Case Report

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Severe diabetic ketoacidosis and coronavirus disease 2019 (COVID-19) infection in a teenage patient with newly diagnosed diabetes

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Abstract

Objectives: Recently, World Health Organization has declared coronavirus disease 2019 (COVID-19) infection a pandemic. Patients with diabetes may be at an increased risk of developing COVID-19 infection, as well as increased risk of morbidity and mortality. Although the current data have shown that the coronavirus infection generally has a milder course in children.

Case presentation: In this case report, we present a teenage patient with severe diabetic ketoacidosis (DKA) as the first manifestation of his diabetes and COVID-19 infection.

Conclusions: He was treated for DKA and COVID-19 infection, and fortunately, had a good response to the treatment.

Keywords: COVID-19; diabetic ketoacidosis; newly diagnosed diabetes.

Background

The novel coronavirus disease 2019 (COVID-19) infection is a public health emergency of international concern. In

March 2020, the World Health Organization announced that COVID-19 infection is a pandemic [1].

Different clinical pictures of COVID-19 range from self-limiting respiratory tract illness to severe progressive pneumonia, multiorgan involvement, and death [2].

Recent research indicated that the case-fatality rate (CFR) was higher among patients with COVID-19 with preexisting comorbid conditions, including cardiovascular disease (CFR, 10.5%), diabetes (7.3%), chronic respiratory disease (6.3%), hypertension (6%), and cancer (5.6%) [3]. Also, the CFR was shown to be higher among older patients [4]. However, most of the confirmed cases of patients with COVID-19 infection are adults; it seems that the incidence rate among children is the same as adults, but symptoms are less developed or milder than adults; therefore the rate of diagnosis may be underestimated [5].

In this paper, we report a 16-year-old boy presented with severe diabetic ketoacidosis (DKA) as the first manifestation of his diabetes. Diagnosis of COVID-19 infection was made after admission as a possible precipitating factor of DKA [6].

Case presentation

A 16-year-old teenage boy presented to the emergency room of our hospital with a seven-day history of fatigue, weakness, nausea, polyuria, polydipsia, and abdominal pain. He complained of a 2-kg weight loss in the recent two weeks. He was a student staying at home since the last month due to the epidemic of coronavirus. He denied the use of any drugs, opioids, cigarette smoking, or alcohol abuse. He had no known drug allergy, and he had no history of diabetes; however, there was a family history of type 2 diabetes in his grandmother and uncle. On physical examination, he displayed mild dyspnea and productive cough.

Upon admission, his temperature was 38.2 °C, with a respiratory rate of 24 deep breaths per minute, heart rate of

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97 pulses per minute, blood pressure of 121/80 mmHg, and oxygen saturation of 94% on room air. He had no respiratory distress; his mucous membranes were dry, and he was drowsy. Lungs were bilaterally clear to auscultation. The cardiac exam was normal. He displayed mild abdominal tenderness and the guarding without a rebound. Musculoskeletal and neurologic examinations were normal. His weight was 58 Kg, and his height was 181 cm (body mass index [BMI] = 17.7 kg/m²).

The patient's laboratory findings confirmed DKA—blood glucose 512 mg/dL, 3+ urine ketone, 3+ glucose in the urine, venous blood gas: pH = 6.9, and bicarbonate = 8 meq/L.

Complete blood cell count showed white blood cells (WBCs) = 21,700, hemoglobin (Hb) = 18.6, hematocrit = 53%, platelet = 3,27,000, lymphocyte = 12%, and polymorphonuclear neutrophils (PMNs) = 85%. Liver function test was normal. Creatinine: 2 mg/dL, urea: 57 mg/dL, K: 5.6 meq/L, Na: 144 meq/L, uric acid: 8.2 mg/dL, amylase: 42 U/L lipase: 12 U/L, calcium: 8.9 mg/dL, phosphorus: 3.7 mg/dL, magnesium: 2.5 mg/dL, C reactive protein: 44 mg/L, erythrocyte sedimentation rate: 6 mm/h, Hb A1c: 12.9%, c-peptide: 0.25 ng/mL (Table 1).

Chest computed tomography (CT) scan was normal.

The patient got admitted to the intensive care unit, and hydration started with isotonic saline, which switched later to half saline. Potassium chloride was added when the potassium level dropped below 5 meq/L. After the initial hydration, he received six units of regular insulin bolus. The treatment continued with an infusion rate of 6 units/h. Also, an empiric broad-spectrum antibiotic was prescribed. After two days, when his blood pH was 7.38 and bicarbonate was 26 meq/L while he tolerated oral intake, his treatment switched to a subcutaneous insulin regimen. The blood and urine cultures were negative.

The sample which was sent for polymerase chain reaction of COVID-19 came back positive on the third day, and the patient was isolated. Hydroxychloroquine and Kaletra (lopinavir/ritonavir) as the protocol of our hospital for COVID-19 at that time were prescribed.

Final laboratory tests showed creatinine: 1.2, urea: 30, blood culture and urine culture was negative, fetal bovine serum: 112, complete blood count: WBC = 10,400, Hb = 14.6, lymphocyte = 15.8%, PMN = 81%, and uric acid = 3.5.

The patient was discharged with a basal-bolus insulin regimen when he had no fever and dyspnea.

Discussion

Here, we report a 16-year-old boy presented with severe DKA and COVID-19 infection as the first manifestation of his diabetes. He was managed for DKA and received treatment for COVID-19. He was discharged with a good condition after 10 days of admission.

Patients with diabetes may be at a higher risk of developing COVID-19 infection with an increased risk of morbidity and mortality [7], and these patients may require special care. Most of the patients with diabetes suffering from COVID-19 are patients with type 2 diabetes. They usually have comorbidities such as hypertension and cardiovascular diseases which make patients more vulnerable against COVID-19 infection.

There are no reliable data suggesting that children with well-managed type-1 diabetes mellitus be at an increased risk of COVID-19 infection. Also, the coronavirus illness generally has a milder course in children [8]; however, a poorly controlled diabetes mellitus can weaken the immune system and thereby increase the risk of infection.

Li et al. [6] have reported a higher incidence of ketosis among patients with COVID-19 infection in both patients with or without diabetes mellitus. Also, they reported three adult COVID-19 patients with DKA. Viral infections, such as COVID-19, can cause serious complications in people with

Table 1: The laboratory values at admission.

Parameter	Value	Reference values
Blood glucose	512	70–100 mg/dL
Blood pH	6.95	7.35–7.45
Bicarbonate	8	22–26 meq/L
Urine ketone	3+	Negative
Urea	57	15–50 mg/dL
Creatinine	2	0.7–1.4 mg/dL
Potassium	5.6	3.5–5 meq/L
Sodium	144	135–145 meq/L
Uric acid	8.2	3.4–7 mg/dL
Amylase	42	<100 U/L
Lipase	12	23–85 U/L
Calcium	8.9	8.6–10.2 mg/dL
Phosphorus	3.7	2.5–5 mg/dL
Magnesium	2.5	1.6–2.6 mg/dL
CRP	44	<10 mg/L
ESR	6	0–15 mm/h
Hb A1c	12.9	4–5.7%
C-peptide	0.25	0.78–1.89 ng/mL
Cell blood count		
WBC	21,700	4,000–10,000/mm ³
Neutrophil (85%)	1,8445	(55–70%) 2,500–8,000/mm ³
Lymphocyte (12%)	2625	(20–40%) 1,000–4,000/mm ³
Platelet	32,7000	14,5000–45,0000/mm ³

CRP, C reactive protein; ESR, erythrocyte sedimentation rate; WBC, white blood cell count; Hb, hemoglobin.

diabetes, such as DKA. During periods of illness, blood sugar levels may rise. DKA occurs when there is not enough insulin available to deal with this elevation. Fats breakdown for energy production and ketones increase in the blood resulting in acidosis which can quickly cause serious health problems. Breakdown of glycogen stores, hydrolysis of triglycerides from adipose tissues, and mobilization of amino acids from muscles result in the production of glucose and ketone bodies by the liver and the development of metabolic acidosis [9]. Our patient was a new case of diabetes presented with severe DKA with venous blood gas pH of 6.9 and bicarbonate of 8. Also, there was an acute kidney injury due to prerenal azotemia. In the first visit, the patient had leukocytosis with dominant neutrophils in cell blood count, and lymphocyte counts did not decrease. Neutrophil-to-lymphocyte ratio (NLR) declined from 7 to 0.95 in six days. The NLR combines both changes in neutrophils and lymphocytes in physiologic stress. Endogenous cortisol and catecholamines may be major causes of leukocytosis and lymphopenia. Cytokines and inflammatory markers may also be involved [10, 11].

Our current knowledge of COVID-19 in patients with diabetes is limited. Our patient showed a good response to treatment despite the severity of ketoacidosis.

What is new?

This is the first report of COVID-19 infection in newly diagnosed diabetes in a teenage patient presented with severe DKA.

Learning points

- Novel coronavirus infection (COVID-19) is a public health emergency.
- Patients with diabetes may be at increased risk of developing COVID-19 infection.
- Viral infections such as COVID-19 can cause severe acute complications in people with diabetes such as DKA.

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