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Thrombocytopenia as diagnostic and prognostic biomarker for severity of COVID-19 infection

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Coronavirus disease 2019 (COVID-19) is a highly contagious disease spread through contact and aerosol transmission, including severe acute respiratory syndrome-coronavirus 2 (SARS-CoV-2), which has affected more than 3000 globally 10,000 people and caused approximately 1 million deaths since December 2019 [1]. On March 12, 2020, the World Health Organization declared it a global pandemic [2]. As of March 28, 2021, WHO has received 126,359,540 confirmed cases of COVID-19, including 2,769,473 deaths [3]. Sudan reported its first confirmed case of COVID-19 on February 26, 2020. As of March 30, 2021, there have been 29,825 confirmed cases and 2041 deaths [4].

The coronavirus has seven subtypes in humans, including 229E, NL63, OC43, HKU1, Middle East respiratory syndrome (MERS)-CoV, severe acute respiratory syndrome (SARS)-CoV, and 2019 novel coronavirus (nCoV) [5]. SARS-CoV-2 is a single-stranded RNA enveloped virus that targets cells through the viral structural spike protein (S) that binds to angiotensin-converting enzyme (ACE2) receptor 2. Once the receptor binds, the virus particle uses the host cell's receptor and endosome to enter the cell. The host type 2 transmembrane serine protease TMPRSS2 promotes cellular entry through protein S. Once in the cell, the viral polyprotein encoding the replication-transcriptase complex can be synthesized. The virus then synthesizes RNA through its RNA-dependent RNA polymerase. The synthesis of structural proteins leads to the completion of assembly and the release of virus particles [6].

The common manifestation of the disease is a febrile illness related to respiratory symptoms (e.g., cough and dyspnea), but other atypical manifestations of infection have been observed. Several cases of COVID-19 have been diagnosed with fever, thrombocytopenia, bleeding, diarrhea, confusion, and renal insufficiency, without evidence of lung involvement. In a narrow sense, only patients with fever and pulmonary symptoms are regarded as potentially infected, which will lead to many missed diagnoses and delayed diagnosis. This is why all doctors should consider these atypical manifestations [7]. Although for most COVID-19 patients, the clinical symptoms are mild and the prognosis is good, it becomes a severe case after about 20 years, accompanied by pneumonia, pulmonary edema, septic shock, metabolic acidosis, acute respiratory distress syndrome Seizures, and even death. Therefore, timely diagnosis and accurate assessment of symptomatic treatment are very important and are the key to improving prognosis and reducing mortality [8].

In addition, hematological changes are common in COVID-19 patients, including low lymphocyte and platelet counts, but normal white blood cell counts. The activated partial thromboplastin time is prolonged, with 26% of D-dimer levels elevated, and the prothrombin time is normal in most patients [9]. In order to optimize patient care and resource allocation during this pandemic, biomarkers are urgently needed to stratify patient risk and actively monitor disease severity. In addition, low platelet counts are associated with higher disease severity scores, such as Multiple Organ Dysfunction Score (MODS), Simplified Acute Physiology Score (SAPS) II, and Acute Physiology and Chronic Health Assessment (APACHE) II.

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According to reports, the incidence of thrombocytopenia in severe acute respiratory syndrome (SARS) is as high as 55%, and it has been identified as an important risk factor for death [10]. The thrombocytopenia caused by SARS-CoV-2 infection is similar to the thrombocytopenia caused by SARS-CoV and HCoV-229E infection. Based on this phenomenon, it is speculated that SARS-CoV-2 also inhibits hematopoiesis in the bone marrow through certain receptors, resulting in a decrease in primary platelet formation and leading to thrombocytopenia, as well as thrombocytopenia due to increased destruction, due to COVID-19 May increase thrombopoietin level. Autoantibodies and immune complexes lead to specific destruction of platelets by the immune system and ultimately caused by increased consumption of platelets because viral infection and inflammation can cause lung damage. Damaged lung tissue and lung endothelial cells can activate platelets in the lungs, leading to microthrombus aggregation and formation, increasing platelet consumption [9].

We concluded that the platelet count is an easy-to-obtain biomarker that is independently related to the severity of illness and the risk of death in the intensive care unit (ICU). More importantly, this observation illustrates the fact that low platelet count (thrombocytopenia) can be used for early detection and risk stratification of COVID-19.

Keywords: Biomarker, COVID-19, SARS-CoV-2, Thrombocytopenia

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Author Contributions

Khalid Abdelsamea Mohamedahmed – Conception of the work, Design of the work, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Maha Mohamed Abbas – Conception of the work, Design of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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