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LETTER TO THE EDITOR

SARS-CoV-2 infection-related diabetes mellitus

1 | INTRODUCTION

We read with interest the commentary entitled "Diabetes and COVID-19" from the *Journal of diabetes*, discussing the relationship between Middle East respiratory syndrome coronavirus (MERS-CoV) infection in 2012 and diabetes mellitus (DM).[1]

Recent reviews have raised the possibility of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)-related DM by suggesting the hypothesis that SARS-CoV-2 infection may have a predilection for beta cells of the pancreas and possibly cause insulin deficiency and DM.[2] Similarly, studies from the SARS-CoV outbreak in 2003 highlighted the relationship between SARS-CoV and angiotensin-converting enzyme 2) receptors in different organs, including the pancreatic islet cells, destroying the insulin-producing cells, and resulting in acute DM.[3]

We describe a patient who developed hyperglycemia and DM after documented exposure to the SARS-CoV-2.

2 | CASE REPORT

Informed consent for this case report was provided by the mother of the patient.

A previously healthy 13-year-old female of Mexican descent was found to be hyperglycemic on routine blood work at her annual visit. The patient endorsed polyuria and polydipsia for 3 weeks, and weight loss for 2 to 3 weeks. Three months prior to the patient's presentation, her mother became ill and tested positive for SARS-CoV-2 by polymerase chain reaction (PCR) analysis. The patient had no SARS-CoV-2-associated symptoms.

The patient's vital signs and physical examination were normal. She had no acanthosis nigricans, and her BMI was at the 78th percentile for age and sex. She was fully pubertal (Tanner stage V), and her family history was noncontributory.

Initial blood glucose was 729 mg/dL, potassium of 4.4 mmol/L, sodium of 133 mEq/L, bicarbonate of 20.6 mEq/L, pH 7.45, anion gap of 14 mEq/L, and she had large ketones in the urine. Her concomitant C-peptide level of 1.0 ng/mL was low in the setting of hyperglycemia. Her glycosylated hemoglobin (HbA1c) was 14.3%.

TABLE 1 Laboratory results

	At diagnosis	At 5-week follow-up
Insulin requirement (total daily dose, μ/kg/d)	0.7	0.5
HbA1c (%) (n = 4.8-5.6)	14.3	9.3
Serum glucose level (mg/dL)	729	
C-peptide of insulin (0.9-7.1 ng/mL)	1.0	
Insulin Ab (uU/mL)	<5.0	
Zinc transporter 8 Ab (U/mL)	<15	
IA-2 autoantibodies (U/mL)	<7.5	
GAD65 (0.0-5.0 U/mL)	<5.0	
Antihuman tTG-IgG ELISA (0.0-20.0 U)	4.4	
TPO (microsomal) Ab (0.0-5.6 IU/mL)	<0.5	
Thyroglobulin Ab (0.0-4.1 U/mL)	1.1	

Abbreviations: Ab, antibody; ELISA, enzyme-linked immunosorbent assay kit; GAD65, glutamic acid decarboxylase 65; HbA1c, glycosylated hemoglobin; IA-2 autoantibodies, insulinoma-associated-2 autoantibodies; TPO, thyroperoxidase; tTG-IgG, tissue transglutaminase immunoglobulin G.

Diabetes-related autoantibodies, celiac, and thyroid disease antibodies were negative (Table 1). Her SARS-CoV-2 antibodies were positive with a negative PCR.

The patient was treated with a basal-bolus regimen of subcutaneous insulin with a maximal total daily dose of 0.7 μ/kg/d. Five weeks after her diagnosis, her insulin requirement and HbA1c were both lower. (Table 1).

3 | DISCUSSION

This patient was found to be significantly hyperglycemic with an elevated HbA1c and ketonuria. Her symptoms of hyperglycemia started shortly after her exposure to the SARS-CoV-2. She had no features consistent with type 2 DM. While type 1 DM (T1DM) without evidence of humoral islet autoimmunity (type 1B) and monogenic DM could not be fully excluded, we postulate that the patient developed SARS-CoV-2-associated DM.

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Our patient has no serological evidence of DM-related autoimmunity, thus being different from reports of new-onset T1DM with confirmed pancreatic autoimmunity presenting during the SARS-CoV-2 pandemic.[4]

Similar to the case described by Hollstein et al,[5] we postulate that the viral infection is implicated in the development of her diabetes because of documented exposure to SARS-CoV-2 and the presence of antibodies against the virus. While we share the lack of direct evidence of causation, we postulate that more patients with similar presentations will be reported during the current pandemic.

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KEYWORDS
COVID-19, diabetes mellitus, transient diabetes mellitus

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