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Cardiology

Atrioventricular Block of Dyasautonomic Origin Complicated by Ventricular Asystole: A Case Report

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Abstract **Case Report**

Atrioventricular (AV) block is an interruption or delay to the electrical conduction due to conduction system abnormalities in the atrioventricular node or in the His-Purkinje system. In addition to intrinsic innervation, the conduction system receives an extrinsic innervation from the two contingents of the autonomic nervous system. In this paper, we present this case on atrioventricular block complicated by asystole owing to increased vagal tone to provide an insight into the contribution of the autonomic nervous system in the etiopathogenesis of conduction disorders.

Keywords: Atrioventricular (AV), asystole, nervous system, etiopathogenesis.

INTRODUCTION

Atrioventricular (AV) block is interruption or delay of electrical conduction due to conduction system abnormalities in the atrioventricular node or the His-Purkinje system. We present a case report on atrioventricular block complicated by asystole owing to increased vagal tone to provide an insight into the contribution of the autonomic nervous system in the etiopathogenesis of conduction disorders.

CASE REPORT

A 51-year-old women without medical history, was admitted for weakness, orthostatic intolerance and palpitations especially at night that started 6 months ago. Physical examination, electrocardiogram and transthoracic echocardiogram did not show any abnormalities. 24-hour Holter monitoring objectified paroxysmal asymptomatic second-degree atrioventricular block. She was referred to cardiology A department unit to explore her autonomic nervous system. She was initially placed in calm. We measured blood pressure, heart rate on admission and we did baseline electrocardiogram. Then, we preceded to cardiovascular autonomic tests interspersed with periods of rest, as describing by Ewing David [1, 2] and detailed by Phillip Low [3].

In the Deep Breathing test, we ask the patient to breathe deeply at a frequency of six breaths for one minute. It evaluates the vagal response. Electrocardiogram have showed, few second after the beginning, a first degree atrioventricular block (PR from 0,2s to 0.32s) (See Figure-1), then a passage in complete atrio-ventricular block (See Figure-2), then, a ventricular asystole during 3.2se (See Figure-3) reasons for stopping the test. Clinically, the patient hadn't felt any symptoms.

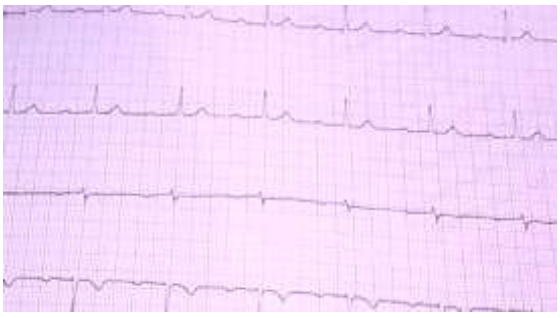

Figure-1: Electrocardiogram showing passage to first degree atrioventricular block (PR space at 0.32seconds)

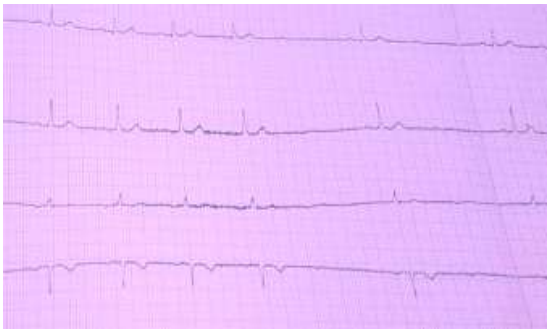

Figure-2: Electrocardiogram showing progression to complete atrioventricular block (30beat/min)

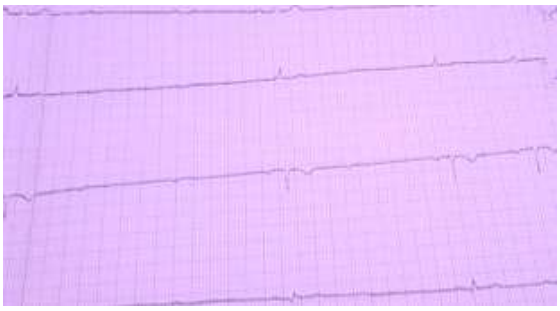

Figure-3: Electrocardiogram showing progression to ventricular asystole of 3,2 seconds

We then carried out the hand grip test, which consists of the patient applying a maximum manual pressure of 50% assisted by a dynamometer. It calculates peripheral sympathetic response α and can also evaluate vagal response. The mental stress test is based on doing arithmetic calculations and evaluate the central sympathetic nerves activities. We have obtained a value of 9% in favor of α deficiency and 7% in favor ß central sympathetic deficiency. The Table-1 summarizes the results from the different tests and interpretation of each one.

Table-1: Results of different tests and interpretation of each one.

Test		Result (%)	Interpretation
Vagal response	Deep breathing	74	Vagal hyperactivity
	Hand grip	25	
	Orthostatic test	20	
Peripheral sympathetic response α		11	Preserved peripheral sympathetic response
Peripheral sympathetic response ß		12	
Central sympathetic response α		9	Central sympathetic deficiency
Central sympathetic response ß		7	

The normal value is 30% for the Deep breathing test and 10% for remaining tests.

The autonomic profile of the patient is in favor of a vagal hyperactivity, as assessed by the three tests that evaluate vagal response (Deep Breathing, Hand grip and Orthostatism), and is responsible of high degree atrioventricular block and ventricular asystole during deep breathing test. Peripheral sympathetic response is preserved and there is a central sympathetic deficiency.

DISCUSSION

The autonomic nervous system is divided into two components: sympathetic and parasympathetic. The cardiac conduction system is densely innervated by each of these two divisions. The parasympathetic contingent acts by decreasing the contractility of the heart (negative inotropic effect), the heart rate (negative chronotropic effect), and the conduction velocity within the cardiac conduction system (negative dromotropic effect). It innervates the sinus node and the atrioventricular node via the two vagus nerves. The right vagus nerve supplies the sinoatrial (SA) node and slows its pacemaker, while the left vagus innervates the atrioventricular (AV) node [4].

Vagally mediated atrioventricular block is a functional and paroxysmal conduction disorder related to the parasympathetic influence on atrioventricular conduction [5] and is characterized by narrow QRS complexes [6]. It can occur acutely, during stimulation of the parasympathetic system (during vagal

stimulation, carotid sinus massage, emotion, etc.), or during the recovery phase after physical exercise. Clinically, it may manifest as discomfort or syncope. Vagally mediated atrioventricular block can be chronic and can be observed in young people and in high-performance athletes. In this case, it is asymptomatic and considered physiological.

Cardiovascular exploration of the autonomous nervous system is based on various cardiovascular autonomic tests that are performed while measuring the variability in the heart rate and blood pressure. Among these tests is the deep-breathing test, which is considered the main test for the exploration of parasympathetic function [7, 8]. It explores the variability of heart rate (HR) over 6 cycles of inspiration and deep expiration lasting 5 seconds each. During inspiration, the vagal tone is lifted, and the heart rate rises. The opposite happens on expiration. Measuring this variability allows a quantitative approach to assessing vagal activity. Indeed, the dysautonomic origin of certain conductive disorders related to vagal hyperactivity, such as atrioventricular block, can be revealed thanks to cardiovascular reactivity tests without having to resort to invasive tests.

In our clinical case, the deep-breathing test induced the appearance of high-grade atrioventricular block, which was complicated by asystole, indicating a hyperactivity of the left vagus nerve, which innervates the atrioventricular node. The increased vagal tone slows conduction in the atrioventricular node but do not affect His-Purkinj conduction. Thus, the vagally mediated atrioventricular block is benign because it is localized within the atrioventricular node and not in the His-Purkinj system [6]. It is thus a functional conduction disorder and not an expression of anatomical involvement [6]. On the other hand, our patient remained asymptomatic during the test and follow-up. For these reasons, we believe that there is no indication for pacemaker implantation as stated by the guidelines [9]. We decided to monitor the patient closely and over a long period of time, with the view that if symptoms appear and are clearly attributable to this conduction disorder, pacemaker implantation may be reasonable [10].

In order to treat vagal hyperactivity, two promising molecules were suggested for our patient: phenobarbital and maprotiline. Phenobarbital is a barbiturate, with sedative, hypnotic properties. It has been shown that it has a beneficial effect on the functional disorders of dysautonomia during vagal hyperactivity [11]. It reduces this vagal activity by damping the cortical centers [11, 12]. A preliminary study was conducted at the Autonomic Nervous System Unit of Cardiology Department A [13]**.** This study involved 16 patients who received phenobarbital at a dose of 30 mg/day for 3 months. We found a reduction in vagal activity in the deep breathing test from 56.5%±31 to 35.1% ±10.7 with a significant p = 0.02. We found a similar result for the hand grip test and orthostatism. Maproptilline is an imipraminic antidepressant, non-selective monoamine reuptake inhibitor, Noradrenaline reuptake inhibitor, an anticholinergic and histaminergic, decreases the presynaptic reuptake of noradrenaline and facilitates sympathetic transmission. Its anticholinergic effect contributes to the elevation of the heart rate. In a previous study [14], five patients with major vagal hyperactivity associated with severe symptomatic bradycardia underwent autonomic nervous system exploration after treatment with maprotiline (50 mg daily for three months). We had observed a spectacular functional improvement, with the regression of all signs (lipothymia malaise, vertigo, headache, etc.). Also, we had noted an increase in the basic heart rate by an average of 10 additional beats and a regression of the vagal hyperactivity by an average decrease of 50% compared to the initial average value. Thereafter, the invention was published describing a novel application of maprotilline in the treatment of autonomic dysregulation with severe symptomatic bradycardia related to its anticholinergic effect [14].

CONCLUSION

This case illustrates the contribution of autonomic nervous system exploration by using cardiovascular reactivity tests, in the detection of dyasautonomic origin (vagal hyperactivity) of a high degree conductive disorder. Future and larger studies are desirable to confirm the encouraging effects of phenobarbital and maprotiline in the treatment of severe vagal hyperactivity.

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