

COVID-19 and Diabetes in Cardiometabolic Disease

Manfredi Rizvi*

Department of Endocrinology, University Clinical Center Ljubljana, Ljubljana, Slovenia

Perspective

For over two years, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic (coronavirus disease 2019 [COVID-19]) has raged with few evidence of abatement or remission. It has impacted not just the bulk of the world's population's daily life, but also the delivery of medical care to our patients. The presence or onset of diabetes in affected people is a significant factor to the severity of COVID-19 and its repercussions, according to epidemiologic research. COVID-19, on the other hand, has had a significant negative impact on the health of diabetics. Diabetes affects more than a third of COVID-19 patients who require hospitalisation, with a case fatality rate of over 25%. Even after accounting for sociodemographic characteristics and concomitant illnesses, patients with diabetes had a 250% increased risk of severe morbidity and mortality, with total mortality rates 50% higher than before COVID.

Comorbid disorders like coronary artery disease, congestive heart failure, and chronic kidney disease have all been linked to poor outcomes. Obesity, metabolic syndrome, and poor glucose control are all factors to consider. Disruption of glucose metabolism, immune modulation, coagulation and inflammatory responses, as well as exacerbation of endothelial dysfunction by acute hyperglycemia and the proinflammatory state have all been proposed as potential pathways underlying the bidirectional and mutually deleterious relationship between glucometabolic perturbations and COVID-19. Endothelial dysfunction is known to be a powerful predictor of future cardiovascular events, and some writers have even proposed that COVID-19 is an endothelial illness in the end.

The malfunction of the vascular endothelium is a crucial element in the development and progression of atherosclerosis at the molecular level, where there is a close interplay between increased inflammation, endothelial dysfunction, and pro-atherogenic lipid changes. Indeed, due to several specific physico-chemical and metabolic properties, smaller and more dense low-density lipoproteins (LDL) have greater atherogenicity than larger counterparts, including prolonged plasma residency time due to reduced affinity for the LDL receptor, greater entry and retention into the arteries, and increased oxidative susceptibility. Several authors have emphasised that inflammation and endothelial dysfunction caused by obesity and diabetes have a significant impact on disease severity in COVID-19 patients, and it has recently been discovered that low levels of high-density lipoproteins (HDL) combined with high levels of triglycerides predict COVID-19 severity. These two lipid changes are generally accompanied by a predominance of tiny, dense LDL, forming the so-called lipid trio or atherogenic lipoprotein phenotype, a lipid feature that is quite common in people with high cardiovascular risk and particularly noticeable in some ethnic groups.

Moreover, endothelial inflammation and atherosclerosis are closely

***Address for Correspondence:** Manfredi Rizvi, Department of Endocrinology, University Clinical Center Ljubljana, Ljubljana, Slovenia; E-mail: manfredi.rizvi@hotmail.com

Copyright: © 2022 Rizvi M. This is an open-access article distributed under the terms of the creative commons attribution license which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Received: 01 January, 2022, Manuscript No. jdcM-22-54392; **Editor assigned:** 03 January, 2022, PreQC No. P-54392; **Reviewed:** 17 January, 2022, QC No. Q-54392; **Revised:** 21 January, 2022, Manuscript No. R-54392; **Published:** 29 January, 2022, DOI: 10.37421/2475-3211.2022.7.164

linked in patients with diabetes or obesity to changes in cytokine biomarkers, which are also strictly associated with atherogenic small, dense LDL; thus, obesity and diabetes patients with concomitant molecular alterations, such as those in inflammatory adipokines and atherogenic lipoproteins, have an increased overall cardiometabolic risk. Investigating the molecular mechanisms that underpin atherosclerosis formation and progression can aid in the development of better treatment strategies; for example, concurrent management of multiple lipid alterations can lower the risk of cardiovascular events and slow the progression of atherosclerosis. This is especially true during the present epidemic, when we've seen a considerable increase in cardiovascular problems in diabetic and obese individuals. Some novel antidiabetic medications, such as glucagon-like peptide (GLP)-1 receptor agonists, appear to play a role in COVID-19 development and severity due to their anti-inflammatory and anti-atherogenic/thrombotic properties, with a potential direct mechanism. In contrast to what has been observed with the administration of some established antidiabetic medicines, these innovative agents can also lower tiny, dense LDL.

A comprehensive examination of the diabetes and obesity-related hazards of exposure, infection, and hospitalisation will assist a coordinated response to the ongoing pandemic. Notably, the pandemic's indirect consequences on chronic illness management, health-care delivery, health-behavioral changes, and speedy response to acute complications have not been thoroughly evaluated. COVID-19 patients' cardiovascular and renal systems deteriorate, both of which can lead to major consequences in the context of diabetes and obesity; therapies must be closely managed, keeping in mind that adherence and duration are crucial for achieving the therapeutic aim. An international panel of experts recently stated that up to 50% of those who died from COVID-19 had metabolic and vascular diseases, emphasising the importance of carefully managing and effectively treating patients with diabetes and obesity now more than ever [1-5].

References

1. Huang, Chaolin, Yeming Wang, Xingwang Li, Lili Ren, and et al. "Clinical features of patients infected with 2019 novel coronavirus in Wuhan Chin.a." *Lancet* 395 (2020): 497-506.
2. Vensentini, Natalia, Ezequiel J. Zaidel, Adrian Charask, Simon Salzberg, and et al. "lo.filo..aciun.ellardi.olra.s..G.IJJaLe_s_en_Ullida.d..e.s...JJ.ida.d.J1s Intensivo durantela pandemia par COVID-19." *Medicina* 80 (2020): 425-432.
3. He, Wenjuan, Lei Chen, Li Chen, Guolin Yuan, and et al. "COVID-19in persons with haematological cancers." *Leukemia* 34 (2020): 1-9.
4. Phrommintikul, Arintaya, Srun Kuanprasert, Wanwarang Wongcharoen, Rungsrit Kanjanavanit, and et al. "Influenza vaccination reduces cardiovascular events inpatients with acute coronary_s_Y[[III0III]. " *Eur Heart J* 32 (2011): 1730-1735.
5. McLaughlin, John M., David L. Swerdlow, Farid Khan, Oliver Will, and et al. "Disparities in uptake of 13-valent pneumococcal conjugate vaccine among older adults in the United States." *Hum Vaccines Immunother* 15 (2019): 841-849.