**Medical Report for User: 68d7b5cacbe6485b12e35c42**

**1. Present Illness**

The patient is a 48-year-old right-handed female with a history of migraines who presents with a 5-day history of progressive, ascending sensorimotor deficits. The onset was subacute, beginning with paresthesias in the distal bilateral lower extremities, described as a "pins and needles" sensation. Over the subsequent 2-3 days, this sensory disturbance ascended to the level of the mid-thighs and was accompanied by the development of significant lower extremity weakness, causing marked gait instability and requiring her to use furniture for support. She reports the weakness is symmetric and more pronounced proximally. Two days prior to presentation, she noted the onset of similar paresthesias in her fingertips bilaterally. She denies any facial weakness, diplopia, dysarthria, or dysphagia. There is no clear bowel or bladder dysfunction, though she reports some difficulty initiating a urinary stream. She denies any preceding febrile illness, vaccination, trauma, or back pain. The patient's functional status has declined significantly, and she is now largely wheelchair-dependent due to the profound leg weakness.

**2. Analysis and Plan**

Assessment
This is a 48-year-old female presenting with a subacute, progressive, ascending, and symmetric sensorimotor polyneuropathy. The clinical presentation, characterized by distal paresthesias that have ascended from the feet to the thighs and now involve the hands, accompanied by profound, predominantly proximal lower extremity weakness leading to loss of ambulation, is highly suggestive of Guillain-Barré Syndrome (GBS), most likely the acute inflammatory demyelinating polyradiculoneuropathy (AIDP) variant. The presence of early autonomic involvement, evidenced by difficulty initiating urination, further supports this diagnosis. While the classic areflexia is not documented, it is strongly suspected. The primary differential diagnosis includes an acute process of the spinal cord, such as transverse myelitis or a compressive myelopathy; however, the absence of a distinct sensory level on the trunk, lack of significant back pain, and the classic ascending nature of the deficits make a primary myelopathy less probable, though it must be emergently excluded. Other considerations such as toxic, metabolic, or vasculitic neuropathies are less likely given the rapid and symmetric progression.
Plan
The patient requires immediate admission to the hospital for urgent diagnostic evaluation and initiation of treatment, with close monitoring for potential respiratory and autonomic compromise. We will first obtain an urgent MRI of the entire neuraxis (cervical, thoracic, and lumbar spine) with and without contrast to definitively rule out any structural cord compression or inflammatory myelitis. Following imaging, a lumbar puncture will be performed to analyze the cerebrospinal fluid, with the expectation of finding albuminocytologic dissociation (elevated protein with a normal cell count) which would further support a diagnosis of GBS. Concurrently, we will send a comprehensive panel of blood work, including inflammatory markers, and arrange for nerve conduction studies and electromyography to confirm the presence of a demyelinating polyneuropathy and assess the severity of nerve injury. Given the high clinical suspicion and rapid functional decline, we will not wait for all diagnostic results and plan to initiate immunomodulatory therapy with Intravenous Immunoglobulin (IVIG) at a total dose of 2 g/kg administered over five days. The patient will be placed on continuous cardiac monitoring and will undergo frequent serial measurements of her negative inspiratory force (NIF) and vital capacity (VC) to monitor for impending respiratory failure. She will be started on deep vein thrombosis prophylaxis due to her immobility. We will also consult Physical and Occupational Therapy for an early assessment to facilitate rehabilitation planning and ensure her safety.