

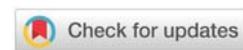


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Letter to Editor

Metabolic Wellbeing and Covid-19

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Abstract

The effects of infection with SARS-CoV-2 on host health are complex and major differences in the severity of the disease are observed. The best choices, nevertheless, are prevention and management. The main functions of genomic metabolic processes regulated by various physiological mechanisms are defined by the metabolic health of a person. Disordered genomic metabolic processes and a reduction in metabolic health are triggered by disruption of these mechanisms. Weaknesses health is the main risk factor for extreme COVID-19. It is probable that potential physiological changes induced by metabolic syndrome and T2D will overlap with COVID-19, potentially exacerbating the course of the disease. Although chronic diabetes hyperglycaemia contributes to harm that can exacerbate the path of the disease in COVID-19, recent research indicates that in patients with COVID-19 and T2D, dysregulated glucose by itself is counterproductive. To regulate the host inflammatory response, to reduce tissue susceptibility to inflammatory damage signals and to maintain physiological function during the extreme and critical phase of infection, glycaemic control is essential.

Introduction

The effects of infection with SARS-CoV-2 on host health are complex and major differences in the severity of the disease are observed. It is possible to identify COVID-19 cases as either asymptomatic or symptomatic. For 40–45 percent of infected persons, asymptomatic persons include carriers who test positive for SARS-CoV-2 and display no symptoms, such as those who test negative for the virus but display seroconversion suggesting a previous infection without ever showing symptoms [1]. These individuals maintain their wellbeing over time during infection, displaying a phenotype of improving health [2]. Symptomatic people include those with no symptoms who exhibit illness after a presymptomatic period. It is very difficult to distinguish between true asymptomatic and pre-symptomatic states. Asymptomatic people are identified as persons that test positive for RT-PCR but lack symptoms that would suggest infection with SARS-CoV-2 [3]. Although some people may go through the whole infection course and never experience symptoms, days or weeks later, some people who initially present as asymptomatic may begin to develop symptoms [3]. The people who will experience symptoms later are described as being presymptomatic. Around 80% of these individuals have a 'mild' course of illness, while the other 20% proceed to serious and severe conditions correlated

with respiratory failure, Acute Respiratory Distress Syndrome (ARDS) and pneumonia, septic shock and multi-organ failure [4].

The clinical manifestations can be split into three phases for symptomatic individuals. When a person becomes symptomatic, phase 1 begins. Usually, individuals experience a dry cough and fever, and they can lose their taste and smell senses, and feel general malaise. The infection is restricted to this phase for most individuals, with sufficient self-care [5]. The pulmonary phase of the infection is identified in phase 2. Pulmonary inflammation and pneumonia occur in individuals who reach this stage, either without hypoxia or with hypoxia [5]. Those individuals need hospitalization. Mechanical ventilation is usually needed for patients with prolonged hypoxia [5]. Then, patients can advance to phase 3. ARDS and extrapulmonary systemic hyperinflammation syndrome may emerge in these patients who are in severe situation. Furthermore, shock, vasoplegia, respiratory failure, cardiopulmonary collapse, myocarditis, acute kidney injury, and other extrapulmonary complications may grow [5]. For most of these patients, the prognosis is negative, and some begin to drop towards death, while others reach stage 4, the period of recovery, and survive and show a phenotype of healthy resistance [6].

The best choices, nevertheless, are prevention and management [7]. It was examined that, based on the situation and symptoms of the patient, a mixture of antiviral drugs with hydroxyl-chloroquine and azithromycin (with a medical practitioner's consultation) could be the best choice to treatment the patients [7]. It is urgently advised and demanded that the preventive steps, management and quarantine should be strictly followed by all individuals without any religious disparity, otherwise the situation could be the worst [7].

Metabolic processes and physiological mechanisms

The main functions of genomic metabolic processes regulated by various physiological mechanisms are defined by the metabolic health of a person. Disordered genomic metabolic processes and a reduction in metabolic health are triggered by disruption of these mechanisms. Weaknesses health is the main risk factor for extreme COVID-19. T2D is among the most popular co-morbidities in infected people in reported coronavirus infections [8,9]. T2D, obesity and hypertension tend to be significant comorbidities in people with COVID-19 in line with this observation, and are associated with more serious and essential courses of COVID-19 disease [10-12]. It is possible that the reasons for this result are multi-factorial and are traditionally assumed to include immune dysfunction. In particular, for instance, people with metabolic syndrome and T2D have impaired immune function, and their antiviral response to SARS-CoV-2 could also be impaired [12]. In addition, physiological complications of T2D and metabolic syndrome are also likely to play a synergic role in the pathophysiology of SARS-CoV-2, rendering patients more vulnerable, regardless of viral strain, to the development of extreme pathology. Additionally, young obese individuals tend to need hospitalization and to contract more serious and important diseases among people with COVID-19 in the world [13]. This finding indicates that in young people, obesity can move COVID-19 towards more serious and important conditions. One potential reason may be that by obstructing diaphragm excursion, obesity imposes physical stress on breathing. In addition, the risk of pulmonary fibrosis, chronic obstructive pulmonary disease and decreased respiratory function is increased by diabetes. These disorders further exacerbate the breathing and hypoxic conditions of patients, contributing to multiorgan injury, as patients with COVID-19 advance to the pulmonary stages of the disease and produce pneumonia and ARDS.

It is probable that potential physiological changes induced by metabolic syndrome and T2D will overlap with COVID-19, potentially exacerbating the course of the disease. Chronic inflammatory conditions include T2D and metabolic syndrome. The increased underlying inflammatory state in patients with pre-existing impaired metabolic health may raise the danger that the immune process will exceed pathogenic stages, as well as the risk of physiological damage, since the extreme and important phases of COVID-19 are guided by an increased immune response to inflammation (cytokine storm) [12]. Likewise, risk factors for kidney failure are diabetes and hypertension together. Diabetes progresses to diabetic kidney disease, in which a reduction in the function of the kidney

results in the accumulation of toxic metabolites that could really damage multiple organs [14,15]. The harm caused by hypertension to the blood vessels reduces the flow of blood to the kidneys and ultimately results in kidney injury. The kidney suffering inflicted by these currently existing metabolic conditions can make the kidneys more vulnerable to illness during SARS-CoV-2 infection, since kidney injury is a frequent cause of COVID-19 [16].

The risk of stroke and cardiovascular complications, which are also found in serious and important cases of COVID-19, is increased by obesity, diabetes and hypertension [17,18]. The blood vessels are weakened by prolonged hyperglycaemia and obesity and are possible causes for plaque build-up, which is itself necessary to induce blood clots. Individuals with COVID-19 have an increased coagulation reaction, which could increase the risk of stroke or pulmonary embolism in conjunction with pre-existing weakened blood vessels with plaques. The impact on the blood vessels of obesity and diabetes could contribute to hypertension, which in turns increases haemodynamics and changes the heart structure. This change will make the heart more vulnerable to damage related to the inflammatory response of the host, ARDS-induced hypoxia, virus infection of the heart, or other cardiac effects in response to extrapulmonary multi-organ damage, thereby potentially explaining why some patients with COVID-19 display signs of heart damage and heart attack. After this, in persons with diabetes, damage to blood vessels and nerves in the brain can cause vascular cognitive decline, dementia and stroke. Seizures are also caused by dysregulated glycaemia in diabetics. There are seizures, strokes and brain inflammation in people with COVID-19, and those with impaired metabolic health could be more vulnerable to developing these diseases due to pre-existing factors that have a detrimental effect on brain health [19].

In the pathogenesis of these diseases, the prevalence of hypertension, cardiovascular disease, nephropathy and retinopathy in people with diabetes has impaired the renin-angiotensin system (RAS). Angiotensin II, the active metabolite of RAS and a potent vasoconstrictor that enhances vascular peripheral resistance, also induces insulin resistance and controls the absorption of sodium in the kidneys. Angiotensin-converting enzyme 2 (ACE2), a receptor that regulates the entry into host cells of SARS-CoV-2, antagonizes the actions of vasodilation-inducing angiotensin II. ACE2 expression in several target tissues of SARS-CoV-2 is increased in patients with T2D [20], and this enhancement has been suggested to be an adaptive mechanism that defends the body against diabetes-related physiological dysfunction [21]. This increased expression will boost the entry and resulting replication of SARS-CoV-2, as well as its distribution throughout the body in T2D patients. Thus, in the sense of COVID-19, a physiological defence technique used to promote metabolic health in T2D could be deleterious.

Conclusions

Although chronic diabetes hyperglycaemia contributes to harm that can exacerbate the path of the disease in COVID-19,



recent research indicates that in patients with COVID-19 and T2D, dysregulated glucose by itself is counterproductive. To regulate the host inflammatory response, to reduce tissue susceptibility to inflammatory damage signals and to maintain physiological function during the extreme and critical phase of infection, glycaemic control is essential. Therefore, in addition to providing to recognizing the pathophysiology of COVID-19 and how inadequate metabolic health exposes patients to a more serious course of disease, it could help to manipulate organismal metabolic variables in patients to shift the symptoms of a disease to a more desirable one, but in addition with other treatment mechanisms. After all, prevention and management seem to be the best solutions.

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