How would you treat: glucagon-like peptide 1 receptor analogue or sodium-glucose cotransporter 2 inhibitor in chronic coronary syndrome and heart failure in type 2 diabetes?

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Abstract

Background. The most common cause of early death in acute myocardial infarction with ST elevation are malignant heart rhythm disorders, generally occurring in the first four hours of myocardial infarction. The incidence of ventricular fibrillation is greatest in the early stage of the myocardial infarction, and sudden cardiac deaths occur most often in outpatient conditions.

Case reports. This paper presents a patient whose first manifestation of coronary artery disease was myocardial infarction with ST elevation complicated by early ventricular fibrillation. Rapid measures of cardiopulmonary resuscitation enabled quick establishment of normal sinus rhythm. Primary percutaneous intervention was performed, with revascularization of artery responsible for acute myocardial infarction. In order to reduce ischemic brain damage, therapeutic hypothermia was applied since the patient was presented in post-reanimation coma.

Conclusion. Better treatment of patients with cardiac arrest in outpatient conditions and faster revascularization of the infarct artery are crucial for a reduction of mortality in acute myocardial infarction.

Key words: acute myocardial infarction, cardiac arrest, modern treatment

Introduction

Patients with type 2 diabetes mellitus (T2DM) are at high risk of vascular complications and heart failure (HF). We present a case with T2DM, coronary artery disease (CAD) and heart failure with preserved ejection fraction (HFpEF).

Case presentation

A 58 years old male with T2DM was admitted to regional hospital with dyspnea and chest pain, elevated level of Troponin T and ECG changes interpreted as non-ST-elevation myocardial infarction. Invasive coronary angiography revealed diffuse epicardial coronary disease, with 50% diameter focal stenosis on all of three coronary arteries and stress echocardiography was recomended. Several weeks later, he develops hypertensive crisis and pulmonary oedema. Afterwards, a single-photon emission computerized tomography was performed and showed only 7% of ischemic myocardium in two segments: basal posterior and apical. At hospital readmission, patient had symptoms and signs of HF with elevated arterial blood pressure of 160/90mmHg and unremarkable ECG. Laboratory tests showed elevated natriuretic peptides (NTproBNP) of 2553pg/ml, and preserved renal function by creatinine clearance (ClCr) of 74.4ml/min. Transthoracic echocardiography revealed left ventricular (LV) ejection fraction of 53%, measured from biplane images, with LV hypertrophy, left atrial enlargement and mild mitral regurgitation. LV diastolic dysfunction showed the ratio (E/e') of the peak velocity of mitral inflow (E) and the average of septal and lateral mitral annular early diastolic peak velocities (e') of 18.75 (Figure 1). Based on tricuspid regurgitation peak velocity of 3.1m/s, pulmonary artery systolic pressure was estimated to 38mmHg. Left ventricular global longitudinal systolic strain (GLS) was reduced: -14.3%. According to the current guidelines, clinical and echocardiographic features correspond to HFpEF. Dobutamin stress echocardiography with GLS analysis showed inducible ischaemia in basal segment of posterior wall and post-systolic thickening of apical segment of interventricular septum (Figure 2) with appearance of ultrasound lung comets suggestive for pulmonary congestion (Figure 3). On repeated coronary angiography an occluded posterolateral branch from left circumflex artery with collateral filling was found (Figure 4). Medical therapy
Figure 1. The ratio of the peak velocity of mitral inflow during early diastole (E), recorded by pulsed Doppler between the tips of the mitral leaflets (left), over the average of septal (right) and lateral (middle) mitral annular early diastolic peak velocities (e') recorded by pulsed tissue Doppler is equal to 18.75

Figure 2. Postsystolic shortening (PSS) during Dobutamin stress echocardiography: speckle tracking echocardiography in basal segment of posterior wall and reduction of longitudinal strain (left); PSS on M-mode in apical segment of the septum

Figure 3. Ultrasound lung comets during stress echocardiography: clinically useful sign of extravascular lung water as a sign of acute pulmonary congestion during stress

was recomended for arterial hypertension, HF and CAD with upgrade of antiabetic therapy with sodium-glucose cotransporter 2 inhibitor (SGLT2i).

Discussion

Heart failure with preserved ejection fraction is a significant clinical problem for patients with DM. Current clinical data suggest that between 30% and 40% of patients with HFpEF suffer from DM. Pathophysiological processes in HFpEF in diabetics include reduced LV compliance with increased enddiastolic stiffness, chronotropic incompetence, reduced LV long-axis systolic function with both epicardial coronary artery disease and microvascular and endothelial dysfunction. A secondary analysis of the CANVAS trial showed similar reduction in heart failure with reduced ejection fraction (HFrEF) and HFpEF (HR: 0.83;95% CI: 0.55 to 1.25) in diabetics. In the DECLARE-TIMI 58 trial, all patients with HFrEF and HFrEF had similar reduction in hospitalization, but reduced cardiovascular death was observed only in patients with HfrEF. Patobiological mechanisms are still unclear, but patients treated with SGLT2i demonstrated reduction of left ventricular mass, delay in rise of natriuretic peptides as well high sensitive Tropinone I. On the other hand, therapy with glucagon-like peptide 1 receptor analogue (GLP-1 RA) in patients with T2DM who were at high risk for cardiovascular events showed lower rates of CV events and death from any cause with neutral effect on HF risk in the general population of T2DM patients with established CV disease or with multiple risk factors. Despite these intriguing possibilities, no clinical trials yet have evaluated the long-term effects of combined use of the two drugs. Given their commercial availability and the fact that both classes have approved indication to reduce CV risk, the dilemma remains how to
choose between SGLT2i and GLP-1 RA in a T2DM patient with HF and CAD? In conclusion, ongoing trials are addressing the role of SGLT2i by reducing adverse cardiovascular outcomes in patients with HFpEF and chronic kidney disease, with and without T2DM while the role GLP-1 RA remains to be defined in individuals with established HF.

References