TAKOTSUBO CARDIOMYOPAPTHY AND PANHYPOPITUITARISM – CASE REPORT

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Introduction

Takotsubo cardiomyopathy or Stress-Induced Cardiomiopathy (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 akynesia 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, mutivessel coronary vasospasm and catecholamine-mediated cardiotoxicity have been suggested as etiologic factors.

Case Report

AM, 74 years old caucasian male
Retired Police officer
Portuguese
Pacemaker user since February 1st, 2012

February 20th, 2012

Addmited in the Emergency Room of a Public Hospital

-Odontalgy since few days ago, medicated with Acetanomiphen
-Constrictive chest pain one hour before that persisted for 15minutes, while resting, with no irradiation.
-No nausea, no vomits, no hypersudoresis, no dyspneia, no visual fields defects

Physical Examination:
-AP: 126/88mmHg; HR: 69bpm; No signs of respiratory dystress
-Cardiac auscultation: rythmic S1+S2

EKG: Pacing rhythm, 70bpm
Generalized invertion of T waves

Parameter	Reference values (RV)	Value (Fist evaluation)	Value (6 hours after beginning pain)
Hemoglobin	13-17,5g/dL	14	
WBC	4-11x10^9/L	11,49	
C Reactive Protein	<0,5 mg/dL	1	
Glucose	70-110 mg/dL	129	
Na+	135-145 mEq/L	139	
K+	3,5-5,1 mEq/L	4,5	
Creatinine	0,7-1,3 mg/dL	1,6	
Troponine	<0,07 ng/mL	**0,37**	**0,79**

Table 1: Analytic evaluation performed in the emergency room

Transthoracic Echocardiogram:
Akynesia of the apex and apical segments of all walls.
Heart base hypercontractility.
Global systolic function severely impaired.

Coronary Percutaneous Intervention:
No lesions documented.

Parameter	value	RV
Urinary adrenaline	**85,6**	0,6-19,9ug/24h
Urinary noradrenaline	61,3	15-80ug/24h
Urinary dopamine	135	65-399ug/24h
Urinary metanephrine	**382**	105-354ug/24h
Urinary normetanephrine	155	74-294ug/24h
Urinary Vanilmandelic acid	3,2	1,8-6,7mg/24h
Urinary Homovanilic acid	2,65	0,0-6,2mg/24h

Table 1: Analytic evaluation performed in the inpatient cardiology department

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

May, 2012

Other important information were highlighted from his past history:
-Multiple syncopal episodes since the previous year that were not accompanied by chest pain, dyspneia, aura or involuntary movements, that lead to the pacemaker implantation
-Some hypertensive paroxysms during last year each one lasting for 20-30minutes. Patient denied headache, palpitation, hypersudoresis, visual disturbance, pallor or anxiety during the episodes
-No adinamia, anorexia, 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, Omeprasol 20mg p.o. 1id,

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

Parameter	Value	RV	Parameter	Value	RV
Hemoglobin	13	13-17,5 g/dL	FSH	1,7	1,4-18,1 U/L
WBC	8,47	4-11x10^9/L	LH	**0,25**	3,1-34,6 U/L
Creatinine	1,1	0,7-1,3 mg/dL	T. Testosterone	**0,1**	240-820 ng/dL
Na+	141	135-145 mEq/L	Total PSA	<0,01	<4 ug/L
K+	4,8	3,5-5,1 mEq/L	ACTH	9,5	0-46 pg/mL
Glucose	94	70-110 mg/dL	Cortisol	**3,8**	4,3-23 ug/dL
T. Cholesterol	**208**	<190 mg/dL	SDHEA	**<15**	80-560 ug/dL
LDL Cholesterol	105	<110 mg/dL	Renin	5,5	1,6-14,7 pg/mL
TSH	0,72	0,55-4,78 uU/mL	Aldostenone	56,5	40-310 pg/mL
FT4	**0,68**	0,8-1,76 ng/dL	PRL	12,4	2,1-17,7 ng/mL
Anti-TPO Ab	48	<60 U/mL	GH	0,1	<3ng/mL
Anti-Tg Ab	<20	<40 U/mL	NSE	**17,1**	0-16,3 ug/L

Table 3: Basa analytic evaluation performed in the Endocrine Department. Urinary cathecolamines and metanephrines were again determined, now in the normal range (results not presented)

Parameter	0 min	30 min	60 min	90 min	120 min
Glucose	106	58	36	74	55
GH	0,15	0,12	0,18	0,17	0,13
PRL	21	32	35	32	29
ACTH	31	37	46	36	32
Cortisol	1,5	2,3	2,6	4,1	2,9
TSH	3,87	6,97	15,5	16	18
FSH	2,2	2,8	2,7	2,6	2,3
LH	0,3	0,6	1,0	1,1	1,1

Table 4: Pituitary multiple reserve test. Administration of TRH 200ug, LHRh 100ug and 7U of regular insulin, all as ev bolus

Sellar CT scan:
No pituitary lesions documented.

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 that possible causes were searched for. This evaluation included Cathecolamines 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. Cathecolamines and Metanephrines were reevaluated, now in less stressing conditions, after treatment of the odontal pathology. Results were within the normal reference range. However, other endocrine disturbances were evident on basal evaluation: a panhypopiyuitarism that was confirmed by the pituitary multiple reserve test, whose cause was not clearly determined. We can hypothise that this disease may justify , at least in part, all the clinical complaints that patient presented during the previous year. Treatment with hydrocortisone 30mg/day p.o., levotiroxine 100ug/day p.o. and testosterone 250 mg 1administration/month IM was offered.

Several case reports in the literature describe the association between Hypopituitarism and SICM and mainly between hypocortisolism 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 hyponatremia on the cardiac membrane sodium-calcium pumps dysfunction, however, in other reports (as in ours) these disturbances were not verified. Yamanaka O et al (1994) also speculated that transient cardiac damage may be induced by overt catecholamine production, which is partially regulated by glucocorticoids. Moreover glucocorticoids seem to play a role in the cardiac myocytes protection and maintain its contractile function by controlling membrane calcium transport. Animal studies also demonstrated depleted microsomal phosphorylase activity after adrenalectomy. This fact impairs glycogenolysis and induces disruption of excitation-contraction cycle.

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