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Case Report

Idiopathic Pancreatitis, Newly Diagnosed Diabetes Mellitus I, and Hypothyroidism in a Young Patient with Recent COVID-19 Infection

Amin Elfituri*, Firas Warda, Leena Shahla, Faisal Bukeirat

College of Medicine, University of Florida, Jacksonville, Florida, United States

ABSTRACT

We present a young patient who came into the emergency department initially with abdominal pain, diagnosed with pancreatitis, but was found to have diabetic ketoacidosis, and later was diagnosed with hypothyroidism. We suspected the patient's diagnoses to be related to autoimmunity that was induced by his recent COVID-19 infection. Our case examines the relationship between COVID-19 and autoimmunity with special attention to the pancreas.

Keywords: Autoimmunity; Molecular mimicry; Coronavirus; Pancreatitis; Diabetes; Hypothyroidism

INTRODUCTION

Coronavirus disease 2019, caused by severe acute respiratory syndrome coronavirus 2 (SARS-COV-2), can vary in presentation depending on individual patients. Most patients who contract the disease are symptomatic carriers; however, a minority of patients may develop life-threatening multi-organ damage. Autoimmune modulators, commonly used in autoimmune disease, have been used to control the progression of coronavirus disease. Multiple reports have linked coronavirus to autoimmune disease in patients who survived the infection. Autoimmune conditions such as Guillain-Barre Syndrome and Systemic Lupus Erythematosus [1,2] have been reported following coronavirus infection. Direct involvement of the virus has also been reported in cases of acute thyroiditis [3,4]. Pancreatic involvement in autoimmunity and the development of autoimmune forms of diabetes mellitus are poorly studied.

CASE PRESENTATION

The patient is a 22-year-old male who presented to the emergency department with nausea, vomiting, and abdominal pain. He denied any history of diabetes mellitus, hypercholesterolemia, hypertension, thyroid disease, alcohol use or cholelithiasis, however, he reported a recent COVID-19 pneumonia, for which he required treatment with prednisone and azithromycin. He was never hospitalized or required supplemental oxygen during his infection period. Pertinent history included unintentional 20-pounds weight loss, polyuria, and polydipsia, for the 3 months prior to presentation. Vital signs did not show any abnormalities. Oxygen saturation was 99% on room air. Physical exam was significant for moderate epigastric tenderness on light palpation. Labs were consistent with diabetic ketoacidosis, acute kidney injury, and acute pancreatitis (please see Table 1). Triglyceride (TG) level was 387 mg/dL, and glycosylated hemoglobin was 10.7%. Patient was transferred to intensive care units and started on insulin drip and aggressive Intravenous (IV) hydration. His condition improved and abdominal pain level had decreased by the second day of admission. He transitioned to subcutaneous Multiple Daily Injection (MDI) of insulin regimen and started on an oral diet. As part of the investigation, Thyroid Stimulating Hormone (TSH) was checked and found to be 11.7 mUnits/L. Free T4 and free T3, were found to be low at 0.4 ng/dL and 0.8 pg/mL, respectively. Patient was discharged on a weight-based dose of levothyroxine and MDI insulin regimen. On further evaluation by the endocrinology team in the outpatient setting, C-peptide level was found to be 0.3 ng/mL when blood sugar was 134 mg/dL. Glutamic acid decarboxylase and thyroid peroxidase antibodies were positive, confirming the diagnosis of type I diabetes mellitus and autoimmune thyroiditis, respectively. On repeat screening, the patient's TSH was normal on T4 replacement and glycosylated hemoglobin percentage had fallen to 7.1%. TG level also decreased to 167 mg/dL. Patient is currently doing well and is being evaluated for insulin pump therapy.

Correspondence to: Dr. Amin Elfituri, College of Medicine, University of Florida, Jacksonville, Florida, United States, Tel/Fax: 3049728774; E-mail: amin.elfituri@jax.ufl.edu

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Lab	Patient's value	Normal value
Sodium	133 mEq/L	135-145 mEq/L
Glucose	300 mg/dL	90-125 mg/dL
Carbon dioxide	5 mmol/L	23-30 mmol/L
Anion gap	18 mmol/L	10-12 mmol/L
Creatinine	1.79 mg/dL	0.74-1.35 mg/dL (adult men)
Glomerular filtration rate	48 mL/min/1.73 m^2	>59 mL/min/1.73 m^2
Lipase	2,322 units/L	0-160 units/L

Table 1: Pertinent labs

DISCUSSION

Coronavirus disease affects the respiratory tract mainly, but gastrointestinal involvement has been reported in the form of viral gastroenteritis and acute pancreatitis. The mechanism is thought to be initiated by Angiotensin Converting Enzyme-2 receptor (ACE-2) which aids in viral entry into the host cell [5-8]. These receptors are highly expressed in both exocrine glands and islet cells of the pancreas. Therefore, pancreas could be a potential target, but the exact process remains unknown. In our case, the patient's pancreatitis and thyroiditis could have been due to direct cytopathic effects of local SARS-COV-2 replication, or indirectly by toxic systemic immune response. In one case series of 52 patients infected with COVID-19, serum lipase was found elevated mildly in 17% of patients without any symptoms. Another case series of 67 patients diagnosed with COVID-19 pneumonia reported a 17% incidence rate of pancreatitis, although only 7% of patients had significant pancreatic damage on imaging. Another case report presented a patient who was diagnosed with acute pancreatitis based on clinical and imaging findings after being treated for COVID-19 pneumonia, five days after being discharged from the hospital. Therefore, pancreatitis should be high on the differential diagnosis in a patient with active or recent COVID-19 viral infection. Regarding thyroid disease in patients with COVID-19 infection, direct damage of the virus to thyroid tissue causing subacute thyroiditis may actually present with hyperthyroid symptoms at first given the release of thyroid hormone from the destroyed gland. As the disease process progresses, hypothyroidism may persist, especially in those with avidity to autoimmune thyroid disease, like our patient since he had positive TPO antibodies [9-12]. Due to similar mechanisms, type I diabetes mellitus may arise in patients who have the antibodies that predispose them to developing it, especially after an inciting event such as a viral infection. In some rare cases, the virus may infect the islet cells directly and destroy them permanently. Coxsackie virus and enterovirus have been well studied in their connection to developing type I diabetes mellitus at a young age, due to the phenomenon of molecular mimicry [13,14]. There is a similarity in molecular make-up and characteristics between some viral proteins and cellular antigens. Therefore, the immune system is unable to distinguish between viral antigens and cellular proteins, leading to increase in autoimmunity. This phenomenon is poorly studied in SARS-COV-2 but it would be of no surprise if such phenomenon is present in infected patients. It would be beneficial to monitor for autoimmune processes in patients who survived coronavirus disease as such processes can increase their morbidity and mortality rate if left unrecognized and untreated.

CONCLUSION

We present a patient who was hospitalized for the management of acute pancreatitis and diabetic ketoacidosis, and was later found to have hypothyroidism. Six months prior to his presentation, he was treated for COVID-19 infection but never required hospitalization. It was suspected that his viral infection could have been linked to the development of all three conditions. Indeed, having the autoimmune antibodies has made him more prone. The mechanism through which COVID-19 infection can predispose patients to developing autoimmune disorders remains an area of interest in future studies.

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