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Letter to the Editor

Diabetic ketoacidosis precipitated by Covid-19 in a patient with newly diagnosed diabetes mellitus

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There is scarce data on diabetic ketoacidosis (DKA) in Covid-19 infection. We report a case of DKA precipitated by Covid-19 in a patient with newly diagnosed diabetes mellitus.

A 37 year-old, previously healthy man presented with 1 week history of fever, vomiting, polydipsia and polyuria.

On admission, his temperature was 38.5 °C. He was haemodynamically stable but mildly tachycardic. He did not display Kussmaul's breathing and did not require supplemental oxygen. His body mass index was 22.6 kg/m^2 with no evidence of insulin resistance.

Given positive contact history, he was tested and confirmed to be infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Laboratory investigations (Table 1) were significant for hyperglycemia, high anion gap metabolic acidosis and ketonemia, confirming the diagnosis of DKA.

He received 6 L of intravenous fluids and intravenous insulin infusion in the first 24 h. Serum electrolytes were closely monitored. DKA resolved the following day and he was transitioned to subcutaneous insulin therapy.

DKA occurs as a result of insulin deficiency and increased counterregulatory responses, which favour the production of ketones. The interactions between SARS-CoV-2 and the renin-angiotensin-aldosterone system (RAAS) might provide another mechanism in the pathophysiology of DKA.

Angiotensin-converting enzyme 2 (ACE2), a key enzyme in the RAAS, catalyzes the conversion of angiotensin II to angiotensin (1–7) [1]. ACE2 is highly expressed in the lungs, pancreas and serves as the entry point for SARS-CoV-2 [1]. After endocytosis of the virus complex, ACE2 expression is downregulated [2]. There are 2 implications of these interactions. Firstly, entry of SARS-CoV-2 into pancreatic islet cells may directly aggravate beta cell injury [3]. Secondly, downregulation of ACE2 after viral entry can lead to unopposed angiotensin II, which may impede insulin secretion [4]. These 2 factors might have contributed to the acute worsening of pancreatic beta cell function and precipitated DKA in this patient.

In addition, the relationship between SARS-CoV-2 and the RAAS can complicate DKA management. Excessive fluid resuscitation may potentiate acute respiratory distress

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Table 1 – Laboratory results.

Investigation	Result	Reference Range
Venous glucose (mmol/L)	39.7	–
Arterial blood gas		
pH (mmHg)	7.28	7.25–7.35
Bicarbonate (mmol/L)	12	22–28
pCO2 (mmHg)	25	35–45
Sodium (mmol/L)	128	135–145
Chloride (mmol/L)	86	95–110
Anion gap	30	8–16
Ketones (mmol/L)	6.4	<0.6
Creatinine (umol/L)	95	67–112
Glycated haemoglobin (%)	14.2	–

syndrome as angiotensin II increases pulmonary vascular permeability and worsens damage to lung parenchyma [5]. Furthermore, angiotensin II stimulates aldosterone secretion, potentiating the risk of hypokalemia, which may necessitate more potassium supplementation in order to continue intravenous insulin to suppress ketogenesis.

In conclusion, it is possible that SARS-CoV-2 may aggravate pancreatic beta cell function and precipitate DKA. Further studies will help delineate the pathophysiology. We also highlight the pertinent clinical considerations in the concurrent management of two life-threatening conditions – DKA and Covid-19.

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