



Metabolic Acidosis due to Ketoacidosis in an Euglycemic Diabetic Patient with COVID-19 Infection: A Case Report

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Abstract

Diabetes is the one of the most frequently detected comorbidities in patients infected with COVID-19. A few case reports also were published that COVID-19 may cause acute hyperglycemic crises (DKA/HHS) in patients with inadequately controlled diabetes, as well as newly diagnosed diabetes. We report a metabolic acidosis due to ketoacidosis in an euglycemic diabetic patient with COVID-19 pneumonia. COVID-19 infection may cause fat breakdown and induce ketosis, ketoacidosis may develop at the end of the process.

Keywords: COVID-19; Diabetes mellitus; Ketoacidosis

Introduction

The coronavirus SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2), which causes the COVID-19 disease, has infected over 9.5 million people and has resulted more than 480,000 deaths on the world, since June 2020 [1]. Although SARS-CoV-2 is known to be responsible for primary pulmonary disease, including pneumonia and Acute Respiratory Distress Syndrome (ARDS), many extrapulmonary manifestations of COVID-19 have been detected by clinicians [2]. We report a metabolic acidosis due to ketoacidosis in an euglycemic diabetic patient with COVID-19 pneumonia.

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Case Presentation

A 55-year-old woman applied to emergency unit with fever and cough. She had a history of type 2 diabetes mellitus, obesity and hypertension. Her blood glucose was regulated with oral anti-diabetic drugs at home. At the time of hospital admission her blood glucose was: 248 mg/dL, creatinine: 0.62 mg/dL, CRP: 127.62 mg/L, leukocyte: 4630 (lymphocyte: 730). Her D-dimer: and procalcitonin results were detected 305 ng/mL and 0.14 respectively. Her infection was due to coronavirus 2 (SARSCoV-2) infection that confirmed by computed tomography of chest (Figure 1,2) in connection with a positive Reverse-Transcriptase-Polymerase-Chain-Reaction in line with (RT-PCR) assay. In the absence of another etiology, we concluded she had pneumonia possibly associated with COVID-19 infection, and started on Hydroxychloroquine (HCQ) 400 mg per day and ceftriaxon 1 gr twice daily. Due to higher D-Dimer levels, low molecular weight heparin 4000 unit per day subcutaneously was also added to her treatment regimen. On the sixth day of admission her dyspnea, shortness of breath, repetitive cough with fever were worsened and she was transferred to intensive care unit. Treatment was continued with favipiravir, piperacillin-tazobactam and low molecular weight heparin. On the seventh day her venous blood gas analysis as follows; pCO₂ was 20.2 mmHg; pH was 7.28; HCO₃ was 10.5 mmol/L., serum lactate was 1.9; glucose was 151 mg/dL, creatinine: 0.53 mg/dL, sodium: 135; color: 104 mEq/L and anion gap: 20.5. Her blood glucose monitoring was done and glucose levels were between 150 mg/dL to 200 mg/dL; glucose was negative and ketone was +3 in her urine test. On the ninth day of hospitalization her dyspnea progressed and she had hypoxemia consistent with respiratory failure. Thus, she was intubated her arterial blood gas analysis as follows; pO₂ was 54.3 mmHg, pCO₂ was 48.1 mmHg; pH was 7.22 HCO₃ was 19.4 mmol/L, serum lactat was 1.53; procalcitonin: 2.67. Kidney and liver functions were normal. Immune plasma treatment was also given to the patient. Her respiratory failure was improved during ICU follow up and then she was extubated. The patient was transferred to the service after 25 days hospitalization in ICU. Total 37 days of hospitalization she recovered and discharged from hospital.

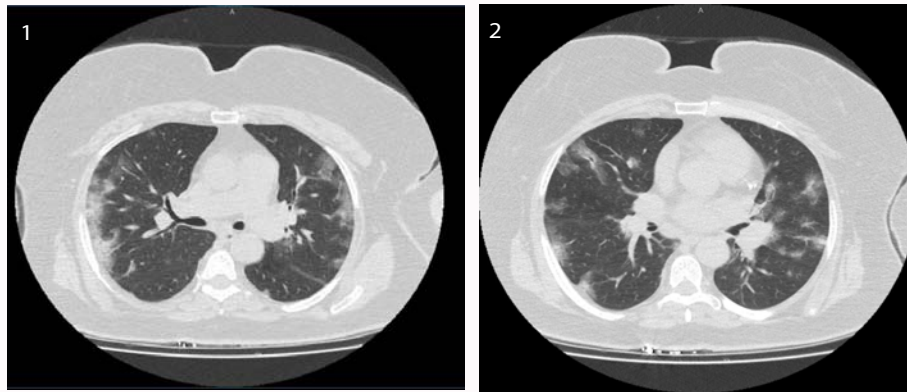


Figure 1,2: Axial images of torax CT at the time of admission.

Discussion

Diabetes is the one of the most frequently detected comorbidities in patients infected with COVID-19. When current data are examined, increased risk of COVID-19 has not been detected in diabetic patients compared to the general population. Nevertheless, diabetes mellitus can increase the risk of severe and critical forms of COVID-19, as a result of this the need for an intensive care unit and/or use of invasive mechanical ventilation might increase, with high mortality rates [3].

The clinical symptoms of COVID-19 infection vary but mainly cause fever and cough. Although mild upper respiratory disease and gastrointestinal disease are mostly observed in COVID-19 infection, severe viral pneumonia with systemic organ failure, or even death might be seen.

Recently some studies have reported that older age or medical comorbidities, such as cardiovascular disease, diabetes mellitus, hypertension, and obesity are risk factors for severe illness and mortality among patients with COVID-19 [4]. A few case reports also were published that COVID-19 may trigger acute hyperglycemic crises (DKA/HHS) in patients with inadequately controlled diabetes, as well as newly diagnosed diabetes [5,6]. In a retrospective study from China, Li et al. [7] reported that; 42 (6.4%) patients admitted with COVID-19 had ketosis out of which 15 (35.7%) had diabetes. Three (20%) out of 15 patients with diabetes had DKA, five patients (26.7%) with diabetic ketosis died, and one of these (25%) presented with acidosis. Two (7.4%) and four (14.3%) of the non-diabetic ketotic patients developed severe acidosis and died, respectively, one of these (25%) presented with acidosis. These findings showed that COVID-19 infection triggered ketosis or ketoacidosis, and caused diabetic ketoacidosis for those with diabetes [7].

Ketones are synthesized from fatty acids in the liver. Ketosis occurs as a result of reduced ketone consumption and this causes increased blood level of ketone bodies [8,9].

Ketoacidosis, a severe metabolic disorder characterized by the accumulation of ketone bodies and acidosis, is mostly seen in people with diabetes and is rarely induced by other pathological conditions [10]. Our patient had diabetes mellitus but she was not hyperglycemic at the time of ketoacidosis.

Worsened hyperglycemia, euglycemic ketosis, and classic diabetic ketoacidosis can be detected in patients hospitalized due to COVID-19 [2]. Accelerated fat breakdown in patients with COVID-19 has also been proposed as a possible mechanism, but this

requires further investigation [7]. Factors not specific to COVID-19 in patients with diabetes and infections include an altered immune response and increase in counter-regulatory hormones that promotes hepatic glucose production, decreased insulin secretion, ketogenesis, and insulin resistance [2,11,12].

Conclusion

Several mechanisms may lead to more severe disease course, including worsened hyperglycemia and ketoacidosis, observed in patients with COVID-19 and diabetes. In conclusion, we observed that COVID-19 infection caused ketoacidosis without hyperglycemia in our diabetic patient. The mechanism of COVID-19 associated ketosis, ketoacidosis or DKA not fully elucidated and needs further research.

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