

Episode 59 – Diabetes and COVID-19 April 29, 2022

Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is associated with worsening diabetes symptoms, and individuals with diabetes are at high risk for developing severe COVID-19. Individuals with type 1 or type 2 diabetes often have comorbidities, such as hypertension, obesity, and cardiovascular disease, which have also been associated with increased morbidity and mortality from COVID-19. Even in individuals without diabetes, SARS-CoV-2 infection has been associated with glucose elevations and worse outcomes. In addition, several studies have reported new-onset diabetes associated with COVID-19. [1] [2] [3] [4]

SARS-CoV-2 binds to angiotensin-converting enzyme 2 (ACE2) receptors,[1] which are expressed in key metabolic organs and tissues, including pancreatic beta cells (β-cell), adipose tissue, the small intestine, and the kidneys. Consequently, it is possible SARS-CoV-2 may have multiple effects on glucose metabolism that could complicate the pathophysiology of preexisting diabetes or lead to new-onset diabetes. [4]

Overview of diabetes

Diabetes is a chronic, metabolic disease characterized by hyperglycemia. Uncontrolled diabetes can lead to serious damage to the heart, blood vessels, eyes, kidneys, and nerves. The most common is type 2, which occurs when the body becomes insulin resistant or produces insufficient insulin. More than 95% of individuals with diabetes have type 2. Type 2 diabetes mainly results from excess body weight and physical inactivity. Symptoms may be similar to those of type 1 diabetes but are often less pronounced. Consequently, the disease may be diagnosed several years after onset, after complications have already occurred. [5] [6]

Historically, type 2 diabetes was only seen in adults. It is now increasing in frequency in children. Rising childhood obesity is a major factor for this increase. However, researchers do not fully understand why some children develop diabetes and others do not, even if they have similar risk factors. Type 2 diabetes in children occurs gradually, with 40% of children diagnosed with no signs or symptoms.[2] [7]

[1] Refer to Episode 28 for additional information on ACE2 receptors and COVID-19.
[2] Possible signs and symptoms of type 2 diabetes in children include polydipsia, polyuria, polyphagia, fatigue, unexplained weight loss, and blurred vision. [7]

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Risk factors increasing the incidence of type 2 diabetes among children include:

- Obesity – being overweight is the primary risk factor for developing type 2 diabetes. Adipose tissue, especially excess abdominal fat, contributes to insulin resistance.[3]
- Sex – young females are more likely to develop type 2 than young males.
- Age – type 2 is more likely to present in the early teens. One reason is hormones during puberty make the body less sensitive to insulin, especially in young females.
- Family history – risk increases if a child has a parent or sibling with diabetes or was born to an individual with gestational diabetes.[4]
- Inactivity – physical activity helps with weight management and helps cells be more responsive to insulin, decreasing insulin resistance. [7] [8]

Adults are at risk for developing type 2 diabetes if they have any of the following conditions:

- High BMI or are overweight, especially if the weight is carried around the abdomen.
- Age ≥40 years.
- Prediabetes (i.e., blood glucose level is higher than normal, but not high enough to be classified as diabetes).
- Have a parent or sibling with type 2 diabetes.
- Physically active less than 3 times a week.
- Developed gestational diabetes while pregnant or gave birth to a baby who weighed over 4 kg (9 pounds).
- Non-alcoholic fatty liver disease.
- Polycystic ovary syndrome.
- High blood pressure.
- High levels of cholesterol and triglycerides.
- Obstructive sleep apnea.
- Acanthosis nigricans (darkened patches of skin).
- Being of African, Arab, Asian, Hispanic, Indigenous, or South Asian descent.
- Long-term use of corticosteroid medication (e.g., >3 months). These medications are widely prescribed to treat inflammation, autoimmune disorders[5] (e.g., lupus, rheumatoid arthritis), and cancer. [9] [10] [11]

Type 1 diabetes is a chronic condition in which the pancreas produces little or no insulin and requires daily administration of insulin. Symptoms of type 1 diabetes may occur suddenly and include polyuria, polydipsia, polyphagia, weight loss, vision changes, and fatigue. Other symptoms include frequent or recurring infections, cuts and bruises that

[3] Insulin resistance usually does not have any symptoms, though some children develop acanthosis nigricans (patches of thickened, dark, velvety skin), usually in body folds and creases (e.g., armpits, back of the neck, or between fingers and toes). As many as 70% of children with type 2 diabetes have this condition. They may also have other conditions related to insulin resistance, such as high blood pressure, high cholesterol, and polycystic ovary syndrome. [8] [43]

[4] Gestational diabetes occurs during pregnancy and is characterized as hyperglycemia with blood glucose values above normal but below levels diagnostic of diabetes. Gestational diabetes is diagnosed through prenatal screening, instead of through reported symptoms. Individuals with gestational diabetes are at an increased risk of complications during pregnancy and at delivery. These individuals, and possibly their children, are at increased risk of type 2 diabetes in the future. [6]

[5] Refer to Episode 45 for additional information on autoimmune disorders.

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are slow to heal, tingling or numbness in the hands or feet, and diabetic ketoacidosis (DKA).[6] The cause and means to prevent type 1 diabetes are not known. However, having a parent or sibling with type 1 diabetes slightly increases risk and it is suspected genetics and the environment may play a role. Investigational studies are ongoing to determine the etiology. [5] [6] [9] [12]

COVID-19 and diabetes

Individuals with underlying medical conditions, such as type 1 and type 2 diabetes, are at higher risk of developing severe outcomes of COVID-19 (e.g., hospitalization, intensive care unit [ICU] admission, intubation or mechanical ventilation, or death). [13] For example, results of a large population study by Barron et al. (2020) showed a significant increased risk of death from COVID-19 in individuals with type 1 and type 2 diabetes. [14]

Several studies have also demonstrated worse COVID-19 outcomes in individuals with poorly controlled diabetes. [15]

Research by Critchley et al. (2018) demonstrated poor glycemic control was associated in general with serious infections and hospital admission. It has been hypothesized hyperglycemia may amplify the hyperimmune response associated with severe COVID-19. [16] [17]

Hayek et al. (2021) showed individuals with poorly controlled type 2 diabetes were much more likely to have severe outcomes from COVID-19. [18]

Holman et al. (2020) found an association between poor glycemic control and COVID-19-related mortality in individuals with type 1 and type 2 diabetes. The risk increased with higher hemoglobin A1c (HbA1c) levels.[7] [17]

Zhu et al. (2020) found well-controlled type 2 diabetes was associated with lower mortality in individuals hospitalized with COVID-19 than those with poorly controlled diabetes. [19]

Diabetes and other glycometabolic abnormalities have been commonly reported during the acute phase of COVID-19. For example, severe metabolic complications of preexisting diabetes, including DKA and hyperosmolarity[8] which require extremely high doses of insulin, have been described in individuals hospitalized with COVID-19. [4]

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[6] Diabetic ketoacidosis (DKA) is a serious, life-threatening complication of type 1 diabetes (much less common in type 2 diabetes). It occurs when insulin deficiency inhibits the ability of glucose to enter cells for use as fuel, resulting in the liver rapidly breaking down fat into ketones to use as a fuel source. The overproduction of ketones causes them to accumulate in the blood and urine and turn the blood acidic. DKA results in the characteristic acetone (fruity) breath odour. [44]

[7] Refer to Episode 31 for additional information on HbA1c.

[8] Diabetic hyperosmolarity (diabetic hyperosmolar syndrome) is a serious condition involving extremely high blood glucose levels, which draws the water out of the body's organs, including the brain to try to remove excess blood glucose via urine. The condition most commonly occurs in individuals with uncontrolled type 2 diabetes and is often triggered by illness or infection. If left untreated, it can lead to life-threatening dehydration. [45] [46]

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There is growing evidence suggesting individuals with COVID-19 could experience a wide range of post-acute sequelae (i.e., long COVID),[9] including diabetes. [20]

A study by Montefusco et al. (2021) revealed glycemic abnormalities could be detected at least 2 months after recovery from COVID-19 in individuals without any preexisting history or diagnosis of diabetes. In almost 35% of these individuals, persistent hyperglycemia continued to be observed for 6 months. [21]

It is not known whether COVID-19 can accelerate preexisting unrecognized diabetes or prediabetes, or directly or indirectly lead to new-onset diabetes. [22]

Additionally, it is not known whether COVID-19 accelerates the onset of permanent diabetes more than other triggering factors. For example, community-acquired pneumonia is frequently associated with hyperglycemia in individuals without diabetes, but it can be a precursor of diabetes, particularly in older individuals, demonstrating hyperglycemia is not unique to COVID-19. [23]

Further, any inflammatory state can cause insulin resistance and increase hepatic glucose production[10] through increased counterregulatory hormones[11] (e.g., glucagon, epinephrine, cortisol), release of cytokines[12] and lipids, and direct hepatocyte[13] injury. [23]

New-onset diabetes

Research has shown an association between SARS-CoV-2 infection and risk of new-onset diabetes. Other viruses (e.g., severe acute respiratory syndrome [SARS] enteroviruses, retroviruses, rubella, mumps, cytomegalovirus, Epstein-Barr virus, varicella zoster virus) have also been associated in the development of diabetes. Whether new-onset diabetes will remain permanent is not known, as the long-term follow-up is limited. Longitudinal research will be required to understand the lasting effects from SARS-CoV-2 infection. [24] [25] [26] [27] [28]

A meta-analysis by Satish et al. (2020) of 8 studies with over 3,700 participants found on average, 14% of adults hospitalized with COVID-19 had new-onset diabetes. [24]

Xie and Al-Aly (2022) reported a significant increased risk of diabetes in the post-acute phase (i.e., after >30 days) of COVID-19 in a cohort of US veterans, including those

[9] Refer to Episodes 28, 38, & 48 for additional information on post-COVID-19 condition (long COVID).
[10] Hepatic glucose production is achieved by glycogen breakdown (glycogenolysis) and new glucose production from available precursors (gluconeogenesis). The liver releases glucose to the blood by glycogenolysis, a process by which glycogen, the primary carbohydrate stored in the liver and muscle cells, is broken down into glucose. Gluconeogenesis involves making glucose from the breakdown of lipids or proteins. [47] [48]
[11] Counterregulatory hormones work against the action of insulin, raising blood glucose levels in response to hypoglycemia. The main counterregulatory hormones are glucagon, epinephrine (adrenaline), cortisol, and growth hormone.
[12] Refer to Episodes 44 &45 for additional information on cytokines.
[13] Hepatocytes are the main functional cells of the liver and perform a number of metabolic, endocrine, and secretory functions.

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who had mild infections and no previous known risk factors for diabetes. The risks were substantial among those who were non-hospitalized and increased with the severity of infection (i.e., hospitalized or admitted to ICU). The analysis found individuals who had COVID-19 were about 40% more likely to develop diabetes up to a year later than were veterans in the control groups. Individuals who were hospitalized or admitted to the ICU had roughly triple the risk compared with the control group who did not have COVID-19. Almost all cases of diabetes detected were type 2. The authors concluded the results suggest diabetes is a component of post-COVID condition (long COVID) and post-acute care after SARS-CoV-2 infection should include diabetes identification and management strategies. [20]

Research by Wander et al. (2022) also showed COVID-19 increased risk of developing diabetes, months after SARS-CoV-2 infection and the risk increased with hospitalization. The risk appeared to be higher in men. The authors hypothesized this may be due to sex differences in immune responses; however, more research is required. [29]

The risk of developing diabetes was also seen in younger populations. Barrett et al. (2022) found youth aged <18 years with COVID-19 were 166% more likely to receive a new diabetes diagnosis >30 days after infection than those without COVID-19, and 116% more likely than those who had an acute respiratory infection prior to the pandemic. Non-SARS-CoV-2 respiratory infections were not associated with an increased risk for diabetes. The observed increased risk for diabetes related to SARS-CoV-2 infection highlights the importance of COVID-19 prevention strategies, including vaccination, for all eligible individuals and screening for diabetes symptoms in children and youth[14] with a history of SARS-CoV-2 infection. [1]

Even adults who had a mild case of COVID-19 were more likely to develop type 2 diabetes than those who had an acute upper respiratory infection according to a recent study by Rathmann et al. (2022). The authors analyzed electronic health records from general and internal medicine practices across Germany. There were 35,865 individuals with documented COVID-19 in the study period (March 2020 to January 2021). The incidence of diabetes after COVID-19 was compared with a cohort of individuals who were diagnosed with an acute upper respiratory tract infection (AURI), but not COVID-19, within the same timeframe. The relative risk of developing type 2 diabetes in the COVID group was 28% higher than in the AURI group. If confirmed through additional research, this study supports monitoring for signs and symptoms of diabetes after recovery from mild COVID-19. [30]

Cromer et al. (2022) conducted a retrospective review of electronic health records of adults admitted to a US hospital with COVID-19. Among 1,902 individuals admitted with COVID-19, 31% (n=594) had diabetes and 13% (n=77) of these individuals had newly diagnosed diabetes. Newly diagnosed diabetes was more common in younger individuals compared to those with preexisting diabetes.

[14] Refer to Episodes 28, 33, & 34 for additional information on COVID-19 in the pediatric population.

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Individuals with newly diagnosed diabetes had lower glycemic parameters (e.g., HbA1c) and higher inflammatory markers (e.g., C-reactive protein [CRP],[15] ferritin, lactate dehydrogenase). Newly diagnosed diabetes was associated with lower insulin requirements, longer length of stay, and ICU admission, but not death. Of the 64 individuals with newly diagnosed diabetes, 56% (n=36) continued to have diabetes and 41% (n=26) regressed to normoglycemia or prediabetes during follow-up (median, 323 days). Of those with evidence of diabetes at follow-up, 44% had diet-controlled diabetes; 42% used noninsulin medications (e.g., metformin); and 14% required insulin.

Based on the findings, the authors suggested COVID-19 infection may not directly cause diabetes but push prediabetes into diabetes and expose undiagnosed diabetes. Also, the findings suggested the primary driver of newly diagnosed diabetes during COVID-19 infection may be stress hyperglycemia[16] rather than direct β-cell injury. Specifically, almost half (41%) with newly diagnosed diabetes experienced regression of hyperglycemia either to the normal or the prediabetic range, suggesting the very high blood glucose seen in these individuals during hospital admission for COVID-19 may represent stress hyperglycemia rather than a permanent change in their physiology. Among those who continued to have evidence of diabetes upon follow-up, the majority were diagnosed with type 2 diabetes and managed without insulin, suggesting permanent β-cell injury or β-cell death was not a major driver of newly diagnosed diabetes at the time of COVID-19 hospital admission. [31]

Furthermore, the risk of mortality appears to be higher in individuals with new-onset diabetes than with individuals with known diabetes. For example, Li et al. (2020) conducted a retrospective study of hospitalized individuals with COVID-19. Results found individuals with newly diagnosed diabetes were more likely to be admitted to the ICU; require invasive mechanical ventilation; had a high prevalence of acute respiratory distress syndrome (ARDS), acute kidney injury, or shock; and had the longest hospital stays, followed by individuals with known diabetes and individuals with hyperglycemia, compared with individuals with normal glucose.

Data also showed glucose levels at hospital admission in individuals with newly diagnosed diabetes and in those with a history of diabetes were both associated with an increased risk of mortality. Patients with newly diagnosed diabetes had a higher mortality than patients with known diabetes, hyperglycemia, or normal glucose. [32]

Maestre-Muñiz et al. (2021) investigated the long-term outcomes of 766 individuals with COVID-19 1 year after hospital discharge, showed 1% (n=7) of the 543 patients who were alive after 1 year had developed new-onset diabetes, and 10% (n=11) of the 109 individuals who had diabetes had worsened glycemic control. [33]

Potential mechanisms for glycemic abnormalities

The precise pathophysiological mechanisms for glycemic abnormalities including new-onset diabetes in individuals with COVID-19 are not known. However, it is likely a

[15] Refer to Episode 31 for additional information on C-reactive protein.

[16] Stress hyperglycemia is a transient increase in blood glucose levels that spontaneously resolves after overcoming of a stressful condition (e.g., acute illness or physical or psychological stress).

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number of complex interrelated processes are involved, such as previously undiagnosed diabetes, stress hyperglycemia, or steroid-induced hyperglycemia due to substantial usage of corticosteroids to treat[17] severe COVID-19. Other possible causes may be adipose tissue dysfunction and insulin resistance, or SARS-CoV-2 triggering β-cell destruction and β-cell autoimmunity. [3]

Preexisting undiagnosed diabetes

Salmi et al. (2022) reported an increase in the number of children admitted to pediatric ICU in Finland due to new-onset type 1 diabetes with severe DKA, and a smaller increase in the incidence of new-onset type 1 diabetes in 2020. This increase took place when there was a low incidence of COVID-19 infections in the pediatric population. Also, 35% of the children diagnosed were tested for SAR-CoV-2, with all being negative. The authors suggested the increase in incidence and severity were due to changes in the healthcare system, the availability of healthcare services, and from parental fears over contracting SARS-CoV-2 rather than direct effects of COVID-19. These changes may have created barriers to accessing healthcare, leading to a delayed diagnosis and aggravated presentation of type 1 diabetes. [34]

A survey by the World Health Organization of 155 countries showed 53% of countries had reduced their preventative- and service-level access for noncommunicable diseases, such as treatment for diabetes and diabetes-related complications either partly or completely in 2020. The full impact of healthcare disruptions on individuals with noncommunicable diseases may take some time to be completely known, including the effect on diagnosis and management of diabetes. [35]

Further, lifestyle changes associated with the pandemic, such as recent weight gain due to self-isolation, social distancing, reduced physical activity, and poor diet as a result of mental health issues could worsen hyperglycemia in those with prediabetes or undetected diabetes. This could lead to insulin resistance, further triggering inflammatory pathways, leading to an increase in diabetes diagnosis upon hospital admission for COVID-19. [3]

Stress hyperglycemia

Individuals with COVID-19 and without preexisting diabetes may present with stress hyperglycemia and may not require long-term antidiabetic treatment following COVID-19 recovery. [15] [23]

Stress hyperglycemia refers to a temporary increase in blood glucose levels during acute illness or physical or psychological stress, which subsides when the stressful event resolves. It is considered a normal metabolic response to acute stress. Regardless, research in both children and adults have associated stress hyperglycemia with adverse clinical outcomes and complications. [36]

For example, stress hyperglycemia in hospitalized patients is associated with longer length of stay, longer ventilator management time, and increased mortality in critically ill

[17] Refer to Episode 57 for additional information on COVID-19 treatment.

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patients. [37] In COVID-19, stress hyperglycemia could be more severe on account of the cytokine storm. [3] [15]

Even though newly diagnosed diabetes in individuals with COVID-19 could be attributed to stress hyperglycemia, studies show exceptionally high insulin requirements in severely or critically ill patients with COVID-19. The prevalence is disproportionate when compared with critical illness caused by other conditions. Thus, the diabetogenic effect of COVID-19 causing persistent blood glucose elevations should be considered. [3] [24]

Several studies have reported stress hyperglycemia after critical acute illness. However, only a few studies have followed these individuals beyond hospitalization to determine if the stress hyperglycemia was temporary or resulted in new-onset diabetes. [3]

For example, Ali Abdelhamid et al. (2016) conducted a meta-analysis of 4 cohort studies with 2,923 participants to evaluate whether stress hyperglycemia increased risk of developing diabetes. Among the participants, 24% (n=698) experienced stress hyperglycemia. On follow-up >3 months after hospital discharge, 19% (n=131) of participants with stress hyperglycemia were identified with newly diagnosed diabetes. The authors concluded stress hyperglycemia was associated with an increased incidence of diabetes. However, the strength of this association should be cautiously interpreted because of statistical and clinical heterogeneity between the studies. [38]

Steroid-induced hyperglycemia

Steroids are associated with a number of side effects including the development of new-onset hyperglycemia or diabetes. Studies have revealed 53-70% of hospitalized individuals without diabetes developed steroid-induced hyperglycemia. [3] [15] [39]

A meta-analysis by Liu et al. (2014) of 13 studies demonstrated 32% of individuals developed steroid-induced hyperglycemia and 19% developed diabetes. [40]

The use of steroids in the context of COVID-19 has increased, especially after the publication of the RECOVERY trial with the use of dexamethasone[18] in hospitalized individuals with COVID-19. Subsequently, a greater use of steroids may increase the risk of developing diabetes, primarily due to the delayed or blunted recovery of β-cell damage. [3] [15] [23] [41]

Steroids elevate blood glucose levels by:

- Increasing hepatic glucose production
- Inhibiting glucose uptake into muscles
- Impairing β-cell function
- Reducing the body's sensitivity to insulin (long-term steroid use can lead to insulin resistance)

The pathophysiology of steroid-induced diabetes and the relative contributions of both β-cell dysfunction and insulin resistance are still not clear. High blood glucose

[18] Refer to Episode 57 for additional information on dexamethasone in the treatment of COVID-19.

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levels may subside after the medication is stopped; however, some individuals develop type 2 diabetes, which will need life-long management. [42]

SARS-CoV-2 infection and the pancreas

Viral infections may have a direct or indirect effect on the pancreas. Studies have reported acute inflammation in the pancreas due to viruses, such as human immunodeficiency virus, mumps, measles, cytomegalovirus, herpes simplex virus, and hepatitis virus.[19] Research has shown certain viral infections were significantly associated with type 1 diabetes-related autoimmunity and functional impairment or β-cell death. [3]

The pancreas is a target of SARS-CoV-2. Acute hyperglycemia has been linked to SARS-CoV-2 binding to the ACE2 receptor in the pancreatic islet cells. ACE2 expression has been shown to be higher in the pancreas than the lungs and expressed in both exocrine glands and the islets of the pancreas, including β-cells. By attaching to ACE2 receptors in β-cells, SARS-CoV-2 could cause acute impairment in insulin secretion. Following SARS-CoV-2 infection, reduced numbers of insulin secretory granules in β-cells and impaired glucose-stimulated insulin secretion have been reported. The virus may damage β-cells by triggering proinflammatory cytokines (e.g., interleukin-6 [IL-6])[20] or by enhancing autoimmunity in genetically predisposed individuals. Also, proinflammatory pathways leading to chronic low-grade inflammation in adipose tissue play a central role in the pathogenesis of insulin resistance and type 2 diabetes.

ACE2 receptors are expressed in the liver, adipose tissue, and skeletal muscle, and the binding of SARS-CoV-2 to these receptors may impair responses to insulin. Consequently, new-onset hyperglycemia and insulin resistance have been reported in individuals with COVID-19 without prior history of diabetes. However, it is not clear whether these metabolic changes are temporary or whether individuals with COVID-19 are at increased risk of developing persistent diabetes. [3] [24] [30]

Take home messages

- Large and well-controlled epidemiological studies with long-term follow-up of ≥1 year will be needed to determine the association between COVID-19, glycemic abnormalities, and new-onset diabetes, and to assess potential causal relationships.
- Oral healthcare professionals should be aware of new symptoms and conditions that may develop after SARS-CoV-2 infection, particularly among those hospitalized. It will be essential to screen clients of all ages for signs and symptoms of diabetes even after recovery from mild COVID-19 and remind clients with previous SARS-CoV-2 infection to monitor for signs and symptoms of diabetes, such as fatigue, frequent urination, and increased thirst, and to promptly seek treatment.
- It is important to be aware of the effects of COVID-19 and its complications will probably be seen for many years to come.

[19] Refer to Episode 53 for additional information on hepatitis B virus (HBV).
[20] Refer to Episodes 44 & 45 for additional information on interleukins.

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- The observed increased risk for diabetes highlights the importance of COVID-19 prevention strategies, including vaccination for all eligible individuals and following public health measures.
- It is important to remember the bidirectional association between diabetes and periodontal disease and educate clients accordingly.

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