TAKOTSUFO CARDIOMYOPATHY AND PANHYPOPITUITARIS - CASE REPORT

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Introduction
Takotsubo cardiomyopathy or Stress-Induced Cardiomyopathy (SICM) is a rare condition most commonly described in postmenopausal Japanese women, in a setting of severe emotional and/or physical stress. Although the condition is often misdiagnosed as an acute coronary syndrome, all the following criteria need to be met to establish the diagnosis of SICM: 1) transient left ventricular wall motion abnormalities, mainly akinesia of the apex with systolic ballooning that usually resolves within few days to few weeks; 2) absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture; 3) new ECG abnormalities or troponin elevation.

Pathogenic mechanisms such as coronary microvascular dysfunction, multivessel coronary vasospasm and catecholamine-mediated cardiotoxicity have been suggested as etiologic factors.

Case Report

Addmitted in the Emergency Room of a Public Hospital
- Odontalgia since few days ago, medicated with Acetanomiphen
- Constrictive chest pain one hour before that persisted for 15 minutes, while resting, with no irradiation.
- No nausea, no vomits, no hypersudorosis, no dyspnea, no visual fields defects

Physical Examination:
- AP: 126/88mmHg; HR: 60bpm; No signs of respiratory distress
- Cardiac auscultation: rythmic S1+S2


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The exclusion diagnosis was Takotsubo cardiomyopathy, so that patient was sent to Endocrine Outpatient Department

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Other important information were highlighted from his past history:
- Multiple syncopal episodes since the previous year that were not accompanied by chest pain, dyspnea, aura or involuntary movements, that lead to the pacemaker implantation.
- Some hypertensive paroxysms during last year each one lasting for 20-30 minutes. Patient denied headache, palpitation, hypoversudorosis, visual disturbance, pallor or anxiety during the episodes.
- No adinamia, anorxia, weight loss, cold/heat intolerance or sexual erectile dysfunction

Current medication: Diazepam 5mg p.o. 1id, Carvedilol 6,25mg p.o. 1id, Lisinopril 50 p.o. 1id, Pitavastatin 1mg p.o. 1id, Omeprazol 20mg p.o. 1id.

Physical examination:
- Yellowish skin color. Pale mucous membranes
- AP: 100/60mmHg; HR: 60bpm
- Scarse body hair

Discussion and Conclusion
We describe the clinical case of a man who was admitted in the emergency room of a public tertiary hospital because of chest pain. Although the first diagnostic hypothesis was an acute coronary syndrome, analytic evaluation, echocardiogram and Coronary percutaneous intervention excluded it, leading to SICM as the most probable diagnosis. This syndrome is not common in men neither in the western countries. So it possible causes were searched. This analysis included Cathocleumines and Metanephrines, regarding the toxic cardiomyopathy that often complicates Pheochromocytoma. As plasma adrenaline and metanephrine were slightly elevated, patient was sent to Endocrine Outpatient Department. Cathocleumines and Metanephrines were reevaluated, now in less stressing conditions, after treatment of the endotal pathology. Results were within the normal reference range. However, other endocrine disturbances were evident on basal evaluation: a panhypopituitarism that was confirmed by the pituitary multiple reserve test, whose cause was not clearly determined. We can hypothese that this disease may justify, at least in part, all the clinical complaints that patient presented during the previous year. Treatment with hydrocortisone 20mg/day p.o. levotiroxine 100ug/day p.o. and testosteron 250 mg/month/month was offered.

Several cases reports in the literature describe the association between Hypopituitarism and SICM and mainly between hypercortisolism and SICM. Some abnormal electrocardiogram findings are observed in adrenal failure even in patients with no cardiac signs or symptoms: QT prolongation, ST depression or deep inverted T. Authors speculate about the consequences of hypoglicemia or hyperglycemia on the cardiac sodium-calcum pumps dysfunction, however, in other reports, as in ours, those disturbances were not verified. Yamaoka O et al (1994) also speculated that transient cardiac damage may be induced by over catecholamine production, which is partially regulated by glucocorticoids. Moreover glucocortocoids seem to play a role in the cardiac myocytes protection and maintain its contractile function by controlling membrane calcium transport. Animal studies also demonstrated deplected mesional phosphorylase activity after adrenalec-tone. This fact impairs glycoInogenolysis and induces disruption of excitation-contraction cycle.