

Aterotrombosi e Tromboembolismo arterioso:
aspetti di laboratorio

TIME COURSE OF LP(A) LEVELS AFTER ACUTE CORONARY SYNDROME: A LONGITUDINAL STUDY

G. Barbieri[1], A.M. Gori[1], G. Ciarrocca Taranta[1], M. Di Gioia[1], F. Alfano[1], E. Lotti[1], A. Rogolino[1], F. Cesari[1], B. Giusti[1], M. Berteotti[1], R. Marcucci[1]

[1]Atherothrombotic Diseases Center, Department of Experimental and Clinical Medicine, University of Florence - Careggi Hospital, Firenze, Italy

Background and Aims. Lipoprotein (a) [Lp(a)] is a well-established, independent, atherosclerotic cardiovascular risk factor. Several studies have demonstrated its prognostic value, alongside traditional risk factors, in patients with acute myocardial infarction (AMI), contributing to improved risk stratification and therapeutic management. Lp(a) levels vary markedly across populations, whereas in each individual they are largely determined by genetics of *LPA* gene, encoding apolipoprotein (a), and are minimally influenced by other factors: for this reason, current guidelines recommend measuring serum Lp(a) at least once in a lifetime. However, emerging evidence suggests that certain clinical conditions, such as acute inflammatory states, may alter Lp(a) concentrations due to the interference with acute phase responders such as interleukin (IL)-6. This raises the question of whether Lp(a) measurement should be reassessed in these scenarios. Therefore, we conducted a longitudinal study assessing Lp(a) concentrations in patients with AMI who were followed for up to one year after the acute event.

Methods. We analysed Lp(a) concentration in serum or plasma samples of 288 patients, admitted to the coronary units of the six hospitals of the Florence health district between 2008 and 2010, enrolled in the frame of the Florence Acute Myocardial Infarction (AMI-Florence 2) registry, using a particle enhanced immunoturbidimetric assay on Cobas c 311 platform (Roche). Serum and plasma samples were stored at -80°C until analysis.

Results. The study cohort included 218 men and 70 women with a median age of 69 years (IQR 59–77). Lp(a) concentrations were first assessed at admission for the acute event (T0), showing a median value of 32.5 (11.0–82.8) nmol/L. Follow-up measurements were performed when available: at T1 (2.2±1.2 months, n=100) with a median Lp(a) of 14 (5–43) nmol/L, at T2 (6.5±1.0 months, n=196) with a median of 19.0 (5.0–49.5) nmol/L, and at T3 (12.6±1.6 months, n=203) with a median of 15 (5–48) nmol/L. Overall, Lp(a) levels showed a statistically significant decrease from the acute phase to all subsequent follow-up time points ($p<0.001$). At T3, in 62/203 (30.5%) patients tested we observed a reduction of at least 25 nmol/L. Stratifying patients according to Lp(a) levels above or below 125 nmol/L (50 mg/dL), we observed a progressive decline in the proportion of individuals classified in the high risk category from T0 to T3. By the 12 month follow up, 22 patients who initially presented with above threshold values had shifted to below threshold levels, an evolution that may meaningfully influence clinical risk stratification and long term management. When examining the cohort by sex, we also confirmed a well established pattern: women consistently exhibited significantly higher Lp(a) concentrations than men at T0 and T1 ($p=0.036$ and $p=0.038$, respectively), a difference that remained evident, though not significant, at the other time points.

Conclusions. Our data demonstrate that Lp(a) levels are significantly affected by the acute phase in patients with an acute myocardial infarction, as levels drop significantly over the months following AMI. Reassessment during follow-up may therefore be necessary to ensure more accurate risk stratification in this clinical setting.

Abstracts printing tool powered by pagepress.org