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Ventricular arrhythmia during rehabilitation of cervical spinal cord injury

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Abstract

Patients with cervical spinal cord injury have a high incidence of cardiac arrhythmias, especially in the first 14 to 30 days after traumatic event (acute phase). Electrophysiological abnormalities described in the acute phase are most often bradycardia, which is spontaneous or triggered by various stimuli. In the chronic phase, varied arrhythmias are described, but ventricular arrhythmias as a result of autonomic dysregulation in chronic SCI are rare and isolated. We present the case of a patient with a C5-C6 incomplete spinal cord injury (ASIA-B grade) in which symptomatic ventricular arrhythmia is described one year after the traumatic event.

Key words: *spinal cord injury, autonomic nervous system, ventricular arrythmias,*

Introduction

Incidence of traumatic spinal cord injury (SCI) varies around the world between 13 to 53 cases per million population, higher in males (male-to-female ratio of 2:1) and in young adults (20-29 years) (1). In SCI survivors, secondary conditions such as circulatory system impairments, could influence mortality and morbidity (1).

Heart rate and blood pressure control depends on the activity of supraspinal centers.In cervical spinal cord injuries, there is an imbalance between decreased or abolished sympathetic output and preserved parasympathetic output. The supraspinal control of sympathetic output originates in the medulla, with preganglionic fibers travelling through the spinal cord to reach the lateral horns at T1 to L3 levels, then synapse in the paravertebral sympathetic ganglionic chain and are distributed to the heart through sympathetic cardiac nerves (2, 3, 4). The dorsal vagal motor nucleusand nucleus ambiguus exert their influence on the heart by efferent parasympathetic output through the vagus nerve, which has a extra-spinal position, and is generally spared in cervical spinal cord injuries.

The spectrum of cardiovascular responses to SCI can be divided in two phases: the acute response, lasting from the time of injury to approximately 4 weeks following injury, and the chronic phase which follows.Within the acute phasethe neurogenic shock occurs, as the severe manifestation of sympathetic withdrawaland vagal over-activity, with consequent severe bradycardia and hypotension. Bradyarrhythmias,especially persistent sinus bradycardia, aredue to parasympathetic predominance, and appear in 66 to 100% of cervical SCI patients. The high level (cervical vs dorsal) and the severity of spinal cord injury increases the risk of bradycardia (3, 5).

In the chronic phase of SCI, below the lesion, some of the sympathetic spinal reflexes recover partially,independently of supraspinal control. In complete SCI lesion, the entire sympathetic efferent activity will be separated from cerebral control (6).In the absence of supraspinal inhibitory input, abnormal activation of sympathetic spinal cord reflexes by various stimuli induces pathologic cardiovascular effects, part of the syndrome of autonomic dysreflexia (7).

Autonomic dysreflexia (AD) is an abnormal spinal reflex-mediated response to stimuli delivered below the level of injury that consists of severe hypertension, dysrhythmias and vasoconstriction below lesion level (8). AD is caused by sympathetic hyperactivity as a response to a strong sensory stimulus below the level of injury (pain, infection, bladder / bowel distension).AD occurs in SCI patients with injury above T6 level, commonly

in the chronic phase, months or years after injury. Sympathetic-induced vasoconstriction generatesacute blood pressure elevation and bradycardia, rarely tachycardia or arrythmias (8). Coronary arteries spasm with secondary myocardial ischemia have also been described during AD (9).Excessive release of endogenous catecholamines during AD could lead to structural changes in myocardial fibers, increasing the oxidative stress of myocardial cells (10,11). Thisexplains why AD is a life-threatening conditionwith potentially severe consequences like stroke or death, if untreated (1), (8).

The pathologic neuronal signaling caused by the SCI predispose to arrythmias. Tachyarrhythmias like atrial fibrillation (AF) and ventricular fibrillation have been described in SCI patients with structurally normal hearts, remote from the time of injury, attributed to dys-synchronous autonomic discharge of AD, with bursts of sympathetic hyperactivity driven by ordinary stimuli (3, 12). More recently, the importance of the parasympathetic nervous system in inducing ventricular arrhythmias has been emphasized, but further studies are needed to clarify its role (13).

Case presentation.

We present the case of a 31year old male patient, which suffered a cervical spinal cord injury at C5 level in 2014 after a car accident. He was operated for spinal cord decompression and further stabilization with screw and rod system at C4-C5 and C5-C6 levels.The severity of SCI was rated ASIA-B on the American Spinal Injury Association (ASIA) Impairment Scale (AIS) (14).An acute upper gastro-intestinal bleeding with hematemesis occurred as early complication from a stress peptic ulcer, and emergency laparotomy with hemostatic ulcer excision was performed. Patient's evolution was further complicated by a severe urosepsis, recovered after antibiotic therapy. The patient started progressively an intensive rehabilitation program one month later.

One year after the spinal cord injury, the patient was admitted to our rehabilitation hospital. Neurological examination showed the picture of an incomplete C5-C6 spinal cord injury, converted to an ASIA-C grade: lower motor neuron deficit in upper limbs withimpaired wrist and fingerextension (2/5 on Medical Research Council MRC scale for muscle strength), less impaired wrist and finger flexion (3/5 on MRC scale), with hypotonus and muscle atrophies, upper motor neuron deficit in lower limbs (3/5 proximally and 2/5 distally on MRC scale) withspasticity and brisk reflexes, bilateral Babinski sign, decreased all sensory modalities below D4 level, incomplete micturition with self-catheterizations. The patient was able to walk with a rollator, for short distances.

At admission, the patient complained for intermittent irregular palpitations having no relation with effort or emotions.His blood pressure was 110/70 mmHg, heart rate 68 beats/min and no abnormal signs on physical examination of the cardiovascular system were noted. The 12-lead electrocardiography (ECG) has shown sinus rhythm, right axis deviation, without any other pathological changes (Figure 1).

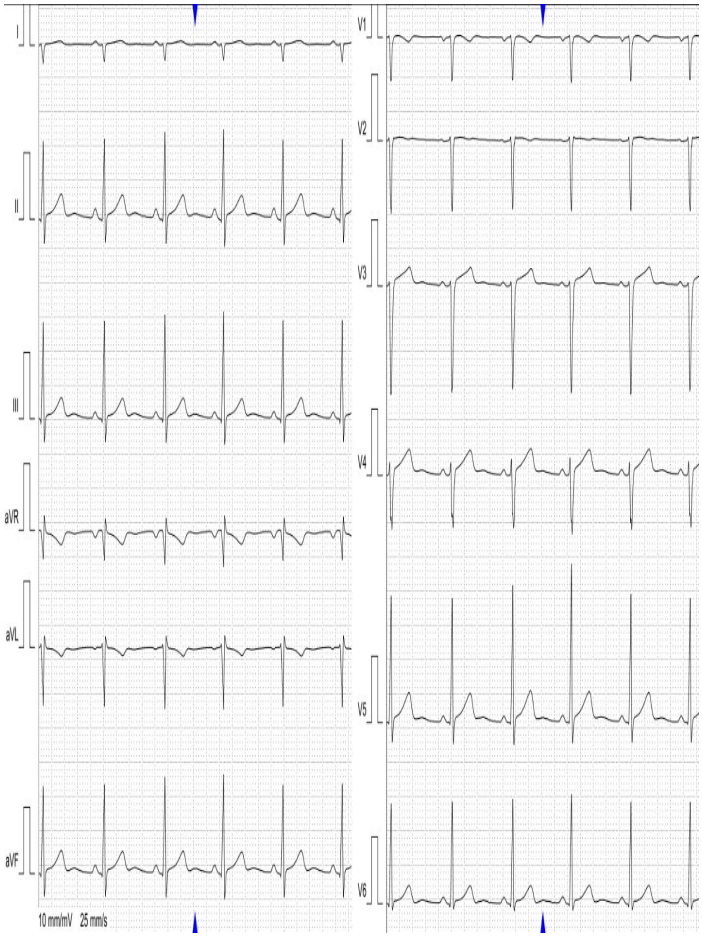

Fig. 1. Baseline ECG on admission

The 24-hours ECG Holter monitoring recorded 1937 ventricular premature beats (VPB) that had five morphologies and were isolated or coupled (Figure 2).

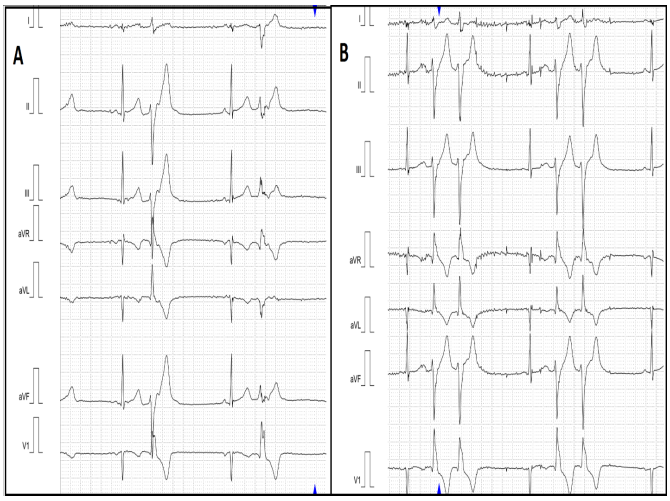

Fig. 2. Two samples of the first ECG Holter monitoring: polymorphic VPB (A) and some couplets (B). Note the low heart rate when VPB occurs.

VPB occurred mainly during the night (between 0:00 and 6:00) and accounted for about 2% of the total number of daily heart beats. Along the whole ECG monitoring period the mean heart rate was 61 beats/min but during night (when the VPB occurred) the mean hart rate was 50 beats/min. We must note normal chronotropic adaptation to effort (the maximum sinus rate was 167 beats/min). The cardiac ultrasound ruled out any structural cardiac disease.

He did not recognize alcohol or other drug consumption.Electrolytes levels in the serum were in the normal range, and thyroid function was normal (normal TSH and FT4 levels).The cardiac ultrasound ruled out any structural cardiac disease. A treatment with 25 mg od metoprolol tartrate was initiated and the ECG Holter monitoring performed 8 days later revealed significant improvement of the arrhythmia burden (72 VPB during 24-hours). The beta-blocker treatment did not influence the mean and minimum heart rate which remained similar.The patient was discharged on 25 mg metoprolol tartrate.

One year later the patient was admitted for follow-up.He did not have any cardiac symptom and the medication was interrupted at least three months before the follow-up visit. Reoccurrence of palpitations was noted in the last month, during a rehabilitation program in a balnear resort, which included daily sessions of hydrotherapy in thermal water. The Holter ECG monitoring performed at follow-up visit revealed around 500 VPB which had a single morphology (Figure 3), different from those recorded one year before, which tended to occur during night on bradycardia; the betablocker treatment resumption was recommended (nebivolol 2.5 mg od).

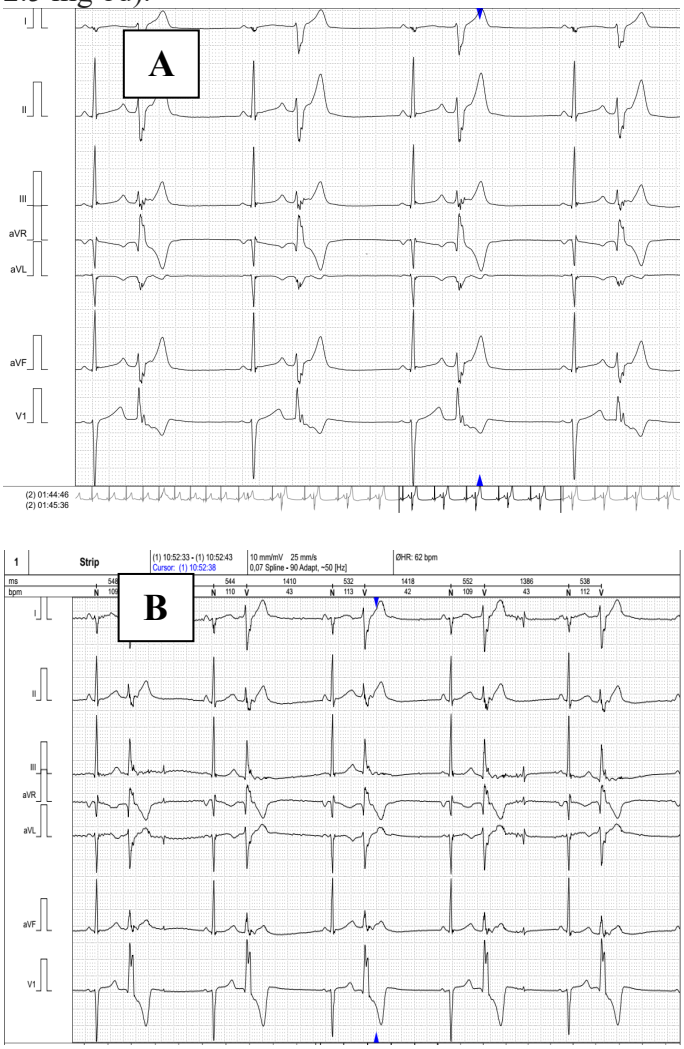

Fig. 3. The 3 years follow-up Holter ECG monitoring has shown arround 500 VPBs (A) having the same morphology (B).

Because of hypotension, the nebivolol had to be interrupted soon and Propafenone 150 mg bid was recommended. It was well tolerated and significantly reduced the VPB number.

Discussion

Ventricular premature beats (VPBs) or ventricular extrasystoles could be a common finding in individuals without any structural heart disease, and are considered a benign condition in the majority of cases, not requiring treatment, if they are clinically silent, their number is low, and their origin is notin the subepicardial layer.

However, sometimes the VBPs are symptomatic (palpitations, feeling of "skipped" heart beats), or, if they account for more than 24% of the total heart beats number, could result in tachyarrhythmia cardiomyopathy (15). That's why the mechanism is important to be known in order to assess the risks and to start the mostappropriate treatment.

The occurrence of VPBs in patients with cervical spinal cord injuries has not been described yet. The only recognized heart rhythm disturbance in the settings of cervical spinal cord injuries is sinus bradycardia which results from reactive hypervagotonia at the impairment of the cervical medullary sympathetic pathways leading to the distribution of preganglionic fibers that exit the central nervous system at the first through fourth thoracic levels of the spinal cord. Heart rate and blood pressure control depends on the activity of supraspinal centers. Disruption of descendent pathways results in sympathetic hypoactivity and disinhibited parasympathetic outflow. Within acute phase of spinal cord injuries, the neurogenic shock consists of severe bradycardia and hypotension. After spinal shock resolution, during the chronic phase, autonomic dysreflexia appears, and it is a syndrome of unopposed parasympathetic discharge with bursts of sympathetic hyperactivity induced by visceral stimuli. Our young patient had along the whole assessment period mild bradycardia (around 50 beats/min) and hypotension tendency which was counterbalanced by adequate hydration and orthostatic training.

The VPBs was an unexpected finding in our patient. They were associated with symptoms (palpitations), which the patient noted after the injury, so one could assume their relation with this event. Some possible mechanisms could explain this: a concomitant acute cardiac or metabolic condition (acute myocarditis with sequel, an electrolytic imbalance).

Myocarditis is a myocardial disorder often unrecognized, characterized by infiltration of inflammatory cells from infectious or autoimmune disorders. Even if often its cause remains unknown, myocarditis is reported to be the underlying mechanism in up to 50% of the patients with ventricular arrhythmias (16). The symptomatic premature ventricular contractions (PVCs) in the setting of supposed myocarditis are treated with beta-blockers or class III antiarrhythmic drugs; in our patient betablockers were effective even at low doses, but clinical intolerance due to hypotension lead to the treatment cessation. Class IC antiarrhythmic drugs could be an alternative in patients with non-ischemic etiologies and without significant myocardial scar. Even if the patient had e severe infectious episode in the subacute SCI phase, the normal systolic and diastolic left ventricular performance make a significant left ventricular scar less probable, even if it wasn't definitely ruled out by cardiac MRI or myocardial scintigraphy. The reoccurrence of VPBs after a one year free-of-symptoms interval makes this hypothesis less probable. The electrolytic imbalance should be easily ruled out by the biochemical tests at each follow-up in-hospital assessment. The follow-up standard 12-leads ECG was without QRS duration prolongation, thus we considered the pro-arrhythmic risk of propafenone quite insignificant.

A less-studied hypothesis is related to the autonomic imbalance as a favoring factor for VPBs. Autonomic influences are frequently involved in arrhythmogenesis and the relationship of autonomic tone to clinically significant arrhythmias are complex and different for specific arrhythmias (17). Both increased vagal tone or hypersympatheticotonia, could trigger arrhythmias. Using heart rate variability (HRV) by Holter ECG monitorization one has documented the correlations between increased vagal tone and some types of ventricular PBs (18). Autonomic imbalance could be a common pathophysiological pathway of many of well recognized triggers (postprandial state, alcohol consumption, caffeine containing beverages, exercise, anxiety or postprandial state, alcohol, caffeine containing beverages, energy drinks, exercise, stress, anxiety). The interval between the peak and end of the T-wave on the ECG and the QT variability index (QTVI) were found to be increased in high level SCI patients with consecutive autonomic lesions, underlying the increased risk for ventricular arrhythmias induced by autonomic impairment (19).

Parasympathetic parameters of HRV underscore the possible role of vagal control in enhancing ventricular ectopic activity.

Moreover it is reported that increased vagal activity could facilitate the occurrence of idiopathic ventricular tachycardia in some patients and even to favor the genesis of idiopathic ventricular fibrillation (18). That's why, the VPBs occurrence in our patient seems to be related to the reactive hypervagotonia, and possible triggered by other additional factors as transient electrolytic imbalance or increased myocardial inflammatory state. The specific circadian distribution pattern of VPBs does support this hypothesis.

Balnear therapy, based on the combined effect of natural therapeutic factors, mineral waters and hydrotherapy, is an important rehabilitation tool in increasing the quality of life inchronic SCI patients (20, 21). Aquatic immersion in warmwater temperatures influences the activity of autonomic nervous system, by decreasing sympathetic modulation while increasing vagal activity. In our spinal cord injured patient with previous autonomic dysregulation, the parasympathetic hyperactivity induced by thermal water could trigger the reoccurrence of ventricular arrythmias.

Conclusion.

The cardiac complications of spinal cord injuries are not negligible, their underlying mechanism could rely on autonomic imbalance or on myocardial (micro)injuries and sometimes, the treatment could be a challenge due to the predisposition to bradycardia and hypotension.Systematic assessment of the autonomic nervous system impairment in SCI patients should be implemented with standard tests (22). The SCI patients should be constantly monitored for cardiac arrythmias even in the chronic phase post-injury. Specific rehabilitation programs designed for SCI patients should take into account the dysregulation of the autonomic nervous system function and its consequences.

Conflict of interest

No conflict of interest for any of the authors regarding this paper.

Informed consent

An informed consent was obtained from the patient included in this article.

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