Phosphatidylserine inside out: inflammation and coagulation abnormalities in COVID-19

Gustavo A. Argañaraz
Julys da Fonseca Palmeira
Enrique R. Argañaraz

Video Byte

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Abstract

COVID-19, caused by the virus SARS-CoV-2, is a significant challenge to health systems worldwide. Its severity appears to be most likely caused by acute inflammation and widespread intravascular coagulation. A recent review examines the molecular basis underlying coagulation and inflammation in COVID-19. Phosphatidylserine (PtdSer) typically lies in the inner leaflet of the plasma membrane in healthy cells. Under some physiological conditions, PtdSer exposure on the outer leaflet of platelets promotes coagulation factor aggregation. However, in pathophysiological conditions, PtdSer exposure may cause excessive activation of coagulation and in viral infections, this may increase infectivity and viral spread. Evidence suggests that SARS-CoV-2 exposure may upregulate PtdSer on cell surfaces throughout the body, causing disseminated coagulation throughout the body. In addition, the virus may upregulate the activity of the protein ADAM-17, which has roles in protecting the heart but can also promote inflammation. This level of inflammation can be connected to acute respiratory distress syndrome (ARDS), another leading cause of death among patients with COVID-19. Although more clinical evidence is needed to confirm a causal connection between SARS-CoV-2 infection and PtdSer translocation, better understanding of these mechanisms will help to identify new therapeutic targets against COVID-19.