NEW INSIGHTS INTO THE PATHOPHYSIOLOGY OF CARDIOGENIC SHOCK: THE ROLE OF THE MICROCIRCULATION

Ashruf JF, Bruining HA, Ince C[1]

Abstract

Purpose of review: The ultimate goal of therapy for cardiogenic shock is to restore microcirculatory function and thereby restore the oxygen supply to sustain cellular function. Therapeutic measures mainly focus on improving pressure-derived macrocirculatory parameters. However, it is increasingly clear that to achieve significant progress in treatment, microcirculatory physiopathological mechanisms must be considered.

Recent findings: Microcirculatory function deteriorated during cardiogenic shock and improved after treatment. Postcardiogenic shock microcirculatory disturbances, both myocardial and peripheral, were a prognostic factor for the long-term outcome. Hypothermia, whether pharmacologically or physically induced, improved postresuscitation myocardial and cerebral function, an effect associated with improved postresuscitation microcirculation. The impact of cardiogenic shock on cerebral and myocardial microcirculation could be evaluated with MRI. In severe heart failure, pharmacological interventions improved microcirculation. An assessment of the microcirculation was often performed using handheld video microscopy for direct observation of the sublingual microcirculation, which proved to be useful for evaluating the effects of interventions during cardiogenic shock. A large multicenter study on critically ill patients is now being conducted using this technique.

Summary: Cardiogenic shock induces microcirculatory disorders that can be monitored and influenced in various manners, both pharmacologically and physically. In addition to global hemodynamic optimization, interventions must also ameliorate the microcirculation.

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VASOMOTOR REGULATION OF CORONARY MICROCIRCULATION BY OXIDATIVE STRESS: ROLE OF ARGINASE

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Abstract

Overproduction of reactive oxygen species, i.e., oxidative stress, is associated with the activation of redox signaling pathways linking to inflammatory insults and cardiovascular diseases by impairing endothelial function and consequently blood flow dysregulation due to microvascular dysfunction. This review focuses on the regulation of vasomotor function in the coronary microcirculation by endothelial nitric oxide (NO) during oxidative stress and inflammation related to the activation of L-arginine consuming enzyme arginase. Superoxide produced in the vascular wall compromises vasomotor function by not only scavenging endothelium-derived NO but also inhibiting prostacyclin synthesis due to formation of peroxynitrite. The upregulation of arginase contributes to the deficiency of endothelial NO and microvascular dysfunction in various vascular diseases by initiating or following oxidative stress and inflammation. Hydrogen peroxide, a diffusible and stable oxidizing agent, exerts vasodilator function and plays important roles in the physiological regulation of coronary blood flow. In occlusive coronary ischemia, the release of hydrogen peroxide from the microvasculature helps to restore vasomotor function of coronary collateral microvessels with exercise training. However, excessive production and prolonged exposure of microvessels to hydrogen peroxide impairs NO-mediated endothelial function by reducing L-arginine availability through hydroxyl radical-dependent upregulation of arginase. The redox signaling can be a double-edged sword in the microcirculation, which helps tissue survival in one way by improving vasomotor regulation and elicits oxidative stress and tissue injury in the other way by causing vascular dysfunction. The impact of vascular arginase on the development of vasomotor dysfunction associated with angiotensin II receptor activation, hypertension, ischemia-reperfusion, hypercholesterolemia, and inflammatory insults is discussed.

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COMPONENTS OF THE COMPLETE BLOOD COUNT AS RISK PREDICTORS FOR CORONARY HEART DISEASE: IN-DEPTH REVIEW AND UPDATE

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Abstract

Atherosclerosis is an inflammatory disease, and several inflammatory biomarkers, such as C-reactive protein, have been used to predict the risk of coronary heart disease. High white blood cell count is a strong and independent predictor of coronary risk in patients of both sexes, with and without coronary heart disease. A high number of white blood cells and their subtypes (for example, neutrophils, monocytes, lymphocytes, and eosinophils) are associated with the presence of coronary heart disease, peripheral arterial disease, and stroke. The coronary heart disease risk ratios associated with a high white blood cell count are comparable to those of other inflammatory markers, including C-reactive protein. In addition, other components of the complete blood count, such as hematocrit and the erythrocyte sedimentation rate, also are associated with coronary heart disease, and the combination of the complete blood count with the white blood cell count can improve our ability to predict coronary heart disease risk. These tests are inexpensive, widely available, and easy to order and interpret. They merit further research.

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