Does People Infected With Coronavirus are Prone to Thrombosis

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Introduction

- Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has become a global pandemic disease.
- It's an enveloped RNA virus, and the seventh member of the human coronavirus family.
- COVID- 19 has been linked to several critical cardiovascular complications, and even individuals without a history of cardiovascular disease are at risk of cardiovascular complications.
- Many viruses rather than coronavirus, including hepatitis C virus (HCV) and influenza virus, can directly lead to platelet hyperactivity.
- Patients with severe COVID-19 commonly experience thrombotic disorders, sepsis, and disseminated intravascular coagulation (DIC), so the mortality rate will be higher.
- Currently, whether the COVID-19 virus can directly activate platelets, and therefore promote its pro-thrombotic function remains unclear.[1]

Materials and Methods

It's a literature review.

Discussion

- Thrombocytopenia is found in 18.8% to 36.2% of patients on admission, according to large-scale researches. Furthermore, COVID-19 patients in the ICU had a 31% cumulative incidence of thrombotic problems, whereas only 1.3% of non-COVID-19 ICU patients had thrombotic issues.
- SARS-CoV-2 uses its Spike protein to enter host cells by binding to angiotensin converting enzyme 2 (ACE2) on the host cell membrane, Meanwhile, transmembrane protease serine 2 (TMPRSS2), proteolytically cleaves and activates the Spike protein to facilitate SARS-CoV-2 virus- cell membrane fusions.
- Although, it has been reported that the Spike protein from SARS-CoV-2 binds to ACE2 and distorts many cell activities, it is still uncertain whether platelets express these factors.
- COVID-19 appears to predispose people to thromboembolic illnesses.

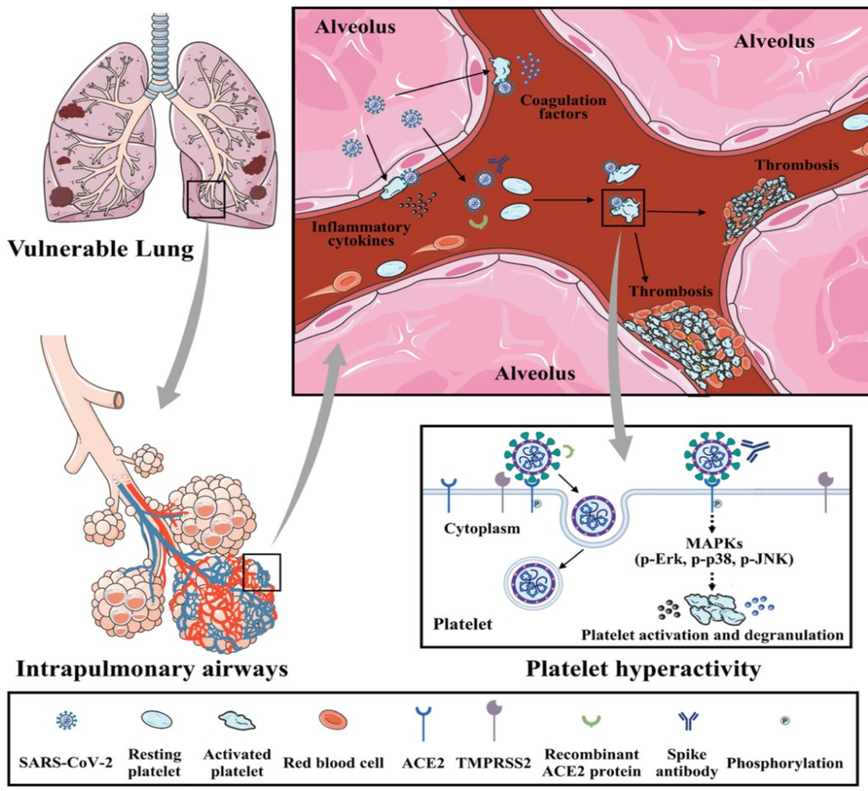

Figure1 adapted from Zhang S 2020

- In this study, they noticed that: **1.**platelets express ACE2 and TMPRSS2; **2.**SARS-CoV-2 and its Spike protein promote platelet function and thrombus formation through the MAPK pathway downstream of ACE2; and **3.**treatment with recombinant human ACE2 protein and anti-Spike monoclonal antibody can block SARS-CoV-2 induced platelet activation and thrombus formation.
- They provide proof that the platelets and key mediators of thrombosis, are hyperactivated in patient with COVID-19, also other recent studies have demonstrated that numerous platelet activation events, including aggregation, adhesion, infiltration, and inflammatory response, contribute to lung damage and microvascular thrombosis in SARS-CoV-2 associated pneumonia.
- These findings, along with those from this investigation, highlight the importance of platelet activation in COVID-19 development.

Conclusion

- In conclusion the study has shown an increased correlation between the thrombotic incidence among covid-19 patients, alongside, decreasing the risk of thrombotic formation with the use of anticoagulants during covid-19 phase.

Reference

1. Zhang S, Liu Y, Wang X, et al. SARS-CoV-2 binds platelet ACE2 to enhance thrombosis in COVID-19. *J Hematol Oncol*. 2020;13(1):120. doi:10.1186/s13045-020-00954-7.