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Cardiovascular Diseases and Infection: Mechanistic Insight on Inflammation, Pathophysiological Mechanisms, and Potential Therapeutic Targets

Short Communication
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Short Communication

Heart injuries and commonly recognized general pathophysiological characteristics and mechanisms are:

A. Septal rupture, free wall rupture, coronary artery thrombosis, cardiac failure, minor electrocardiographic cardiac enzyme abnormality, or complex arrhythmia with cardiac failure [1],

B. Myocardial injury, ventricular rupture, valve disruption, septal rupture, and commotion cordis,

C. Pericardial injury, valvular injuries, coronary artery injuries, cardiac chamber rupture, myocardial contusion, and ischemia,

D. Heart Infection (Endocarditis, Myocarditis, Pericarditis) [2].

Agreeing to etiology, dealing with an ischemia-reperfusion injury is a therapeutic challenge for physicians, because it is interlinked with many clinical manifestations such as systemic inflammatory response syndrome, cerebral dysfunction, gastrointestinal dysfunction, myocardial hibernation, acute heart failure, and multiple organ dysfunction syndromes [3]. The route of uncoupling of Nitric Oxide Synthase (NOS) also generates ROS (Reactive Oxygen Species) and promotes the phenomena of ischemia-reperfusion injury, and overall, these mechanisms inductee cell death. Additionally, ischemia-reperfusion injury generates hypoxic stress that initiates the extrinsic- and intrinsic pathways. By promoting and commencing each other, these routes and paths finally assisted cells to achieve death.

Prolonged ischemia-reperfusion injury-induced various cell damage and further initiates apoptosis, necroptosis, autophagy, and necrosis [4]. The answers to these queries will be helpful for further elucidation of other cellular events i.e., the role of Nicotinamide Adenine Dinucleotide Phosphate Hydrogen (NADPH) oxidase system, xanthine oxidase system, and nitric oxide synthase system [5]. Various systems such as NADPH oxidase, nitric oxide synthase, and xanthine oxidase promote oxidative stress, and after mitochondrial damage and electrolyte imbalance in reperfusion, the state followed it [6]. Hypoxanthine oxidized into uric acid in the presence of xanthine oxidase and as well as initiate Reactive Oxygen Species (ROS) production [7]. At the outset, it is the conversion of ATP into hypoxanthine. In a lower concentration of ATP, xanthine dehydrogenase is converted into xanthine oxidoreductase and supports ROS generation and the above-mentioned conversions [8,9]. In ischemia-reperfusion injury, the 'Nitrogen Oxides' (NOx) enzymes produce superoxide and hydrogen peroxide, and the activation reactions and routes of HIF-1α (Hypoxia-Inducible Factor 1-alpha), phospholipase A2, TNF-α (Tumour Necrosis Factor alpha), IL-1β (Interleukin-1β), IFN-γ (Interferon gamma), and angiotensin II, further promote the aforementioned conversion. Furthermore, the ligands, TNF-α, TRAIL (TNF-Related Apoptosis Inducing Ligand), TL1A (Tumor Necrosis Factor (TNF)-like cytokine 1A), and TWEAK (Tumour Necrosis Factor (TNF)-like weak inducer of apoptosis (TWEAK)), Fas Ligand, which represent death actively participate with proteolysis, recruit the caspase cascade for initiating cell death [4,10]. The routes of catalysing the activities and involvement of caspase cascade initiated by a log chain of cellular events such as ROS stimulate pro-apoptotic Bcl-2 family (B-cell leukemia/lymphoma 2 protein) and do some changes to the veracity of the mitochondrial sheath.

In the end, all these cellular events enforced mitochondrial cleavage and fusion. Therefore, cellular events such as mitochondrial membrane potential, cytochrome c release, and cell death are interlinked. The cellular routes, intracellular adapter molecules, ROS-induced DNA damage, a materialization of necrosis, and necroptosis are achieved in an alternative fashion, wherein, necrosome stimulates various stimuli and catalyze necroptosis [11-13]. Low concentration of ATP inductee an irreversible cellular energy failure. The originated oxidative stress during ischemia-reperfusion injury hinders several cellular activities, events, and routes and as a result, the formation of autophagosomes initiated, which fuse with a lysosome [14-16]. Myocardial Infarction

(MI) is the most common cause of heart injury, and after reperfusion, the activation of innate and adaptive immune responses and cell death programs are further enhanced [17]. MI and reperfusion damage are characterized by inflammation and inflammatory cell infiltration. Ischemic heart damage increases chemokine and cytokine3 expression, triggering the innate immune response through toll-like receptors [18]. The process of attracting inflammatory cells is dynamic and expertly planned [19]. The infiltration of neutrophils, monocytes, macrophages, dendritic cells, and lymphocytes into the damaged heart aids in angiogenesis, ventricular remodeling, and inflammation.

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Conflict of Interest

The authors declare no conflict of interest, financial or otherwise.

Declaration of Competing Interest

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Data Availability Statement

Due to the nature of the research, [ethical, legal/commercial] supporting data is not applicable and thus not available.

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