Mechanisms & implications of coronary microvascular dysfunction in patients without structural heart disease or obstructive atherosclerosis.

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Investigations of coronary artery disease(CAD) in humans are most often limited to the 'tip of the iceberg' i.e. to the large epicardial coronary arteries. Actually, conductive arteries, prearterioles (500-100 μ), arterioles (<100 μm)form a rich network. The microcirculation controls coronary resistance and myocardial blood flow. Function of coronary microvasculature can be approached by measurement of CBF (IC Doppler velocity, Positron-emission tomography, magnetic resonance etc..). The coronary flow reserve (CFR) can be studied by studies of CBF at rest and during hyperaemia. In patients with angiographically normal coronary arteries and decreased CFR(<2)one can suspect that the symptoms are related to microvascular disease. There are different types of microvascular disease in the absence of obstructive CAD (1) With or without myocardial diseases (2) iatrogenic coronary microvascular dysfunction.

In this presentation we will consider microvascular dysfunction without myocardial diseases (cardiomyopathy), i.e. related to hypertension, diabetes, and Syndrome X. The latter is frequent in women complaining of angina-like chest pain. The mechanisms are still not completely understood. The main hypothesis would be Ischemia in small myocardial areas surrounded by normal zones with normal / increased contractility and located on small prearterioles. Another hypothesis is abnormal pain perception. The vital prognosis is not engaged (no risk of death or MI) but the functional prognosis is very bad: Recurrence of pains poorly controlled by the treatment. In all these cases, the treatment of hypertension, dyslipidemia, the use of statins, oestrogen therapy and ACE inhibitors is usually proposed with various results