COVID-19 pneumonia in an infant with a hemodynamically significant ventricular septal defect

Utkarsh Kohli1, Julia C. Rosebush2, Nicola M. Orlov3 and Ahmeneh Ghavam4

1Section of Pediatric Cardiology, Department of Pediatrics, Comer Children’s Hospital and Pritzker School of Medicine of University of Chicago, Chicago, IL, USA; 2Section of Infectious Diseases, Department of Pediatrics, Comer Children’s Hospital and Pritzker School of Medicine of University of Chicago, Chicago, IL, USA; 3Section of Hospital Medicine, Department of Pediatrics, Comer Children’s Hospital and Pritzker School of Medicine of University of Chicago, Chicago, IL, USA and 4Section of Critical Care, Department of Pediatrics, Comer Children’s Hospital and Pritzker School of Medicine of University of Chicago, Chicago, IL, USA

Abstract

Reports thus far suggest a mild course for acute COVID-19 infection in children; however, its effects in vulnerable paediatric populations, including children with haemodynamically significant congenital heart disease, have rarely been reported. We therefore report on a 4-month-old Hispanic male with a moderate sized conoventricular ventricular septal defect and pulmonary overcirculation who presented with COVID-19-associated pneumonia.

We are currently in the midst of a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) mediated coronavirus disease 2019 (COVID-19) pandemic. In addition to pneumonia and damage to several other organ systems, SARS-CoV-2 infection has been associated with deleterious effects on human myocardium.1–3 Acute COVID-19 infection has been reported to run a milder course in children; however, severe myocardial involvement in children with multisystem inflammatory syndrome-children, a post COVID-19 likely immune mediated entity, has been reported.4–11 Adults with underlying cardiovascular disease, however, have been found to sustain cardiorespiratory damage leading to increased morbidity and mortality due to COVID-19 infection.6 Children with haemodynamically significant congenital heart disease are at an increased risk of decompensation and hospitalisation when concomitantly infected with other respiratory viruses such as respiratory syncytial virus and influenza,12–14 lending credence to the notion that COVID-19 could run a more severe course in these children. As the effects of COVID-19 have rarely been reported in this population,15 we report on the clinical course of an infant with COVID-19 pneumonia and a moderate-sized haemodynamically significant conoventricular ventricular septal defect.

Case report

An otherwise healthy 4-month-old (6.8 kg, 27th percentile for weight) Hispanic boy with a moderate-sized conoventricular ventricular septal defect was transferred to a quaternary care centre from an outside emergency room where he had tested positive (polymerase chain reaction assay) for COVID-19. Both of his parents were also found to be COVID-19 positive and were self-isolating. He was being cared for by his grandmother who noted the patient to be fussy and febrile (102 F) a day prior to presentation. She also reported decreased oral intake and several episodes of non-bilious emesis.

Upon admission to the paediatric intensive care unit, the patient’s heart rate was 163 bpm, blood pressure 83/55 mmHg, respiratory rate 40–52 breaths/minute with increased work of breathing, and oxygen saturation of 99% on supplemental oxygen was noted. Cardiorespiratory examination was remarkable for a grade 3/6 holosystolic murmur best heard at the left lower sternal border and diffuse ronchi heard over bilateral lung fields. His liver was palpable 2 cm below the costal margin, and his chest radiograph demonstrated cardiomegaly, increased pulmonary vascularity, and a left-sided retrocardiac opacity suggestive of pneumonia (Fig 1). An electrocardiogram was performed and only showed sinus tachycardia. The patient was also continuously monitored on telemetry and no arrhythmias, heart block, or ectopy were noted. A trivial patent foramen ovale, a moderate-sized conoventricular ventricular septal defect, moderate left atrial and left ventricular enlargement, and increased flow velocity across pulmonary and mitral valves (suggesting pulmonary overcirculation) were noted on transthoracic echocardiogram (Fig 2). Biventricular function appeared normal and unchanged from the past. The patient was started on high flow nasal cannula (5 L per minute with 60% FiO2) and continued on his home dose of furosemide (6 mg every 12 hours). Serum aspartate aminotransferase (44 U/L; normal: 8–37 U/L) and D-dimer...
Figure 1. The chest radiograph shows levocardia, cardiomegaly, increased pulmonary vascularity, left-sided aortic arch, normal visceral situs, and a left-sided retrocardiac opacity suggesting pneumonia.

Figure 2. The echocardiogram shows moderate left atrial and left ventricular dilation (Panels a, b, and c). The moderate-sized con-overtricular ventricular septal defect is profiled in apical 4-chamber, parasternal long, and parasternal short axis views (red arrows, Panels b, c, and d). Interrogation with Colour Doppler reveals the shunt across the ventricular septal defect to be left-to-right. Continuous wave Doppler interrogation of the pulmonary valve (Panel e) reveals an increase in flow velocity (peak velocity 3.2 m/second, peak gradient 41 mmHg) across a normal-sized pulmonary valve annulus (1.0 cm, Z score = 0.29). An increase in flow velocity (Peak E wave velocity = 1.54 m/second, mean gradient = 3.3 mmHg) is also noted across a normal-sized mitral valve annulus (1.9 cm, Z score = 1.7) upon continuous wave Doppler interrogation suggesting functional mitral stenosis (Panel f).
(7.18 μg/ml; normal < 0.40 μg/ml) concentrations were elevated while his platelet count was mildly decreased (143,000/μl; normal: 150,000–450,000/μl). The rest of his laboratory parameters including ferritin and C-reactive protein were within the normal range. A nasopharyngeal swab was obtained for the SARS-CoV-2 RNA polymerase chain reaction assay (Roche cobas SARS-CoV-2 assay on the cobas 6800, Roche labs, Geneva, Switzerland) and was positive. A nasopharyngeal swab was obtained for other common respiratory pathogens and was found to be negative. Due to resolution of fever, rapid symptomatic improvement, and normalisation of laboratory abnormalities, he was able to be weaned from high flow nasal cannula to room air within 48 hours of admission and discharged home a day later with close follow-up with his primary care physician and cardiologist.

**Discussion**

We report on the case of a 4-month-old boy with a moderate-sized conoventricular ventricular septal defect and left-sided volume overload secondary to pulmonary overcirculation, who developed COVID-19 pneumonia. The patient exhibited mild respiratory symptoms without signs of cardiac decompensation during his hospitalisation. These findings are of interest as COVID-19 pneumonia has not been previously reported in a child with unrepaired, haemodynamically significant congenital heart disease, though there are reports in adults with repaired congenital heart disease and an adult with haemodynamically significant congenital heart disease, though there are reports in adults with repaired congenital heart disease and an adult with unrepaired congenital heart disease. Although we are unable to draw any significant conclusions based on the clinical course of a single patient, the rapid improvement of this patient following admission does raise the possibility that COVID-19 infection may not necessarily be associated with marked decomposition in children with haemodynamically significant congenital heart disease, particularly left-to-right shunts. Given the probable paucity of these patients at any single paediatric centre, there is a dire need for collaborative research efforts on a global scale to characterise the clinical features and outcomes of COVID-19 in children with haemodynamically significant congenital heart disease as well as other vulnerable paediatric populations.

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