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Spotlight on Special Topics

SARS-COV-2 PLATELET FUNCTION & THROMBOTIC COMPLICATIONS: EFFECTS OF ASPIRIN THERAPY IN COVID-19

Poster Contributions
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Background: Coronavirus Disease-19 (COVID-19) is associated with an increased risk of thrombotic events such as myocardial infarction (MI), stroke, and venous thromboembolism (VTE). SARS-CoV-2 utilizes viral spike proteins to bind the host transmembrane angiotensin-converting enzyme 2 (ACE2) and a membrane serine protease TMPRSS2 to coordinate host cell viral entry. Activated platelets play a critical role in thrombus formation. However, their influence in COVID-associated thrombosis and the benefit of anti-platelet therapies in SARS-CoV-2 infection are unknown.

Methods: Both ACE2 and TMPRSS2 protein expression was evaluated in healthy human platelets by immunoblotting (n=20, each) and confirmed by confocal microscopy (n=6 ACE2, n=3 TMPRSS2). ACE2 and TMPRSS2 expression was also evaluated by immunoblotting (n=10, each) in patients with coronary artery disease (CAD). Review of 22,072 patients tested for COVID-19 at Cleveland Clinic and propensity matching of 248 COVID positive patients treated/not exposed to the platelet inhibitor aspirin or other non-steroidal anti-inflammatory drugs (NSAIDs) was used to determine if anti-platelet therapy protected patients from death, MI, or VTE.

Results: ACE2 and TMPRSS2 are present on human platelets. There is no association between ACE2 (r^2=0.004, p=0.79) or TMPRSS2 (r^2=0.058, p=0.30) expression and age. There was a numerically higher expression of ACE2 and TMPRSS2 in patients with CAD vs controls. In propensity-matched analyses of aspirin use the incidence of MI and VTE were not different, however aspirin therapy was associated with increased risk of thrombotic stroke (3.6% vs 0.40%, p=0.036) and the composite endpoint of MI, VTE, and stroke (9.3% vs 2.8%; OR 3.52, 95% CI: 1.48-8.40, p=0.005). NSAID therapy was associated with risk of the composite endpoint (3.8% vs 1.6%; OR 2.49, 95% CI: 0.58-1.62, p=0.046). Neither aspirin (OR 0.52, 95% CI: 0.51-1.41; p=0.52) nor NSAIDs (OR 0.97, 95% CI: 0.58-1.62; p=0.90) were associated with mortality effects.

Conclusion: Human platelets express the requisite SARS-CoV-2 receptors to permit viral access but anti-platelet therapy consisting of aspirin does not protect from thrombotic events or mortality.