Sino-atrial block due to Guillan–Barré syndrome: a rare case

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SOUHRN

Sinoatriální blokáda je stav charakterizovaný zpomaleným (nebo přerušovaným) přenosem vzruchů ze sinoatriálního uzlu do síní.[1] Dysfunkce sinoatriálního uzlu vede potenciálně k sinusové zástavě, bradykardii až asystolii, synkopě a srdeční zástavě. Za těchto podmínek je nezbytná implantace dočasného nebo trvalého kardiostimulátoru. Ke vzniku sinoatriální blokády dochází z nejrůznějších příčin, častěji je důsledkem hypertonie vagu, změn v koncentracích iontů nebo myokarditidy, případně blokáda vzniká z iatrogenních příčin.[2–9] Popisujeme případ 60letého muže s Guillainovým–Barrého syndromem, jemuž bylo nutno implantovat kardiostimulátor pro sinoatriální blokádu II.–III. stupně a symptomatické pauzy v délce 2,8 s.

Keywords:
Guillain–Barré syndrome
Pacemaker
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ABSTRACT

Sino-atrial node block is a condition characterized by a slowed (or interrupted) electrical impulse transmission from the sino-atrial node to the atrium.[1] The sino-atrial dysfunction potentially leads to sinusal arrest, bradycardia up to asystole, syncope and cardiac arrest. In such situations, a temporary or permanent pacemaker implantation is necessary. Different causes may be responsible for sinus atrial block, more frequently vagal hypertone, ions alterations, myocarditis and iatrogenic causes.[2–9] We report the case of a sixty-year-old man with Guillain–Barré syndrome who required pacemaker implantation for II to III degree sino-atrial block and symptomatic pauses lasting 2.8 s.

Introduction

Guillain–Barré syndrome (GBS) is an autoimmune-mediated, inflammatory, demyelinating, peripheral polyneuropathy which affects motor, sensitive and autonomic nerves. Typically, symptoms include a symmetric, ingravescent weakness starting from the lower limbs which worsen to flabby paralysis with proximal nerves involvement. Up to 10% of patients with GBS require intensive care management and mechanical respiratory support as consequence of diaphragm and respiratory muscles paralysis with respiratory failure.[10–12] Cardiovascular involvement is extremely rare, but cases of hemodynamic instability and bradyarrhythmias have been described.

Despite symptoms severity, majority of patients with GBS have full recovery in a period variable from weeks to months.[10–13] According to the European federation of Neurological Societies and Peripheral Nerve Society Guidelines, different GBS types are distinguishable on the base of nerves involved, recovery time and typical features: Acute inflammatory demyelinating polyradiculoneuropathy (AIDP), Miller Fisher syndrome (MFS), acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN), Bickerstaff encephalopathy, and acute autonomic neuropathy (AAN). The latter is probably the most rare and affects both the sympathetic and parasympathetic nervous system inducing orthostatic hypotension, anhidrosis, reduced salivation, and lacrimation. Cardiovascular involvement is common among patients with AAN, frequently with sinusal or atrial tachycardia, ventricular tachycardia or sinusal / atrio-ventricular block to asystole, in these latter cases giants T wave, ST alteration and presence of U wave are common features at electrocardiogram (ECG).[14–28] This GBS form is burdened by a slow, and frequently not full, recovery.[13] An infection with excessive

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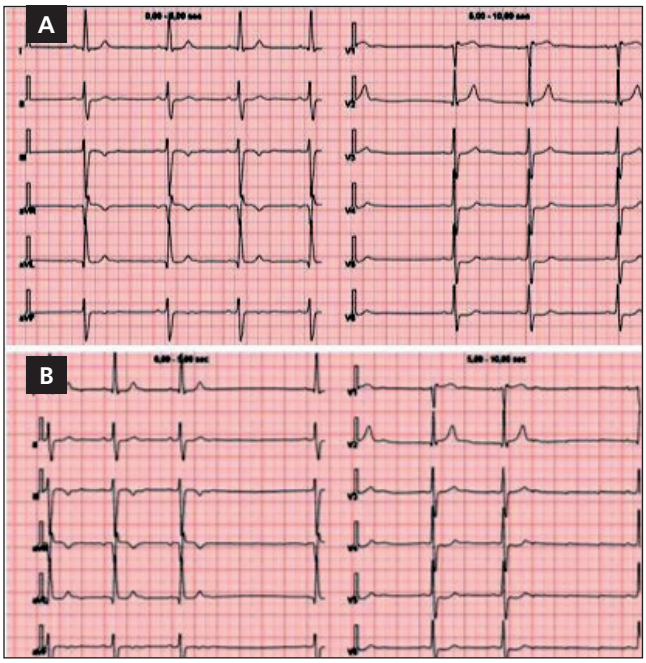

Fig. 1 – ECGs at admission (A, B). (A) SAB II. degree type 1 (progressive PP interval shortening followed by a pause; (B) SAB II. degree type 2 (3 : 1) (constant PP interval with pause lasting double of the preceding PP interval). In both A and B aspecific ST alteration and U wave, frequent features in bradycardia secondary to GBS, are observable.

immune response and antibodies cross-reaction against self-antigens, specifically gangliosides present on the Schwann cells, is usually the trigger of GBS.[14,15] Interestingly, lactose-containing gangliosides are also present in cardiac conduction system. This may in part explain cardiovascular involvement in GBS, however, autonomical dysregulation, consisting in overactivity of either sympathetic or parasympathetic system, seems to have the most important role in cardiovascular dysfunction among these patients.[17,18,24] Therapy for GBS consists of intravenous immunoglobulin administration and symptomatic therapy, with intesive care support if necessary. Although, conduction disturbances may persist in up to 50% despite immunoglobulin therapy. In this scenario a pacemaker (PM) implant may be the only solution.[27]

Case report

A sixty-year-old man was sent to our hospital emergency room because of worsening weakness and dizziness which had begun two weeks before. He had history of coronary artery disease (CAD) (previous elective surgical revascularization with triple coronary artery bypass) in annual monitoring using stress echocardiography. Single episode of psoriasis at a young age and GBS, for which he dismitted therapy on his own and dropped out follow up two years before. He underwent ultrasound Doppler evaluation for suspected venous insufficiency a few months earlier.[29–36] The first resting ECG showed an extremely low heart rate, 32 bpm, due to the second degree type I sino-atrial block (SAB) (Luciani–Wencheback) determining pauses up to 2.8 s; aspecific ST alteration and U wave were also present. Successive ECGs showed an alternance between SAB second degree type I and type II (Mobitz), the latter with a 3 : 1 conduction pattern.

Hemodynamics were stable (blood pressure 112/75 mmHg) and there were no signs of respiratory failure (peripheral oxygen saturation was 97% without oxygen support and his respiratory rate was 16 acts per min).

Blood chemistries were normal, including high-sensitive T troponin, thyroid and renal function, and showed no electrolytes alteration (Na^+ 136 mEq/L, K^+ 3.7 mEq/L, Mg 0.99 mEq/L and Ca^+ 9 mg/dL).

Then he was admitted to our cardiology unit to further investigate SAB origin. Once in our departement, any drug possibly interfering with SA node was stopped, in particular nebivolol was dismitted but without improvement on SAB. Transthoracic echocardiography was performed enlightening normal function and dimension of both left and right ventricle (ejection

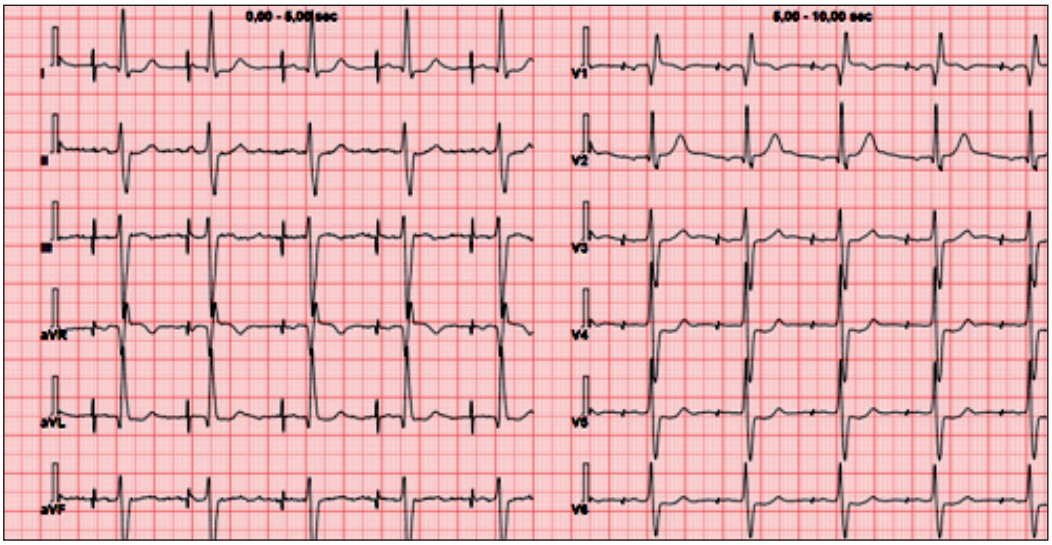

Fig. 2 – ECG after bicameral pacemaker implant, with 100% atrial paced.

fraction 55%, TAPSE 23 mm) without significative valvular disease.[37,38]

Putting together all the piece in the box, after collegial discussion with neurologists, an "ad adiuvantibus" diagnosis of SAB secondary to GBS flare-up was then hypothesized. Patient underwent permanent bicameral PM implant few days later and immunoglobulin therapy was restarted according to neurological indication. One month follow-up showed 100% paced atrial beats, confirming the importance of a permanent atrial pacing despite immunoglobulin therapy.

Discussion

Several causes may determine SAB: increased vagal tone, myocarditis, drugs (beta-blockers, calcium-blockers, procainamide, digoxin), acute or chronic myocardial infarction.[39–41] However, despite very rare, SAB GBS-related cases have been described as well in literature. According to our opinion, patient poor compliance to GBS therapy led to a syndrome flare up with cardiac involvement.[42,43]

Conclusion

GBS is a potential, albeit extremely rare cause of SAB. Dysfunction in GBS is usually reversible, however, improvement may require up to months and, especially in AAN GBS cases, sometimes may not be present irrespective to therapy, resulting in a permanent damage. In light of this consideration, a permanent PM implant may be the correct answer in patients with GBS and conduction systems disorders.

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