AN INTERESTING ETIOLOGY OF VENTRICULAR TACHYCARDIA

R. Roşulescu*

*“Carol Davila” Central Military Hospital, Dept. of Cardiology, Bucharest, Romania***Abstract**

Intermittent hypoxia and ventilatory intermittence due to sleep disorders such as obstructive sleep apnea can lead to cardiac arrhythmia, coronary artery disease, conductance disturbances and cardiac failure. Sub-clinical and overt hypothyroidism is an important physiopathological complex that is linked with cardiovascular risk, alteration of lipidic profile promoting atherogenic mechanisms. The two pathologies combined can exponentially increase the cardiovascular event risk.

This case report demonstrates a young patient with obesity and arterial hypertension accusing irregular heart beats. Despite a normal resting ECG and echocardiography the patient presented malignant ventricular arrhythmias revealed on the 24h Holter ECG. The Epworth scale was 20 and the patient presented numerous episodes of hypopnea with an increased apnea-hypopnea index associated with ventricular arrhythmias on the polisomnography. The endocrine profile of the patient revealed a subclinical hypothyroidism that untreated can lead to accelerated atherosclerosis and arrhythmic risk. CPAP treatment alongside the levothyroxine treatment at TSH levels above 10mUI/L have controlled the malignant arrhythmic events without complex antiarrhythmic drugs sustaining the pathogenic contributions of this two pathologies in arrhythmogenesis process.

Key words: cardiac arrhythmias, sleep disorder, hypoxia, subclinical hypothyroidism.

INTRODUCTION

Sleep disorders such as obstructive sleep apnea are known to increase the global cardiovascular risk. Intermittent hypoxia and ventilatory intermittence can lead to cardiac arrhythmia, coronary artery disease, conductance disturbances and cardiac failure. Subclinical and overt hypothyroidism is an important physiopathological complex that is linked with cardiovascular risk, alteration of lipidic profile promoting atherogenic mechanisms (1, 2). The two pathologies combined can exponentially increase the cardiovascular event risk. Severe obstructive sleep

apnea benefits from continuous positive pressure therapy (CPAP) and subclinical hypothyroidism can be amended by using levothyroxine substitutive therapy.

In patients with subclinical hypothyroidism even if the sleep apnea and the resulting hypoxia status is corrected by CPAP, without the substitutive treatment with levothyroxine the patient cardiovascular risk is still relatively high (4, 8).

CASE REPORT

We present a case of a 48 years male patient that was admitted at the emergency room accusing irregular heartbeats associated with chest pains and fatigue. These symptoms started four months ago and the intensity has increased between the starting point and the moment of presentation in the emergency room. The routine anamnesis revealed that he is non-smoker with partially controlled primary arterial hypertension using three antihypertensive classes of drugs (angiotensin converting enzyme inhibitors, calcium blockers and thiazidic diuretics), atherogenic dyslipidemia with high levels of triglycerides and abdominal obesity having a body mass index of 34.65 kg/m² and an abdominal circumference of 128 cm.

The clinical examination was not contributive for the main diagnosis. The heart rate was approximately 115 per minute rhythmic, without cardiac failure phenomena, and normal respiratory frequency.

The patient vitals are in normal range having a blood pressure of 135 over 80 mmHg; oxygen saturation measured by pulseoximetry was above 95%. The surface 12 lead standard electrocardiogram revealed sinus rhythm, a normal QRS axis, without ventricular depolarization anomalies, arrhythmia or conduction issues (Fig. 1). The biological data revealed from the blood analysis showed a high level of triglycerides unknown until admission and a hormonal pattern of subclinical hypothyroidism. All the haematology and biological data are presented in Table 1.

*Correspondence to: Razvan Roşulescu MD, “Carol Davila” Central Military Hospital, Dept. of Cardiology, 88 Mircea Vulcanescu street, Bucharest, Romania, E-mail: rosulescu.razvan@gmail.com

In addition to the thyroidian hormone status we should mention that adrenal and gonadal endocrine status was in normal range values.

The transthoracic echocardiography illustrated normal cardiac cavities, with a normal functioning valvular apparatus, without pericardial fluid, endocardial vegetations or left ventricular wall kinetic dynamics during the cardiac cycle.

A 24 hours Holter EKG revealed premature ventricular depolarization with the tendency of ventricular bigeminism, trigeminism and ventricular tachycardia, while the 48 hours Holter EKG depicted the same ventricular arrhythmia (Fig. 2 A-C) focused in the first part of it during the second half of the night.

Considering the cardio metabolic features of the patient, the pharmacological partial response to the antihypertensive medication presented, the evaluation of Epworth sleep apnea quantification scale was highly positive score – 20 points.

Taking into account the predominantly nocturnal cardiac arrhythmic events the patient was submitted to a polysomnographic evaluation (Fig. 3) which revealed over 16 apnea episodes lasting about 90 seconds each with a lowering of oxygen saturation down to 87% and 20 hypopnea episodes.

The apnea – hypopnea index was above 15, the patient being diagnosed with obstructive sleep apnea and continuous positive airways pressure - therapy (CPAP) was recommended. In addition, the pharmacologic treatment included L-Thyroxin, fibrates, antihypertensive drugs.

After 30 days of CPAP therapy and beta blockers therapy the polysomnographic results improved

considerably with the significant lowering of hypopnea and apnea episode from 20 to 5 and from 16 to 3.

Also we have to report the absence of palpitations and fatigue symptoms and the absence of ventricular systematic premature depolarization on the 48 hours Holter EKG after 30 days and 60 days of CPAP therapy.

DISCUSSION

It is a well known fact that sleep disorders can induce accelerated atherosclerosis, altered inflammatory and coagulation responses, cardiovascular diseases such as coronary artery disease, hypertension and arrhythmias.

The most common arrhythmias related to sleep disorders such as sleep apnea are represented by atrial fibrillation, atrio-ventricular conduction blocks

Figure 1. Emergency room admission EKG.

Figure 2. A: Ventricular tachycardia strip from Holter EKG; B: Ventricular bigeminy strip from Holter EKG; C: Ventricular trigeminy strip from Holter EKG.

Table 1. Biological data

Hb	14.8g/dL
L	7656/mm ³
PLT	270000/mm ³
Cho	170mg/dL
LDL - Cho	97mg/dL
HDL - Cho	56mg/dL
Glu	97mg/dL
Triglycerides	278mg/dL
Serum Urea	37mg/dL
Serum Creatinine	1.00mg/dL
Na ⁺	139mEq/L
K ⁺	4.5mEq/L
Mg ²⁺	2.01mg/dL
Ca ²⁺	4.33mg/dL
Serum TSH	6.4mUI/L
T4	7.9microg/dL
T3	1.3ng/mL
ATPO	20UI/mL

and ventricular events such as premature ventricular depolarization up to non-sustained or sustained ventricular tachycardia.

Several studies have shown a statistically significant correlation between obstructive respiratory events such as hypoxia, arousals and cardiac arrhythmias (1).

The DREAM study had the main objective to determine if sleep disordered breathing is connected with cardiovascular diseases and in particular with cardiac arrhythmia (1, 2).

The study population was represented by veterans who underwent polysomnographic studies according to which were categorized using the apnoea-hypopnoea index in severe (>15), moderate ($<5<15$) or none (<5) sleep disorders. From the total recordings were selected the nocturnal cardiac arrhythmias represented by complex ventricular premature depolarization, bigeminy, trigeminy, non-sustained and sustained ventricular tachycardia, atrial fibrillation, supraventricular tachyarrhythmia. The patients with an increased apnea-hypopnea index, above 15, after adjusting according to age, gender, cardiovascular diseases had frequent cardiac arrhythmias. Moreover, the arrhythmias were represented by ventricular complex events such as trigeminy and non-sustained ventricular tachycardia. Also atrial fibrillation was a cardiac event frequent in the patients with moderate and severe sleep obstructive apnea (3, 4).

Several studies revealed that sleep disorders such as sleep apnea can alter the repolarization physiology of the cardiomyocyte by altering the potassium channel normal function inducing an instability in the electrical potential of the cardiomyocyte increasing the risk of QT prolongation and cardiac arrhythmic events up to malignant ventricular events (5).

Padeletti *et al.* have demonstrated that hypoxia can induce a hyperactivation of the autonomic sympathetic system that can lead in alteration of the cardiac excitability and it may have a contributive role in the induction of cardiac arrhythmias (6).

In addition the patient has a subclinical hypothyroidism recently diagnosed. It is well known that clinical and subclinical hypothyroidism can be responsible for the growing intima-media thickness of carotid artery. Bozkus *et al.* in their study have demonstrated that patients with primary and subclinical hypothyroidism have a greater intima-media thickness of the common and internal carotid artery that can result in cerebral hypoxia and an increased risk of cardiovascular events such as malignant ventricular arrhythmias (7).

Figure 3. A sequence of polysomnographic evaluation.

This subclinical condition could have a contributing effect alongside with the obstructive sleep apnea in the cardiac arrhythmic events of our patient. Sleep deprivation is a very important health problem in modern world societies so we must recognize the presence of a potential sleep disorder and think to investigate further this possibility.

The common approach in subclinical hypothyroidism (SCH) is to initiate the levothyroxine treatment according to the levels of serum TSH. The main protocol is to begin the levothyroxine treatment at TSH levels above 5mIU/L (8). Ersoy *et al.* proposed that levothyroxine treatment in subclinical hypothyroidism should be initiated at values between 5 and 10mIU/L to prevent the cardiovascular complications of SCH. The secondary objective was to stop the increase in mean carotid artery intima media thickness (CIMT) in SCH patients with TSH level between 5 and -10 mIU/L.

A total of 60 patients with TSH level between 5 and -10 mIU/L were included in this study divided equally in two main groups with or without levothyroxine treatment.

The variables studied were the modulation of the cardiovascular risk factors during a 6 months study between the two groups and also the variation of the mean carotid artery intima media thickness. Blood pressure was measured at the beginning of the study and after 6 months in sitting position after 10 minutes of resting status. LDL cholesterol, HDL cholesterol, serum fibrinogen and homocysteine were determined from a standard 15mL venous blood sample. The carotid artery intima media thickness variation was determined by using a 2D high accuracy cervical ultrasound exam.

The results obtained regarding the systolic and diastolic blood pressure values, homocystein and fibrinogen levels in both groups were similar but hsCRP levels, LDL cholesterol and the mean carotid artery intima media thickness were significantly lower

($p < 0.001$) than the values obtained at baseline in the treated group (8).

Considering that high sensitive CRP is a surrogate parameter of atherogenic process and LDL cholesterol are the main atherogenic molecules linked to the foam cells development corroborated with the lowering of the mean carotid artery intima media thickness we can conclude that our patient, having a 6.4mUI/L TSH level, can benefit from levothyroxine treatment.

Conflict of interest

The author declares that he has no conflict of interest concerning this article.

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