Systemic lupus erythematosus concurrent with COVID-19: is platelet the right target?

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Abstract

In December 2019, Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), is widely spread and causes a critical threat to clinical burden and public healthcare. The outbreak of COVID-19 has aroused widespread concern in the rheumatology community.

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In December 2019, Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), is widely spread and causes a critical threat to clinical burden and public healthcare. The outbreak of COVID-19 has aroused widespread concern in the rheumatology community[1].

Consideration its alarming levels of spread and severity, new biomarkers are urgently needed to stratify patients' risk and assess disease severity. Hematological changes are common in COVID-19 patients, which include reduced lymphocyte count and platelet count but normal white blood cell count[2]. Platelet count is a fast, simple, inexpensive and easily available biomarker. Henry et al indicated that low platelet count is associated with increased risk of severe disease and mortality in COVID-19 patients. Platelet count increased the ability to assess COVID-19 patients, and should serve as a practical tool to assess the worsening illness of COVID-19 patients during hospitalization[3]. Li et al reported that platelet count was an independent risk factor for COVID-19 mortality. Baseline platelet levels and dynamic changes were closely associated with subsequent mortality in COVID-19 patients at admission. Close monitoring of platelets during hospitalization can help clinicians develop targeted therapies and provide timely critical care to patients[4].

Co-occurrence between rheumatic diseases and COVID-19 is a topic worth considering in clinical rheumatology. Systemic lupus erythematosus (SLE) is a common and worldwide autoimmunity disease, and thrombocytopenia is a common clinical hematological disorder in SLE. A longitudinal observational study showed that patients with SLE-related thrombocytopenia have high disease activity, bad prognosis, and decreased survival rate[5]. Dong et al suggested that an increased risk of mortality and end organ damage in patients with SLE were related with thrombocytopenia[6]. Nevertheless, the correlation between thrombocytopenia, COVID-19, and SLE has not been reported.

When SLE patients are infected with SARS-CoV-2, and accompanied by thrombocytopenia, is it the cause of SLE or SARS-CoV-2 infection, will SARS-CoV-2 increase the disease activity and severity of SLE patients? How to continue to manage SLE in the context of the COVID-19 pandemic? How rheumatologists use their own basic knowledge and rich clinical experience to judge and choose drugs to achieve the best therapeutic effect?

Platelet is an important biomarker and should be monitored when co-occurrence between SLE and COVID-19. The mechanism of thrombocytopenia in the development of COVID-19 and SLE is still unclear and needs to be clarified by further clinical and basic research.

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