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Severe COVID pneumonia complicated with transient Diabetic ketoacidosis in a diabetes-naive patient: A case report and literature review

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Abstract

A very few cases with Coronavirus disease (COVID-19) inducing Diabetic ketoacidosis in non-diabetic patients are reported. Hyperglycaemia caused by transient damage to pancreatic cells by the virus and cytokine storm is identified as the underlying pathophysiology. Here, we present a patient who presented with transient Diabetic ketoacidosis while being treated for COVID-19 pneumonia and is the first reported case in Sri Lanka as per literature. We highlight the approach to diagnosis, monitoring and management of this patient which was challenging for the treating physician.

Case: A 34-year-old previously well Sri Lankan male presented with severe covid pneumonia. He was found to have diabetic ketoacidosis and managed as per protocol with fluid and insulin. His glycosylated haemoglobin was normal. Even though his Covid pneumonia worsened leading to death, he recovered from diabetic ketoacidosis and did not require exogenous insulin later in the course of the illness.

Conclusion: Transient hyperglycemia with ketoacidosis, such as in our patient, associated with Covid infection has been reported very rarely in the literature. The treating physician should have a high index of suspicion to diagnose these types of patients and to manage them timely. Otherwise, it will be an added but a preventable factor to increase mortality in patients with Covid pneumonia.

Keywords: coronavirus disease; COVID-19; non-diabetes; diabetes-naive; hyperglycemia; diabetic ketoacidosis.

Abbreviations: COVID -19: Coronavirus Disease; Fio2: Fraction Of Inspired Oxygen; Pao2: Partial Pressure Of Oxygen; Paco2- Partial Pressure Of Carbon Dioxide; HCO3: Bi Carbonate, DKA: Diabetic Ketoacidosis; IV: Intra Venous; KCL: Potassium Chloride; VBG: Venous Blood Gas; ICU: Intensive Care Unit; HFNO: High Flow Nasal Oxygen; NIV; Non-Invasive Ventilation; PEEP: Positive End-Expiratory Pressure; CRP: C-Reactive Protein; ARDS: Acute Respiratory Distress Syndrome; ECMO: Extracorporeal Membrane Oxygenation; ACE-2: Angiotensin Converting Enzyme-2; TNF-A: Tumour Necrosis Factor; IL-6- Interleukin 6.

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Introduction

Coronavirus disease (COVID-19) causing ketosis and ketoacidosis among patients with underlying type-1 or type-2 diabetes are reported from many countries [1,2]. However, cases with COVID-19 infection causing ketoacidosis in non-diabetic patients are scarce [1,3].

Herein, we present a case of severe COVID-19 pneumonia inducing transient diabetic ketoacidosis in a non-diabetic patient and is the first reported such case in Sri Lanka.

Case report

34-year-old male, previously well, presented to a local hospital with a history of fever and sore throat for 6 days. He was positive for SARS-CoV-2 rapid antigen test and transferred to a COVID-19 treatment centre. On admission, he was conscious and rational with blood pressure of 123/78 mmHg with a pulse rate of 110/min and an on-air saturation of 84%. His saturation was 92% on FiO_2 of 60% via venture mask and a PaO_2 of 70 mmHg and $PaCO_2$ of 22 mmHg. On admission, chest radiograph showed bilateral patchy opacifications (Figure 1). His random blood sugar was 413 mg/dl and denied a history of diabetes mellitus. pH was 7.26 with HCO_3 of 11 mmol/L and had significant ketonuria (+3). A diagnosis of severe covid pneumonia with Diabetic Ketoacidosis (DKA) was made and the patient was fluid resuscitated as per DKA guidelines. He was started on intravenous (IV) soluble insulin 0.1 u/kg (6 units) per hour regimen, IV dexamethasone 6mg daily and S/C enoxaparin 40 mg daily dose. The patient was also started on IV ceftriaxone 1 g twice daily and IV levofloxacin. His potassium level was 3.2 mmol/l and was replaced with added potassium chloride (KCL) to intravenous 0.9% saline. His blood sugar and Venous Blood Gas (VBG) were done as per protocol. The results of his blood sugar, venous gases and ketones were given in Table 1.

On day 2 of admission to Intensive Care Unit (ICU), the patient's oxygen requirement increased requiring High Flow Nasal Oxygen (HFNO) with FiO_2 of 70% with a flow of 60 l/min. He remained on HFNO for 6 hours but showed a marked increase in work of breathing and started on Non-Invasive Ventilation (NIV) with pressure support of 8 mmHg and PEEP of 5 mmHg. A repeat chest radiograph was performed and it showed worsening of bilateral peripheral opacifications. At this point, a repeat C-Reactive Protein (CRP) showed a value of 180 with a procalcitonin of 0.01. A decision was made to give a bolus dose of IV Tocilizumab 600 mg. The insulin 6 units/hour infusion was continued for 48 hours. 0.9% saline was continued and a 5% dextrose infusion was started once blood sugar reduced below 250 mg/dl. VBG showed clearing of acidosis with an absence of ketones in the urine.

On the third day of the ICU stay, prior to stopping insulin infusion the patient had worsening glycaemic control and reappearance of DKA. IV insulin infusion was increased to 8 units per hour and was maintained on the same for another 12 hours. On day 4 of ICU stay, complete resolution of DKA status was observed. Thereafter he was started on S/C insulin three times a day as per blood sugar levels. He did not require insulin after the 6th day of the ICU stay and insulin had to be stopped as his blood sugar became persistently normal. His HbA1c level on admission was 4.5%.

On the 7th of the ICU stay, he desaturated on NIV, requiring intubation and invasive ventilation. A repeat chest radiograph showed an Acute Respiratory Distress Syndrome (ARDS) like picture. He was referred for Extracorporeal Membrane Oxygenation (ECMO) due to his resistant hypoxia. He was paralysed and ventilated in prone position and despite this, he remained hypoxic on FiO_2 100% with a PEEP of 15 mmHg. He did not have further episodes of ketoacidosis. He succumbed on day 10 due to severe hypoxemia while awaiting ECMO.

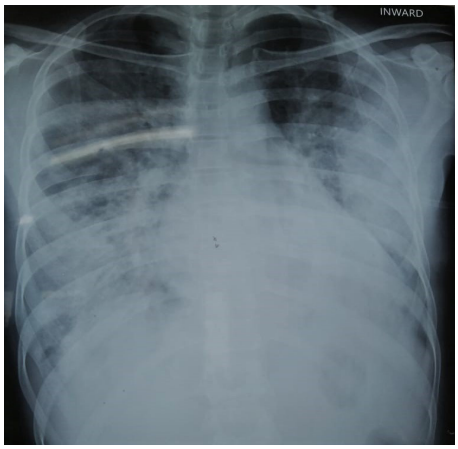

Figure 1: Chest radiograph on admission showing bilateral patchy opacifications.

Table 1: Laboratory profile during ICU stay.

	On admission	6 hrs after admission	12 hrs after admission	24 hrs after admission
pH	7.210	7.239	7.317	7.357
HCO_3 (mmol/l)	12	13.3	16.5	21.3
Base excess	-13	-12	-7	-5.4
Serum sodium (mmol/l)	132	133	134	135
Serum pottasium	3.2	3.1	3.5	4.5
Serum glucose	413	318	257	201
Urine ketone bodies	+++	+++	++	+
Lactate	3.2	3.3	1.1	0.5

Discussion

Although Diabetic Ketoacidosis (DKA), a potentially fatal metabolic complication, seen among people with diabetes mellitus, viral infections including Coronavirus disease are reported to cause ketoacidosis in non-diabetic patients [1,3]. Similarly, our patient developed DKA in the absence of a past history of diabetes and with a normal HbA1C level.

SARS-CoV-2 binds to ACE-2 (Angiotensin Converting Enzyme-2) receptors to enter into cells. These receptors are present on pancreatic beta cells as well. Direct damage to pancreatic cells by the virus leads to insulin deficiency and subsequent DKA [2]. In addition, cytokine storm caused by Coronavirus leads to increased production of pro-inflammatory cytokines

such as TNF-α and IL-6 that are known to induce hyperglycemia regardless of patient's diabetic status [1,4]. Our patient had clinically severe COVID-19 pneumonia so that the stress caused by activation of systemic inflammatory response was severe enough to precipitate DKA.

Previous studies have documented the effective use of Tocilizumab, a monoclonal antibody against IL-6, for patients who had severe COVID-19 disease due to cytokine storm[5]. Unfortunately, our patient did not respond well to Toclizumab. After the resolution of ketoacidosis, our patient had good glycaemic control without exogenous insulin.

The mechanism of resolving DKA and normalizing blood sugar without exogenous insulin while his covid 19 infection continued to deteriorate remains to be ascertained. Our case highlights the importance of monitoring blood sugar and look for DKA even in non-diabetic patients. Hence, the possibility of ketosis and DKA should be considered in all hospitalized COVID-19 patients as they can increase the morbidity and mortality risks. However, the insulin requirement may reduce drastically, as was the case in our patient, where insulin treatment needed to be withdrawn to prevent hypoglycemia. However, our patient passed away due to severe hypoxemia.

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Authors' contribution: All authors have equal contributions in this case. All authors were involved in the management of the patient and read and approved the final manuscript.

Consent for publication: Written informed consent was obtained from the patient's wife for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials: Not applicable

Competing interests: The authors declare that they have no competing interests.

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