

Is COVID-19 the 'Adult' Form of Kawasaki Disease? Implication for Treatment

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Coronaviruses constitutes a large family of positive-sense, single-stranded RNA viruses. Novel coronavirus species have caused widespread disease among humans around 2002 and 2012. The current pandemic of Covid-19, caused by yet another novel species, SARS-CoV-2, besides shaking the economies worldwide, has brought special challenges for the medical management.

Covid-19 is classified, based on the severity of the presentation, into mild, moderate, and severe forms, leading to shock [1]. That its severity is explained by an upsurge of cytokines is widely accepted. In its severe form, it presents like Kawasaki Disease Shock Syndrome (KDSS) in children.

Kawasaki Disease (KD) is considered a type of systemic vasculitis syndrome, primarily affecting the medium-sized muscular arteries, including coronary arteries. The diagnosis of KD is based on characteristic clinical signs and symptoms and laboratory findings [2]. The term of KDSS was proposed for a small subset of children with KD who manifested hemodynamic instability leading to shock [3].

The question is, is Covid-19 an adult form of the KDSS? The parallel is based on the similarities in their manifestations as compared below.

Aetiology

Epidemiological data indicate involvement of an infectious agents like coronavirus also in KD [2].

Clinical features

Severe form of COVID-19 resembles KDSS in clinical features. Both can have pneumonia, acute respiratory distress syndrome (ARDS), multi-organ dysfunction and shock. Signs include severe dyspnoea, hypoxemia, oliguria, hypotension, cold extremities, skin mottling, and lethargy [1]. Multi-organ dysfunction results from dysregulated immune response and other host defences to the triggering factor, possibly the infection with a virus [2].

Laboratory findings

Both the conditions share laboratory findings of hyper-coagulability and hyperlipidaemia, elevated levels of D-Dimers, C-reactive protein (CRP), creatinine kinase (CK), ferritin, and higher levels of interleukins such as IL- 2, IL-7, granulocyte colony-stimulating factor, interferon gamma-induced protein-10, monocyte chemotactic protein-1 and tumour necrosis factor- α (TNF- α). Lymphopenia and neutrophilia are also detected in both the conditions [1,2,4].

Pathophysiology

Like in KDSS, shock and multi-organ dysfunction in severe COVID-19 are due to upsurge of pro-inflammatory cytokines (TNF- α , IL-6 and IL-1 β), known as cytokine storm, associated with capillary leak and multi-organ dysfunction, leading to death with sustained high cytokine levels over time [5].

Proposal

High dose 2 g/kg of intravenous immunoglobulin (IVIG) over 12 hours is the gold standard treatment of KDSS. In addition, high dose of acetylsalicylic acid as an anti-inflammatory agent is given in the first few days till inflammation has subsided, followed by acetylsalicylic acid at a lower dose used, as anti-platelet, for at least 6 weeks [2].

Since there is no recognised treatment for the severe infection due to COVID-19, based on the similarities between KDSS and Covid-19, the rationale for taking the same line of intervention as in KDSS in the management of Covid-19 is justifiable. Since the use of non-steroidal anti-inflammatory (NSAID) has been questioned in Covid-19, use of acetylsalicylic acid might need exploration and caution.

Conclusion

In addition to supportive treatment, we recommend considering high dose (2 grams/Kg) of IVIG over 12 hours as a treatment for severe form of COVID-19 and high dose of acetylsalicylic acid as an anti-inflammatory agent (with caution) given in the first few days until the inflammation has subsided and then low dose of acetylsalicylic acid as anti-platelet for at least 6 weeks.

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