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COVID-19 and congenital heart disease: *Cardiopulmonary interactions for the worse!*

To The Editor,

As I read through a recent exemplary description of the management concerns emanating as a result of COVID-19 infection in the pediatric age-group featured in the journal,[1] the cardiac anesthesiologist in me feels stimulated enough to elucidate upon the peculiar repercussions of the novel coronavirus (SARS-CoV-2) in congenital heart disease (CHD) patients wherein the cardiopulmonary liaison compounds the clinical situation furthermore.

While a restrained cytokine storm as a consequence of inadequately developed adaptive immune mechanisms and a reduced function of angiotensin-converting enzyme (ACE2) have been suggested to render the pediatric patients less susceptible to a severe form of respiratory disease, the existing experience with viral illnesses such as influenza and respiratory syncytial virus in CHD patients is sufficient enough to raise a sense of caution.[2,3] Moreover, the initial attempts at the characterization of COVID-related disease burden in pediatrics such as the retrospective analysis of 48 confirmed COVID-19-infected pediatric patients admitted in North American intensive care units highlight the importance of preexisting morbidities in dictating the clinical trajectory of the disease.[4] With particular regard to a co-existent CHD, the complexity of the underlying congenital cardiac lesion, syndromic associations with potential multi-systemic involvement and compromised functional and nutritional status present additional predispositions.

In this context, a recent statement issued by the British Congenital Cardiac Association identifies the subset of CHD patients presenting a heightened risk of severe disease following COVID-19 infection which includes[5] the following:

- Single ventricle patients post-Fontan procedure
- Chronically cyanosed patients
- Severe cardiomyopathy with a diminished cardiac performance
- Underlying severe pulmonary artery hypertension (PAH)
- Postorthotopic heart transplant
- Uncorrected significant CHD (ventricular septal defect [VSD], Tetralogy of Fallot [TOF], etc)
- Co-existing morbid conditions (chronic lung, liver, or kidney disease)
- Impaired immune response such as in Down, DiGeorge, and asplenia (heterotaxy) syndrome.

On the one hand, where the concomitant immunosuppression and organ involvement present obvious concerns, and on the other hand, anticipation and mitigation of the altered or the aggravated disease process in unrepaired CHD mandates a sound understanding of the underlying pathophysiology and the prevailing cardiopulmonary interactions. In addition to the systemic effects of infection like increased metabolic demand, tachycardia, and myocardial depression, the tenuous balance of the systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) is disturbed by the ongoing inflammatory process with a consequent elevation of the PVR/SVR ratio.[3] The elevated PVR/SVR ratio favors an enhanced right-to-left shunting which alongside pulmonary venous desaturation leads to the accentuation of cyanosis and precipitation of cyanotic spells in children with TOF. Moreover, the procoagulant milieu potentiates the thrombo-embolic sequel in background of hyperviscosity syndrome with elevated chances of cerebral abscess formation in absence of meticulous air bubble precautions. At the same time, the

children with an increased pulmonary blood flow (PBF) such as VSD are particularly prone to repeated respiratory infections where even a mild degree of pulmonary involvement can exacerbate decompensation. The aforementioned PVR elevation (accentuated by hypoxemia, mechanical ventilation, and stiff lungs in COVID-related acute respiratory distress syndrome) also enhances the risk of right ventricular failure by presenting an augmented afterload to the ventricle owing to the PAH exacerbation. Similarly, PBF following Fontan palliation is inherently sensitive to rise in PVR and the concomitant risk of circuit thrombosis can also not be undermined. Pharmacological control of the PVR by administering inodilators like milrinone or nitric oxide therapy can prove to be pivotal in such circumstances.[3]

These unprecedented times have also raised unique diagnostic and management dilemmas, alike. To name a few, the association between the Kawasaki disease (KD) and SARS-CoV-2 is intriguing with a global increment in the children manifesting KD-like symptoms in the era of this ongoing pandemic.[6] Moreover, ethical and logistic concerns have grown surrounding the smooth provision of interventions such as extracorporeal cardiopulmonary resuscitation (ECPR), particularly in background of overwhelmed critical care resources.[7]

To conclude, the panorama of the epidemiological data on the populations at risk in the current COVID-19 pandemic continues to evolve. With the results of a large prospective observational study on the "impact of COVID-19 in CHD" (COVID-CHD) being ardently awaited, the cohort of our pediatric patients with sick hearts presenting diverse concerns throughout the palliative or corrective course demands a whole-hearted effort on our part, to provide the best clinical care in these challenging times.

CONFLICT OF INTEREST

We do not have any conflict of interest and any commercial or financial interest in this material and agree to abide by the rules of your journal regarding publication of this article.

AUTHOR CONTRIBUTION

Rohan Magoon conceptualized and wrote the original draft.

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The American Academy of Pediatrics; its structure; mission and the importance of getting involved and supporting individual subsections

The American Academy of Pediatrics (AAP) was founded 90 years ago by a group of 35 pediatricians to serve as an independent body to address the needs of children. Specifically, at a time in which children were seen and treated as "small adults," these early visionaries paved the way for adopting much of what is now seen as standard practice. From preventative healthcare visits to the adoption of