known for a long time, the pathophysiologic reasons and allergic angina syndrome have diagnosed in the last two decades. Kounis Syndrome is an unusual reason of myocardial ischemia. Allergic and anaphlactic reactions are the reasons of Kounis Syndrome. Although it is not a rare disease, its diagnosis is difficult and easily overlooked.

Case Report: Fifty-seven-years-old female patient was admitted Emergency Department for allergic reaction with urticarial rash on whole body. In her medical history there are hypertansion, diabetes mellitus, hyperlipidemia and chronic renal failure. Her complaint was began after the injection of proton pump inhibitor at the end of hemodialysis two hours ago. In her physical examination there were uvulae edema, dispnea and common ronchus. After applying adrenaline 0,5 mg intramusculary twice per fifteen minutes the patient complained about a chest pain. Subacute anterior myocardial infarctus was detected in her ECG (Fig. 1). The laboratuary test results were BNP:4438 pg/ml (0-100), myoglobin:1482 ng/ml(14-66), CK/MB:16 ng/ml (0,6-6,3), Troponin I:13,5 ng/ml (0-0,04), creatinine: 6,5 mg/dl (0,5-0,95). Transtorasic echocardiogram and CAG were performed by cardiologists. In the echocardiogram the mid anterior and septume were acinetic, ejection fraction was %33. By the CAG, plaque formations in Cx artery and RCA, a lesion in LAD which doesn't cause significant stenosis. And the patient discharged on the fifth day.

Discussion: Kounis Syndrome is caused by inflammatory mediators released during mast cell activations in allergic reactions. In the severity of Kounis Syndrome; the patients sensitivity, comorbidities, the allergen concentration and the route of allergen enterance are important. Kounis' ECG changes are; ST segment changes, any degree of heart block or arrhythmias. Three varianst of Kounis Syndrome are vasospastic allergic angina, allergic myocardial infarction and stent thrombosis. The type 1 variant includes normal coronary arteries without risk factors for coronary artery disease. In the type 2 variant the infarction depends on already existing atheromatous disesase. The type 3 variant includes coronary stent thrombosis with eosinophils and mast cells. The patient with the type 1 variant treatment of allergic reaction may abolish symptoms and CAG may not be performed. But on the other variants CAG must be performed. In the treatment adrenaline should not be applied because of aggravating ischemia and worsening coronary vasospasm in Kounis Syndrome. Antihistaminic agents and corticosteroides can be used in the treatment. In our case the patient was in the group of type 2 variant. Although CAG performed in the early period, applying epinephrine may worsen the lesion of infarction. And in the earlier period inhibition of mast cell degranulation may be efficacious in preventing the acute coronary events of Kounis Syndrome.

Conclusion: Allergic and anaphylactic reactions are the reasons of Kounis Syndrome which is unusual reason of myocardial ischemia. Although it is not a rare disease, its diagnosis is difficult and easily overlooked.

Keywords: Kounis Syndrome, myocardial ischemia, anaphylaxis

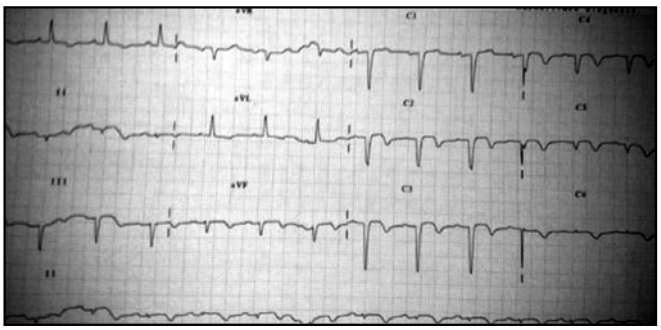

Figure 1. Subacute anterior MI, ST segment elevation on V1-V2-V3-V4 derivation

P-044 **Kardiyovasküler Aciller**

HYPOXEMIA CAUSED BY A RARE ACUTE CARDIAC PATHOLOGY

Murat Yeşilaras, Nur Zafer, Öner Özdoğan, Özge Duman Atilla, İbrahim Toker

Department of Emergency Medicine,İzmir Tepecik Training and Research Hospital, İzmir

Introduction: Acute hypoxemia is a clinical condition frequently encountered in the ED. Acute pulmonary edema, pneumonia, COPD, pulmonary VTE are the most common pathologies associated with this situation. In this case, we presented a young patient with acute hypoxemia.

Case: A 35 years old female presented to the ED with new onset nausea and vomiting. In the medical history, she had a cardiac surgery for total repaired of ASD (atrial septal defect) fifteen days ago. On admission, she had a blood pressure of 96/46 mmHg, heart rate of 102/min, body temperature of 36^0C, respiratory rate of 33/min, peripheral oxygen saturation of 84%. There were no rales, rhonchus or murmurs on cardiopulmonary examinations. She was mildly cyanotic. There wasn't any pathology on electrocardiogram. On arterial blood gases analysis, pH, PCO2, PO2, HCO3 and lactate were found to be 7.603, 12.8 mmHg, 43 mmHg, 12.4 mmol/L and 4.47 mmol/L respectively. There was mildly a leukocytosis (12.7x109/L). BUN, electrolytes, cardiac markers and D-dimer were normal. There was no pathology on chest x-ray. On bedside echocardiography (ECHO), EF was normal and a right structure was not dilated. Pulmonary thromboembolism was suspected and CT angiography of thorax was ordered. At first while giving IV contrast, transition was not seen on pulmonary artery (PA) in contradistinction to superior vena cava (SVC) and aorta (Figure 1). By giving additional contrast and waiting for contrast transition, there was no thromboembolism on the images. Because of this and no contrast passage on PA, we thought about right-to-left shunt and consultated the patient to cardiologiest. On transthorasic ECHO, LVEF measured as %60, sistolic PA pressure as 9mmHg and there was no shunt. She hospitalized to coronary care unit (CCU). In CCU, she was still hypoxemic. On contrast TTE by giving agitated saline from bilateral antecubital veins, contrast transition was directly from SVC to left atrium (Figure 2). So she operated for repairing of sinus venousus type ASD by patch and dilating SVC by pericardial patch. After operation, the patient's complains and hypoxemia regressed and she discharged from hospital.

Conclusion: The most significant sign of right-to-left shunt (RLS) is hypoxemia, but before whole pathologies that frequently found with hypoxemia, diagnosing RLS is very hard. Patent