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Role of Endothelial Shear Stress in Post-Viral Cardiopulmonary Remodeling

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Description

Viral infection causes major endothelial dysfunction disorders that involve the immune system, the inflammatory response and the apoptosis-angiogenesis interdependency which is a major process in tissue repair and regeneration [1]. Restoration of the unbalanced angiogenesis-apoptosis pathway in viral infection, as well as in wound healing, depends on endothelial functions which are regulated by shear stress [2].

As a reminder, Endothelial Shear Stress (ESS) controls and maintains endothelial functions [3], as well as vasculogenesis, cardiogenesis, embryogenesis, organogenesis through the angiogenesis-apoptosis interdependency process, from the 8th day of gestation until death. Also, ESS stimulates chemical facilitators like monocyte chemo attractant protein-1 (MCP-1); TNF-α; bFGF and MMPs and promote potent vasodilators like Nitric Oxide (NO) [4,5].

Despite the ability of endothelial cells to adapt to various pathological conditions [6], the outcome of post-viral infection depends on the host biological responses, which are governed by the diversity of patients' conditions, viruses and target organs [7-10]. This is clearly demonstrated by the controversial results of post-viral remodeling and metabolic processes of vital organs such as liver and brain [11,12], which become even worse with dynamic vital organs like the cardiopulmonary system. As restoration and maintenance of endothelial functions, the key vector to complete recovery of the hosts metabolic processes, depends on ESS-inducing circulatory driving forces, causing the therapeutic dilemma of cardiopulmonary viruses [13].

Taking the example of the Covid-19 virus which invades host cells *via* Angiotensin Converting Enzyme Receptor 2 (ACE2) [14], we can recognize that most of the patients succumb to multiple organ failure and/or Sudden Cardiac Arrest (SCA) as a result of aggravated endothelial dysfunction disorders, whether in the form of comorbid conditions e.g., arterial hypertension, mediated by the virus, e.g., inflammatory response and/or iatrogenic, e.g., thromboembolic syndrome [15]. Likewise, the outcome of viral myocarditis like almost all types of Dilated Cardiomyopathy (DCM) remains a potentially life-threatening condition [16,17]. The restoration of the unbalanced angiogenesis-apoptosis pathway in post-viral DCM that requires neovascularization with a full-thickness myocardial reconstruction, is principally regulated by vasculogenesis, angiogenesis and cardiogenesis endothelial function processes [18].

As is known, the early vasculogenesis process begins at the embryonic endometrial implantation around the 6th day of gestation, to create the first blood vessels from blood islands, to be followed by sprouting-splitting angiogenesis to construct the whole cardiovascular system: heart, vessels and blood components [19,20]. Similarly, early cardiogenesis process which employs the constitution of endocardium, myocardium (atrioventricular myocytes and Purkinje fibers) and epicardium from the original cardiac mesoderm precursor cells (cardiogenic mesoderm) [21], depends on both Endocardial Endothelium (EE) and Myocardial Capillary Endothelium (MyoCapE), and additionally to circulating endothelial cells rather than coronary vascular endothelium [22].

In the intrauterine life, ESS is provided principally by myocardial contractility of the fetal heart [23]. In the early postnatal period, after suppression of the placental circulation, drop of pulmonary afterload and closure of physiological shunts, angiogenesis-cardiogenesis processes depend principally on the respiratory pump, cardiac contractility and vascular resistances. For example, in the postnatal period, successful right or left myocardial remodeling occurs independently of coronary network by the direct effect of ESS on the endocardium as a result of increased afterload of the contractile ventricle. This is obviously demonstrated in pathophysiologic remodeling such as Cor pulmonale [24], severe aortic stenosis [25] and pulmonary artery banding [26], which occurs due to increased intraventricular ESS (Laplace's law). Similarly, an increased intravascular ESS can seriously disturb the physiological remodeling at the right heart side, promoting serious hemodynamic conditions and irreversible damage, such as Eisenmenger syndrome and vein graft disease [27-29]. We should emphasize that in most of viral dilated cardiomyopathy patients show symptoms and signs of left ventricular failure which subsequently leads to congestive heart failure. The right-heart side including the pulmonary tract is a highly compliant circuit, in which the Right Ventricular (RV) mass is approximately 1/6 of the left ventricular mass. This makes the RV vulnerable to sudden mechanical overload which is a rapidly fatal hemodynamic condition that cannot withstand the time-consuming remodeling processes, refuting claims of acute myocardial dilation in Covid-19 patients [30].

In conclusion, disruption of potential ESS-inducing circulatory driving forces, as in viral cardiomyopathy, can promote irreversible damage due to compensation of apoptotic cardiomyocytes by fibrous tissue. Likewise, post-viral pulmonary fibrosis, most often occur in patients with Acute Respiratory Distress Syndrome (ARDS), treated with mechanical ventilation. As ventilators prevent the respiratory pump from performing its crucial role as a potential ESS generator, along with their interference with coronary perfusion flow causing further hemodynamic deterioration in ARDS. Besides, heterogeneity of extra-alveolar and alveolar endothelial cells of the fragile alveolar system can promote barotrauma, and most likely alveolar fibrosis [31,32]. Smooth and uneventful recovery of cardiopulmonary viruses may require the employment of ESS therapy which controls hemodynamics, tissue oxygenation, remodeling and metabolic processes *via* a plurality of endothelial mediators [33]. The clinical application of ESS could be induced properly with pulsatile CAD, in correspondence with pathophysiology and cardiovascular biophysics to maintain a fully functioning respiratory pump and avoid the creation of

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a vicious circuit of energy losses and endothelial dysfunction raised by opposing hydraulic circuits [13,34].

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