THE ROLE OF ANGIOTENSIN-CONVERTING ENZYME 2 INTO THE PATOGENESIS OF COVID-19 INFECTION

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Introduction

The angiotensin-converting enzyme 2 (ACE2) is an exopeptidase that catalyzes the conversion of angiotensin II to angiotensin 1-7. ACE2 receptor has recently been identified as the entry point of the severe acute respiratory syndrome 2 coronavirus (SARS-CoV-2) in human cells.

Keywords

„ACE2", SARS-CoV-2", „COVID-19 pathogenesis".

Purpose

To understand the role of ACE 2 receptors in the pathogenesis of new type coronavirus disease – 2019 (COVID-19).

Material and methods

A literature search on the topics of receptor ACE2 was conducted using the MEDLINE database with the PubMed interface. Relevant articles were selected using the following keywords: ACE2, SARS-CoV-2, COVID-19 pathogenesis

Results

ACE 2 is widely expressed in human tissues, especially in type II pneumocytes, endothelial cells, enterocytes. SARS-CoV-2 uses the Angiotensin converting enzyme 2 (ACE2) as a binding receptor for protein S (spike), which allows it to infect host cells. For the viral and cell membranes to be able to fuse, protein S must be cleaved by TPRSS2 (transmembrane-serine protease 2), a change that facilitates the entry of viral particles into the cell. During infection, tissues expressing ACE2 become direct targets, resulting in serious pathological changes and progressive dysfunction of several organs or even death.
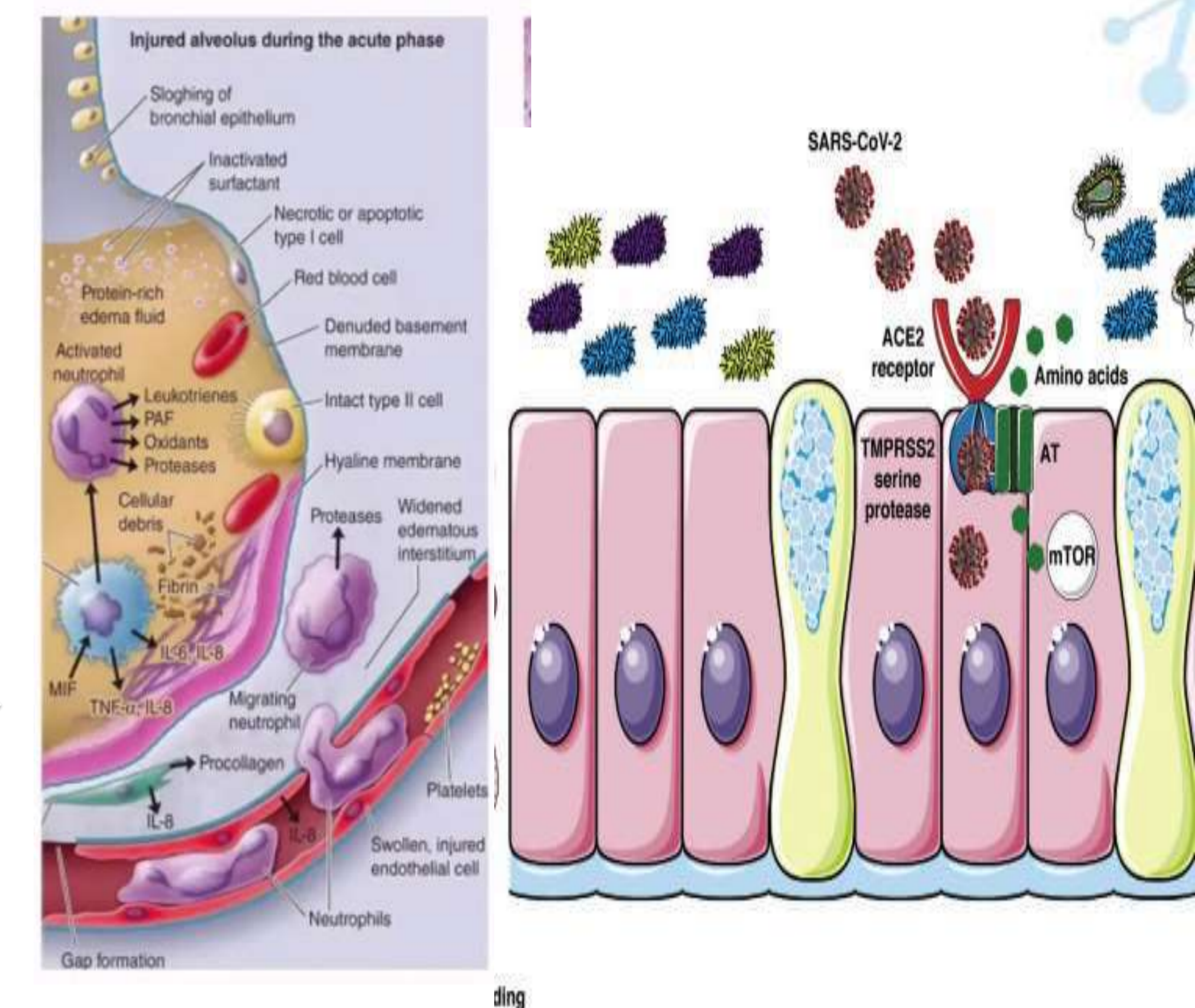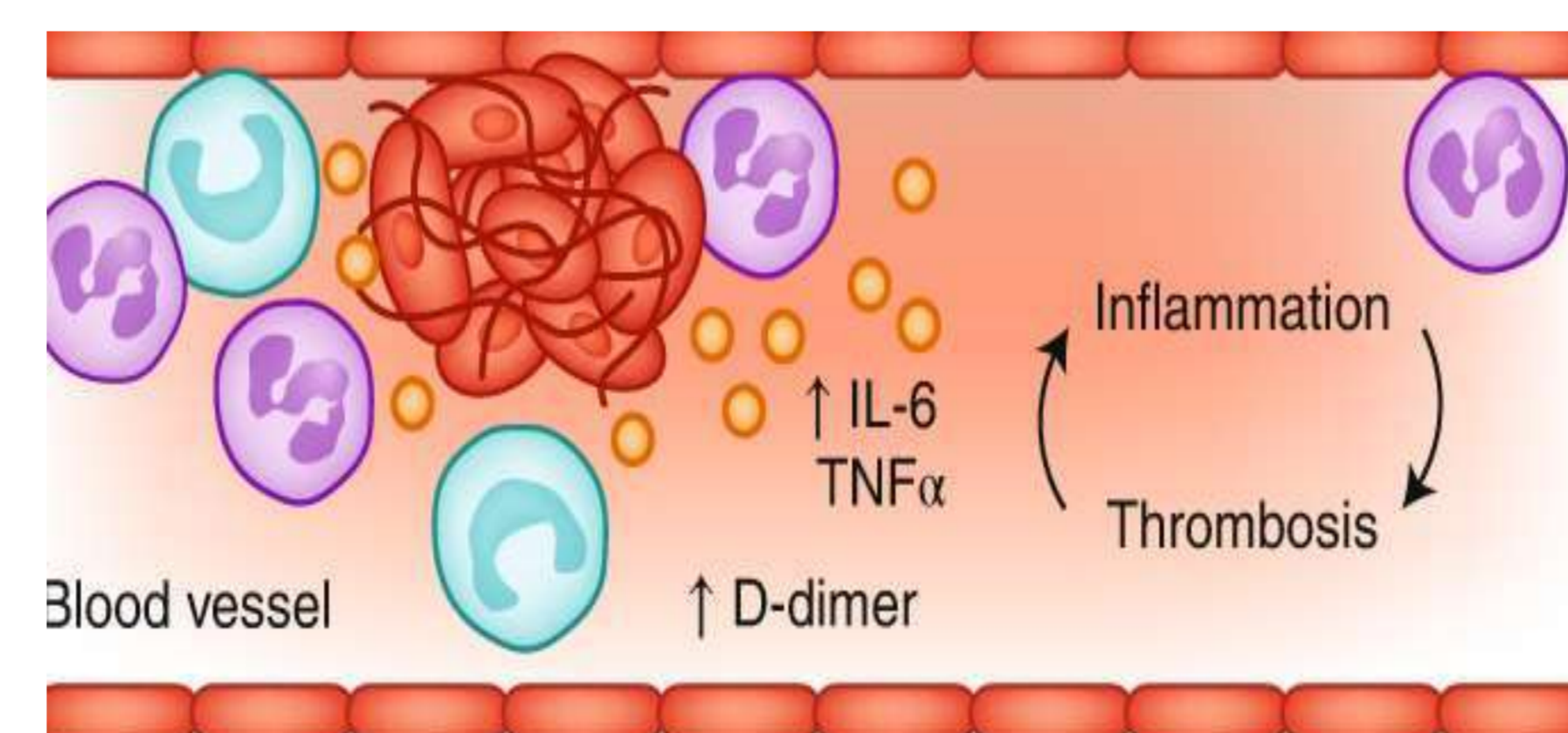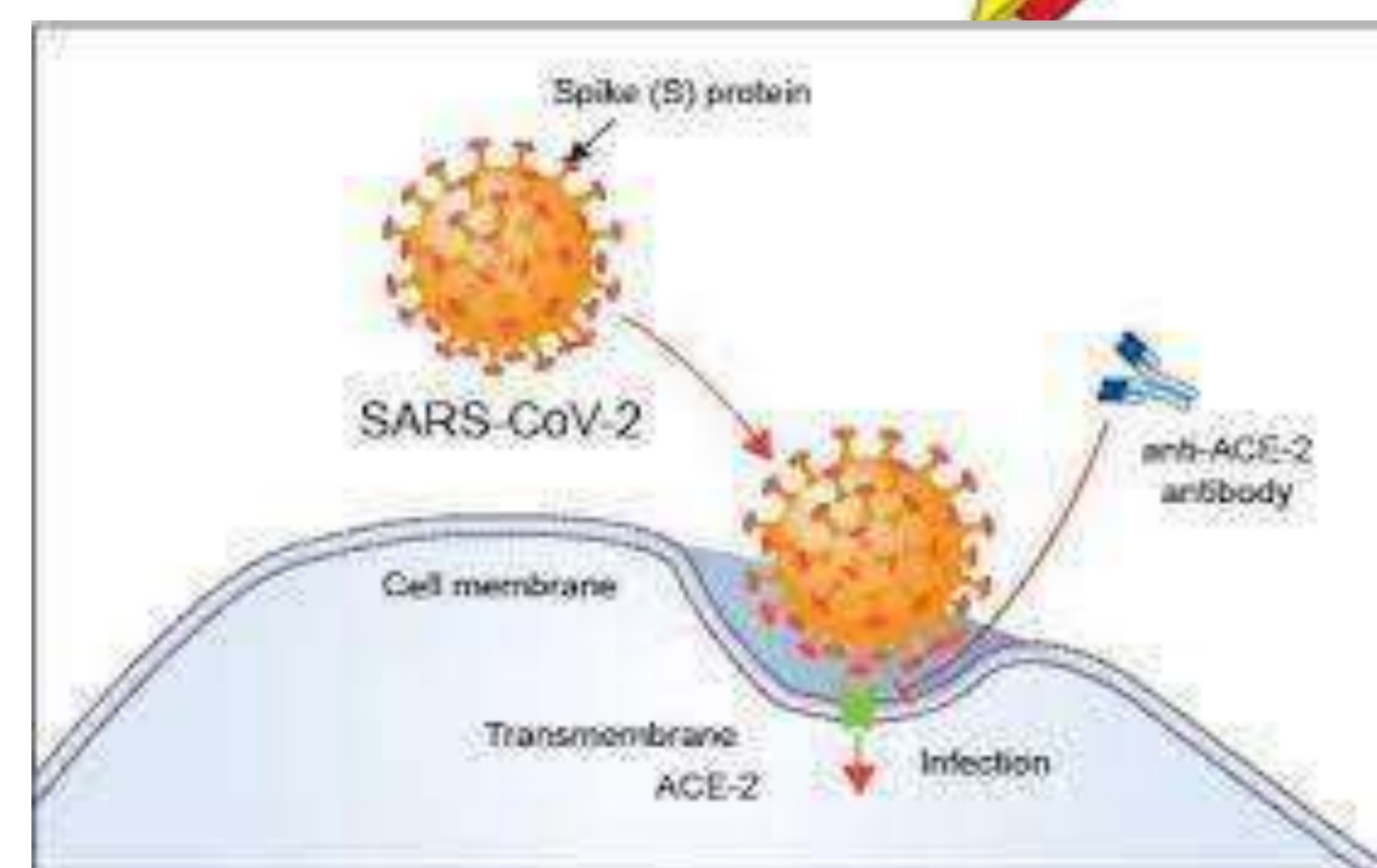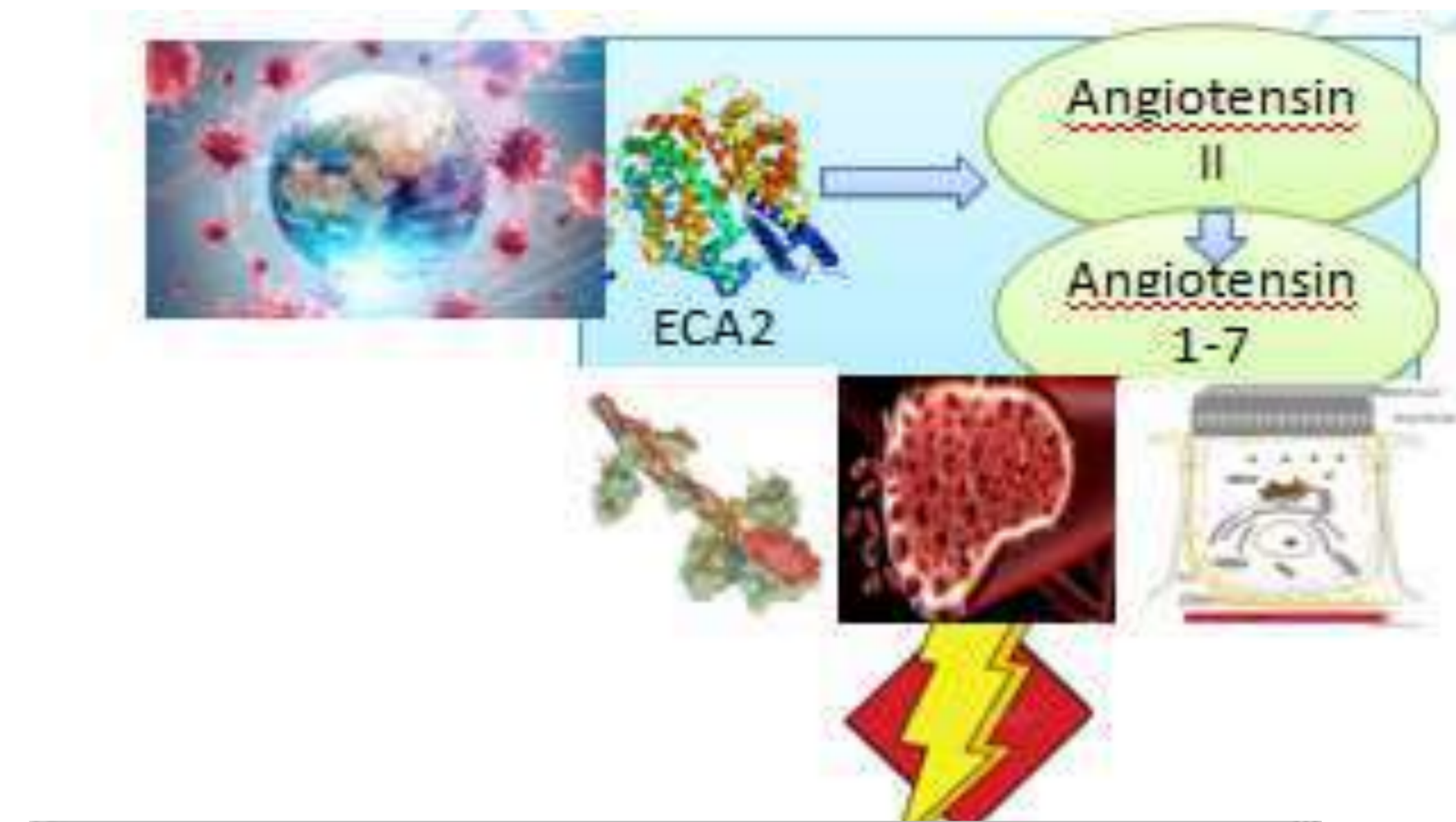
Direct cytotoxic effect

Endothelial cell damage and thromboinflammation
(↑Thrombin production ↓fibrinolysis)

Dysregulation of the RASS:
Tissue injury/remodeling
Inflammation
Vasoconstriction
Vascular permeability

Disregulated immune response:
T cell lymphopenia
Inhibition of interferon signaling SARS-CoV-2
Hyperactive innate immunity

Conclusions

The distribution of ACE2 in different organs is significantly associated with clinical symptoms of SARS-CoV-2 infection. Knowledge of pathogenesis of infection with COVID-19 leads to a proper assessment of the severity and risk of SARS-CoV-2 and to an optimal treatment strategy.