

Case Report

JMR 2021; 7(2): 54-56

March- April

ISSN: 2395-7565

© 2021, All rights reserved

www.medicinarticle.com

Received: 16-01-2021

Accepted: 09-03-2021

COVID-19 Infection with Refractory Diabetic Ketoacidosis- A case Report

Reshmi Mishra¹, Jyoti Ranjan Behera², P. Ramkumar³, Mukesh Kumar Jain²

¹ Associate Professor, Department of Pediatrics, Kalinga Institute of Medical Sciences, Bhubaneswar, Odisha, India

² Assistant Professor, Department of Pediatrics, Kalinga Institute of Medical Sciences, Bhubaneswar, Odisha, India

³ Post Graduate Student, Department of Pediatrics, Kalinga Institute of Medical Sciences, Bhubaneswar, Odisha, India

Abstract

Diabetic ketoacidosis is an acute life-threatening complication of type 1 diabetes. Sometimes it is the first presentation in an undiagnosed child. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) disease (COVID-19) and diabetes mellitus are very much interrelated as diabetes mellitus is associated with an increased risk of severe COVID-19 at the same time, many cases of new-onset diabetes had been diagnosed. Hyperglycemia, metabolic acidosis, and ketonemia are classical presentations. It is essential to correct the acidosis and fluid correction and insulin therapy in these patients, leading to vital organ dysfunction. In refractory metabolic acidosis, renal replacement therapy may help.

Keywords: COVID-19, Refractory DKA, Renal replacement therapy in DKA.

INTRODUCTION

The whole world has been affected by a serious and fatal pandemic, the severe acute respiratory syndrome coronavirus 2 (SARS-COV2; COVID-19). The severity of the diseases is more in diabetic patients. SARS-CoV-1 binds to the ACE2 receptor in the pancreatic islets, causing damage and acute diabetes [1].

There are reports of COVID-19 to accelerate the fat breakdown and induce ketosis, further developing ketoacidosis, even in non-diabetic patients [2]. Type 1 DM diagnosis is usually suggested by a high level of HbA1C, which indicates a longstanding duration of the disease. There are many reports of COVID -19 with type II DM, but few reports with DKA as the only presentation of COVID-19.

CASE REPORT

A seven-year-old girl presented to the emergency department with complaints of acute onset of abdominal pain associated with vomiting for two days and breathing difficulty along with abnormal behavior for six hours. She had received some primary care in the form of intravenous fluids and antiemetics before being referred to our institute. There was no antecedent history of fever, cough, loose motion, polyuria, polydipsia, or weight loss.

She was admitted to the pediatric intensive care unit with a GCS of 8/15, heart rate of 160 per minute, blood pressure 88/54 mm Hg, and prolonged CFT. Her respiratory was 50 per minute, and she had Kussmaul breathing. The oxygen saturation was 90% with a non-breathing mask. The random blood sugar was more than 500 mg/dL with a large serum ketone body (3.8mmol/lit). The ABG showed severe metabolic acidosis with a high anion gap (Table 1). Thus the diagnosis of severe DKA was made.

A bolus of normal saline (20ml/kg) was given initially. The fluid deficit was calculated by taking a 10% deficiency and was replaced with the maintenance fluid. Antibiotic, as well as insulin infusion at a rate of 0.1 unit/kg/hour, was initiated. Because of severe refractory acidosis, sodium bicarbonate correction was also given. As per the need, the child was intubated and put under a ventilator, anticonvulsants and inotropes were started. The investigation reports raised the suspicion of COVID 19 infection (Table 2). The chest X-ray showed opacities in the right upper and middle zone, which further strengthened our suspicion compounded by the history that both the parents were positive for COVID-19. However, the rapid antigen test was negative. The reverse transcriptase-polymerase chain reaction test was not available at our institute at that point in the night, so planned for the next morning.

***Corresponding author:**

Dr. Reshmi Mishra

Associate Professor,
Department of Pediatrics,
Kalinga Institute of Medical
Sciences, Bhubaneswar,
Odisha, India

Email:

docreshmis[at]gmail.com

Table 1: Serial ABG findings

	Admission	6 hrs	12 hrs
pH	6.69	6.76	6.89
PCo2	30.3	58.2	58.4
HCO3	3.4	5.4	7.2
Base Excess	-28.5	-23.9	-20.5
Anion Gap	30.4	26.5	19.6

Table 2: Investigation Reports

Investigation	Patient Value
TLC	34520 cell/cumm (N - 72%, L-23%)
Hb	14.8 gm/dl
Platelet count	5,18,000 cell/cumm
CRP	30 mg/L
S.urea	48 mg/dl
S.creatinine	1.48 mg/dl
S.sodium	144 mmol/L
S.potassium	3.2 mmol/L
S.calcium	9.3 mg/dl
PT	16.6 sec
INR	1.51
aPTT	27.9 sec
Sr Ferritin	850
D.Dimer	1.27 µg/ml

However, there was little improvement in blood sugar and ketone body level despite five hours of insulin infusion. Hence tube priming was done, fluid and electrolytes were adjusted, and the insulin infusion was increased to 0.15 U/kg/Hr after changing the brand of insulin. Because of the high TLC, CRP, and right upper and middle zone opacities in X-ray the antibiotics were upgraded.

Because of the possibility of cerebral edema, 0.5 mL/kg/hour of 3% sodium chloride infusion was started, and fluid intake was optimized. To improve blood pressure, multiple ionotropes were gradually added. This, however, proved to be of little benefit, and the child succumbed within 12 hours of admission despite all efforts.

DISCUSSION

As the COVID 19 pandemic is grappling the whole world, an increase in the number of newly diagnosed diabetes cases is being seen. When infected by this deadly virus, the known diabetics are experiencing poor glycemic control despite treatment [1]. However, these data are primarily limited to type- 2 diabetes. Data regarding SARSCov2 infection in patients with type 1 diabetics are limited to a few case reports [2]. The Angiotensin-converting enzyme 2 (ACE2), which serves as the entry portal for SARS-CoV-2, is highly expressed in the pancreatic beta cells. Virus mediated cellular injury resulting in decreased insulin secretion is the proposed pathogenesis of ketoacidosis [3]. Studies have also reported that COVID-19 might accelerate the fat breakdown and induce ketosis, further developing ketoacidosis even in non-diabetic patients [4]. Type 1 DM diagnosis is usually suggested by a high level of HbA1C, which indicates a longstanding duration of the disease [5]. In our case, the HbA1C level was 10.5%, suggesting that the child had Type 1 DM, but it was not evident until she was affected by COVID-19.

Several proinflammatory cytokines, whose release has been found to be triggered by the SARS Cov 2 virus, can lead to third spacing and fluid

refractory shock in children [5]. Moreover, the refractory metabolic acidosis in such patients leads to catecholamine-resistant shock [6]. The acute renal injury precipitated by the gross hemodynamic derangements was further complicated by the COVID 19 infection in our case [2]. The child had multisystem inflammatory syndrome features in children (MIS-C), an entity that is typically being observed in children infected with COVID 19, and its incidence is on the rise worldwide [7]. Shock in MIS-C is usually due to poor myocardial contractility, which is diagnosed by low left ventricular ejection fraction (LVEF) in echocardiography [8]. Unfortunately, echocardiography could not be done in this child due to a very short stay. The role of bicarbonate to manage the persistence of acidosis is not clearly defined [9]. There are few adult reports which show a rapid reversal of acidemia with renal replacement therapy in DKA patients [10, 11].

CONCLUSION

Renal replacement therapy should be considered as an early intervention in children with DKA with persistent acidemia, even with fluid and insulin therapy. We can consider peritoneal dialysis in the case of hemodynamically unstable patients to prevent poor outcomes. More extensive studies are required to establish the possible association of COVID 19 infection and presentation of DKA in a previously undiagnosed type-1 diabetes and look for the factors leading to such resistance to insulin therapy in DKA usually respond excellently.

Funding

None.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

REFERENCES

1. Yang JK, Lin ss, Ji XJ, Guo LM Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. *Acta Diabetol.* 2010;47(3):193-199
2. Li J, Wang X, Chen J, Zuo X, Zhang H, Deng A. COVID-19 infection may cause ketosis and ketoacidosis [published online ahead of print, 2020 Apr 20]. *Diabetes Obes Metab.* 2020;10.1111/dom.14057.
3. Chee YJ, Ng SJH, Yeoh E. Diabetic ketoacidosis precipitated by Covid-19 in a patient with newly diagnosed diabetes mellitus. *Diabetes Res Clin Pract.* 2020;164:108166.
4. Daniel S, Gadhiya B, Parikh A, Joshi P. COVID-19 in a Child With Diabetic Ketoacidosis: An Instigator, a Deviator or a Spectator. *Indian Pediatr.* 2020 Jul 15:S097475591600211.
5. Yang J, Lin S, Ji X, Guo LM. Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. *Acta Diabetol.* 2010;47:193-99.
6. H Su, M Yang, C Wan, Yi LX, Tang F, Zhu HY, et al. Renal histopathological analysis of 26 postmortem findings of patients with COVID-19 in China. *Kidney Int.* 2020;98:219-27.
7. Mitchell JH, Wildenthal K, Johnson Jr RL. The effects of acid-base disturbances on cardiovascular and pulmonary function. *Kidney Int* 1972;1(5):375-389. DOI: 10.1038/ki.1972.48.
8. Pawar MS. Multi system inflammatory syndrome in children and adolescents temporally related to COVID-19. Available at: <https://www.who.int/publications-detail/multisysteminflammatory-syndrome-in-children-andadolescents-with-covid-19.%20Accessed%2015%20May%202020> <https://www.who.int/publications-detail/multisysteminflammatory-syndrome-in-children-andadolescents-with-covid-19>. Accessed 15 May 2020.
9. Belhadjer Z, Méot M, Bajolle F. Acute heart failure in multisystem inflammatory syndrome in children (MIS-C) in the context of global SARS-CoV-2 pandemic [published online ahead of print, 2020 May 17]. *Circulation.* 2020;10.1161/CIRCULATIONAHA.120.048360.
10. Chua HR, Schneider A, Bellomo R. Bicarbonate in diabetic ketoacidosis - a systematic review. *Ann Intensive Care* 2011;1(1):23.

11. Singh H, Krishna RHS, Jain A, Sharma N. Peritoneal dialysis for the rescue in critical refractory metabolic acidosis in diabetic ketoacidosis. *J Family Med Prim Care* 2019;8(5):1792–1793. DOI: 10.4103/jfmpc.jfmpc_138_19.