CR23 Refractory ventricular fibrillation in a patient with ST-elevation myocardial infarction (STEMI) Petra Relota^{*a*}, Martina Periša^{*a*}, Lorena Remenar^{*a*}, Karla Schwarz^{*a*}, Tomislav Letilović^{*a,b*}

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KEYWORDS: Hypokalemia; Myocardial infarction; Ventricular fibrillation

INTRODUCTION/OBJECTIVES: Ventricular fibrillation (VF) is a life-threatening arrhythmia that can lead to sudden cardiac death by compromising cardiac output. It is a common complication of acute myocardial infarction (MI) due to the electrical instability of the myocardium. Refractory VF is defined as sustained VF that persists despite appropriate interventions. CASE PRESENTATION: A 52-year-old male with a history of arterial hypertension and smoking presented with symptoms of acute coronary syndrome (ACS) and was diagnosed with inferolateral ST-elevation myocardial infarction (STEMI). During the initial examination, the patient suffered cardiac arrest with ECG findings of VF and was promptly resuscitated and defibrillated multiple times. Percutaneous coronary intervention (PCI) was performed on the right coronary artery (RCA) with the insertion of two stents. During the two-hour procedure, the patient experienced recurrent and sustained VF, requiring multiple defibrillations. Laboratory results indicated the presence of hypokalemia despite acidosis and multiple defibrillations, both of which elevate potassium levels. This finding, along with a good response to the magnesium infusion treatment, suggests the possibility of torsades de pointes. Post-procedure, the patient's condition stabilized and he was admitted to the coronary intensive care unit. CONCLUSION: Hypokalemia can induce arrhythmias such as torsades de pointes, which can convert to VF. Acidosis is a common finding in post defibrillation time, but it is not usually linked to hypokalemia. The question remains if hypokalemia is the main reason for development of VF, but in all probability VF is caused by multiple factors including electrical instability of myocardium post MI.

CROSS

CR24 Renal artery stenting in a patient with renovascular hypertension

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KEYWORDS: endovascular procedure; renal artery stenosis; renovascular hypertension

INTRODUCTION/OBJECTIVES: Renal artery stenosis can be caused by fibromuscular dysplasia or, more often, atherosclerotic disease. It can result in hypertension, recurrent heart failure, pulmonary edema, acute coronary syndrome or progressive renal failure. Renal artery stenting is an interventional radiology procedure, which is performed in cases resistant to conservative treatment.

CASE PRESENTATION: A 38-year-old female patient was evaluated for resistant arterial hypertension, hypertensive pulmonary edema and renal insufficiency, resulting in frequent hospitalizations. Computed tomography (CT) angiography and color doppler imaging showed left main renal artery (anatomical variation with two left renal arteries) and right renal artery subocclusion. Dynamic renal scintigraphy revealed afunctional right kidney. Left renal artery endovascular stenting was proposed. The procedure was performed via right common femoral artery access under local anesthesia. Catheterization of left renal artery was performed, the subocclusion was crossed with the atraumatic wire and balloon expandable stent was placed across stenotic part. The postinterventional angiogram showed good arterial lumen with no residual stenosis. The patient was stable and discharged the following sad with dual antiaggregation therapy in first 3 months, then acetylsalicylic acid lifelong. On the follow-up visit she no longer complained on any symptoms and was not hospitalized since.

CONCLUSION: Renal artery stenosis can cause secondary hypertension which can be resistant to conservative treatment, therefore stenting procedure is a potential alternative. Renal artery stenting is minimally invasive procedure which can result in improved renal status as well as hypertension management.