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Diabetes with New-onset and Hyperglycemia Linked to COVID-19

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Introduction

COVID-19 symptoms range greatly in intensity, from the absence of symptoms to fatality [1]. The length of the symptoms is another source of variation. Patients in hospitals may develop persistent weariness and shortness of breath [2]. Global partnerships have aided in the creation of COVID-19-fighting vaccinations and treatments [3]. After an intense COVID-19 infection for four weeks, the Organizations for Prevention and Control of Disease (CDC) defined post-COVID-19 illnesses as new health disorders that cannot be attributed to any other causes or chronic or persistent COVID-19 symptoms [4]. People with COVID-19 frequently have certain severe comorbidities, such as high blood pressure, heart disease, being overweight, diabetes, and renal disease. Many symptoms and ailments are connected to post-COVID disorders. Following COVID-19, the CDC lists chest discomfort, difficulty focusing, headache, problem sleeping, dizziness, exhaustion, breathlessness, coughing, changes in smell or taste, diarrhea, stomach pain, and muscle or joint pain as the most prevalent symptoms of the disorder [5]. Hospitalized patients, individuals with medical conditions prior to COVID-19 infection, those who were not immunized, and those who developed multisystem inflammatory syndrome after acute COVID-19 infection are among the patient populations who are at an increased risk for developing post-COVID-19 disease. After COVID-19, advanced age, a high body mass index, and being female are additional risk factors for illness [6]. These people's symptom duration was compared to the symptom duration of symptomatic controls with negative COVID-19 tests who were matched for age, sex, and body mass index. The emergence of new diabetes patients during hospitalization has previously been linked to other viral infections and severe diseases. Although the precise mechanisms by which COVID-19 patients acquire new diabetes are unknown, it is probable that a number of intricately interconnected processes, such as previously undetected diabetes, stress hyperglycemia, microtubule-high blood sugar, and direct or indirect impacts (Figure 1) [7], are involved. SARS-CoV-2 in cells, the serious acute respiratory syndrome coronavirus. Because there is lengthy follow-up for these individuals, new hyperglycemia is likely to persist. Further metabolomic investigations in the context of acute COVID-19 infection are required to identify the causal agent, prognosis, and therapeutic choices, and individuals with diabetes appear to have worse outcomes [8].

Figure 1: Potential mechanisms for development of new-onset diabetes in people with COVID-19 [7].

Methods

For an oral glucose tolerance test, patients diagnosed with diabetes were defined as having hemoglobin A1C levels of 6.5%, a fasting blood glucose level of 126 mg/dL, or a two-hour blood glucose level of 200 mg/dL [9]. Critically ill individuals frequently have severe hyperglycemia, which is frequently used as a gauge of illness severity [10]. The majority of COVID-19 patients were older, and 21.6% of them had diabetes; 20.8% had just been diagnosed (fasting glucose 7.0 mmol/L and/or HbA1c 6.5%); and 28.4% had dysglycemia. This represents an 80% rise in new T1Ds during the pandemic compared to earlier years. Moreover, there seems to be a higher risk of death among young people with T1D, DKA, and hyperosmolar hyperglycemia, which has been observed to be exceptionally common in COVID-19 patients with established diabetes [11,12]. Compared to COVID-19 participants with diabetes who had just developed the disease, although the precise processes behind the emergence of a new diagnostic in COVID-19 individuals are unknown, it is probable that there are several different, intricately linked etiologies at play, including prediabetes, stress hyperglycemia, abnormalities of both insulin production and glucose uptake, and steroid-induced diabetes. These patients may have had undiagnosed diabetes prior to treatment due to recent weight gain, lifestyle changes, and increased hyperglycemia, largely as a result of social isolation, limited physical activity, and bad eating habits as a result of mental illness. After being

hospitalized for an acute illness, people can develop hyperglycemia and new-onset diabetes. This occurrence has been seen before, most notably during the SARS-CoV-1 outbreak. Increased mortality has also been linked to diabetes upon admission without administration of glucocorticoids, and cytokine storms' acute inflammation may make insulin resistance worse. Obesity affects glucose metabolism, immunological responses, and inflammation in addition to being a risk factor for the serious side effects of diabetes and COVID-19 [13]. The association of coronavirus with the ACE2 protein in islet cells of the pancreas has been linked to acute hyperglycemia brought on by coronavirus infection. ACE2 is expressed in glandular tissue and pancreatic islets, including cells, and has been demonstrated to be more abundant in the pancreas than in the lungs. Humans infected with SARS-CoV-2 have a prothrombotic and very inflammatory cytokine storm. Research on the expression of genes and proteins in live cultures of human pancreas and postmortem pancreatic tissue from COVID-19 patients found that SARS-CoV-2 can infect pancreatic cells, suggesting that hypothalamic, pituitary, and adrenal pancreatic islets and exocrine acinar and duct cells allow entry of SARS-CoV-2. SARS-CoV-2 is a clinical condition that can affect pancreatic cells both directly and indirectly [14]. Another study found that COVID-19 patients had pancreatic cells that are abundantly expressed with the SARS-CoV-2 protein, ACE2, and related invasion factors, which invade cells, alter pancreatic insulin levels and synthesis, and result in beta cell death.

Conclusion

Millions of individuals throughout the world have been impacted by COVID-19, and as new versions of the SARS-CoV-2 virus appear, it continues to change. Diabetes is one of the post-infectious consequences of COVID-19 that may pose a considerable public health burden given the large population burden. It is unclear if increased blood sugar (in non-diabetics) or new diabetes is brought on by inflammatory and immunological responses, a direct impact of SARS-CoV-2 on cells, or a complicated interplay of processes. While we are still in the middle of a worldwide COVID-19 pandemic, we are expected to witness an even larger spread of the novel diabetes linked to SARS-CoV-2 infection, which likewise looks to be a complicated illness involving several pathophysiological processes and new cases of diabetes globally [15]. A global effort is required to study a large number of individuals in order to discover novel forms of diabetes linked to COVID-19. Most studies have focused on COVID-19 patients who were hospitalized; there is little to no data on patients with milder illnesses who were handled in the community. Also, nothing is known regarding the long-term prognoses of diabetics with COVID-19 and their protracted COVID-related illnesses.

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