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Case Report

A Case of Acute Fulminant Presentation in a Term Neonate with COVID-19

Ramanathan V, Binoy Vadakke Nellissery*, Sandeep Kuchi, Sai Srinivas, Sridhar Kalyanasundaram, Deepu Abraham and Nishath Ahmed

Aster Hospital, Al Qusais, Dubai, UAE

***Corresponding Author:** Binoy Vadakke Nellissery, Aster Hospital, Al Qusais, Dubai, UAE.

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Abstract

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has led to a global pandemic affecting 213 countries as of April 26, 2020. Reports of neonatal cases of COVID-19 remain sparse. we report a case of neonatal COVID-19 with a fulminant course, in the UAE and provide a review of published cases. A 2 days old neonate presented to the emergency room with fever, poor oral intake, reduced activity, and vomiting. On Examination baby was febrile, encephalopathic and had tachycardia and Tachypnoea. During hospital stay baby had Focal seizures and Arrythmias which were treated appropriately. Echo Suggested severe cardiac dysfunction. The PCR test for SARS-CoV-2 was positive. Blood test results revealed marked elevations of troponin I, D dimer, creatinine phosphokinase (CPK) and ferritin, with severe lactic acidosis. Refractory coagulopathy was observed. Blood and urine cultures were sterile. Severe metabolic acidosis that was intractable to treatment was observed. Empirical administration of intravenous immunoglobulin (IVIG) and methyl prednisolone did not show any positive response. Unfortunately, the patient in our case did not respond to all the supportive measures we provided. As Per the currently available literature available, most infants with COVID-19 are asymptomatic or mildly symptomatic. Till date, only one case of neonatal COVID-19 with cardiac involvement has been reported. Our case report could help to alert the medical fraternity that neonatal cases may present with sickness, especially as a trigger to a fulminant presentation due to predisposing conditions.

Keywords: *Troponin I; D Dimer; Creatinine Phosphokinase (CPK); Ferritin*

Introduction

In December 2019, cases of pneumonia caused by the novel severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) began to emerge in Wuhan, Hubei province, China. The SARS-CoV-2 belongs to the genus Betacoronavirus and causes an acute infection termed coronavirus disease or COVID-19. Reports of neonatal cases of COVID-19 remain sparse; however, such cases with information on the clinical course in infected neonates are increasingly being reported. Many aspects of the disease characteristics of COVID-19 in this population remain unknown. Here, we report a case of neonatal COVID-19 with a fulminant course, in the UAE.

Case Presentation

The patient in this case was an infant who was born by ventouse-assisted vaginal delivery at 38 weeks gestation, with a birth weight of 2.77 kg and without any complications. The mother had an uneventful antenatal course and both parents were asymptomatic for COVID-19. The infant was discharged home within 24 hours of life and was active and feeding well at home, as reported by the parents. The following day, at 2 days of life, the infant was presented to the emergency room with one episode of fever, poor oral intake, reduced activity and vomiting.

Physical examination showed that the infant was febrile and lethargic, and displayed reduced responsiveness with decreased tone and sluggish reflexes suggestive of encephalopathy. The anterior fontanelle was normal, with no evidence of abnormal movements or focal neurological deficits. The infant was tachycardic, but showed normal perfusion and cardiac rhythm, at the time. Additionally, the infant

was tachypnoeic and showed increased work of breathing. All other examination findings were normal. The infant had no obvious dysmorphic features, and had a head circumference of 32 cm.

The infant was admitted to the neonatal intensive care unit (NICU) in the negative pressure isolation room, and humidified high-flow nasal cannula therapy was initiated. Baseline investigations, sepsis screen and SARS-CoV-2 polymerase chain reaction (PCR) test were conducted. The infant was administered intravenous antibiotics, due to suspected early onset sepsis with meningitis. One episode of right focal seizure was noted; following this, phenobarbitone was administered.

Persistent fever was noted, with ongoing seizure and worsening neurological status. The infant was electively intubated and ventilated. Intravenous acyclovir was administered due to the worsening clinical and neurological status.

In the NICU, the infant experienced cardiac arrhythmia, with a rhythm suggestive of ventricular tachycardia. Two-dimensional echocardiography scanning by a paediatric cardiologist suggested severe cardiac dysfunction, with regurgitation in the mitral and aortic valves, an ejection fraction of 30%, and normal structure with normal coronaries. The arrhythmia was managed with antiarrhythmic treatment, by using adenosine and amiodarone. Subsequently, the infant developed hypotension with worsening perfusion; this was treated with the initiation and escalation of inotropic support for the persistent hypotension.

The PCR assay conducted to test for SARS-CoV-2 at Authorised Laboratory, Dubai, UAE showed positive results after the day of admission. In view of the above history and clinical presentation, a presumptive diagnosis of viral myocarditis with severe cardiac dysfunction secondary to COVID-19 was considered.

Blood test results revealed marked elevations of troponin I, D-dimer, creatinine phosphokinase (CPK), and ferritin, with severe lactic acidosis. Refractory coagulopathy was observed, despite the administration of multiple fresh frozen plasma transfusions. Blood and urine cultures were sterile. Chest X-ray scans showed bilateral haziness of the lung fields. Computed tomography chest scans could not be performed due to the poor clinical condition of the patient.

In the father's family, a previous baby born of a different partner (first wife) had died due unexplained causes, possibly asphyxia and sepsis. The mother of the baby had lost 2 of her male siblings during their neonatal periods. The present marriage was not consanguineous. In view of the previous sibling deaths (in both mother's family and father's first baby), the presence of an inborn error of metabolism as the underlying condition, with COVID-19 precipitating the worsening of clinical symptoms was considered as a likely possibility. Newborn screening results were normal, except for elevated alanine levels. The laboratory confirmed that urea cycle disorders could not be fully excluded. The infant had elevated serum ammonia levels and very low urea levels.

Due to the worsening cardiac function and arrhythmia, the infant was relocated to another centre to receive intensive paediatric cardiac support and monitoring. The cardiac function and hypotension deteriorated further; therefore, the infant required maximum inotropic support. Empirical administration of intravenous immunoglobulin (IVIG) and methyl prednisolone did not show any positive response. Severe metabolic acidosis that was intractable to treatment was observed. Ammonia levels decreased during this period without specific treatment; however, they remained high. Serum amino acid profile showed high levels of multiple amino acids; the implication of this is unclear. Serum and urine organic acids were normal. Cranial ultrasound scanning showed Grade I germinal matrix haemorrhage on the left side.

The infant died on day 7 of life due to severe cardiac dysfunction and refractory hypotension. A repeat COVID-19 test conducted by endotracheal suction, which likely resulted in an inadequate sample, on the day prior to that of death was reported as negative.

SR. No.	Characteristics	Data
1.	**Patient Characteristics**	
	Gestational age	38 weeks
	Age at presentation	2 days
	Male	
	Mode of delivery	Vaginal
	Resuscitation at delivery	None
	APGAR Scores (1 and 5 min)	8, 9

2.	**Vital signs (on admission)**	
	Temperature (rectal)	38.0°C
	Heart rate	175/min
	Respiratory rate	68/min
	Blood pressure	80/46 mmHg
	Oxygen saturation	98%
3.	**Laboratory values**	
	Complete blood count (Day 2 of life)	
	Haemoglobin (g/dL)	17.4
	White blood cells (x 10^3/µL)	14.4
	Neutrophils (%)	67
	Lymphocytes (%)	24
	Monocytes	7
	Platelets (x 10^3/µL)	3.52
	Complete blood count (Day 3 of life)	
	Haemoglobin (g/dL)	13.6
	White blood cells (x 10^3/µL)	4.44
	Neutrophils (%)	56
	Lymphocytes (%)	31
	Monocytes	10
	Platelets (x 10^3/µL)	1.6
	Prothrombin Time (sec) (Day 1)	
	Control (sec)	11.6
	Value	**36.7**
	INR	1.56
	Activated Partial thromboplastin time (sec)	
	Control	35
	Value	29
	Prothrombin Time (sec) (Day 2)	
	Control (sec)	11.6
	Value	36.7
	INR	3.20
	Activated Partial thromboplastin time (sec)	
	Control	35.2
	Value	29.0
	SGPT (U/L)	17
	SGOT (U/L)	80
	Influenza	Negative
	RSV	Negative
	SARS-Cov-2 PCR (Nasal swab)	Positive
	Ferritin(ng/ml)	> 5000
	D-dimer	14565
	Troponin-I (Normal range: 0-0.0156 ng/ml)	2.0 ng/ml
	Procalcitonin	1.090 ng/ml
	CPK (U/L)	801
	C-Reactive Protein (mg/dl)	3.84

CPK: Creatinine Phosphokinase, INR: International Normalized Ratio, RSV: Respiratory Syncytial Virus, SGPT: Serum Glutamic Pyruvic Transaminase, SGOT: Serum Glutamic Oxaloacetic Transaminase

Discussion

Worldwide, the numbers of COVID-19 cases are rapidly increasing. The UAE government has been actively trying to limit the spread of COVID-19, and even though the number of cases has been rising here (close to 50,000 cases by the end of June 2020), the mortality rate is not very high. We report a case of neonatal COVID-19 presenting with acute cardiac dysfunction and a fulminant course. Reports of COVID-19-related newborn deaths are scarce. This case may have been complicated by a possible inborn error of metabolism; however, since we cannot unambiguously confirm the same, the possibility of a significant role of the infection should be considered, due to the very high levels of procalcitonin, ferritin, and D-dimer, and the decreasing levels of ammonia, despite no specific interventions for this outcome.

The possibility of vertical transmission was considered here as the infant became symptomatic within the first 48 hours of life, despite the mother being asymptomatic. The prevalence of COVID-19 in the community was high during this time period.

Per the currently available literature available, most infants with COVID-19 are asymptomatic or mildly symptomatic. Till date, only one case of neonatal COVID-19 with cardiac involvement has been reported [1]. This case was considered to be vertically transmitted, as the mother tested positive for COVID-19. The infant in this case was symptomatic immediately after birth, with respiratory distress and later developed cardiac dysfunction. This case was reported as congenital myocarditis, and was treated with the administration of inotropes, IVIG, and diuretics. The condition of the infant gradually improved, and the infant was discharged home after 10 days of life [1].

In the present case, we noticed abnormal laboratory parameters, such as, markedly elevated ferritin, troponin I, D-dimer and CPK levels. The presence of refractory coagulopathy was also noted.

These abnormal parameters support the indication of COVID-19 infection. The infant experienced persistent fever throughout the hospital stay; this was managed symptomatically. All blood and urine cultures were sterile. Although the result of the repeat test for COVID-19 on day 6 of life (just prior to death) using an endotracheal suction sample was negative, this may have been a false negative result. A third sample could not be obtained because the infant died.

Munoz., *et al*. reported the case of a 3-week-old neonatal patient presenting with fever, lower respiratory tract symptoms, and significant chest radiographic changes. This infant required ventilatory support and intercostal drain insertion for treatment of pneumothorax. The patient was treated with intravenous antibiotics and administered a 5-day course of hydroxychloroquine and azithromycin [2].

Conclusion

Information on COVID-19 in neonates is limited; therefore, our case report could help to alert the medical fraternity that neonatal cases may present with sickness, especially as a trigger to a fulminant presentation due to predisposing conditions. In this case, the possibility of inborn errors of metabolism contributing to the clinical condition cannot be ruled out. Unfortunately, the patient in our case did not respond to all the supportive measures we provided, and we await further information on specific guidance on successful management of the myocardial dysfunction in such cases.

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