Non-ST-segment elevation myocardial infarction in patients with no significant lesions in coronary arteries and non-contributory CMR: utilization and efficiency of secondary prevention therapies

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Introduction: Utilization and Efficiency of Secondary prevention therapies in patients presenting with NSTEMI associated with normal or near-normal findings on angiography and non-contributory CMR is still unknown

Objectives: The aim of our study is to compare the rates of secondary prevention medication prescription and outcomes in patients with approved ischemic cause on cardiac MRI versus patients with non-contributory CMR

Methods: A retrospective monocentric study enrolling all subsequent patients hospitalized for NSTEMI without significant lesions in coronary arteries (stenosis < 50%), between January 2004 and January 2010. Patients were divided into two groups, depending on CMR findings. In the first group, 43 patients with confirmed myocardial infarction and the Group 2 consisted of 22 non-contributory CMR patients. Patients with confirmed acute myocarditis were excluded from the study. We measured rates of aspirin, statin, β-blocker, and angiotensin-converting enzyme inhibitor (ACEI)/angiotensin II receptor blocker (ARB) prescription at hospital discharge and a mean follow up of 55 months among eligible patients.

Results: Baseline clinical, electrocardiographic and echographic characteristics of the patient population were similar between the 2 groups. At hospital discharge, patients with non-contributory CMR had significantly lower rates of aspirin (68.1% versus 90.9%, p<0.05), statin (36.3% versus 76.8%, p<0.001), β-blocker (31.8% versus 64.3%, p<0.001), and ACEI/ARB (36.3% versus 76.2%, p<0.001) prescription as compared with confirmed ischemic patients. After a mean follow up of 55 months, patients with non-contributory CMR remained significantly less likely to receive prescriptions of secondary prevention therapies compared with the ischemic group (all probabilities values <0.001). MACE (Death, non fatal MI and TLR) was significantly higher in the ischemic group (12.2% vs 0%) (p<0.001).

Conclusion: Our study demonstrates that patients with non-contributory CMR have an excellent prognosis although they received a lower rate of secondary prevention medication prescription at hospital discharge and long term follow up, as compared with patients with confirmed myocardial infarction. These findings highlight an opportunity to clarify the care of this group.

Materials and Methods: it is a retrospective study including 15 patients hospitalized in our service over a period of seven years from 2006 to 2013, presenting a pericarditis.

Results: It was of 15 patients including 5 men and 10 women, with a sex ratio of 0.5. The average age of our patients was 49 years (26 to 83 years). The pericarditis was revealed by: dysphonia (86% of cases), a chest pain (20% of cases), a flu-like syndrome with a moderate fever (13% of cases) and edema of the lower limbs (26% of cases). All patients had ultrasound a pericardial effusion well tolerated (moderate to abundant) except four patients who were in tamponade. She was of viral origin in 4 cases. It was in relation to a systemic disease in 6 cases: a systemic lupus erythematosus isolated in 3 cases, a rheumatoid arthritis in one case and a Sjögren syndrome in one case. In three cases the pericarditis was of renal origin. In addition, the pericarditis was secondary to radiotherapy for thymus in one case. In a single patient no etiology has been found. The evolution under specific medical treatments was good except of three patients who needed a surgical treatment with transition to chronicity in two cases.

Conclusion: The pericarditis is often easy to diagnose. A complete etiological investigation allows an earlier and adapted therapeutic. A good collaboration between an interist and cardiologist would allow an effective prevention of the acute risk of tamponade and chronic evolution toward the constriction.

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The uterine and vascular actions of estetrol delineate an original distinctive profile of estrogen receptor α modulation, uncoupling nuclear and membrane activation

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Estetrol (E4) is a natural estrogen produced by the human fetal liver only during pregnancy. Its distinctive biological profile offers potential benefits in women’s health, such as contraception and hormone replacement therapy. The crystal structures of the estrogen receptor α (ERα) ligand binding domain with 17beta-estradiol (E2) and E4 were very similar, whereas these two estrogens showed distinct positioning within phospholipid bilayers. Using in vivo approaches, we demonstrated that high doses of E4 stimulate genomic, ERα-dependent effects in the uterus leading to epithelial proliferation and prevent atheroma in hypercholesterolemic mice to a similar extent as E2. In contrast to E2, however, E4 failed to promote membrane-initiated ERα signaling such as acceleration of endothelial healing. Moreover, E4 antagonized this endothelial effect of E2. We conclude that E4 is a weak estrogen able to modulate the nuclear transcriptional activity of ERα, and is not only devoid of membrane-initiated steroid signal, but also able to antagonize the membrane effects of E2, thereby delineating a distinctive profile of ERα activation.