Background: Lipoprotein (a) is a molecule similar to LDL-Cholesterol with thrombogenic and atherogenic capacity. Plasminogen activator inhibitor (PAI-1) is the primary physiological inhibitor of the fibrinolytic system. Genetic polymorphisms of PAI-1 determine their plasma concentrations levels; 4G/4G and 4G/5G as opposed to 5G/5G mutations diminish fibrinolytic activity, predisposing to thrombotic events.

We aim to establish whether there is a relationship between levels of Lp-a and polymorphisms of PAI-1 with the extension of coronary artery disease (CAD), presence of intracoronary thrombus and abnormal TIMI flow.

Methods: Transversal observational study in which patients admitted with ACS and underwent a coronary angiogram were evaluate measuring Lp-a levels and PAI-1 polymorphisms.

Results: A total of 102 patients were evaluated. The prevalence of elevated levels of Lp-a was 71%. The prevalence of PAI-1 polymorphisms 4G/4G, 4G/5G, and 5G/5G was 19%, 62%, and 19% respectively. Patients with the combination of elevated Lp-a and mutated genotype 4G/4G had more extended CAD (OR 4.5, 95% CI 1.5-15, p = 0.04), increased presence of intracoronary thrombus (OR 3, 95% CI 1-8, p = 0.04), and higher incidence of abnormal TIMI flow (OR 5, 95% CI 1.1-17, p = 0.01). Compared to patients with non-ST elevation ACS, those with ST-elevation ACS had a higher prevalence of 4G/4G mutation (64.7% vs. 35-3% RR 5.9, 95% CI 1,3 – 26,7 p< 0.039).

Conclusion: The prevalence of elevated Lp-a levels and mutated genotypes of PAI-1 is high in patients with ACS. Patients with the combination of elevated Lp-a and mutated genotype 4G/4G have more extended CAD, an increased presence of intracoronary thrombus and a higher incidence of abnormal TIMI flow. Patients with ST-elevation ACS have a higher prevalence of 4G/4G mutation than with non-ST elevation ACS.