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CASE REPORT

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Type 1 diabetic manifestations in a young man triggered by COVID-19

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Abstract

COVID-19 could potentially lead to the progression of the course of undiagnosed type 1 diabetes in an infected individuals.

KEYWORDS

COVID-19, hyperglycemia, polydipsia, SARS-CoV-2, type 1 diabetes

1 | INTRODUCTION

We report a critical case of COVID-19 in a young man with overt hyperglycemia. He attended to hospital with weight loss, extreme thirstiness, and large volumes of urine. He had hyperglycemia and proteinuria after COVID-19 and was diagnosed with T1D. COVID-19 could be an infectious trigger for incidence of T1D.

The outbreak and rapidly expanding infectious with coronavirus disease 19 (COVID-19) caused by the SARS-CoV-2 virus has led to a devastating effect on public health and the global economy. There is no clear image of the spectrum of symptoms of COVID-19.[1-3] An emerging area, in which there is little information, is the COVID-19–related diabetic manifestations. Although previous studies showed ketosis or ketoacidosis, and induced diabetic ketoacidosis (DKA) in diabetic patients with COVID-19 infection, but here is no clear image of diabetic manifestations as a presenting symptom for COVID-19 in individuals who had not shown any symptoms of diabetes yet.[2,4] A few studies showed the development of diabetes and severe metabolic complications in patients with COVID-19 infection.[5,6] Recently, a teenage case was reported with severe DKA as the first manifestation of his type 1 diabetes(T1D) and COVID-19 infection.[7] To the best of our knowledge, this is the third report of hyperglycemia as the presenting symptom of COVID-19 in a young man who had not complained of any symptoms of T1D before COVID-19 infection.

Fatemeh Dehghani Firouzabadi and Mohammad Dehghani Firouzabadi contributed equally to this work

2 | CASE PRESENTATION

A 23-year-old man without any past medical history of disease or autoimmune disease (eg, hypothyroidism and adrenal insufficiency) was applied to the hospital with weight loss, feeling of extreme thirstiness, and abnormally passage of large volumes of urine with an increase in urinary frequency for one week. He was a teacher who has lived in Saveh, Iran, and did not use any drugs, opioid, cigarette smoking, and alcohol. He had no history of a drug allergy and no history of diabetes or autoimmune diseases in his families. After initial assessment, we figured out that he had have fever and weakness during one week before admission that was controlled by over the counter use of acetaminophen. Physical examination showed a moderate dehydration (dry mouth, dry and cool skin, and dizziness) with a heart rate of 120 beats/min, temperature of 36.8°C, blood pressure of 100/70 mm Hg, and oxygen saturation of 97% on room air without any respiratory distress, and his BMI was 23 kg/m^2. Lung sounds were normal. No noticeable sign of abnormality in sensory or motor nerve function was seen. Electrocardiogram showed sinus tachycardia at a rate of 115 beats per minute. Laboratory tests showed hyperglycemia (plasma glucose levels were 405 and 352 mg/dL in two successive samples), (3+) glycosuria, and 3+ proteinuria. His 2 hours postprandial glucose level (2 hpp) and HbAlc were 449 mg/dL and 12.2%, respectively, and confirmed the diagnosis of diabetes. Other laboratory tests including complete blood count, blood gases, C-reactive protein, erythrocyte sedimentation rate, liver and kidney function tests, amylase, lipase, calcium, and magnesium were normal (Table 1). Chest computed tomography scan was suspected for COVID-19 infection showing ground glass infiltration in the lateral aspect of right middle lobe (Figure 1). Reverse transcription-polymerase chain reaction (RT-PCR) assay confirmed the diagnosis of COVID-19 infection after 2 days. At admission, patient was given initial bolus of isotonic saline continued by half saline and along with insulin infusion therapy (insulin drip at 0.1 unit/kg of ideal body weight/hour) and his hyperglycemia and tachycardia were improved gradually. Serum electrolytes were closely monitored and treated appropriately. Hyperglycemia resolved the following day, and insulin infusion was transitioned to subcutaneous insulin therapy. After receiving laboratory confirmation for COVID-19, the patient was treated according to the national treatment protocol at the time (hydroxychloroquine 200 mg PO q12h and oseltamivir 75 mg PO q12h, both for five days). He was discharged on a basal/bolus insulin regimen (30 units of insulin Glargine daily and 8 units of insulin Aspart three times daily). He was retested with PCR assay for COVID-19, and the result was normal 2 weeks after discharge. Glycemic control was good and the blood glucose levels were nearly controlled with basal/bolus insulin regimen when he was visited within three days, two and 4 months after hospital discharge.

TABLE 1 Laboratory tests on admission

Laboratory tests	Value	Unit	Reference range
Blood glucose	405 and 352	mg/dL	70-120
FBS	352	mg/dL	Less than 100
2hpp	449	mg/dL	Less than 140
HbA1c	12.2 and 12	%	4.4-5.7
Cell blood count			
WBC	4600	mm^2	4000-10 000
Neutrophil	76%		55%-70%
Lymphocyte	19%		20%-40%
Platelet	124 000	mm^2	140 000-400 000
ESR	17	mm/hr	1-20
CRP	Less than 6		Less than 6
Blood gases			
PH	7300		7.350-7450
PCO2	46	mm Hg	35-45
PO2	31	mm Hg	30-40
HCO3	22.6	mmol/L	22-26
Anion gap	13	mEq/L	8-16
Urea	35	mg/dL	10-50
Creatinine	1.2	mg/dL	0.8-1.4
Sodium	137	mmol/L	135-148
Potassium	3.8	mmol/L	3.5-5.3
Triglycerides	81	mg/dL	Up to 150
HDL-c	31	mg/dL	35-85
LDL-c	94	mg/dL	Less than 130
Total cholesterol	141	mg/dL	Up to 220
PT	13.1	sec	12-14
INR	1.09		1-1.17
PTT	31	sec	27-45
AST	20	U/L	Up to 35
ALT	19	U/L	Up to 45
ALK-P	310	U/L	98-279
Amylase	53	U/L	0-100
Total Bilirubin	0.7	mg/dL	Less than 2
Direct Bilirubin	0.2	mg/dL	Less than 0.4

Abbreviations: 2 hpp, 2 h postprandial; ALKP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; FBS, fasting blood sugar; HbA1c, hemoglobin A1c; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; PT, prothrombin time INR, International normalized ratio; PTT, partial thromboplastin time; WBC, white blood cells.

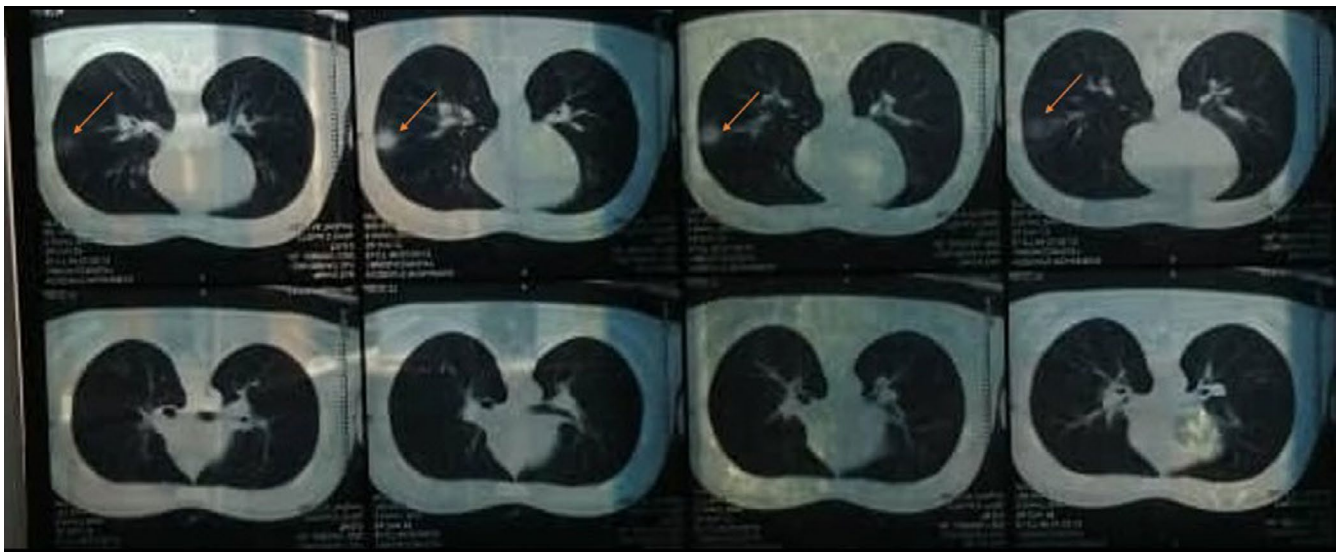

FIGURE 1 Chest CT Scan of patient during admission. Single ground glass infiltration in the lateral aspect of right middle lobe in COVID-19(arrows)

3 | DISCUSSION

In this case report, we presented a young man with no past medical history who attended to the hospital with weight loss and complaining of extreme thirstiness and passage of large volumes of urine after infection with COVID-19 that was diagnosed with T1D. Although the question remains open about COVID-19 and increases risk of the incidence of TID, this case report is a unique initial presentation in a young man with COVID-19 infection who had not complained of any symptoms of T1D before COVID-19.

According to the staging classification of type 1 diabetes by ADA,[8] in the stage 1 individuals are normoglycemic but they have two or more T1D–associated islet autoantibodies. At the stage 2, individuals represent with two or more islet autoantibodies along with the development of glucose intolerance or dysglycemia due to the loss of functional beta-cell mass resulting in abnormal blood glucose tests. The 5-year and lifetime risk of symptomatic disease at stage 2 is approximately 75% and 100%, respectively. Stage 3 includes the presentation of the typical clinical symptoms of T1D, such as polyuria, polydipsia, weight loss, fatigue, DKA, and others.

Our case was in the stage 2 T1D due to HbA1c more than 12% that was checked two times during admission. In stage 2, patients are presymptomatic, and any trigger, such as viruses, host-microbiome, antibiotics, and food or diet, can accelerate the progression of their diabetes to stage 3 defined as the onset of symptomatic T1D.[9] Possible explanation for the diabetogenic effect of COVID-19 and progression of the course of diabetes could be due to the entrance of the virus to the organs and tissues such as pancreatic beta cells and adipose tissue via binding angiotensin-converting enzyme 2 (ACE2) receptors, which are expressed in these organs.[10] The acceleration of destruction of pancreatic beta cells leads to the overt T1D. Another possible explanation may be due to increase immune response to the viral antigen and aggregate autoantibodies generation leading to the autoimmune insulitis and pancreatic beta-cell destruction similar to other viral infections.[11] Recently, a case with acute necrotizing pancreatitis following COVID-19 was shown that COVID-19 may be induced pancreatitis.[12] Therefore, COVID-19 infection could lead to the faster progression of T1D in subjects who are genetically high risk for autoimmune diseases via activation of immune response and cytokine release.

This patient emphasizes that clinicians should be vigilant to the possibility of COVID-19 infection as an infectious trigger for hyperglycemia and the incidence of T1D, even if a patient has subtle symptoms or asymptomatic.

Previous studies have shown hyperglycemia and insulin resistance as manifestations of SARS-Cov-1 and COVID-19.[7,13,14] However, SARS-Cov-1 induced transient hyperglycemia that was relieved after hospital discharge.[14] Here, we are reporting an acceleration in the course of hidden T1D to the overt stage, following COVID-19 infection that needs continuing basal/bolus insulin regimen after 4 months follow-up.

Overall, hyperglycemia and diabetic symptoms may be a poor prognostic factor for COVID-19, which is difficult to detect in early stages and requires swift attention. Hyperglycemia, if left untreated, could cause deterioration of patients' general and metabolic condition. Therefore, further global awareness of this clinical scenarios and longer

follow-ups are needed to determine whether COVID-19 infection can cause or accelerates the destructive process of the pancreatic beta cells leading to rapid progression of subclinical stages to overt type 1 diabetes.

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CONFLICT OF INTEREST

The authors declare that they have no competing interest.

AUTHOR CONTRIBUTIONS

Fatemeh Dehghani Firouzabadi, Mohammad Dehghani Firouzabadi, Alireza Esteghamati: involved in conception and design of work and critical revision of the article. Fatemeh Dehghani Firouzabadi, Mohammad Dehghani Firouzabadi, Fatemeh Moosaie, Saeedeh Rafiee, Alireza Esteghamati: involved in drafting the article. All the authors approved the final version and have the agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

ETHICAL APPROVAL

Informed consent was obtained from the patient before the study.

DATA AVAILABILITY STATEMENT

All data generated or analyzed during this study are included in this submitted article.

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