ELECTROCARDIOGRAPHIC CHANGES IN THE MOST FREQUENT ENDOCRINE DISORDERS ASSOCIATED WITH CARDIOVASCULAR DISEASES. REVIEW OF THE LITERATURE

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ELECTROCARDIOGRAPHIC CHANGES IN ENDOCRINE DISORDERS (Abstract): Cardiovascular abnormalities associated with endocrine diseases are often frequent and due to complex relationships between endocrine glands (with internal secretion) and cardiovascular system (heart and vessels). Certain hormones secreted by the endocrine glands (particularly the thyroid and pituitary gland) excesses or deficiencies, are involved in morphogenesis, growth processes and activity regulation of cardiovascular system, most often in connection with the autonomic nervous system. There are also a lot of electrocardiographic changes caused by hormonal disorders that requires differential diagnosis and represents the source of erroneous diagnosis. Endocrine pathology occurred later than a heart disease, may worse heart function. Ignoring the cardiovascular events that may occur in the evolution of endocrine diseases, may induce increased mortality due to cardiovascular complications. Keywords: ENDOCRINE DISEASES, ELECTROCARDIOGRAM, RHYTHM TROUBLES.

Endocrine pathology offers a large broadband of multiple interference and implications in cardiovascular pathology. These connexions are supported by complex relationships between endocrine glands (with internal secretion) and cardiovascular system (heart and vessels). It is well known that certain hormones secreted by the endocrine glands (particularly the thyroid and pituitary gland) are involved in morphogenesis, growth processes and activity regulation of cardiovascular system, most often in connection with the autonomic nervous system (1, 2).

This complex relationship between endocrine and cardiovascular pathology is not new.

In 1835, Robert Graves (one whose name will carry disease - Basedow Graves) described..."Three cases of prolonged violent palpitations in women with thyrotoxicosis...". Twenty years later, in 1855, Thomas Addison described in a highly suggestive phrase (which later would clearly outline the clinical picture of the disease)... "patients with small adrenal capsule have weak and compressible pulse and as a progress of the disease, the body loses weight, pulse becomes smaller and weaker, and the patient, after a long time and grad-
Electrocardiographic changes in the most frequent endocrine disorders associated with cardiovascular diseases. Review of the literature

Usually falls off... ".

Therefore since the mid-nineteenth century were known or suspected effects of thyroid hormone and adrenal gland diseases of the cardiovascular system (2).

The problem is up-dated now. A meta-analysis published in 2007 in the European Journal of Endocrinology emphasized that thyroid hormone excess would increase cardiovascular mortality of about 1.7 times. Another study published in 2011 confirmed the same fact, speaking about increasing cardiovascular mortality with 20% in persons with hyperthyroidism.

The interrelations between endocrine and cardiovascular pathology are important due to several reasons: 1. The cardiovascular symptoms may mask the endocrine disease. This is the major source of confusion and diagnoses errors 2. After diagnosis, the endocrine disease will require specific treatment. 3. Ignoring the cardiovascular events that may occur in the evolution of endocrine diseases, may induce increased mortality due to cardiovascular complications. Endocrine pathology occurred later than a heart disease, may worse heart function. There are also a lot of electrocardiographic changes caused by hormonal disorders that requires differential diagnosis and represents the source of erroneous diagnosis (1, 3, 6, 20).

Pituitary gland pathology

The excess of growth hormone (GH) also has implications in cardiovascular disease. Acromegalic cardiomyopathy is the most severe form of cardiac damage. The main clinical aspects are: arterial hypertension and early atherosclerotic signs induced by acromegalic cardiomyopathy.

On ECG the main abnormalities are: left axis deviation, left ventricular hypertrophy with T-wave inversion, septal Q-waves, ST-T wave depression, abnormal QT dispersion and conduction defects that occur in 50% of patients, left bundle-branch block, atrial and especially ventricular arrhythmias (induced by cardiac dilation). These changes are nonspecific. Moreover, sick sinus syndrome may be associated with acromegalic cardiomyopathy (1, 3, 4, 5, 7, 9, 10, 18).

Thyroid gland pathology

Thyroid hormones (thyrotoxicosis) excess induces both cardiac and vascular effects. The thyroid hormones activity may be performed through cardiac specific receptors or mediated by the sympathetic nervous system (which is over stimulated). The excess of thyroid hormones increases the metabolic needs of the tissues (high demand of oxygen in the peripheral tissues) (1, 2, 3, 21).

The main cardiac effects of excessive thyroid hormone are: increased contractility, automaticity and atrial excitability (with consequent risk of supraventricular arrhythmias) (2, 3, 19).

The vascular effects of thyroid hormones are: vasodilatation in skin and muscle and decreased peripheral vascular resistance (2, 3).

Cardiac manifestations of hyperthyroidism may be functional (as hyperkinetic syndrome) and organic (cardiothyreosis) (21).

Hyperkinetic syndrome - often brings the patient to the emergency room. The most common electrocardiographic abnormalities are: sinus tachycardia, hyper voltage, normal morphology of QRS complexes. Each of these entities has clinical and therapeutic particularities. Hyperthyroidism is considered an aggravating and precipitating factor of heart ischemia and heart failure.
As main characteristics:
- Atrial fibrillation from cardiothyreosis is first repetitive and then becomes permanent, with fast ventricular rate and resistance to digital treatment. The sinusial rhythm may be recovered only after achieving the euthyroidian status. Despite high ventricular rate, atrial fibrillation from cardiothyreosis is rarely followed by hemodynamic instability;
- Pectoral angina occurs frequently in patients with hyperthyroidism (hyperthyroidism aggravates or induces angina episodes); in some cases thyroid hormones may induce vasospasm in normal coronary artery. However, angina occurs in patients with subclinical coronary heart disease (coronary bed being affected by atherosclerosis, including microvascular injury);
- Hypothyroidism occurs due to insufficient synthesis, transport and/or reception of thyroid hormones. It’s cardiovascular effects are mainly the consequence of decreasing metabolic needs of myocardial or peripheral tissues (vascular permeability and capillary growth);
- Electrocardiographic abnormality in hypothyroidism (for differential diagnosis in the clinical context) includes: sinus bradycardia, micro voltage, QT prolongation, conduction disturbances, changes in repolarization phase, T wave flattening (1, 2, 3, 5, 6, 9, 10, 11, 12, 17).

Parathyroid diseases
Hyperparathyroidism (excessive secretion of parathyroid hormone), have the following consequences: hypercalcemia, hypercalciuria, hypokalemia, and hyperphosphaturia. The calcium ion increases myocardial contractility and peripheral vascular resistance and induces nephro-
calcinosi. Cardiovascular manifestations of hyperparathyroidism are very polymorphic: arterial hypertension, cardiac calcifications (may induce the occurrence of arrhythmias), accelerated atherosclerosis (pectoral angina, myocardial infarction) (23).

The characteristic ECG modification on hypercalcemia is QT interval shortening but it may induce also severe conduction disturbances: total AV block and sinus arrest, ventricular arrhythmias (PVCs, ventricular tachycardia, ventricular fibrillation) and promotes digitalis intoxication complications.

Hypocalcemia from hypoparathyroidism induces electrocardiographic abnormalities, consisting of: QT prolongation (may induce malignant ventricular arrhythmias) and changes in repolarization phase (ST) without stretching T-wave. Rarely, hypocalcemia may induce dilated cardiomyopathy which becomes reversible once with serum calcium values control (1, 2, 3, 5, 6, 16).

Adrenal diseases
Their correlation with cardiovascular pathology is quite complex, due to the effects of both mineralocorticoid and glucocorticoid hormones.

Hypercortisolism (excessive production of glucocorticoid hormone-dependent or independent of ACTH) has as main cardiac manifestations: moderate arterial hypertension, accelerated atherosclerosis (at coronary level may produce angina attacks). The main ECG abnormalities is secondary left ventricular hypertrophy (related to arterial hypertension or coronary atherosclerosis) (3, 8). Arrhythmias are rare and usually occur on the background of hypertensive left ventricular hypertrophy. Hyperaldosteronism (primary or secondary excess of mineralocorticoid) may induce
cardiovascular emergencies, both related to secondary hypertension or severe hypokalemia (may cause severe ventricular arrhythmias). Severe hypokalemia induces ECG abnormalities: QT prolongation, abnormal repolarization phase (slight depression of ST segment, flat T waves), U waves in precordial leads, prolonged P and PR interval duration (first degree atrioventricular block), wide QRS complex, ventricular arrhythmias (ventricular extrasystoles, ventricular tachycardia with evolution through ventricular fibrillation refractory to defibrillation). The severity of ECG abnormalities correlates with low potassium level (14, 15, 22, 24).

Primary adrenal insufficiency (Addison's disease) has humoral response causing hypoglycemia, hyponatremia, hypochloremia, hyperkalemia. Cardiovascular consequences are represented by hypotension and syncope and specific electrocardiographic changes related to electrolyte disturbances. Hyperkalemia causes various ECG abnormalities related to potassium levels: sharp, narrow base T waves, intraventricular conduction abnormalities (large QRS complexes exceeding 0.12 seconds), left axis deviation and then the decrease of P wave until disappears, ST-segment abnormalities (pseudo - lesion), arrhythmias and atrioventricular conduction disturbances. At high potassium concentrations (above 7.5 mEq / l) - cardiac arrest (14, 15, 22, 24).

Pathology of the adrenal medulla
Pheochromocytoma - chromatin tumor of the adrenal medulla is characterised by excessive secretion of catecolamines (adrenaline and noradrenaline). The clinical manifestations are represented by paroxystic seizures, paroxystic or permanent hypertension, sometimes orthostatic hypotension, arrhythmias, angina episodes. Electrocardiographic abnormalities in pheochromocytoma are nonspecific: negative T waves, left ventricular hypertrophy, sinus tachycardia, short PQ interval, supra ventricular arrhythmia (atrial premature beats, paroxystic or permanent atrial fibrillation) (13).

Adrenergic myocarditis and cardiomyopathy have clinical manifestation as: left ventricular failure or cardiac asthma, pulmonary oedema or severe arrhythmia (atrial fibrillation, premature ventricular beats or ventricular tachycardia). All of them represents emergencies and requires specific therapeutic measures. All these clinical and electrocardiographic signs of pheochromocytoma disappear together with the removal of the tumor (13).

CONCLUSIONS
Electrocardiographic changes in endocrine disorders are quite frequent and may be provoked by hormone excess (ex. hyperthyroidism) or by secondary electrolites disturbances (hypokalemia associated with primary or secondary hyperaldosteronism). Sometimes these electrocardiographic changes may be related to other cardiac abnormalities – such as arterial hypertension or left ventricular hypertrophy often associated with acromegaly or pheochromocytoma. Ignoring the cardiovascular events that may occur in the evolution of endocrine diseases, may induce increased mortality due to cardiovascular complications. In the majority of cases these electrocardiographic signs disappear together with the treatment of endocrine disease.
REFERENCES