

**Abstract Form**

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| <b>Hospital Affiliation:</b>                 | Kern Medical Center  |                                     |                          |                          |                            |                          |                                     |
| <b>Presenter Name (Last, First):</b>         | Davtyan, Edvard, MD  |                                     |                          |                          |                            |                          |                                     |
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| <b>Project Title:</b>                        | Uncovering Dry Beriberi in a Patient with Catatonia                  |                                     |                          |                          |                            |                          |                                     |
| <b>Research Category (please check one):</b> |  |                                     |                          |                          |                            |                          |                                     |
| <input type="checkbox"/>                     | <b>Original Research</b>   | <input checked="" type="checkbox"/> | <b>Clinical Vignette</b> | <input type="checkbox"/> | <b>Quality Improvement</b> | <input type="checkbox"/> | <b>Medical Education Innovation</b> |

**Abstract**

**Introduction:**  
 Dry Beri-Beri involves severely decreased thiamine levels and can lead to issues affecting the central and peripheral nervous systems. Demyelination of the neurons occurs when thiamine, Vitamin B1, reaches critically low levels subsequently affecting neuronal conductivity and causing peripheral neuropathy. Catatonia is a neuropsychiatric syndrome characterized by motor and behavioral abnormalities, such as immobility, stupor, and mutism. There can be diagnostic confusion when uncovering a neuromuscular condition such as Dry Beri-Beri in the setting of a patient with catatonia.

**Case Presentation:**  
 A 43-year-old woman with medical history of schizoaffective disorder and methamphetamine use presented to the emergency department from a local community behavioral health hospital due to poor oral intake for two days and severe catatonia. On arrival, the patient was mute, stuporous, immobile, and resistant to instructions, meeting DSM-5 criteria of catatonia. Initial labs were notable for Thiamine <7. LP performed showed elevated protein of 110 and normal white blood cell count supporting diagnosis of Guillain-Barre. Patient received IVIG x7 days with no resolution of symptoms. Nerve conduction study demonstrated normal F waves, making Guillain-Barre less likely. Nerve conduction significant for diffuse sensory neuropathy, severe in lower limbs and moderate in upper limbs and moderate bilateral tibial motor axonal and mild bilateral peroneal motor demyelinating neuropathy. On physical exam the patient continued with facial and lower extremity weakness with absent reflexes and decreased positional sense. Based on the aforementioned labs and diagnostic studies, diagnosis of Dry Beri-Beri was established and the patient was subsequently started on Thiamine therapy. Patient continues to remain stable.

**Discussion:**  
 Thiamine deficiency leads to beriberi, further classified as dry or wet. Dry Beri-Beri is characterized by neuropathy with the duration and magnitude of the thiamine deficiency correlating to the severity of its presentation. The presenting symptoms of both Dry Beri-Beri and Guillain-Barré syndrome are similar and difficult to distinguish due to the overlap of their signs, symptoms, and diagnostic features. Our patient presented with lower extremity weakness and cerebrospinal fluid (CSF) findings consistent with Guillain-Barré prompting treatment with IVIG. CSF in patients with beriberi can also show a mild elevation in protein, furthering the difficulty in diagnosis. Distinguishingly, neuropathic pain is not usually a symptom of GBS, as in beriberi neuropathy. Additionally, GBS is characterized by demyelinating features like conduction blocks and prolonged F waves with preserved sensory responses. This is unlike our patient who had sensory and motor neuropathy on electromyography and normal F waves. These clinical features are helpful in determining GBS from Dry Beri-Beri, especially in patients at risk for thiamine deficiency.

**Conclusion:**  
 This particular case of Dry Beri-Beri is remarkable as the patient presented with catatonia obscuring the patient's significant neuromuscular deficits and ultimately delaying appropriate treatment. The complexity of differentiating Dry Beri-Beri from Guillain-Barre syndrome in a patient with catatonia emphasizes the need for a multidisciplinary approach involving psychiatrists and neurologists in patient care.