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# Guillain-Barré Syndrome Presenting with Sinus Node Dysfunction and Refractory Shock

Authors' Contribution:  
Study Design A  
Data Collection B  
Statistical Analysis C  
Data Interpretation D  
Manuscript Preparation E  
Literature Search F  
Funds Collection G

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**Conflict of interest:** None declared

**Patient:** **Male, 76**  
**Final Diagnosis:** **Guillain-Barré syndrome**  
**Symptoms:** **Bradycardia • refractory hypotension**  
**Medication:** —  
**Clinical Procedure:** —  
**Specialty:** **Cardiology**

**Objective:** **Unusual clinical course**





**Background:** Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyneuropathy that is usually associated with preceding respiratory or gastrointestinal infection and has the hallmark manifestation of ascending flaccid paralysis. We report an atypical presentation of GBS.

**Case Report:** A 76-year-old male presented with acute onset of diaphoresis and altered mental status. He subsequently developed severe bradycardia and refractory hypotension, which initially responded to dopamine infusion. A temporary pacemaker wire was placed to stabilize the heart rate but hypotension persisted. Acute autonomic dysfunction was suspected. Head and chest imaging was unrevealing. Lumbar puncture revealed albuminocytologic dissociation that was consistent with a diagnosis of GBS. Hospital course was complicated with acute kidney injury and metabolic acidosis. Plasmapheresis was initiated. The patient eventually died of multi-organ failure.

**Conclusions:** Autonomic dysfunction is a known but rare presentation of GBS. In patients presenting with refractory bradycardia and hypotension, GBS should be considered in the differential diagnosis.

**MeSH Keywords:** **Autonomic Nervous System Diseases • Guillain-Barré Syndrome • Sick Sinus Syndrome**

**Full-text PDF:** <http://www.amjcaserep.com/abstract/index/idArt/902668>

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## Background

GBS is an acute inflammatory demyelinating disorder of the peripheral nervous system with an incidence of 1.11 per 100,000 persons each year [1,2]. GBS is strongly associated with a preceding infection, most commonly with *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus and *Mycoplasma pneumoniae* infection [3]. The molecular mimicry of the antigens of these pathogens and the subsequent development of anti-ganglioside antibodies are an important part of the pathogenesis of GBS. Patient usually report symptoms of an upper respiratory tract or gastrointestinal infection prior to hospital admission. The cardinal clinical feature of GBS is progressive and symmetrical ascending muscle weakness, which usually starts in the legs and, in some cases, may progress to respiratory failure [1,2].

We report a very atypical manifestation of GBS where sinus node dysfunction and refractory shock were the presenting symptoms. The absence in this patient of the hallmark ascending paralysis resulted in delayed diagnosis and treatment.

## Case Report

A 76-year-old male was admitted to the emergency department (ED) for diaphoresis, slurred speech, and both urinary and fecal incontinence. Prior to admission, he was in his usual state of health until the sudden onset of symptoms. He had a history of coronary artery bypass graft surgery two years before this presentation, as well as a history of hypertension, mild dementia, transient ischemic attack (TIA) with no residual deficits, chronic obstructive pulmonary disease, and gastroesophageal reflux disease. The list of home medications included aspirin, dipyridamole 200–25 mg twice daily, calcium carbonate 600 mg once daily, multivitamin tablet once daily, pantoprazole 40 mg twice daily, donepezil 10 mg at bedtime, memantine 28 mg at bedtime, metoprolol succinate 75 mg twice daily, atorvastatin 40 mg at bedtime, losartan 50 mg daily, albuterol inhaler, umeclidinium bromide inhaler, and latanoprost eye drops. There was no recent change in his medication list.

On admission to the ED, his blood pressure was 137/92 mm Hg and he had a heart rate of 59 bpm. His respiratory rate was 19 with oxygen saturation of 98% on 2 liters of oxygen delivered by nasal cannula. He was lethargic but responded appropriately to verbal commands. His altered mental status in the setting of a previous TIA raised concern for cerebrovascular accident and thus computed tomography (CT) of the head was ordered. During the CT scan, he developed severe bradycardia with a heart rate of ~30 bpm and worsening mental and respiratory status. Emergent intubation was performed for airway protection. Intravenous atropine failed to increase his

heart rate. His bradycardia improved with intravenous dopamine infusion. An electrocardiogram revealed sinus bradycardia with an old left bundle branch block.

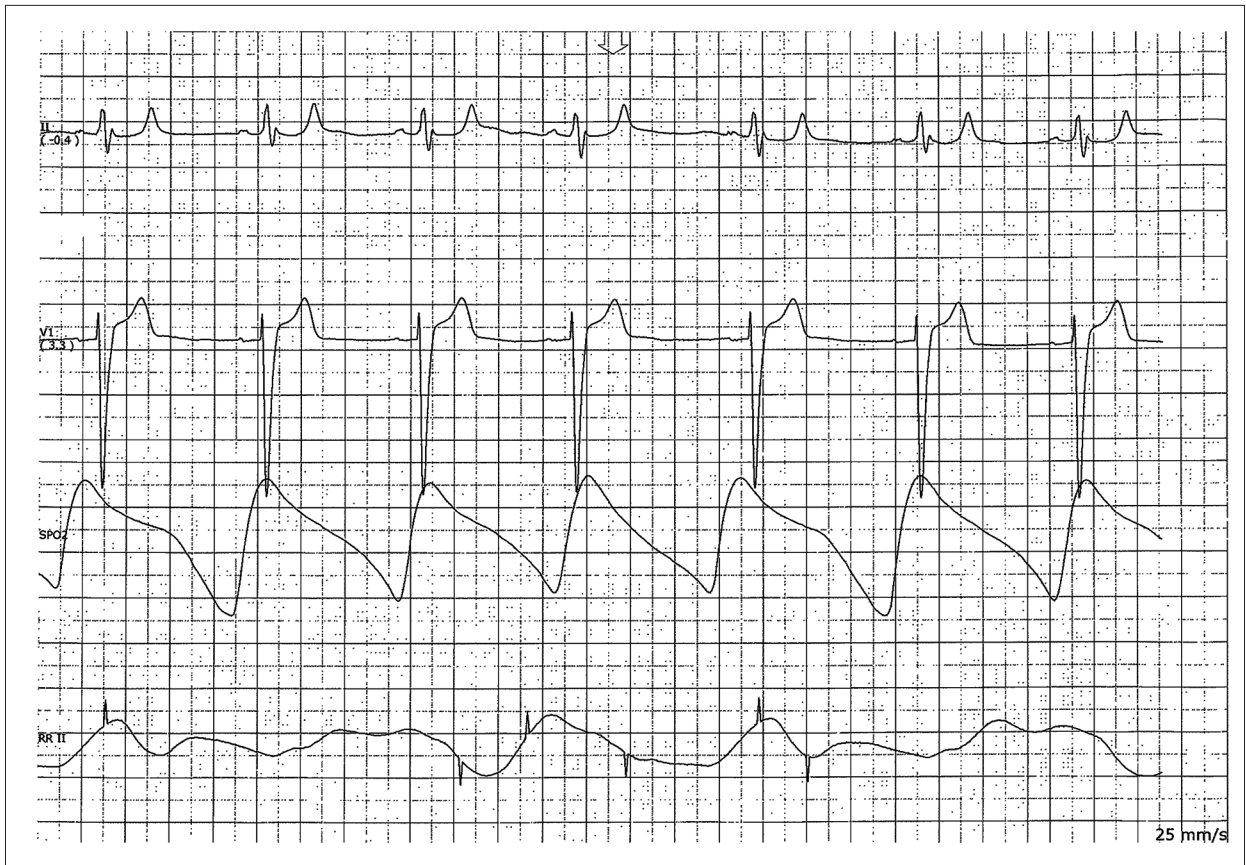
Subsequently, a CT head scan did not show any acute intracranial pathology. The CT chest scan was negative for aortic dissection or pulmonary embolism. Blood tests revealed leukocytosis of  $19.8 \times 10^3/\mu\text{L}$  and a lactic acid level of 3.6 mmol/L with an anion gap of 18 mEq/L. Troponin was negative on two occasions and his thyroid function test was within normal limits. He was transferred to the intensive care unit at our hospital for higher level of care.

Multiple attempts to titrate the patient off a dopamine infusion were unsuccessful with recurrence of severe bradycardia (Figures 1, 2) followed by severe hypotension. An echocardiogram showed a normal left ventricular ejection fraction of 55% with normal systolic function and no evidence of outflow tract obstruction. A temporary pacemaker wire was placed and the output was set asynchronously at 80 bpm. Despite this, he developed worsening hypotension and an epinephrine infusion was added. An electroencephalogram reported continuous generalized slowing, consistent with an encephalopathy of moderate to severe degree but did not demonstrate any epileptiform abnormalities. In order to investigate the possibility of underlying central nervous system infection, a lumbar puncture was performed. Cerebrospinal fluid (CSF) analysis reported albuminocytologic dissociation with the high protein concentration of 62 mg/dL (normal range: 15–45 mg/dL) and no increase in cell count. This finding is consistent with GBS. Neurological examination without sedation demonstrated areflexia in both upper limbs and lower limbs. Five cycles of plasmapheresis were planned.

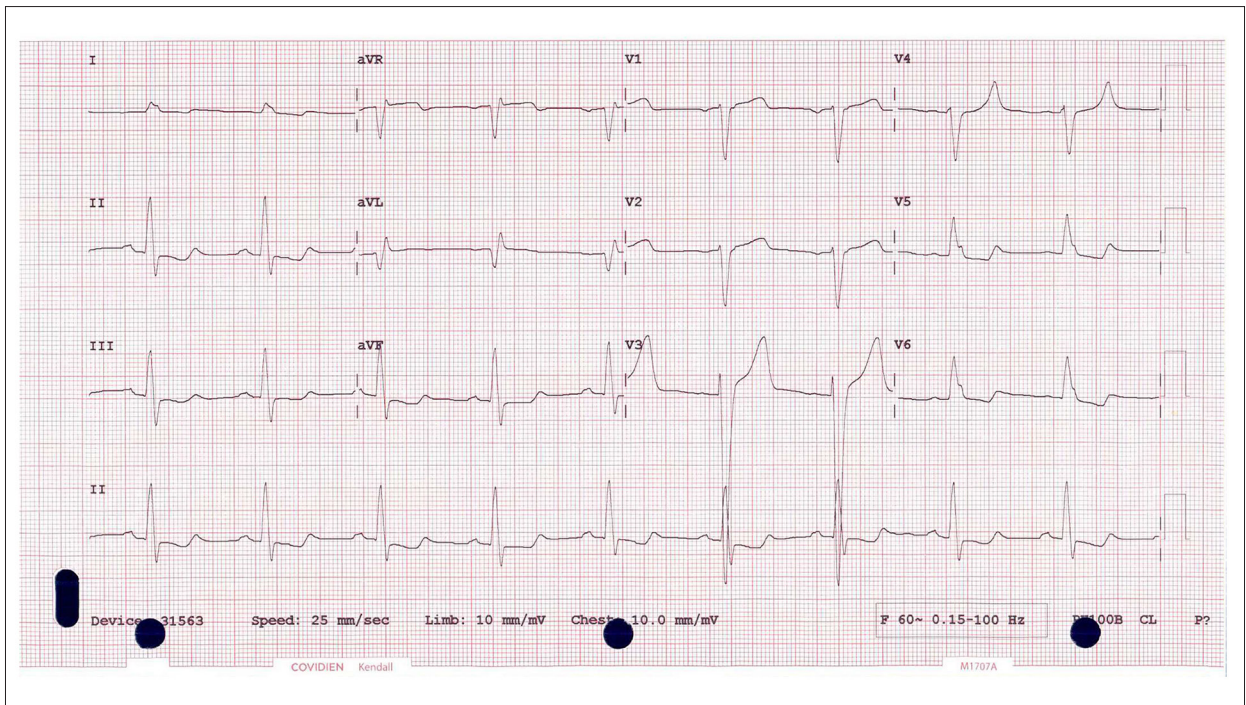
The patient subsequently developed acute kidney injury with creatinine trending up from 1.0 mg/dL to 1.7 mg/dL as well as worsening metabolic acidosis with lactic acid now rising to 4.8 mmol/L, most probably secondary to hypoperfusion. He completed one cycle of plasmapheresis the following day, which did not improve his clinical state. His lactic acid increased to 11.2 mmol/L subsequently. At this point, his family decided to opt for comfort care and he passed away shortly after compassionate extubation.

## Discussion

Autonomic dysfunction is commonly seen in GBS patients due to the involvement of both sympathetic and parasympathetic fibers [1,2]. Recognizing autonomic dysfunction, which can lead to sinus node dysfunction and refractory shock, is the cornerstone of the management of GBS. Severe bradyarrhythmia and recurrent asystolic episodes necessitating pacemaker placement have been reported with GBS patients [4,5]. Our patient had



**Figure 1.** Severe bradycardia with heart rate of 40 bpm without dopamine infusion.



**Figure 2.** Bradycardia responding to dopamine infusion.

severe sinus bradycardia, which was responsive to dopamine infusion, suggesting sinus node dysfunction. Subsequently temporary wire placement was performed to maintain adequate heart rate in order to help maintain adequate perfusion. Despite temporary pacemaker placement, he continued to have hypotension and shock that was most likely related to vasodilatation and autonomic dysfunction. The sinus node dysfunction was probably secondary to autonomic dysfunction.

The presence of significant blood pressure fluctuations is often seen with GBS. Our patient presented with sustained hypotension requiring the support of both dopamine and epinephrine infusions. Plasma exchange has been proven as an effective

treatment for GBS [6]. The severity of our patient's condition did not allow him to complete his treatment. Mortality of GBS is between 3% and 7% and autonomic dysfunction is an important predictor of death [1,2].

## Conclusions

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In summary, in patients presenting with autonomic dysfunction associated with sinus node dysfunction and refractory shock, GBS should be considered. This case highlights the importance of recognizing autonomic dysfunction as a poor prognostic indicator in GBS.

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