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Perspective

Cardiopulmonary Bypass Surgery Patients with Neutrophil Dysfunction

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DESCRIPTION

One of the most significant medical breakthroughs of the twentieth century was open heart surgery. The development of Cardio Pulmonary Bypass (CPB) by Extra Corporeal Circulation (ECC) has been the cornerstone of this remarkable success. After Cardio-Pulmonary Bypass (CPB) surgery, hemorrhage is a well-known complication that causes significant morbidity and mortality. Depending on the clinical circumstances, the incidence ranges from 5 to 25%. Several factors have been identified as causal causes, including platelet count, platelet dysfunction, haematocant, medications utilized, type of pump used, type of oxygenation system used, and certain coagulation problems, but none has been definitively proven. Platelet dysfunction appears to be the most likely explanation for coagulopathy following CPB. Platelet dysfunction caused by CPB is most likely due to a combination of platelet activation, membrane damage, and fragmentation, all of which lead to the creation of micro-particles.

Platelet activation can be caused by a variety of things, including platelet adherence and aggregation to fibrinogen adsorbed on the bypass circuit, mechanical trauma and shear stress, cardiotomy suction, traces of thrombin, plasminogen, and ADP, complement activation, hypothermia, and platelet exposure to the blood-air interface in bubble oxygenators. In some cases, platelet surface membrane damage leads to changes in alpha 2 adrenergic receptors, fibrinogen receptors, and Glyco-Protein (GP) 1b molecules. Some people attribute the effects of Hepann to CPB. CPB causes platelet-derived micro-particles to develop, potentially as a result of physical stress or complement activation. The actual process that causes platelet dysfunction, however, is unknown. The majority of research now focuses on platelet membrane receptor activation and platelet microparticle production as the cause of platelet dysfunction in CPB. Many people are perplexed by the basic pathophysiology of abnormal hemostasis caused by CPB. Obviously, before a practical strategy for rapid diagnosis and effective therapy can be initiated, the basic process of impaired hemostasis associated with CPB must be fully understood and acknowledged. The most prevalent hemostatic consequence after cardiopulmonary bypass is platelet dysfunction. Platelet dysfunction is caused by blood interaction with non-endothelial surfaces of the heart-lung machine, as well as the products of activated granulocytes and endothelial cells during cardio pulmonary bypass. Platelet and other blood constituents' behaviour during cardiopulmonary bypass has led to the identification of medicines that preserve platelet counts and function following heart surgery. The use of CPB to preserve platelet function reduced the risk of hemorrhagic events and the requirement for homologous platelet transfusions after heart surgery. Platelets are activated by a number of triggers produced at the contact between the non-endothelial surface and blood, as well as at the site of damage.

ADP and thrombin levels in the blood when active platelets contribute to thrombogenesis by adhering to a disturbed vascular surface, aggregating to form the platelet plug, and providing co-factors to the enzymatic coagulation cascade, a fibrin clot is formed, and vascular alterations are induced. The tripeptide sequence Arg Gly Asp [RGD] found in many sticky proteins is the best characterized cell binding domain. Protein receptors on the platelet surface that are members of the integrin family of cell surface molecules recognise the RGD domain.

Platelet glycoproteins GP 1 b/ IX complex interact with vWF to mediate platelet mitial attachment to the sub-endothelium. Platelets disseminate on the sub-endothelium after adhesion. Platelet activation is set off by spreading on collagen. The shift in platelet shape from discoid to sphencal with extruded pseudopodia is the first evident response in the activation cycle. The surface GPIIb/IIIa receptor undergoes a conformational change in response to the initial shape change, revealing binding sites for fibrinogen, fibronectin, vWF, and vitronectin, which drive platelet-to-platelet aggregation. Platelet aggregation is caused by the bridging of fibrinogen molecules between several cells, and it necessitates an activation-dependent change in the GPIIb/lIIa complex to allow sticky protein attachment. ADP and TXA2, soluble agonists released by activated platelets, as well as thrombin produced following activation of the coagulation cascade, cause exposure of GPIIb/IIIa binding sites in the sub-endothelium. The nature and concentration of inciting stimuli, as well as the sensitivity of circulating platelets, control the rate and amount of platelet activation. Platelet aggregometry can be used to observe graded platelet behaviour *in vitro*.

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