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

Running Title: *I d T P X PTT C TaP h P E* -

Address for Correspondence:

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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Sources of Funding

Disclosures

References

1.

- *V 9 T*

2.

V 9 T -

3.

P Vh -

4.

- *dT aX*

5. -

I V dP dT T dT I P I X

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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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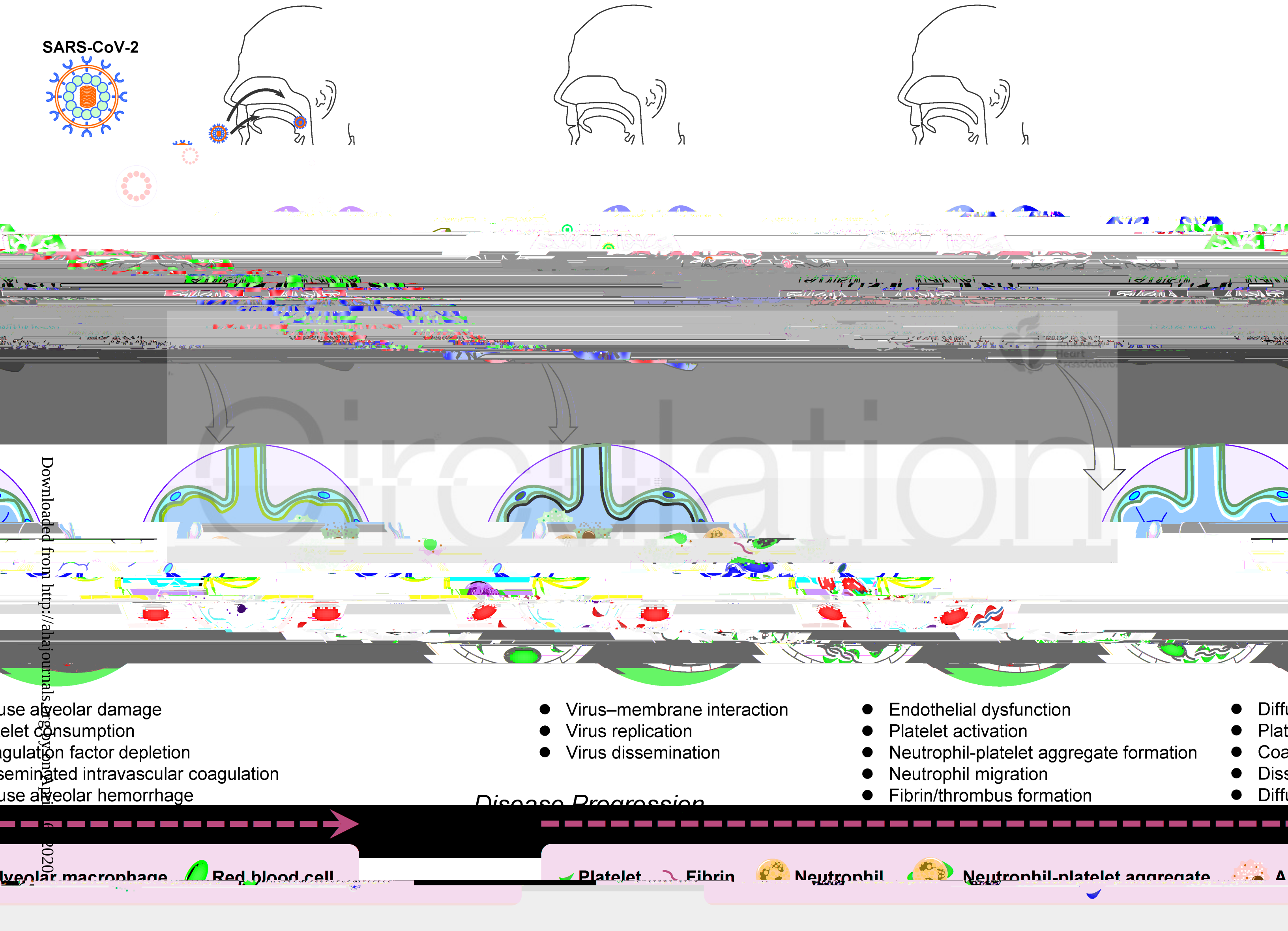
SARS-CoV-2
use alveolar damage
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seminated intravascular coagulation
use alveolar hemorrhage
Virus–membrane interaction
Virus replication
Virus dissemination
Endothelial dysfunction
Platelet activation
Neutrophil-platelet aggregate formation
Neutrophil migration
Fibrin/thrombus formation
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