Case Report

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Acute neurogenic stunned myocardium in a patient with Guillain–Barré syndrome: case report

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Introduction: Autonomic dysfunction is a prevalent symptom of Guillain–Barré syndrome (GBS); cardiovascular involvement in this scenario has been mentioned infrequently in the literature.

Case Presentation: A 65-year-old man with GBS presented with reversible left ventricular systolic failure. On first presentation, our patient had no history or indications of heart malfunction. During the clinical manifestation of his autonomic dysfunction, he had electrocardiographic alterations, modestly increased cardiac enzymes, significant left ventricular systolic dysfunction, and segmental wall motion irregularity. Once the initial episode was over, these anomalies and his symptoms resolved quickly.

Discussion: We believe the reversible left ventricular dysfunction was caused by the toxic impact of elevated catecholamines as well as transiently injured sympathetic nerve endings in the myocardium, which was apparently caused by GBS. We recommend that echocardiography be performed in patients who exhibit clinical signs of autonomic dysfunction, particularly if they are associated with abnormal electrocardiographic findings, cardiac enzyme elevation, or hemodynamic instability, so that appropriate medical therapy can be instituted as soon as possible.

Conclusion: GBS is a not a very rare situation in our context. Thus, doctors are supposed to know the life-threatening complications such as neurogenic stunned myocardium and be prepared to dodge it.

Keywords: coronarography, electrocardiography, Guillain–Barré syndrome, neurogenic stunned myocardium

Introduction

Guillain–Barré syndrome (GBS) is a grave post-infectious immune peripheral neuropathy. It is the consequence of an autoimmune destruction of nerves in the peripheral nervous system. It can affect motor, sensory, and autonomic fibers and is believed to be the most frequent cause of acute neuromuscular paralysis and ventilatory failure[1]. Morbidity and eventual mortality in patients with the GBS are associated with cardiopulmonary instability, including blood pressure (BP) fluctuations, potentially fatal arrhythmias and myocarditis. In the literature, we have found some case reports associating electrocardiogram (ECG) abnormalities and GBS. Generally explained by temporary differences in cardiac innervations or catecholamine cardiotoxicity, ECG abnormalities are frequently regressive.

HIGHLIGHTS

- Cardiovascular involvement of Guillain–Barré syndrome has been mentioned infrequently in the literature.
- Doctors are supposed to know the life-threatening complications such as neurogenic stunned myocardium and be prepared to dodge it.

Presentation of the clinical case

We report the case of a 65-year-old male patient, presenting to the emergency department with increasing flaccid paralysis evolving since a week. The patient had been having escalating symmetrical weakening of the limbs, concluding in tetraplegia with significant swallowing problems.

The patient and his family had no previous medical history, he was not on any medications at the time of his presentation, and he had no allergies. He does, however, mention viral pharyngitis in the prior 2 weeks. He lived in the countryside, which explains the tardiness of his consultation.

At his admission, the patient was afebrile with a BP of 140/70 mmHg, a heart rate of 73 beats/min, a respiratory rate of 14/min, and an oxygen saturation of 97% on room air. The patient

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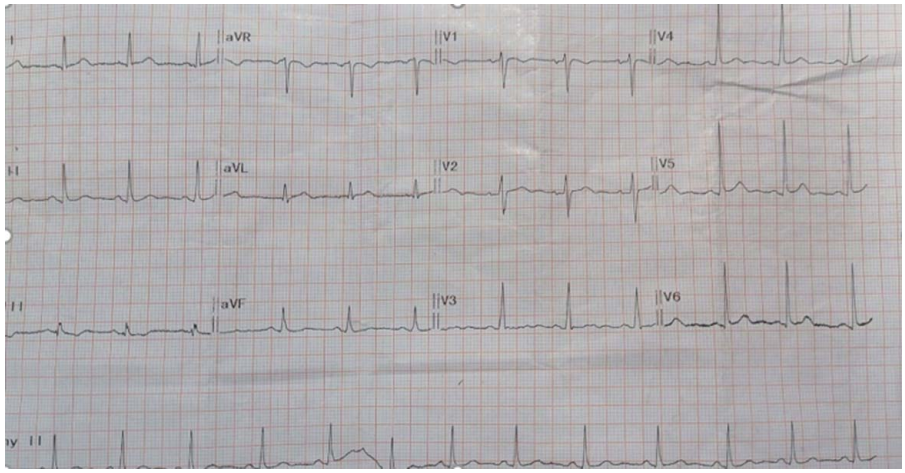

Figure 1. The patient's normal electrocardiogram at admission. aVF, augmented vector foot, aVL, augmented vector left; aVR, augmented vector right.

was conscious, at the time of hospitalization, with no respiratory dyspnea or respiratory symptoms. The muscular strength assessment revealed weakness in four limbs, with an MRC scale of 2/5 in the proximal upper extremities, 3/5 in the distal upper extremities, and 1/5 in the lower extremities. Deep tendon reflexes were absent generally. He had no spine sensory level. No meningeal irritation signs were present, nor was there an upper motor neuron disorder. The ECG was normal (Fig. 1); he also had no hypercapnia [partial pressure of carbon dioxide ($PaCO_2$) = 41 mmHg] in arterial blood gas analysis.

Cervical and brain magnetic resonance imaging (MRI) were preformed and returned normal. A neurophysiological study was performed. Electrodiagnostic parameters demonstrated decreased amplitude at compound muscles action potential and no response at sensory nerve action potential, while electromyography showed a decreased recruitment. All of these symptoms point to acute motor–sensory axonal neuropathy; we also performed a cerebrospinal fluid analysis that showed elevated protein levels (860 mg/l) with normal cells (3/mm^3). The lab test results upon admission were normal.

The patient was admitted to the intensive care unit, where treatment with intravenous immunoglobulin was started at the dose of 400 mg/kg/day over the course of 5 days. On day 3, the patient was intubated because of a progressive respiratory failure due to muscle weakness and mucus plugging. Two days later, the patient presented a sudden tachycardia (160 beats/min) and become hemodynamically unstable (Average Blood Pressure = 44 mmHg) despite fluid loading. In the following hours, additional fluids and a high dose of norepinephrine were administered to stabilize the patient. His Average Blood Pressure remained low (Average Blood Pressure = 62 mmHg), the tachycardia persisted (sinus rhythm of 133 beats/min), and ECG revealed sinus tachycardia with nonspecific ST-T segment changes (Fig. 2), but the urine output remained normal. Laboratory tests revealed normal white blood cells; normal platelets and hematocrit; normal liver, thyroid, and kidney function; normal

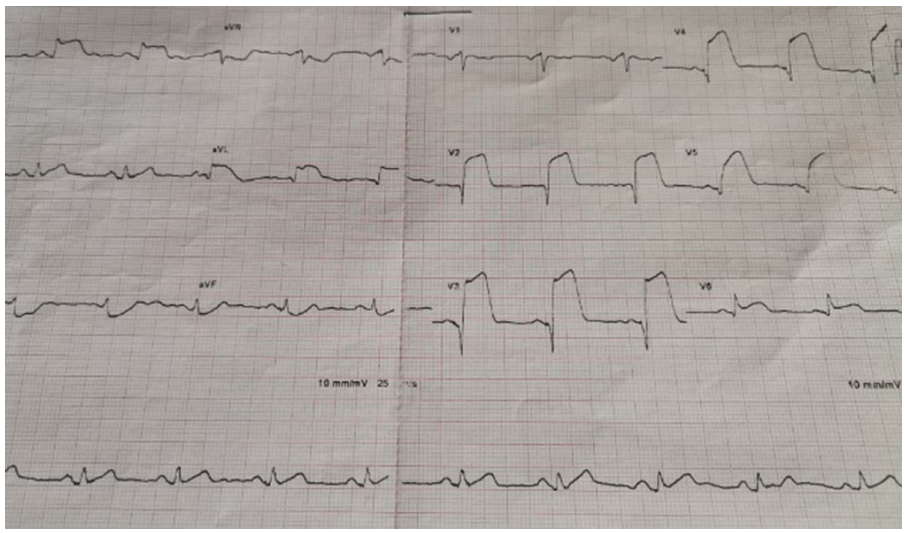

Figure 2. The patient's electrocardiogram after the state of shock where he presented an elevated ST segment in the anterior. aVF, augmented vector foot, aVL, augmented vector left; aVR, augmented vector right.

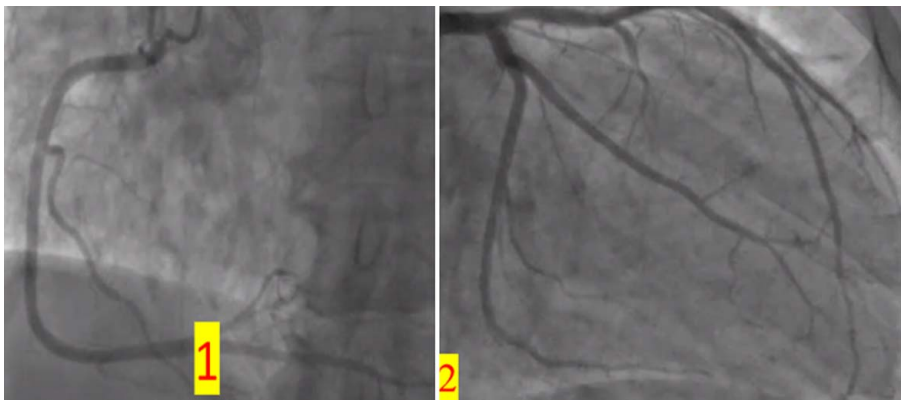

Figure 3. Angiographically normal coronary systems: 1: right; 2: left.

creatine kinase (CK = 56 U/l, normal <145 U/l), but elevated troponin (1590 ng/l, normal <26 ng/l) and N-terminal pro-brain natriuretic peptide (1391 pmol/l, normal <15 pmol/l). Urgent transthoracic echocardiography was performed, which revealed dilated and severe hypokinetic left ventricle, normal heart valves, normal right ventricle, and the lack of pericardial effusion (Fig. 1A, B). The estimated left ventricular ejection fraction (LVEF) was 20%. A new ECG was performed, which showed inverted T-waves in leads I, AVL, and V1–V6. Urgent coronary angiography to exclude coronary artery disease was performed, which was normal (Fig. 3).

The diagnosis of stress cardiomyopathy was confirmed; dobutamine infusion was initiated (5 µg/kg/min) to assist the left ventricular contractility and to reduce the afterload despite persistent tachycardia. This increased the BP. In the next 72 h, dobutamine was tapered because the patient became gradually hemodynamically stable, and due to persistent tachycardia, bisoprolol was introduced to reduce the sympathetic tone and improve myocardial work.

The patient underwent a tracheostomy on day 10 because of difficult weaning. He did not show any signs of improvement regarding his neurological deficit, at day 20, and we opted for the second dose of immunoglobulin with the same dose. On the 32nd day of his hospitalization, the patient was released from the ICU department to the neurological ward on spontaneous breathing, but the patient still had peripheral symmetrical and especially motor polyneuropathy. The repeat flow-up Trans-thoracic Electrocardiogram showed gradual normalization of LVEF.

This case is written following the SCARE guidelines[2].

Discussion

GBS is an acute inflammatory disease that has an effect on the peripheral nervous system. It is considered the most common cause of acute flaccid paralysis in young adults and the elderly and an important cause of admission to intensive care[3]. Manifestations of the GBS vary from monoparesis to life-threatening paralysis of the respiratory muscles. The latter is often punctuated by the presence of cardiac involvement; this ranges from variations of BP to involvement of the myocardium and potentially fatal arrhythmias[1].

The term neurogenic stunned myocardium has been used to sum up these abnormalities in case of central nervous system injury, in the absence of coronary artery disease. Although the pathophysiology of this syndrome is still unknown, up to 50% of patients, at autopsy, were found to have myocardial contraction band necrosis. The contraction band necrosis has been produced with catecholamine infusion; a sympathetic overflow and catecholamine toxicity have been implicated as the triggers[4].

We report a case of reversible left ventricular systolic dysfunction in a GBS patient. He had no history or signs of cardiac dysfunction on admission. During his autonomic dysfunction, he showed electrocardiographic abnormalities accompanied by elevated cardiac enzymes and left ventricular systolic dysfunction and segmental wall motion abnormality on the transthoracic echocardiography. The rapid beginning and resolution of symptoms, together with the abnormalities mentioned earlier, their correlation with the acute phase of neurologic illness, and the absence of other known cardiac risk factors were compatible with neurogenic stunned myocardium.

Massive catecholamine releases due to the stressful event of rapid respiratory deterioration, and the transiently damaged sympathetic nerve endings in the myocardium, presumably a consequence of GBS, have most likely caused the development of cardiomyopathy. In addition, norepinephrine infusion could have aggravated catecholamine excess, which might have contributed to the myocardial dysfunction[5].

One potential problem with our interpretation of the clinical data is that the patient had no baseline echocardiogram before the onset of his acute neuropathy. Therefore, one could argue that the wall motion abnormalities documented on his first echocardiogram might have predated the onset of his neurologic illness. However, he had no history of cardiac disease, and his ECG only began to show abnormalities as his neuropathy worsened. Severe inflammatory myocarditis has been described in GBS, but is extremely rare; although we did not perform a cardiac MRI, it would be unlikely to improve over a period of weeks.

In accordance with this background, the use of β-receptor-stimulating medication in patients with GBS and stress cardiomyopathy who have severe hypotension seems inappropriate. Non-β-receptor-stimulating agents like vasopressin might be beneficial[6].

A small number of cases associating GBS with stress cardiomyopathy have been reported in the literature. In the majority, cardiomyopathy has been signaled within the first week of admission and completely resolved on discharge[7]. Known

complications include heart failure (17.7%), recurrence (3.5%), and mortality (2.7%)[8].

Treatment is based majorly on angiotensin-converting enzyme inhibitors, β-blockers, and diuretics in patients who are stable hemodynamically. β-blockers are thought to lower sympathetic tone and improve the myocardial work/oxygen consumption ratio[9]. In hemodynamically unstable patients, the use of norepinephrine may be ineffective, and treatment must be individualized for each patient.

Conclusion

We report a case of stress-induced cardiomyopathy, which needs timely management in terms of diagnostics and treatment. Neurogenic stunned myocardium is a rare complication during the acute phase of GBS and requires the treating physicians to be alerted in order to limit the casualties linked to it.

Ethical approval

Not applicable, this is a case report.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution

C.B.: study conception; I.A.E.H.: study conception, data collection and analysis, and writing and editing; A.E.: study conception, data collection and analysis, and writing and editing; I.L., H.E.A., and O.H.: contributor; H.Bkiyar: supervision and review data validation; N.E.O.: supervision and review data validation; H.Brahim: supervision and review data validation.

Conflicts of interest disclosure

The authors declare that they have no conflicts of interest.

Guarantor

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Provenance and peer review

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