**CORONARY PLAQUE EROSION AND MYELOPEROXIDASE: INSIGHT FROM A CLINICOPATHOLOGICAL STUDY**

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Inflammation plays a key role in coronary atherosclerosis progression and destabilization. The activation of the innate immune system has been recently shown to be involved in coronary plaque instability. In particular neutrophils are recruited early at the site of coronary thrombosis, as if triggers of neutrophil activation suddenly localize at the culprit plaque. Interstingly, Myeloperoxidase (MPO), a marker of neutrophil activation, has been shown to have a prognostic role in acute coronary syndrome patients independently of traditional marker of inflammation such as C-reactive protein. The introduction of high resolution imaging modalities as optical choerence toography (OCT) is able to define with high precision details of the vessel wall and to characterize mechanisms of coronary instability. In particular, two main mechanism have been identified to be associated with coronary thrombosis: plaque rupture and plaque erosion. This two distinct morphological features of the culprit and thrombosed coronary plaque seem to be associated with specific elevation of inflammatory biomarkers. Indeed, we recently shown that MPO levels were higher in patients with ACS and plaque erosion detected by OCT as compared to those with plaque rupture. This observation open the way to a better understanding of pathophisiology of acute coronary syndromes, may further stimulate research activity and possibly guide a more targeted therapy and prevention of acute coronary syndromes according to the specific pathophysiological pattern operating in the individual patient.