Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Case Report

Newly diagnosed diabetes and diabetic ketoacidosis precipitated by COVID-19 infection

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COVID-19 infections and diabetes have been linked since early reports identified patients with diabetes mellitus having worse clinical outcomes [1]. COVID-19 infections have been shown to cause hyperglycemia in patients with known diabetes [2]. However, there has only been one case reported on COVID-19 infection precipitating a new diagnosis of diabetes mellitus type II [3]. We report a case of an individual without prior history of diabetes presenting in diabetic ketoacidosis after being diagnosed with COVID-19 one week prior.

A 54-year-old male presented to the emergency department (ED) via EMS for worsening shortness of breath. Patient reported a three-week history of fatigue and then developed shortness of breath and a cough one week prior to presentation. Shortly after the patient's shortness of breath developed, the patient was tested as an outpatient for COVID-19 and tested positive. The patient also endorsed loss of taste, lightheadedness and an intermittent cough. His past medical history is significant for hypertension, kidney stones, testicular hypofunction and erectile dysfunction. He had no prior surgeries, occasionally smoked cigars, and denied alcohol or drug use. His vital signs were blood pressure 143/87 mm/Hg, heart rate 110 beats per minute, Temperature 98.1 °F orally, Respirations 26 breaths per minute, SpO2 98% on room air. He had a BMI of 42.56 kg/m^2. On physical exam he appeared ill and was tachycardic and tachypneic. His heart sounds were normal, his lungs clear to auscultation, abdomen was soft and nontender with normal bowel sounds, his legs revealed no swelling, and he had a normal neurological exam. The remainder of his physical examination was unremarkable.

Testing in the ED revealed hyperglycemia, anion gap metabolic acidosis and ketonuria which confirmed the patient to be in diabetic ketoacidosis (DKA). He had a blood glucose of 463 mg/dL, sodium of 126 mmol/L, potassium of 5.5 mmol/L, chloride of 86 mmol/L, and CO2 of 9 mmol/L, creatinine of 1.24 mg/dL and an anion gap of 31. His WBC were 9.3 thou/cmm, with absolute neutrophils of 7.56 thou/cmm. The remainder of his CBC differential was normal. His venous pH was 7.193, pCO2 was 26.9 mmHg, HCO3 was 9.9 mmol/L with a base excess of −17.3 mEq/L and lactic acid was 3.8 mEq/L. Hepatic function revealed an ALT of 66 U/dL and the remainder was unremarkable. Rapid COVID-19 testing was positive. Urinalysis revealed >1000 mg/dL of glucose, >160 mg/dL ketones and 30 mg/dL of protein. His chest x-ray did not show any infiltrates or other abnormalities.

While in the ED he was treated with 2 L of normal saline and an insulin drip was started at 0.1unit/kg of ideal body weight/hour. The patient was admitted to the medical intensive care unit. Further lab testing revealed an elevated Ferritin 1763 ng/mL, his d-dimer was normal at 410 ng/mL, C-reactive protein 3.6 mg/dL, Lactate dehydrogenase was 228 U/L. While admitted, the patient's acidosis resolved and he was transitioned to subcutaneous insulin and a diabetic diet. He was discharged to home on hospital day 5.

There is a paucity of data on diabetic ketoacidosis (DKA) and Covid-19 infection. We report a case of DKA precipitated by Covid-19 in a patient with newly diagnosed diabetes mellitus. There has been one prior case report of DKA and new onset diabetes mellitus in the setting of COVID-19 infection [3]. DKA occurs as a result of insulin deficiency, increased counterregulatory response which results in the production of ketones. The angiotensin-converting enzyme 2 (ACE2) is a key enzyme in the renin-angiotensin-aldosterone system and it catalyzed the conversion of angiotensin II to angiotensin [4]. ACE2 is found in the lungs, pancreas and serves as the entry point for COVID-19 [4]. Once endocytosis of the virus complex occurs, ACE2 expression is downregulated [5]. This allows for entry of COVID-19 into pancreatic islet cells which may cause beta cell injury [6]. The downregulation of ACE2 can also lead to unopposed angiotensin II, which may impede insulin secretion [7]. These factors may have played a role in precipitating DKA in this patient. As emergency physicians continue to treat patients with COVID-19 infection, it is important to understand the implications this disease can have on organ systems. Further studies and reports will help to delineate the exact pathophysiology. Patients with elevated blood sugar and no history of diabetes should be evaluated for the possibility of new onset diabetes mellitus and DKA, especially in the setting of concomitant COVID-19 infection.

Prior presentations

None.

Author contribution statement

AIH and GDG contributed to the medical management of the patient in the emergency department. ELS drafted the manuscript, and all

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Declaration of competing interest

None.

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