

# Inflammatory “Endotheliopathy” with COVID 19 Infections in Children, Unanswered Questions for Management Post Recovery

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## Letter

Recent studies from U.K, US and Canada have reported a unique presentation of the novel corona virus (SARS-CoV-2) in the pediatric age group. High fevers, rash, multiorgan involvement was a recurrent mode of presentation; with an overall case fatality rate in the US-Canadian PICU consortium group was reported to be approximately 4.2% [1-7].

This hyper inflammatory shock syndrome is associated with elevated inflammatory markers (C-reactive protein, procalcitonin, ferritin, triglycerides, D-dimers) and has been labeled Pediatric Multi-System Inflammatory Syndrome (PMSIS) [2].

Coronary endothelium affections like aneurysm and life-threatening long-term morbidity has been long understood as a threat to patients that have long recovered from the initial presentation of Kawasaki Disease in childhood [1].

COVID 19 associated polyvasculopathy, evident from recent literature from adult patient’s, appears to be a unique entity due to the affinity of the SARS-CoV-2 to the ACE-2 receptor, which is widely expressed in the human endothelium. SARS-CoV-2 uses the SARS-CoV receptor ACE2 for host cell entry [2].

A strong inflammatory pathway activation thus ensues and leads to downstream effects such as endothelial dysfunction, intravascular coagulopathy, pulmonary micro thrombi, myocardial injury and elevation of cardiac and coagulopathy biomarkers [3].

It’s fathomable thus those mechanisms of pathophysiology in hospitalized children with COVID 19 would not be much different (Figure 1).

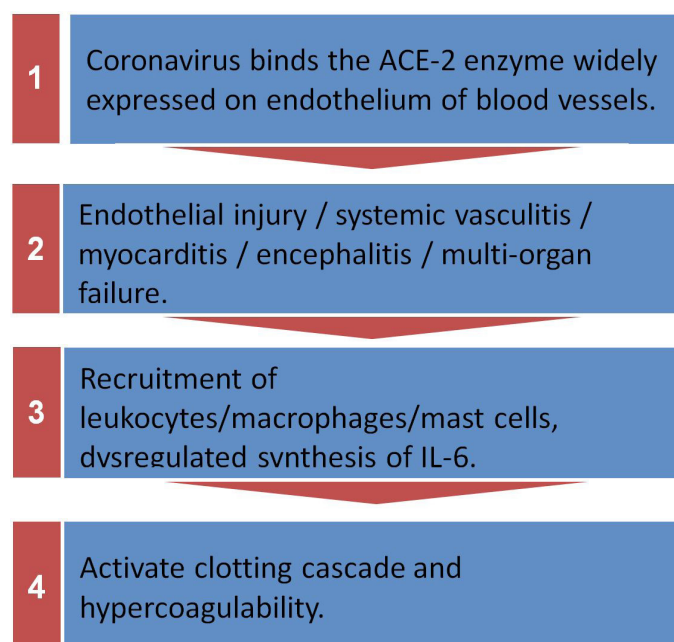


Figure 1: Proposed mechanism of SARS-CoV-2 pathophysiology

A recent study of children from Italy with a Kawasaki-like illness showed that 6 in 10 had abnormal echocardiography but only 2 had abnormal coronary vasculature [6]. The anticoagulant used was aspirin, as is recommended for Kawasaki disease. Aspirin therapy will be continued for 8 weeks and thus far there have been no adverse events reported. This is in short term follow-up.

A correspondence from the United Kingdom reported 8 children with a similar Kawasaki-like illness presenting with shock [7]. One child developed dilated coronaries, and another had an arrhythmia with shock. All of the children were treated with intravenous immunoglobulin but only 6 were given aspirin.

Studies in adult patients have shown a unique clinical phenotype in patients with COVID 19 infection. COVID Coagulopathy is unique due to its predilection for systemic endothelial activation and subsequent thrombosis. 17% of hospitalized patients in France were reported to develop pulmonary embolism and the cumulative incidence of venous thromboembolic event (VTE) in the Netherlands group was 31%. Incidence of bleeding and thrombocytopenia in hospitalized COVID patients was disproportionately low [8,9].

PMSIS cases reported in the UK recently, saw one patient developing a giant coronary aneurysm and another with cerebrovascular infarct [7]. COVID coagulopathy being a non-consumptive coagulopathy in contrast to Disseminated Intravascular Coagulation (DIC) the risk of thrombosis is heightened and one can argue the need of prolonged anticoagulation prophylaxis furthermore due to the prothrombotic nature of this illness.

Post discharge surveillance, choice and duration of anticoagulation, modalities of imaging and assessment of organ function in the pediatric patients with PMSIS are questions that remain unanswered. Thus, aggressive and prolonged anticoagulation in these patients with close cardiac status monitoring for these patients' post recovery would be a prudent.

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