Case Report

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A confirmed COVID-19 in a patient with newly diagnosed hypertension and preexisting type 2 diabetes mellitus: a case report

Gudisa Bereda, B.Pharm

Background and importance: Coronavirus disease 2019 (COVID-19) aggravates preexisting diabetes mellitus and contributes to newly discovered hypertension by increasing blood pressure by inhibiting the activity of angiotensin-converting enzyme 2 in the rennin–angiotensin system. Diabetes patients may be more vulnerable to COVID-19 due to chronic comorbidities such as obesity and cardiovascular disease such as hypertension.

Case presentation: On 23 March 2022, a retired black African woman in her 60s was taken into the emergency room with the chief complaints of frequent midnight urine, hazy vision, headache, fever, and tingling in her hands and feet. A throat swab PCR test that revealed positive results after 28 h was used to confirm COVID-19. Her electrocardiogram showed sinus tachycardia with a heart rate of 105 beats per minute. Fluid resuscitation (0.9% normal saline) of 1000 ml and drip insulin administration commenced as soon as she was brought to an ICU.

Clinical discussion: In this case report, the patient had been previously diagnosed with type 2 diabetes mellitus. COVID-19 affects the beta cells, forcing them to release insulin and increasing insulin insufficiency, which leads to her blood glucose raising. Type 2 diabetes mellitus is therefore the most frequent comorbidity of COVID-19 in this case report.

Conclusion: Poor blood glucose management in the case of COVID-19 may increase the pathogen's susceptibility, the likelihood that patients will be admitted to the hospital, and the likelihood that mortality will be enhanced.

Keywords: case report, COVID-19, hypertension, PCR, type 2 diabetes mellitus

Introduction

Coronavirus disease 2019 (COVID-19) is extremely infectious and is spread by infected people's respiratory droplets. It affects the lungs after entering the body through the upper respiratory mucous membrane[1]. All ages are susceptible to COVID-19, but the disease has harsher implications for the elderly and those with a history of chronic illness (comorbidity)[2]. Heart disease, hypertension, and diabetes mellitus are the three most common comorbidities linked to COVID-19[3,4]. People with type 2 diabetes mellitus are not more likely to contract severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), but if they do, they are more likely to have a more severe illness, which will lengthen their recovery period[5]. The clinical spectrum of SARS-CoV-2 infection ranges from mild to severe, manifesting as asymptomatic infection, mild upper respiratory tract disease, and severe viral pneumonia with respiratory failure and even death[6]. The patient's risk of acquiring a severe form of COVID-19 may be increased by hypertension[7]. In the COVID-19 pandemic, metformin is regarded as the first-line therapy for type 2 diabetes[8]. Antihypertensive medications known as angiotensin-converting enzyme inhibitors are frequently prescribed to people with type 2 diabetes mellitus[9]. The best form of therapy to achieve glycemic control is subcutaneous insulin administered using a basal or multiple daily injectable schedules [10]. In this study, COVID-19 induces newly diagnosed hypertension via two routes, which advances scientific knowledge. First, COVID-19 affects the beta cells, forcing them to release insulin and raising relative insulin insufficiency. This exaggerates type 2 diabetes mellitus that has already been diagnosed, which in turn causes diabetes to increase peripheral artery resistance and, ultimately, raise blood pressure. Second, COVID-19 aggravates preexisting diabetes mellitus and contributes to newly discovered

HIGHLIGHTS

- Coronavirus disease 2019 (COVID-19) exacerbates diabetes mellitus that already exists.
- Diabetes mellitus is the most frequent comorbidity of COVID-19 infection.
- The majority of COVID-19 hospitalized patients will need insulin.

Department of Pharmacy, Negelle Health Science College, Negelle, Oromia Regional State, Ethiopia

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**Corresponding author. Address: Department of Pharmacy, Negelle Health Science College, Negelle 1000, Ethiopia. Tel: +251 913 118 492/+251 910 790 650. E-mail address: gudisabareda95@ gmail.com (G. Bereda).*

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hypertension by increasing blood pressure by inhibiting the activity of angiotensin-converting enzymes 2 in the rennin–angiotensin system. This case study shows a retired female with confirmed COVID-19, newly discovered hypertension, and pre-existing type 2 diabetes mellitus. This case report was submitted in accordance with the Surgical CAse REport (SCARE) criteria[11].

Case presentation

On 23 March 2022, a retired black African woman in her 60s was taken into the emergency room with the chief complaints of frequent midnight urine, hazy vision, headache, fever, and tingling in her hands and feet. She had been experiencing stomach pain, throwing up, and losing strength 1 day earlier. The hospitalized patient had a past medical and drug history but no family history. She had a history of traveling to another city to visit her ill ex-husband. A COVID-19 positive test has been approved as her ex-hospitalization husband had been infected. Three days before her admittance, her ex-husband, who had been exposed to COVID-19, may have been in close contact with her. Despite having prehypertension the previous year, she did not use any medication to manage it. She was making lifestyle changes to lower her blood pressure, including improved eating habits and regular aerobic activity. Her blood pressure was normal up to the point of admission after 3 months of regular exercise and dietary changes. She had had type 2 diabetes mellitus for the previous 15 years. She had a treatment plan that included the prescription drugs metformin (1.5 g twice a day) and glibenclamide (10 mg twice daily), but she had not been consistent in taking them.

Frequent urination, excessive thirst, hyperglycemia, weak muscles, sore throat, fever, headache, nonproductive cough, and shortness of breath lasting a day were the patient's symptoms at the time of admission to the emergency room. She was in excellent health 3 days before her arrival. At the time of her admission to the emergency room, her vital signs included a body temperature of 39.4°C, weight of 82.3 kg, height of 1.58 m, BMI of 32.9 kg/m^2, blood pressure of 147/98 mmHg, respiratory rate of 17 cycles per minute, and oxygen saturation of 88% on room air. She had a 112 beats per minute peripheral pulse and was dehydrated.

Blood tests performed when she was admitted to the emergency room showed that her fasting blood sugar level was 192 mg/dl, her random blood sugar level was 215 mg/dl, her 2-h postprandial blood sugar level was 235 mg/dl, her serum creatinine level was 2.3 mg/dl, her serum sodium level was 151 mEq/l, and her serum potassium level was 5.1 mEq/l. Before being sent to an ICU, she was given intranasal oxygen at a rate of 5 l/min for 2 h. Auscultation of the patient's chest revealed a nonradiating, moderately uncomfortable chest pain that felt like pressure but subsided with relaxation. A throat swab PCR test that revealed positive results after 26 h confirmed COVID-19's discovery, and then she was admitted to an ICU.

She spent 26 h in the emergency room before being transferred to an ICU, where she was diagnosed with the recently discovered SARS-COV-2 virus, pre-existing type 2 diabetes mellitus, and new onset stage I hypertension. There are currently fewer options for exercise due to government restrictions placed during a pandemic to curb the virus' spread, so she stopped. When she was transferred to an ICU, her vital signs showed that she had a febrile body temperature of 39.5 °C, a peripheral pulse rate of 103 beats per minute, a respiratory rate of 16 breaths per minute, and an oxygen saturation level of 90% on room air.

At the time of her admission to an ICU, her blood tests revealed that she had blood urea nitrogen of 39 mg/dl, fasting blood glucose of 230 mg/dl, 2-h postprandial blood glucose of 245 mg/dl, serum creatinine of 2.5 mg/dl, serum sodium of 149 mEq/l, serum potassium of 5.2 mEq/l, Hb 14.5 g/dl, leukocytes of 4620/µl, platelets of 139 200/µl, neutrophils of 41%, pH arterial blood of 7.09 (normal value: 7.32–7.43), anion gap level of 17 mEq/l (normal value: 3–10 mEq/l), partial pressure of carbon dioxide of 23 mmHg (normal value: 38–42 mmHg), serum bicarbonate level of 8.0 mEq/l (normal value: 22–29 mEq/l), serum phosphate level of 2.6 mg/dl (normal value: 2.8–4.5 mg/dl), white blood cell count of 19 400 cells/mm^3 (normal value: 4500–11 000 cells/mm^3), serum chlorine level of 107 (normal value: 96–106 mEq/l), an AST/SGOT (aspartate aminotransferase) level of 68 U/l (normal value: 0–35 U/l), an (ALT/SGPT) alanine aminotransferase level of 95 U/l (normal value: 0–35 U/l), an erythrocyte sedimentation rate of 9 mm/h (normal value: 0–20 mm/h), 45% hematocrit (normal value: 39–49%), lymphocytes 26%, and urine analysis was positive for urine ketones of 3+.

According to an analysis of her arterial blood gas, she had a high anion gap to make up for metabolic acidosis. Bronchoscopic breath sounds with crackles were detected during a physical examination within normal limits in the middle of the right and left lungs. Her electrocardiogram showed sinus tachycardia with a heart rate of 105 beats per minute and ST depression in the anterior–lateral leads. She had a huge, soft, nontender abdomen that was dull all the time and free of organomegaly. On the Glasgow Coma Scale during the evaluation to determine her level of consciousness, she scored 11 out of 15.

Her drip insulin supply and fluid resuscitation (0.9% normal saline) were started as soon as she was brought to an ICU. As soon as she arrived, she began inhaling 5 l of oxygen per minute through a nasal cannula. She has taken thromboprophylaxis and treatment for her confirmed COVID-19 infection with 40 mg of enoxaparin subcutaneously every 12 h. On day 23, the patient's blood sugar began to normalize, so the syringe pump insulin therapy was discontinued, and subcutaneous insulin injections were once more recommended. She then started receiving NPH (neutral protamine hagedorn) insulin 15/30 to better manage her metabolism. She received one dose of sustained-release nifedipine (20 mg), and then her blood pressure was checked again 2 h later. Her blood pressure was subsequently examined and found to be stage I hypertension classification; enalapril (5 mg) was added to her daily regimen to manage her hypertension. As needed, she received 500 mg of acetaminophen to lower her high body temperature.

Good glycemic control was kept under control with the use of subcutaneous insulin and dietary management. After 30 days, NPH was discontinued, metformin 2 g was reintroduced twice daily, and glibenclamide 10 mg twice daily when her blood sugar levels were under control. Her prior antidiabetic treatment had not been adhered to; thus, the metformin dosage was raised. The patient was finally discharged on 23 May 2022, after 30 days in the hospital, following the negative results of two consecutive COVID-19 throat swab tests. Enalapril, 5 mg daily for 30 days, was added as a new antihypertensive medication regimen prior to her release.

Outcome and follow-up

Her blood pressure and blood glucose levels were nearly normal when she was discharged. At least 1 month after being discharged, the patient's blood pressure and blood sugar were under good control; throughout this period, follow-up and monitoring were carried out in an ICU. She was instructed to keep going to the ambulatory clinic for monthly checkups.

Discussion

SARS-CoV-2 is the cause of COVID-19[12]. The endocrine system's metabolic dysregulations of COVID-19 have included thyroid, adrenal, gonadal axis, and, in particular, pancreatic dysfunction, which can result in glucose dysregulations like diabetes[13]. The SARS-CoV-2 interacts with the angiotensin-converting enzyme 2 receptors that are highly expressed in pancreatic beta cells, the lungs, kidneys, and the small intestine. The renin–angiotensin–aldosterone system's angiotensin-converting enzyme 2 enzymes are principally responsible for converting angiotensin II to angiotensin I. A SARS-CoV-2 infection of pancreatic islet cells has the potential to cause immediate damage to beta cells, leading to insulin shortage[14]. In this study, the patient had been previously diagnosed with type 2 diabetes mellitus. Despite the fact that she was noncompliant with her medication regimen, up to the time of her admittance, her blood glucose levels had never risen as high as they had recently. However, COVID-19 affects the beta cells, forcing them to release insulin and increasing insulin insufficiency, which leads to her blood glucose rise. Type 2 diabetes mellitus also has severe peripheral insulin resistance and a relative insulin shortage.

The clinical prognosis and sickness severity of SARS-CoV-2 patients vary, with advanced age and other chronic medical problems having the most impact[15]. Diabetes mellitus, which has been connected to disastrous outcomes such as ICU hospitalization, invasive ventilation, and death[16], is one of the most frequent comorbidities in COVID-19 patients. Diabetes mellitus is one such condition that has been shown to dramatically impact outcomes in COVID-19 patients[17]. Chronic comorbidities such as obesity, cardiovascular disease, and hypertension, as well as altered angiotensin-converting enzyme 2 expression, dysregulated immunological response, and endothelial dysfunction, may make diabetes patients more vulnerable to COVID-19 with greater severity[18]. In this study, the patient had class-II obesity (32.9 kg/m^2 BMI), which made it difficult for her to follow her antidiabetic medication regimen by raising fatty acid and inflammatory levels, which in turn caused peripheral insulin resistance. Her body may be under stress as a result of battling an infection with the COVID-19 virus, which might cause her blood sugar levels to increase. Diabetes mellitus is, therefore, the most frequent comorbidity of COVID-19 in this case study.

Plasma glucose and diabetes mellitus levels both independently predict mortality and morbidity in SARS-CoV-2 patients. Possible mechanisms that could increase susceptibility to COVID-19 in people with type 2 diabetes mellitus include higher cellular binding with greater affinity and efficient virus entry; decreased viral clearance; decreased T cell function; increased susceptibility to inflammation and cytokine storm; and the presence of cardiovascular disease[19]. Diabetes mellitus is associated with a proinflammatory state and the inhibition of the innate immune response. Diabetes has been linked to an increased risk of infections and adverse outcomes, including pneumonia and influenza[20]. High levels of hyperglycemia are still observed in COVID-19-infected diabetic hospitalized patients[21]. In this case report, her high blood sugar levels increase her risk of developing pulmonary infections by changing her levels of inflammatory cytokines. They also directly increase the replication of SARS-CoV-2 through the production of reactive oxygen species in the mitochondria and the activation of hypoxia-inducible factor 1 alpha. She was more hyperglycemic than usual at the time of her hospitalization since COVID-19 itself may also increase stress hyperglycemia because of the cytokine storm it causes.

In most cases, a COVID-19 infection is a minor illness. The oxygen saturation of the lung field during radiography was 50%, the respiration rate was 30 breaths per minute, and the ratio of the partial pressure of oxygen to the percentage of inspired oxygen was 300 in some patients with reduced respiratory function[22]. Reduced neutrophil, macrophage, and monocyte activity, as well as decreased lymphocyte proliferation in response to a variety of stimuli, are all effects of uncontrolled hyperglycemia. Additionally, aberrant delayed hypersensitivity reactions and defective complement activation have been seen in diabetic patients[23]. Herein, the patient had weak muscles, a fever, a headache, a sore throat, a nonproductive cough, and shortness of breath for a day when she was admitted to the emergency room. Due to her respiratory dysfunction, which includes shortness of breath, the COVID-19 infection, in this case, is not mild. Her weakened antimicrobial defense and increased lung inflammation as a result of diabetes mellitus made her more vulnerable to respiratory distress infections like COVID-19. Her class-II obesity puts her at risk for hypoxemia by reducing lung volumes to the point that her lung units close during normal breathing.

The majority of hospitalized COVID-19 patients will need insulin, especially those with respiratory problems[24]. By reducing symptoms, the likelihood of acute complications, the raised risk of infection, and other hyperglycemia-related issues, hyperglycemia can be controlled[25]. For COVID-19 patients, a helpful technique for maintaining glycemic control and improving health outcomes is continuous intravenous insulin infusion[26]. In addition, metformin, a first-line therapy for type 2 diabetes, improve immune responses and protects against acute respiratory distress syndrome in comparison to other oral hypoglycemia-lowering medicines[27]. As soon as she was taken to an ICU, drip insulin delivery and fluid resuscitation (0.9% normal saline) were started. To treat her confirmed COVID-19 and for thromboprophylaxis, she had 40 mg of enoxaparin subcutaneous injections every 12 h. In order to lower her temperature or fever that occurred due to COVID-19, she was given 500 mg of acetaminophen.

Strengths of the case report

The study used face-to-face communication with the patient and was devoid of selection bias, response bias, and information bias. The study was conducted through direct observation; there is no feedback barrier between the investigator and the responder.

Limitations of the case report

There was no follow-up after the patient was discharged to determine whether she was completely recovered and restored to her normal blood pressure and blood sugar. The study was not based on systematic investigations.

Conclusion

Diabetes mellitus is one of the risk factors for escalating COVID-19 infection. Early illness treatment and transmission prevention now face additional obstacles and challenges as a result of early infection care and prevention. The most likely reasons for a greater risk of SARS-CoV-2 infection and a poorer prognosis in diabetes mellitus include the proinflammatory state, inhibition of the innate immune response, probable higher levels of angiotensin-converting enzyme 2, vascular dysfunction, and prothrombotic conditions.

Ethical approval

This case report didn't require review by ethics committee.

Patient consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written informed consent is available for review by the editor-in-chief of this journal on request.

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None.

Author contribution

G.B. contributed to the preparation of the proposal, participated in preparing the first draft of the manuscript, and editing of the manuscript. The author checked and confirmed the final version of the manuscript.

Conflicts of interest disclosure

There are no conflicts of interest.

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