**Introduction:** Epicardial adipose tissue (EAT), located between the myocardium and visceral layer of pericardium is an emerging risk factor for cardiometabolic diseases.

**Methods:** The retrospective study consisted of patients hospitalised for STEMI treated with PCI from 2014 to 2016, EAT thickness was measured from the parasternal long-axis view at end-diastole. Cholesterol levels were determined in a blood sample. According to median patients were divided in two groups: thin EAT group (<2.27 mm, n = 270) and thick EAT group (≥2.27 mm, n = 223). Statistical analysis was performed with SPSS using Mann–Whitney test, T-test, logistic regression analysis. Values of cholesterol levels were evaluated by ROC curves. p < 0.05 was significant.

**Results:** Total 492 patients (332 males, 66.62 ± 12.24 year-old) were enrolled. Groups did not differ by age, gender, morbidity of diabetes mellitus and triglyceride levels. Patients had higher BMI (29.41 ± 4.97 vs. 28.13 ± 4.67 kg/m², p = 0.009), total cholesterol (≥4.82 mmol/l; 35.2 vs. 26.4%, p = 0.024), low density lipoprotein cholesterol (≥2.5 mmol/l; 45.8 vs. 33.3%, p = 0.004) and reduced high density lipoprotein cholesterol (HDL-C) levels (≥1 mmol/l; 24.4 vs. 10.4%, p = 0.009) in thick EAT group. Logistic regression analysis revealed that higher BMI (OR = 1.532, 95% CI 1.008–2.328, p = 0.002) and HDL-C ≤1 mmol/l (OR = 1.777, 95% CI 1.159–2.724, p = 0.008) were associated with thicker EAT. Killip class >III was more frequent (17.6 vs. 10.3%, p = 0.02) in thick than thin EAT group.

**Conclusion:** Increased EAT thickness was associated with obesity, cardiometabolic risk factors and influenced severity of left ventricular dysfunction.

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**Influence of blood inflammatory parameters to erythropoietin resistance in haemodialysis patients**

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**Aim:** To evaluate the correlation between the inflammatory blood parameters and the resistance to EPO among the haemodialysis patients.

**Introduction:** Erythropoietin therapy is considered to be the standard treatment of anemia in chronic kidney disease patients, yet some patients do not respond well to this therapy. This is called EPO resistance and could be generally associated with the chronic inflammation.

**Methods:** A retrospective one single centre study, which analysed medical records of 30 HD patients who had advanced CKD and received EPO treatment in Vilnius University Hospital Santaros Clinics from 2016–2009 to 2016–2011. Data analysed – concentrations of C-reactive protein, neutrophils, lymphocytes, platelets, as well as EPO dose per kilo and hemoglobin concentration (measured at the beginning of the EPO therapy and one month after the treatment).

**Results:** Patients were grouped into 2 categories: 1 group (n = 14) – concentration of hemoglobin increased, 2 group (n = 16) – concentration decreased after treatment. In 1 group average concentration of platelets were statistically significantly (p = 0.039) higher (230.2 ± 73.70) compared to 2 group (174.1 ± 66.96).

Furthermore, platelets concentration among patients with hemoglobin level of >100 g/l (n = 17) after one month of treatment were statistically significantly (p = 0.012) higher (231.06 ± 56.41) compared to those patients with hemoglobin level of <100 g/l (n = 13) (160.08 ± 78.17) after treatment.

Additionally, patients with hemoglobin levels after one month of treatment 100–125 g/l (n = 15) were separated into two groups based on C-reactive protein level: >5 (1 group) and <5 (2 group). Average concentration of erythropoietin was statistically significantly higher in 1 group (n = 9) (223.82 ± 69.15 VV/kg) than in 2 group (n = 6) (116.68 ± 59.68 VV/kg).

Correlation analysis revealed that among patients with hemoglobin levels of <110 g/l after treatment there is a statistically significant positive correlation (r = 0.428) between change of hemoglobin levels before and after treatment and erythropoietin dose and statistically significant (p = 0.023) negative correlation (r = 0.481) with lymphocytes concentration in blood.

**Conclusion:** HD patients with a higher concentration of platelets respond to EPO therapy better than those with a lower concentration. Increased EPO dose results in higher Hgb