

# Guillan-Barre Syndrome in a Patient with Uncontrolled Diabetes and Severe Peripheral Neuropathy

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## Abstract

The complications of diabetes are frequently encountered in the Emergency Department. In contrast, Guillan-Barre Syndrome (GBS) is a relatively rare diagnosis requiring a high index of suspicion which can cause significant morbidity and mortality if not recognized and properly treated. GBS is an acquired condition which is usually preceded by a viral upper respiratory or gastrointestinal (GI) illness which can cause peripheral weakness and potentially diaphragmatic paralysis leading to life-threatening respiratory failure. Herein, we present a case of a 57-year-old male with a history of poorly-controlled diabetes who presented with both sensory and motor weakness of the distal upper and lower extremities; the patient was ultimately diagnosed with Guillan-Barre syndrome. This case illustrated an uncommon disease process that was initially mistaken for an extremely common disease both of which require very different management. This illustrative case is important to the emergency medicine physician because quick identification can stave off untoward complications and increased morbidity and mortality of GBS including respiratory distress and airway emergencies.

## Keywords

Diabetes, Guillan-Barre Syndrome, Neuropathy, Weakness

## 1. Introduction

Diabetes-related complications are frequently encountered in the Emergency Department (ED) including medication reactions, hyperosmotic hyperglycemic syndrome, and diabetic ketoacidosis; moreover the prevalence of diabetes continues to rise leading to even more urgent and emergent diabetes-related issues

[1]. In contrast, Guillan-Barre Syndrome (GBS) is a relatively rare life-threatening condition requiring a high-index of suspicion especially given its intrinsic morbidity and mortality if missed or diagnosis is delayed. GBS is an acquired condition which is usually preceded by a viral upper respiratory or GI illness [2]. It is characterized by symmetric ascending sensory and motor disturbances which can mimic peripheral neuropathy, electrolyte and metabolic abnormalities including hypercalcemia, multiple sclerosis, and tick paralysis [3]. Herein, we describe a case of a 57-year-old man with uncontrolled diabetes who presented with stocking-glove sensory and motor weakness who was ultimately diagnosed with Guillan-Barre syndrome in addition to severe peripheral neuropathy. We discuss management of our patient and underscore the significance for the emergency medicine physician to be vigilant of patients presenting with unusual neurological findings in the face of long standing chronic medical problems. This is important to the emergency medicine physician because quick identification of progressively worsening neurological deficits can stave off untoward complications and increased morbidity and mortality of GBS including respiratory distress and airway emergencies.

## 2. Case Presentation

A 57-year-old man with a history of poorly controlled type-2 diabetes and hypertension presented to the ED with the chief complaint of upper and lower-extremity sensory changes over the past two weeks with associated and weakness in his legs. He noted previous paresthesias in his upper and lower extremities and stated he burned his feet on hot pavement two weeks ago while walking barefoot on the street but did not realize it until several days due to his severe lower-extremity neuropathy. He also endorsed recent stumbling and subjective lower-extremity weakness for which he was seen and subsequently discharged from another ED. At the outside ED, a computed tomography scan (CT) of his brain was determine to be normal and his laboratory studies including a basic metabolic panel and complete blood count with differential were unremarkable.

He presented to our institution several hours after his previous discharge because he was unable to rise from the transport van to walk to his house. On arrival to our facility, the patient was in no apparent distress and his initial vital signs were within normal limits. The patient denied any recent fevers or illnesses, including upper-respiratory or flu-like symptoms. Positive physical exam findings noted the patient had sensory defects from his feet to knees and fingers to wrists bilaterally, which is not unexpected for a diabetes patient. However, he also had 1/5 reflexes in his lower extremities and 2/5 upper extremity reflexes. His strength was intact proximally, but 3/5 distally from his knees to his feet. His blood work was remarkable for a blood glucose of 266 mg/dL and slightly elevated erythrocyte sedimentation rate. A CT of his head was negative for acute processes including ischemia, hemorrhage, multiple sclerosis or demyelinating

disease.

Concern was stemmed due to an asymmetric neurologic exam and motor deficits which prompted an emergent consult with a neurologist who recommended a lumbar puncture (LP) to assess for GBS. An LP was performed and was significant for a cerebral spinal fluid protein level of 122 mg/dL, and glucose level of 113 mg/dL (normal ranges are 14 - 15 mg/dL and 40 - 75 mg/dL, respectively). Opening pressure was within normal limits.

Ultimately, the patient was diagnosed with GBS and started on 5 days of intravenous immunoglobulin therapy. The patient showed improvement and suffered no untoward complications. Subsequent follow-up evaluations over a period of weeks showed the patient had significant improvement in his overall strength. He was discharged to an inpatient rehabilitation facility where he continued to recover and was eventually discharged home with complete resolution of his function status.

### 3. Discussion

GBS is the most common and acute paralytic neuropathy in the world [4] with a wide range of manifestations from mild paralysis to reports of symptomatology mimicking brain death [5]. The most commonly accepted mechanism of GBS is proposed molecular mimicry causing the immune system to attack the myelin or axon of peripheral nerves [6]. This may lead to insidious lower extremity weakness with progressive decline of function. A more serious condition is the Miller-Fisher variant where patients may develop cranial-nerve palsies or diaphragm paralysis requiring intubation and possible prolonged mechanical ventilation.

The treatment of choice for GBS is intravenous immunoglobulin or plasma-exchange therapy both of which are equally successful and while expensive, can be life saving. Oral or intravenous steroids have been shown to be neither beneficial nor harmful in GBS [7]. Though not encountered often, this potentially devastating disease should remain on a provider's differential when assessing a patient with motor weakness and emergent neurology consult should be obtained even if this requires transfer to another receiving hospital.

### 4. Conclusion

The prevalence of diabetes continues to increase in the US [1]. Diabetic peripheral neuropathy is an entity frequently encountered in the ED, and patients with advanced disease often have distal neurosensory but not neuromotor deficits. These deficits may be due to glycosylation of the myelin sheath by excessive circulating glucose, or inhibited repair of peripheral nerve endings [8] [9] [10]. It is easy to attribute seemingly chronic neurologic symptoms to poorly controlled diabetes; physicians should take care to document a thorough neurologic exam to rule more serious etiologies. A miss can lead to potentially catastrophic results for the patient and vigilance is always warranted.

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