VIA MEDICA

LETTER TO THE EDITOR

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POST-COVID-19 HAEMATOLOGIC CHAMGES: POTENTIAL MECHANISMS BEHIND INCREASED ANAEMIA RECOGNITION

Oskar Szymanski, Magdalena Grzesiak

University Medical Hospital in Wroclaw, Poland

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Dear Editor,

recently, I had the pleasure to read the publication by Acar et al. [1] in the Journal of Disaster and Emergency Medicine. The authors discuss the problem of anaemia detection rising between September 2020 and September 2023, specifically haemolytic anaemia and investigate any possible connections to the COVID-19 pandemic aftermath. The authors' conjectures appear to be in accordance with scientific findings concerning long COVID-19. Long COVID-19, also known as Post-Acute Sequelae of COVID-19 (PASC), is a condition where symptoms continue or appear after the initial phase of COVID-19 [2, 3]. The duration of these symptoms can extend for several weeks or even months following the resolution of the initial manifestations of the disease. The mentioned study period encompasses both the highest level of the pandemic's severity and its subsequent gradual decrease, making it a particularly intriguing timeframe for observing long-term COVID-19. With a prevalence rate ranging from 10% to as high as 30% among COVID-19 patients, this condition presents a substantial challenge for healthcare professionals and systems globally [4].

Existing literature has documented haematologic alterations in the erythroid system in individuals after recovering from COVID-19, including anaemia, elevated mean corpuscular volume, and reduced haemoglobin levels [5]. Nevertheless, what mechanisms could be responsible for these alterations? While long COVID-19 does not specifically refer to a distinct disease, it encompasses a range of symptoms, including a vascular immune-mediated response and inflammation [4, 6].

Among the different ways a disease can manifest, the involvement of cytokine-mediated thromboinflammation is especially important in this situation. The presence of microvascular thrombosis, heightened atherosclerosis, and overall endothelial damage are potential factors that could contribute to the heightened awareness of anaemia caused by erythrocyte damage while passing through modified blood vessels. In addition, the literature also documents alterations in the structure of red blood cells in COVID-19 patients that continue for several months after being discharged from the hospital [7, 8]. These modified red blood cells display a noticeable physical characteristic - an elevated standard deviation in their level of flexibility, size, and volume. However, the red blood cells of patients who have recovered do not completely return to the condition of those in the healthy group. Furthermore, certain red blood cells

CORRESPONDING AUTHOR:
Oskar Szymanski, University Medical Hospital in Wroclaw, 6/1 Prudnicka St., 50-503 Wroclaw, Poland
phone: +48692734277; e-mail: osgiliat@wp.pl
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exhibited diminished dimensions and irregular morphology, prompting researchers to hypothesize the presence of fragmented erythrocytes [7].

In addition to the changes in the shape of erythrocytes, COVID-19-induced oxidative stress also impacts the proteins and lipids present in the cell membrane. This leads to a decrease in the stability of the membrane and an increase in glycolysis within the erythrocyte [8]. Mature erythrocytes cannot produce new proteins, so these alterations remain unchanged throughout their lifespan, which may explain the prolonged symptoms. Structural protein damage and membrane lipid changes may make COVID-19 patients more likely to break down red blood cells. The damage caused by oxidative stress may explain this susceptibility. The altered shape and size of some red blood cells may increase spleen trapping and reduce lifespan due to damaged capillary walls.

On the other hand, erythrocytes that have undergone fewer changes and have not been removed from the bloodstream by the spleen are still susceptible to damage because their cell membrane is less stable, their ability to change shape is reduced, and they are believed to be more sensitive to oxidative stress. Patients with chronic fatigue syndrome (CFS), which is another manifestation of long-term COVID-19, have also shown a decrease in red blood cell (RBC) deformability [9]. Erythrocytes obtained from patients with CFS exhibited reduced membrane fluidity, modified surface charge on the plasma membrane, and elevated levels of reactive oxygen species.

Long-term COVID-19 causes haematologic changes. However, the exact development of these changes is complex and the role of different processes and modifications is unclear. This letter covered the main pathophysiological changes that may cause haematologic abnormalities. Additionally, the article hypothesizes mechanisms related to the increased identification of haemolytic anaemia in Acar et al. [1] article.

Article information and declarations

Author contributions

The conceptualization and idea development for the study were carried out by Oskar Szymański. Magdalena Grzesiak was responsible for data preparation and conducting the literature review. The manuscript was written by Oskar Szymański.

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Conflict of interest

All authors declare no conflict of interest.

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