From weak knees to a troubled heart – a case of severe autonomic dysfunction in Guillain–Barré syndrome

Adriana Pacheco[a], Leandra Fagundes[b], José Beato Coelho[c], Raquel Ferreira[a], Ana Briosa[a]

[a] *Cardiology Department, Unidade Local de Saúde da Região de Aveiro, Aveiro, Portugal*
[b] *Neurology Department, Unidade Local de Saúde de Coimbra, Coimbra, Portugal*
[c] *Neurology Department, Unidade Local de Saúde da Região de Aveiro, Aveiro, Portugal*

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SOUHRN

Guillainův–Barrého syndrom (GBS) je autoimunitní zánětlivé onemocnění postihující periferní nervový systém, jehož rozvoji často předchází infekce respiračního nebo gastrointestinálního traktu. Závažnou komplikací je dysfunkce autonomních systémů, zvláště kardiovaskulárního systému, projevující se kolísáním krevního tlaku, arytmiemi a změnami na EKG záznamu. K rozvoji infarktu myokardu dochází vzácně; jeho diagnostika v dané situaci je obtížná.

Popisujeme případ 67letého muže s nedávno prodělanou infekcí horních cest dýchacích, který byl dopraven k lékaři s progredující slabostí dolních končetin. Rychle u něj došlo k rozvoji tetraparezie, dysfagie, respiračního selhání a akutního otoku plic, doprovázených změnami na EKG záznamu odpovídajícími akutnímu koronárnímu syndromu, se zvýšenými hodnotami troponinu. Koronarografické vyšetření nicméně nepotvrdilo obstrukční postižení, zatímco echokardiografické vyšetření prokázalo tranzitorní dysfunkci levé komory.

Pacientovi byl intravenózně aplikován imunoglobulin a současně byla přijata intenzivní podpůrná opatření. Neurologické vyšetření a vyšetření mozkomíšního moku později potvrdily GBS. Průběh onemocnění se prodloužil a zkomplikoval se rozvojem nozokomiálních infekcí, u pacienta však došlo k částečnému zotavení z neurologického hlediska a k úplné obnově systolické funkce levé komory srdeční.

Popsaný případ upozorňuje na význam důsledného monitorování kardiovaskulární funkce u pacientů s GBS, protože dysfunkce autonomních systémů může mít podobné projevy jako akutní koronární syndrom a může významně ovlivnit prognózu.

Keywords:
Acute coronary syndrome
Autonomic dysfunction
Guillain–Barré syndrome

ABSTRACT

Guillain–Barré Syndrome (GBS) is an autoimmune inflammatory disease of the peripheral nervous system, often preceded by respiratory or gastrointestinal infection. A serious complication is autonomic dysfunction, particularly cardiovascular, manifesting as blood pressure fluctuations, arrhythmias, and electrocardiographic changes. Myocardial infarction is rare and its diagnosis may be challenging in this setting.

We report the case of a 67-year-old man with a recent upper respiratory infection who presented with progressive weakness of the lower limbs. He rapidly developed tetraparesis, dysphagia, respiratory failure, and acute pulmonary edema, accompanied by electrocardiographic changes consistent with acute coronary syndrome and elevated troponin. However, coronary angiography revealed no obstructive disease, while echocardiography showed transient left ventricular dysfunction.

The patient was treated with intravenous immunoglobulin and intensive supportive measures. Neurophysiological and cerebrospinal fluid studies later confirmed GBS. His course was prolonged, complicated by nosocomial infections, but he achieved partial neurological recovery and full recovery of left ventricular systolic function. This case highlights the importance of careful cardiovascular monitoring in patients with GBS, as autonomic dysfunction may present with acute coronary syndrome-like features and significantly influence prognosis.

Introduction

Guillain–Barré Syndrome (GBS) is an inflammatory autoimmune disease that affects the peripheral nervous system, characterized by symmetric ascending paralysis and areflexia of sudden onset, generally preceded by a respiratory or gastrointestinal infection in the weeks before.[1,2] A common and serious complication of GBS is autonomic dysfunction, which frequently involves the cardiovascular

Address: Adriana Pacheco, Avenida Artur Ravara 35, 3810-164, Aveiro, Portugal, e-mail: adrianasreip@gmail.com
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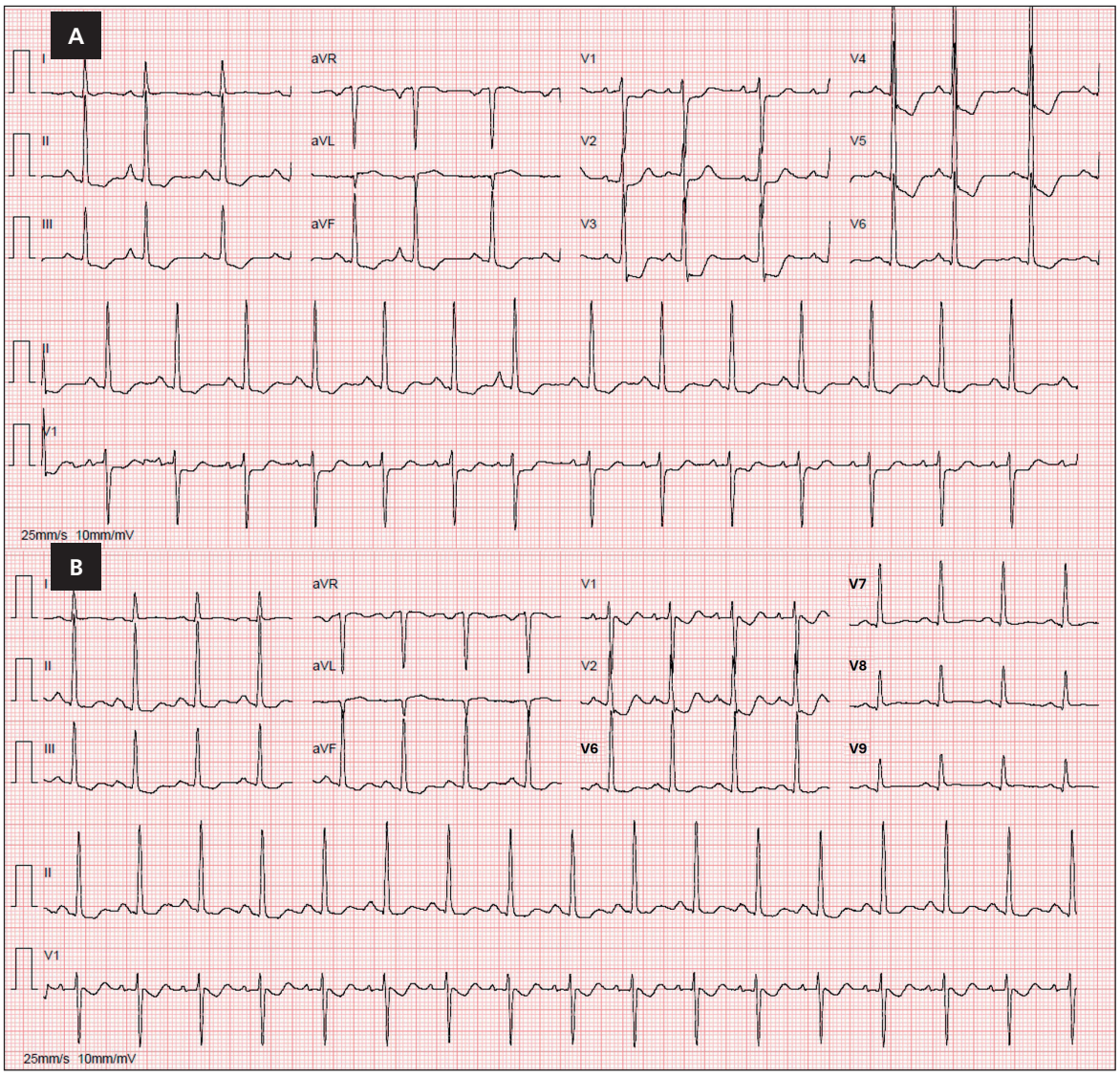

Fig. 1 – ECG during acute pulmonary edema. (A) sinus rhythm, HR 89/min, ST depression from V_2 to V_6; (B) same ECG with ST elevation in the posterior leads.

system, namely with fluctuations in blood pressure, arrhythmias, and electrocardiographic changes.[3] Myocardial infarction is rare, with only a few cases reported, and its diagnosis can be challenging due to the presence of electrocardiographic changes in patients with GBS.

Case report

A 67-year-old man, with a history of mild upper airway infection 3 weeks earlier, presented to the emergency room (ER) due to weakness in the lower limbs. Upon admission, he had a blood pressure (BP) of 169/90 mmHg and heart rate (HR) of 80/min, he was conscious and oriented, with no changes in cardiac and pulmonary auscultation, strength grade 3/5 in the lower limbs and 5/5 in the upper limbs. Sensitivity and reflexes were not tested. The blood tests (including troponin I), arterial blood gas analysis, ECG and head CT were normal. During his stay in the ER, he progressively developed weakness of the upper limbs, dysphagia and dyspnea. He maintained high blood pressure despite captopril. He suddenly developed acute pulmonary edema, diaphoresis, and cognitive impairment, with a BP of 204/110 mmHg, HR 150/min, with crackles on lung auscultation, and SpO_2 65% with FiO2 31%. Arterial blood gas analysis was performed with type 2 respiratory failure and respiratory acidemia: pH 7.28, pC_{O2} 53.1 mmHg, pO_2 45.6 mmHg, Lac 1.6 mmol/L, HCO_3 –24.6 mmol/L. The ECG showed sinus rhythm, HR 89/min, ST depression from V_2 to V_6 and ST elevation in posterior leads (**Fig. 1**). Repeated blood tests showed leukocytosis (19.7 G/L, N 4.1–11.1) with neutrophilia (17.8 G/L, N 2–7.5), normal renal function, elevated troponin I (2030 pg/mL, N <47.3) and NT- proBNP (928 pg/mL, N 0–125). An echocardiogram was performed which showed moderate depression of the left ventricular ejection fraction, with anterolateral and inferolateral akinesia. He was stabilized with non-invasive ventilation and intravenous furosemide and was sent for emergency

coronary angiography, which ruled out obstructive coronary disease.

After the procedure, approximately 12 hours after admission, his deficits worsened, being anarthric, unable to protrude the tongue, strength grade 0/5 in the lower limbs and 1/5 in the upper limbs, arereflexic and with weak chest expansion, with worsening respiratory failure and failure of non-invasive ventilation, and was then intubated and ventilated.

The most likely diagnostic hypothesis was Guillain–Barré syndrome with rapidly progressive onset, with severe respiratory failure and cardiac involvement.

He was accepted into the intensive care unit of the referral hospital for emergent plasmapheresis, however, after discussion with immunochemotherapy, it was considered unsafe to carry out this procedure due to high cardiovascular risk, which is why he was started on immunoglobulin 30 g/day for 5 days. He needed vasopressor support with norepinephrine, which was suspended on the 3rd day of hospitalization. He later developed a hypertensive profile, requiring antihypertensive therapy.

Neurophysiological and cerebrospinal fluid (CSF) examinations were consistent with the diagnosis of GBS. CSF examination showed an elevated protein level (45.7 mg/L, N 15–45) with normal cells (1.6/mm^3). The EMG on the 9th day of hospitalization showed severe, acquired, acute motor demyelinating polyneuropathy. She had a repeat EMG a week later, which showed acute motor axonal polyneuropathy.

The patient had a prolonged hospitalization, punctuated by nosocomial infections. Despite this, he showed slow and partial recovery from the deficits, and full recovery of left ventricular systolic function, and ended up being discharged to a rehabilitation unit.

Discussion

Autonomic dysfunction with cardiovascular involvement of GBS is widely recognized.[1,4–7] The most common electrocardiographic changes reported are sinus tachycardia, followed by ST segment changes, with ST segment elevation being relatively uncommon. Isolated elevation of troponin has also been observed.[4] These abnormal patterns could, in part, be explained by catecholamine-associated myocardial injury theories: a disorder of catecholamine uptake around myocytes,[8] redistribution of coronary blood flow,[9] or denervation hypersensitivity of the myocardium.[3]

An observational study reported that acute coronary syndrome (ACS) developed in 2.08% of patients with GBS.[4] Coronary vasospasm in patients with GBS, as a result of dysautonomia accompanied by increased catecholamines, also contributes to ACS. In our case, vasospasm could justify the patient's presentation, namely ST elevation, high troponin and segmental changes on echocardiogram, the without obstructive coronary disease. Cardiovascular complications are usually short-term, but can be fatal in GBS, being one of the determining factors in clinical outcome.[2,3] Thus, their assessment and treatment is of the utmost importance.

Conflict of interest
Nothing to disclose.

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Informed consent
The patient provided informed written consent for the publication of the study data.

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