Cardiothyrotoxicosis: where are we in Marrakech?

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Background: Hyperthyroidism may complicate preexisting cardiac disease or may cause cardiac complications in individuals with structurally normal hearts defining cardiothyrotoxicosis. They are underdiagnosed, due to its low occurrence in series from Africa. The aim of this study is to assess the incidence, the demographic data, diagnostic features and etiological aspects of cardiothyrotoxicosis among hyperthyroidisms.

Methods and results: Since August 2004 we have prospectively enrolled 348 patients with hyperthyroidism in cardiology and endocrinology departments in 03 hospitals of Marrakech, we have divided them into 2 groups: 58 cases with cardiothyrotoxicosis (group I) and 290 with only hyperthyrotoxicism (group II).

Cardiothyrotoxicosis was observed with an incidence of 16.6%. The mean age was respectively of 45.5±13.3 versus 33.8±11.4 years (p<10^-6). Cardiothyrotoxicosis was related to multinodular goiters (30 cases, 52%), while the principal cause of hyperthyroidism was toxic adenoma (122 cases, 42%). The clinical profiles of cardiothyrotoxicosis were dominated by heart failure in 44 cases (75%) and atrial fibrillation in 33 cases (57%). The other modes of presentation were: The atrial flutter in 02 cases, ventricular extrasytole (trigeminism) in 02 cases, second auriculoventricular block in 02 cases, dilated myocardiopathy in 10 cases, coronary heart disease occurred in 05 cases and severe mitral regurgitation in 02 cases. Treatment requires necessarily return to euthyroidism.

Conclusion: This study confirms the relative frequency of cardiothyrotoxicosis, the proportionally weak place of Basedow disease among hyperthyrotoxicosis causes and the involvement of associated cardiac disease and higher age in cardiothyrotoxicosis.

Pre clinical mutation carriers from families with dilated cardiomyopathy and angiotensin conversion enzyme inhibitor (the PRECARDIA study)

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Context: Dilated Cardiomyopathy (DCM) is familial in at least 30% of cases, usually with autosomal dominant inheritance, and underlying genes/mutations are increasingly identified. Familial DCM is characterized by age-related penetrance, that means that the cardiac expression of the disease is usually absent for a long period and progressively appears with advanced age, usually after 20 years of age.

Aim: Study the impact of ACE inhibitors (ACEi) in subjects who carry a mutation (leading to a genetic form of heart failure) but have not yet developed DCM (cardiac expression).

Design: This is a multicentre European double-blind randomized and controlled trial with perindopril or placebo in the context of a European consortium “INHERITANCE” (FP7 European Union, HEALTH-2009-2.4.2-3, Grant agreement n° 241924). The PRECARDIA trial was recently approved by the Head of medicines Agencies in Europe (HMA) and by AFSSAPS in France. We plan to enroll 200 participants from 7 European centres/countries. Follow-up duration is 3 years after inclusion. Primary Endpoint is a composite end-point: occurrence of DCM or deterioration of LV end diastolic diameter/ volume or Ejection fraction (echocardiographic or magnetic resonance imaging). Secondary Endpoints are related to the evolution of other echocardiographic parameters or hormonal biomarkers (including Mid-Regional pro-Adrenomedullin).

Expected results: If the hypothesis is confirmed, and as a consequence, the knowledge derived from basic research (genes identification in DCM) will be translated into clinical practice (early identification of subjects at high risk of developing heart failure through predictive genetic testing) with the development of new therapeutic management (early ACEi) that will help to decrease the morbidity and mortality associated with the disease. This will constitute a paradigm of the development of preventive medicine thanks to the development of genetics in the cardiovascular field.

Analysis of BNP and troponin I to differentiate apical ballooning syndrome from acute coronary syndrome

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Objectives: We sought to analyze B-type natriuretic peptide (BNP)/troponin I (TnI) ratio and TnI kinetics to differentiate apical ballooning syndrome (ABS) from acute coronary syndrome (ACS).

Methods: In 62 ABS, TnI and BNP measurements were collected prospectively and compared to 90 patients with anterior ST-segment elevation myocardial infarctions (STEMI) (n=47) and anterior non-STEMI (n=43).

Results: In case of persistent ST-segment elevation, BNP/TnI ratio above 165 can identify ABS with accuracy (sensitivity: 95%, specificity: 98%). In the absence of persistent ST-segment elevation, a ratio above 515 can identify ABS (sensitivity: 57%, specificity: 86%). Decreasing TnI profile was found more in ABS than in ACS. TnI peak occurred earlier in ABS than in ACS. A normal TnI on patient admission may exclude ABS.

Conclusions: Analysis of BNP and TnI provides a simple, non-invasive method to differentiate ABS from ACS.