Results and Discussion

In the grand scheme, this patient was effectively diagnosed and treated with complete resolution of symptoms during hospital course. Appropriate diagnostics, labs, imaging and consultants were brought to the case. However, there were areas of improvement on retrospection that could have facilitated the patient's recovery.

This patient was admitted overnight and the night team did a great job in admitting and starting the workup for generalized weakness. Unfortunately, thiamine labs were not ordered and it was the day team who ordered them, with the results coming back later in the evening. Had thiamine been ordered by the night team, the results would have come back faster and IV thiamine would have been started sooner. The patient's hospital stay might have been reduced by a day had thiamine be ordered initially.

Another problem is the lack of follow-up, as the patient did not come to the primary care or neurology outpatient visits. It is uncertain what the reason is for this, but on the medicine end, telephone reminders should be implemented to ensure the patient is seen in a timely manner. Although neurology deemed the patient less likely to have AIDP, an EEG study outpatient would better support their argument and help rule out serious medical conditions. A primary care follow-up would be essential, as the patient would have his labs (especially thiamine levels) regularly checked and repleted to avoid future hospitalizations.

The patient was not adequately counselled on appropriate alcohol intake. Risky behaviours such as alcohol binges and their negative consequences were not adequately talked out with the patient. It is imperative the root of the problem be addressed for future scenarios, as it can help prevent future readmissions. Alcohol anonymous resources and phone numbers should have been provided to the patient. With the patient's approval, his wife should have been part of the medical trajectory as better social support would help.

Current guidelines for dry beriberi treatment involve oral, intramuscular, or IV (if critically ill) 100 to 200 mg three times daily for two to three days, followed by maintenance regimen [5-7]. Maintenance regimen involves 5 to 100 mg once daily until patient is no longer at risk for deficiency [5-7]. Doses less than 100mg may be obtained in the form of multivitamin [5-7]. Thiamine is a relatively safe medication and the only contraindication is prior hypersensitivity to it [5-7].

Conclusion

Thiamine, vitamin B1, is imperative in facilitating human body processes. Commonly found in dietary sources, deficiencies can result in dry beriberi, a condition manifesting as severe impairments in motor and sensory deficits. Deficiencies can also stem from severe alcoholism. This paper explores a unique presentation of dry beriberi with complete resolution of symptoms with aggressive thiamine repletion.

Acknowledgment

None.

Conflict of Interest

The authors declare that they have no conflict of interest.

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