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Case Report

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JUNCTIONAL TACHYCARDIA COMPLICATING A PHEOCHROMOCYTOMA: A CASE REPORT

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ABSTRACT

Pheochromocytoma is a rare and misleading cause of secondary hypertension (HT), with an often varied clinical presentation. It can manifest or be complicated by cardiovascular symptoms independent of hypertension, such as rhythm disorders that can be life-threatening, must be quickly linked to their cause to allow the establishment of appropriate treatment. These cardiac manifestations seem to be the consequence of a "toxic" effect induced by the elevation of catecholamine levels. We report the case of a 15-year-old child in whom a pheochromocytoma was revealed by a junctional tachycardia. The urinary dosage of free catecholamines revealed a very high level of noradrenaline, associated with the demonstration of a right adrenal mass on abdominal CT scan.

KEYWORDS: Pheochromocytomas, Rhythm disorders, Junctional tachycardia.

INTRODUCTION

Pheochromocytomas (PHEO) are rare neuroendocrine tumors arising from the chromaffin cells of the adrenal glands, whose prevalence generally estimated from hospital series in hypertensive patients is of the order of 1–2 in 10,000.[1]

They present a great clinical diversity[2] and cause a release of catecholamines, which can lead to cardiovascular complications such as transient ventricular dysfunction, myocardial ischemia and rhythm disorders.

Literature data show a relatively high incidence of cardiovascular complications (19.3%) in subjects with pheochromocytoma.[3]

Cardiac disorders in patients with pheochromocytoma are due to chronic tachycardiomyopathy and coronary vasospasm.[4]

Diagnosis is difficult because it is complex to link cardiac symptoms to these adrenergic tumors.

We report the case of a 15-year-old child in whom a pheochromocytoma was revealed by a junctional tachycardia.

Clinical Case

This is a 15-year-old child with no cardiovascular risk factors, no particular pathological history; who presented with palpitations with sudden onset and end that had been developing for 3 days associated with profuse sweating and abdominal pain in the epigastric region without irradiation or triggering or aggravating factors, without chest pain or dyspnea, no syncope or lipothymia.

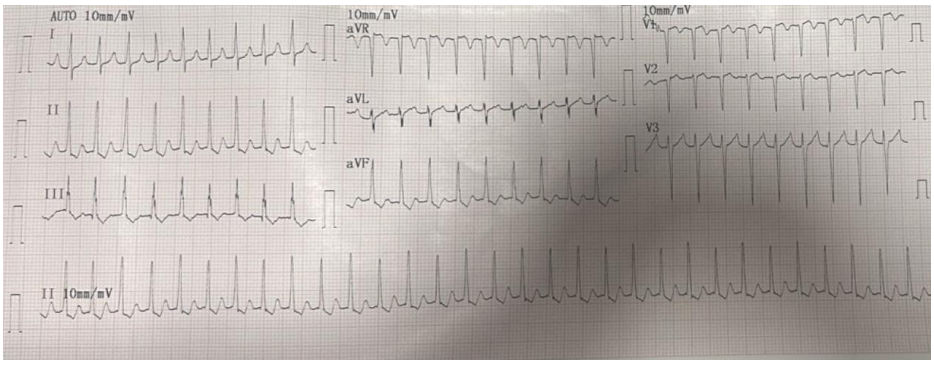
All of this developing in a context of apyrexia with occasional headaches. It should be noted that the patient had the same symptoms 1 week before, prompting him to consult a general practitioner where an ECG was done (no document), an assessment was requested but not done by the patient and he was put on beta-blocker. General examination: patient in fairly good general condition, normal-colored mucosa, no OMI Physical examination: Severe systolic-diastolic hypertension with a BP: 220/150 in the right upper limb and 198/153 in the left limb, Very fast pulse at 154 bpm Sao2 at 98% in room air.

Cardiac auscultation found regular tachycardia, no murmur or added noises Auscultation of the vascular axes was normal without murmur and the rest of the examination without particularity. On biological assessment: Hemoglobin at 16g/l, normal renal function, thyroid assessment and normal CRP The urinary dosage

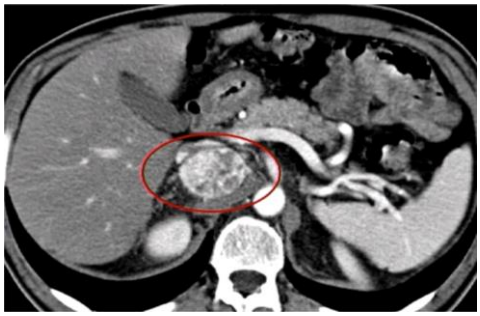
of metanephrine showed a very high Noradrenaline level at 1200ug/l or 15 times normal.

The electrocardiogram revealed a junctional tachycardia at 230 BPM. (well tolerated) Transthoracic ultrasound (performed after slowing down of the HR): non-dilated hypertrophied LV, site of slight global hypokinesia of preserved systolic function LVEF at 50% (SBP), low LVPR, non-dilated right cavity, dry pericardium.

After slowing down of the heart rate and in view of this clinical picture and the strongly positive biological results, an abdomino-pelvic CT scan was performed, objectifying a round right adrenal mass pushing back the upper pole of the right kidney measuring 65 mm in the transverse axis x 47 mm in the anteroposterior axis. After injection of contrast agent, the mass was well filled and heterogeneous, without stenosis of the renal artery. TJ was treated with a combination of alpha-blocker, beta-blocker and calcium channel blocker, which allowed good control of his blood pressure and a decrease in heart rate. The patient underwent surgery and the diagnosis of PHEO was confirmed by histological study of the surgical specimen.



ECG: Junctional tachycardia at 185 bpm with narrow QRS and non-individualized P waves, hidden in the QRS.



Abdominopelvic CT scan: Right adrenal mass measuring 65X47mm pushing back the right kidney filled and heterogeneous.

DISCUSSION

The cardiac manifestations of pheochromocytoma are the consequence either of the repercussions of hypertension or of prolonged catecholaminergic impregnation. In this case, they are often unrecognized and can have dramatic consequences. They can be revealing or complicate the evolution of an unrecognized symptomatic pheochromocytoma. The patient may be totally asymptomatic or have cardiac disorders such as paroxysmal or prolonged hypertension, heart failure and fatal arrhythmias. [2]

Palpitations are the second most common symptom of this disease (50 to 70%), after headaches.[5] Indeed, the excess of catecholamines leading to stimulation of β-adreno-receptors is generally associated with sinus tachycardia. On the contrary, other arrhythmias may also occur, especially supraventricular.[3] (as in the case of our patient) or even ventricular tachycardias.[6] Ventricular arrhythmias are a rare complication of PHEO, but they can also occur. In predisposed individuals, catecholamines cause QT prolongation with a subsequent risk of torsade de pointes, ventricular tachycardia.[7,8]. Occasionally, this potentially fatal complication may be the first presentation of PHEO, making the diagnosis of PHEO very difficult and important.

Furthermore, it is interesting to note that the action of catecholamines on the cells of the sinus node or atrioventricular node may also be associated with bradyarrhythmias, due to a reflex response to a sudden increase in blood pressure.[9]

According to the literature, 9 to 12% of patients with pheochromocytoma have cardiac complications[10,11] These complications are due to the increase in circulating catecholamines, which intensify peroxidative and lipoperoxidative metabolism at the level of cell membranes. This leads to the production of free radicals, thus causing myocardial damage, often reversible.[12]

In addition, some genetic polymorphisms of the beta-adrenergic receptor have been associated with severe left ventricular dysfunction, either by increasing sensitivity to catecholamines or by elevating synaptic noradrenaline levels due to a decrease in negative feedback.[13] This systolic dysfunction of the left ventricle is usually transient and reversible within a few days after the start of treatment with α-adrenergic receptor antagonists.[14]

Drugs that interfere with adrenergic stimuli can worsen a patient's condition, including beta blockers.[2] As in the case of our patient, where a hypertensive crisis occurred after the administration of a beta blocker to treat the tachycardia. This paradoxical reaction often helps guide the diagnosis of PHEO.

The diagnosis of pheochromocytoma as a cause of sinus arrest, atrioventricular dissociation or supraventricular arrhythmia is often late, which sometimes leads to the implantation of a pacemaker or ablation of the His bundle. In our case, the junctional tachycardia was controlled by a combination of alpha blockers, beta blockers and calcium channel blockers.

CONCLUSION

Despite significant progress in PHEO screening techniques, there are still a significant number of undiagnosed cases mainly due to a clinical presentation that is not very suggestive. The possibility of a pheochromocytoma should be kept in mind when faced with a rhythm disorder, particularly in young subjects with paroxysmal hypertension. The most effective prevention of cardiovascular complications related to pheochromocytoma is based on early recognition of this diagnosis "we must think about it!", because many complications, which can be life-threatening, can occur from the first episode, even without warning signs.

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