# Antiplatelet Therapy Following Percutaneous Coronary Intervention in Patients Complicated by COVID-19: Implications from Clinical Features to Pathological Findings

**Running Title:** -

**Address for Correspondence:**

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**Figure**

**Figure**

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ndividuals that are thrombocytopenic would lose the ability to deposit fibrinogen and fail to seal the damaged pulmonary vasculature.

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## Disclosures

## References

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## Figure Legend

**Figure. The potential pathophysiological evolution of SARS-CoV-2 infection in lung tissue and implications for antiplatelet therapy.**

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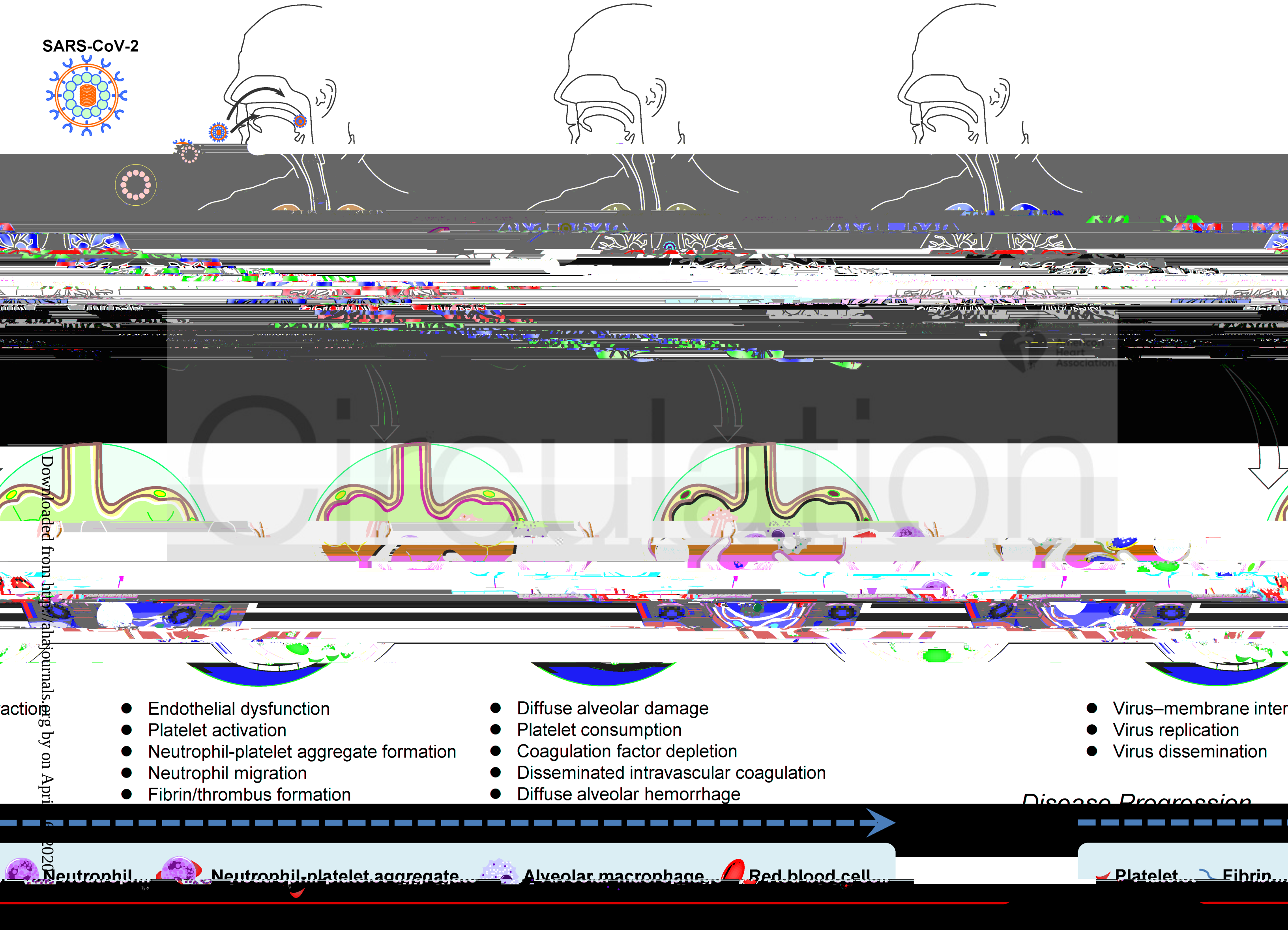
SARS-CoV-2
action
Endothelial dysfunction
Platelet activation
Neutrophil-platelet aggregate formation
Neutrophil migration
Fibrin/thrombus formation
Diffuse alveolar damage
Platelet consumption
Coagulation factor depletion
Disseminated intravascular coagulation
Diffuse alveolar hemorrhage
Virus–membrane inter
Virus replication
Virus dissemination
Disease Progression
Neutrophil
Neutrophil-platelet aggregate
Alveolar macrophage
Red blood cell
Platelet
Fibrin