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

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Figure

Figure

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

Acknowledgments

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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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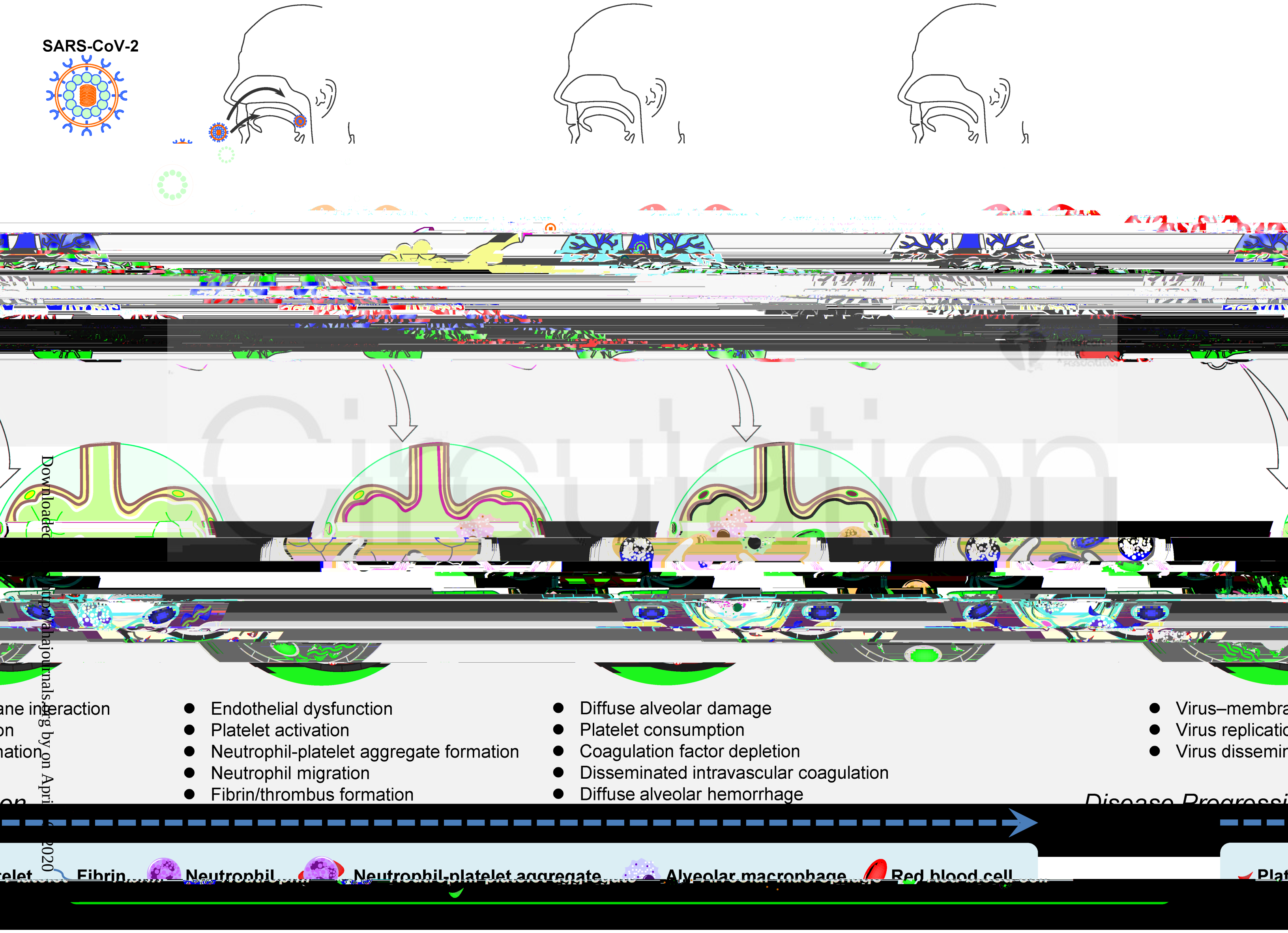

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- Endothelial dysfunction
- Platelet activation
- Neutrophil-platelet aggregate formation
- Neutrophil migration
- Fibrin/thrombus formation

- Diffuse alveolar damage
- Platelet consumption
- Coagulation factor depletion
- Disseminated intravascular coagulation
- Diffuse alveolar hemorrhage

- Virus–membra
- Virus replicatio
- Virus dissemin