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Editorial

Gut Microbiota and COVID-19

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The gastrointestinal tract, like the respiratory system, is affected by COVID-19. Viral particles replicate in enterocytes. In addition, immune system disorders are important in the pathogenesis of COVID-19. Many commensal microbes are known to have immunomodulatory effects, and changes in the composition of the microbiota can lead to immune system dysfunction.

Patients with COVID-19 were significantly more likely to have an abnormal microbiota than patients without the disease (regardless of antibiotic treatment) and these changes persisted for 30 days. The degree of microbiota change was independent of the amount of SARS-CoV2 RNA excreted in the faeces, the intake of corticosteroids, antiviral drugs and proton pump inhibitors. Microbiota changes were most strongly associated with the severity of COVID-19 (mild, moderately severe, severe, critical illness). To a lesser extent, there was an association between microbiota changes and antibiotic intake. The degree of microbiota change was also correlated with elevated concentrations of inflammatory cytokines and blood markers such as C reactive protein, lactate dehydrogenase, aspartate aminotransferase and gamma-glutamyl transferase.

Commensal microbes such as Faecalibacterium prausnitzii, Eubacterium rectale and a range of bifidobacteria, which have an immunomodulatory effect, were less common in patients with COVID-19. At the same time, the patients had more micro-organisms such as Ruminococcus gnavus, Ruminococcus torques and Bacteroides dorei. This imbalance was more pronounced the more severe the COVID-19 symptoms and the more inflammatory markers in the blood. Such persistent microbiota abnormalities after coronavirus infection may predispose to the development of some persistent symptoms and a multisystem inflammatory response after COVID-19 [1].

Angiotensin-converting enzyme 2 (ACE2) deficiency alters the composition of the gut microflora in mice, and patients with COVID-19 develop gut dysbiosis accompanied by a reduction in bacterial diversity and abundance. This dysbacteriosis has serious consequences: gut microflora can remotely stimulate the host's response to viral respiratory tract infections; conversely, dysbiosis can worsen disease outcomes by reducing the number of commensal bacteria, thus promoting the multiplication of pathogenic flora. The role of gut microflora in coronavirus infection remains to be elucidated to determine whether gut microflora can serve as a biomarker of disease severity or as a treatment for this disease [2].

It should be noted that in COVID-19 in persistent microbiota disruption, the cholinergic system, particularly the cholinergic anti-inflammatory pathway, may play a significant role related to the concentration of proinflammatory cytokines in the blood and in the affected organs innervated by N. vagus [3-6].

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