COVID-19 is associated with high blood glucose levels: Diabetic neuropathy during the SARS-CoV-2 pandemic

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Case Report

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Abstract

**Background:** The nerves in the legs and feet are most frequently damaged by diabetic neuropathy. The COVID-19 infection is associated with a high risk of neuropathy symptoms.

**Case Presentation:** On July 12, 2022, a 58-year-old black female retiree with significant symptoms of numbness and muscle weakness in the hands and legs was brought into the emergency room. 17 years prior, she received a type 2 diabetes mellitus diagnosis. Metformin 1.5 g twice a day and glibenclamide 10 mg twice a day were part of her therapy regimen. When she was admitted to the emergency room, she described a one-day history of shortness of breath, frequent urination, excessive thirst, hyperglycemia, excessive appetite, fever, headache, and dehydration. A chest X-ray showed bilateral diffuse, patchy airspace opacities that could be caused by multifocal pneumonia or viral pneumonia. She started receiving 1000 mL of fluid resuscitation (0.9% normal saline) as soon as she was moved to the critical care unit, along with a drip-in insulin infusion.

**Conclusion:** Diabetes, infections like COVID-19, poor vitamin levels, and other factors can all contribute to diabetic neuropathies. According to the Centers for Disease Control and Prevention, patients with type 2 diabetes mellitus are much more likely to experience severe morbidity and death from coronavirus disease-19. Symptoms of diabetic neuropathy continued for months after a COVID-19 infection test resulted in a positive result.

**Introduction**

Patients with type 2 diabetes mellitus frequently experience this bothersome condition called diabetic peripheral neuropathy, which raises the risk of developing diabetic foot ulcers and lower limb amputation [1]. The clinical spectrum of COVID-19 encompasses issues affecting multiple organs and varies from asymptomatic to deadly [2]. Rather than the emergence of new cases of diabetes mellitus, COVID-19 illnesses generate metabolic alterations that lead to diabetes mellitus [3]. Diabetes mellitus raises mortality and has a negative impact on COVID-19 outcomes [4]. Adults with type 2 diabetes are more likely to develop life-threatening conditions or pass away from COVID-19 infections [5]. This case study examines the retired woman's type 2 diabetes, which the COVID-19 infection had aggravated, as well as the connection between diabetic neuropathy and the SARS-CoV-2 virus.

**Case Presentation**

On July 12, 2022, a 58-year-old black female retiree with significant symptoms of numbness and muscle weakness in the hands and legs was brought into the emergency room. One day prior, she had been experiencing stomach pain, vomiting, and increasing exhaustion. Her family members have a long history of type 2 diabetes mellitus, including her grandmother and father. She had never used chemotherapeutic medications, been exposed to pollutants, or struggled with alcoholism. She has a background involving medical and pharmaceutical problems. 17 years prior, she received a type 2 diabetes mellitus diagnosis.
Metformin 1.5 g twice a day and glibenclamide 10 mg twice a day were part of her therapy regimen. She had consistently taken her medications until her admission date. She made it a habit of traveling outside of the city to visit her younger sister, who was ill. Her sister gave consent for a COVID-19 test to be positive while she was in the hospital. She might have come into contact with COVID-19 two days before admission. When she was admitted to the emergency room, she described a one-day history of shortness of breath, fever, pain in the hands and legs, headache, tingling in the hands and legs, excessive sensitivity to touch, and dehydration. Three days prior to being hospitalized, she was in good health. At the time of her arrival at the emergency room, she had the following vital signs: a body temperature of 39.1°C, a weight of 70.4 kg, a height of 1.60 m, a body mass index (BMI) of 27.5 kg/m2, a blood pressure of 127/89 mmHg, a respiratory rate of 19 cycles per minute, a peripheral pulse rate of 101 beats per minute, and an oxygen saturation level of 86% on room air.

Her blood chemistry done upon her admission to the intensive care unit showed blood urea nitrogen of 36 mg/dl, fasting blood glucose of 229 mg/dL, 2-hour postprandial blood glucose of 232 mg/dL, serum creatinine of 2.2 mg/dl, serum sodium of 117 mEq/L, serum potassium of 3.7 mEq/L, hemoglobin of 14.8 g/dL, leukocytes of 4,310/µL, platelets of 139,800/µL, neutrophils of 65%, pH arterial blood of 7.06, anion gap level of 19 mEq/L, partial pressure of carbon dioxide of 30 mmHg, serum bicarbonate level of 9.6 mEq/L, serum phosphate level of 2.7 mg/dL, white blood cell count of 18720 cells/mm3, serum chlorine level of 91, an aspartate aminotransferase level of 83 units/L, alanine aminotransferase level of 89 units/L, an erythrocyte sedimentation rate of 12mm/hour, 46% hematocrit, lymphocytes 25%, and urine analysis was positive for urine ketones of 3+.

A chest X-ray showed bilateral diffuse, patchy airspace opacities that could be caused by multifocal pneumonia or viral pneumonia. Wheeze, as well as crepitations in the right infrascapular region and bilateral airspace consolidations, which were more pronounced on the left side and involved nearly all zones, were all audible when the chest was auscultated. Her electrocardiogram (ECG) showed sinus tachycardia at 101 beats per minute and ST depression in the anterior-lateral leads. There were no abnormalities or abscesses found during the magnetic resonance imaging. No evidence of compressed nerves was found during the computed tomography scan.

She had never before established an approved COVID-19 infection. The result of the SARS-CoV2 reverse transcription polymerase chain reaction was positive. She spent two days in the emergency room with previously well-controlled type 2 diabetes mellitus, the recently diagnosed COVID-19 infection, and newly developed diabetic neuropathy before being transferred to an intensive care unit.

She started receiving 1000 mL of fluid resuscitation (0.9% normal saline) and drip insulin at the critical care unit. She arrived and started breathing five liters of oxygen. Two hours after each meal, she underwent a fasting and random blood sugar check. A fasting blood glucose level of 100 to 140 mg/dL and a 2-hour postprandial blood glucose level of 140 to 200 mg/L were maintained by adjusting the insulin dose in accordance with her blood sugar levels. Enoxaparin 80 mg subcutaneously was given to her every 12 hours to treat her confirmed COVID-19. On day 10, syringe pump insulin therapy was stopped.
and subcutaneous insulin injections were resumed as soon as her blood sugar started to normalize. She received neutral protamine Hagedorn (NPH) insulin 54/22 after ten days of insulin drip to help with her poor metabolic control. She took 25 mg of amitriptyline once a day, at night. She received 500 mg of acetaminophen as needed to reduce her fever brought on by COVID-19. NPH was stopped after 15 days, metformin 1.5 mg twice daily was restarted, and glibenclamide 10 mg twice daily was resumed. With the aid of subcutaneous insulin and nutritional management, good glycemic control was maintained.

**Patient Perspective**

On July 29, 2022, she was finally allowed to leave the hospital after receiving two consecutive negative results from COVID-19 throat swab tests. She was discharged with her current diabetes medications as well as painkillers for her diabetic neuropathy. It was advised that she visit the ambulatory clinic for a monthly checkup.

**Discussion**

The COVID-19 pandemic caused by the SARS-CoV-2 virus has become a major global catastrophe, raising concerns for the diabetic community in particular [6]. By attaching to its cellular angiotensin-converting enzyme receptors, which are widely distributed in adipose tissue and pancreatic beta cells, SARS-CoV2 induces ketosis-prone diabetes by causing abnormal glucose metabolism and pancreatic beta cell death. This mechanism results in diabetes mellitus in SARS-CoV patients [7].

The most common reason for non-traumatic foot amputation and autonomic failure is diabetic neuropathy, which is brought on by damage to the peripheral and autonomic nerve systems [8]. Some people experienced tingling and numbness from the COVID-19 infection. In patients with diabetes mellitus (DM), severe COVID-19 infection combined with hypoxemia is linked to neuropathic symptoms and pervasive sensory impairment. Serious COVID-19 problems are more likely to occur in diabetics [9].

More than 50% of diabetic individuals experience diabetic neuropathy, and these people are also more likely to have impaired immunological and respiratory systems. It has been demonstrated that diabetic patients with microvascular difficulties are more likely to experience more severe side effects from the COVID-19 infection and may also be more likely to experience the onset or progression of neuropathy [10]. Diabetes-susceptible individuals are particularly susceptible to COVID-19 because diabetes impairs the immune system and reduces its capacity to combat infections [11].

Patients with COVID-19 frequently develop neuropathies of the peripheral nerves, which are typically brought on by immune processes or neurotoxic side effects of medications used to treat COVID-19 symptoms [12]. Peripheral nerve compression brought on by protracted bed rest in an intensive care unit also plays a minor role. Diabetes not only increases the risk of serious COVID-19 consequences, but its common comorbidities can also make the infection worse [13].
Neuropathy caused by COVID-19 will be treated with an emphasis on symptom alleviation and virus recovery [14].

Diabetes mellitus is associated with an increased risk of complications, hospitalization, and mortality in people infected with COVID-19 [15]. The patient in this study was hospitalized in the critical care unit due to a COVID-19 infection and had previously been diagnosed with type 2 diabetes, which lengthened hospital stays. In this investigation, in addition to the inflammation brought on by the COVID-19 infection, type 2 diabetes was associated with low-grade chronic inflammation brought on by increased visceral adipose tissue.

Poor diabetes management has been connected to the COVID-19 infection [16]. Extreme hypercoagulability and uncontrolled responses are more common in diabetes patients with COVID-19, which may worsen the prognosis [17]. COVID-19 likely worsened metabolic issues related to pre-existing diabetes mellitus rather than causing people to develop the disease for the first time.

Patients with type 1 and type 2 diabetes mellitus have been connected to the novel COVID-19 pandemic [18]. Furthermore, diabetes mellitus is linked to a greater death rate in COVID-19 patients [19]. The novel COVID-19 was recently found to be related to the interaction of the coronavirus SARS-CoV-1 and angiotensin-converting enzyme in pancreatic cells, which leads to cell damage and exacerbation of diabetes [20].

The worsening COVID-19 prognosis may be caused by additional confounding factors in people with diabetes mellitus, such as obesity, renal disease, cardiovascular disease, and the use of particular medicines [21]. The patient in this study was previously identified as having class I obesity, putting her at risk for worsening type 2 diabetes mellitus and a COVID-19 infection.

One of the risk factors connected to poorer outcomes and mortality from COVID-19 pneumonia is diabetes mellitus [22]. Ketone production is less common in people with diabetes mellitus who reduce their weight and control their diet [23].

**Conclusion**

Due to a deficiency in the autonomic nervous system and the inflammatory reflex, which results in a pro-inflammatory state in diabetic patients with COVID-19, diabetic neuropathy may be a substantial risk factor for severe COVID-19. Diabetes mellitus raises mortality and has a negative impact on COVID-19 outcomes. Adults with type 2 diabetes are more likely to suffer from life-threatening illnesses and pass away from COVID-19 infections.

**Declarations**

*Consent for publication*
Written informed consent was obtained from the patient for publication of this case report.

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**Competing interests**

The author has no financial or proprietary interest in any of the materials discussed in this article.

**References**


