

Reticulated Platelets in COVID 19 Patients

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The corona virus disease 2019 (COVID 19) has caused pandemic from 2019 and still continuing in 2021 with symptoms of acute respiratory distress syndrome and widespread mortality. COVID 19 causes platelet activation and presence of viral RNA is observed along with thrombosis in pulmonary and extrapulmonary vasculature [1,2].

Immature platelets are increased in COVID 19 and other viral infections like HIV and Viral Hepatitis. Immature platelets have rough endoplasmic reticulum fragments and therefore the capacity of protein synthesis. These are also known as Reticulated platelets (RP). These are associated with high platelet turnover and have a increased propensity of thrombus formation [3].

The Immature platelet count (IPC) is calculated by multiplying immature platelet fraction (IPF) and total platelet count, representing the absolute count of immature platelets. The COVID 19 patients have higher levels of IPC and IPF therefore the thrombotic events in these patients may be due to immature platelets. The high platelet turnover may be responsible for increased levels of immature platelets in COVID 19 patients and therefore enhanced thrombus formation [4].

The host immune response to SARS COV -2 is influenced by platelets. The critically ill COVID 19 patients showed shared pathways with platelet gene expression and sepsis along with immune regulation and antigen presentation with expression of proteins with antiviral properties like interferon-induced transmembrane protein 3 [5].

The COVID 19 infection is similar to other viral infections like HIV and viral hepatitis resulting in formation of platelet monocyte complexes via P selectin and activation of extrinsic coagulation pathway [6]. The COVID 19 patients show higher levels of P selectin on surface membranes of platelets than normal controls [3]. There is enhanced platelet monocyte interaction and tissue factor activation in severe COVID 19 patients leading to increased platelet aggregation [4].

In HIV patients the platelet turnover is enhanced due to increased platelet activation leading to increased clearance of platelets [3]. In COVID 19 infection, thrombocytopenia is due to platelet destruction by immune response to virus and increased platelet aggregates in lung leading to consumption. The platelet production is not affected in COVID 19 and therefore compensatory platelet production increases. However, this also increases the number of immature platelets which are more functionally active and have increased propensity for thrombus formation.

The platelet distribution width is increased and mean platelet volume of platelets is decreased in COVID 19 patients and correlated with disease severity and mortality [4]. The immature platelets are increased in sepsis patients before appearance of symptoms and show a decline as symptoms of sepsis are seen and therefore may serve as diagnostic and prognostic markers [7].

Higher levels of RPs were associated with a decreased risk of severe or life-threatening hemorrhage in patients with a very low platelet count. The Prophylactic platelet transfusions with concentrates containing high percentages of RPs seem to be more effective than concentrates with low percentages of RPs, leading to a lower number of required transfusions [8].

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