

27th European Cardiology Conference

October 22-24, 2018 | Rome, Italy



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Endoplasmic Reticulum Stress, Inflammation, Oxidative Stress And Neutrophil Extracellular Traps In Cardiovascular Diseases

The aim of this work is to summarize the understanding of the interrelated roles of endoplasmic reticulum (ER) stress, oxidative stress & inflammation in cardiovascular diseases. Insults interfering with ER function lead to the accumulation of unfolded & misfolded proteins in the ER. An excess of proteins folding in the ER is known as ER stress. This condition initiates the unfolded protein response (UPR). When the UPR fails to control the level of unfolded & misfolded proteins, ER-initiated apoptotic signalling is induced. Moreover, the role of the protective nuclear erythroid-related factor 2 (Nrf2)/antioxidant-related element (ARE) & the activation of the pro-inflammatory nuclear factor-kappa B (NF-kB) are analysed. Current literature data are presented, focusing on three topics of related pathologies: atherosclerotic plaque, coronary artery disease & diabetes. Moreover, current evidence suggests the likelihood of a link between venous thromboembolism (VTE) & atherosclerosis, although they have been traditionally considered as different pathological identities. The contribution of neutrophils to human atherogenesis has been underestimated, if compared to their contribution established in VTE. This is due to the major importance attributed to macrophages in the plaque destabilization. Nevertheless, recently, the role of neutrophils in atherogenesis deserves increasing attention. In particular, neutrophil extracellular traps (NETs) are net-like chromatin fibres which are released from dying neutrophils. The death of neutrophils with NETs formation is called NETosis. During activation, neutrophils produce Reactive Oxygen Species (ROS), through the activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase. The main function of NETs is trapping and killing pathogens. However, NETs formation has been observed in various chronic inflammatory diseases, autoimmune diseases, vasculitis, lung diseases, cancer and VTE. Recent studies suggest that NETs formation could contribute also to atherosclerosis progression. New data report the presence of NETs in the luminal portion of human atherosclerotic vessels and coronary specimens obtained from patients after acute myocardial infarction. Programmed death mechanisms in atherosclerosis such as apoptosis, efferocytosis and also NETosis, share common features & triggers. If defective, they can lead the cells to a switch from programmed death to necrosis, resulting in the release of pro-atherogenic factors, accumulation of cell debris and progression of the disease. This talk aims to analyse the emerging role of neutrophils focusing on NETosis and oxidative stress burden in orchestrating common mechanisms in atherosclerosis & thrombosis.

Biography

Chiara Mozzini pursued her Medical Doctor Degree from the University of Brescia, Italy (2006). She is a Board Certified Specialist in Internal Medicine at the University of Verona, Italy (2012). Section of Internal Medicine University of Verona, Italy Medical Doctor Degree University of Brescia (Italy). She received her PhD certification in clinical and experimental medical sciences. She is an Adjunct Professor (Researcher of type A)- Section of Internal Medicine at the University of Verona, Italy. Her research field of interest includes: atherosclerosis, cardiovascular diseases, coronary artery disease, diabetes, oxidative stress, endoplasmic reticulum stress and ultrasound (cardiac-abdominal-vascular). Her H-index is 11.