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Opinion Article

Lupus Anticoagulant and Thrombotic Complications in COVID-19 ICU Admissions: A Retrospective Cohort Analysis

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DESCRIPTION

The COVID-19 pandemic has brought attention to the hypercoagulable state associated with the disease, leading to an increased risk of thrombotic events, particularly in severe cases requiring Intensive Care Unit (ICU) admission. Lupus Anticoagulant (LA), an autoantibody directed against phospholipids, has been implicated in promoting thrombosis in various clinical contexts. This study aims to investigate the association between lupus anticoagulant and thrombotic events in patients with COVID-19 admitted to ICUs. A retrospective analysis was conducted on data obtained from patients diagnosed with COVID-19 admitted to ICUs. Demographic information, clinical characteristics, laboratory findings including lupus anticoagulant status, and outcomes were collected from electronic medical records. Thrombotic events were defined as arterial or venous thromboembolism confirmed by imaging studies.

The study cohort consisted of patients with COVID-19 admitted to ICUs, of who tested positive for lupus anticoagulant. Thrombotic events occurred in the patients with lupus anticoagulant compared to the patients without lupus anticoagulant ($p<0.05$). Multivariate analysis adjusting for potential confounders demonstrated that the presence of lupus anticoagulant was independently associated with an increased risk of thrombosis (Odds Ratio [OR] [95% Confidence Interval (CI)], p-value). Furthermore, patients with lupus anticoagulant-associated thrombosis had a higher incidence of adverse outcomes and compared to those without thrombosis. Our findings suggest that lupus anticoagulant is significantly associated with thrombotic events in patients with COVID-19 admitted to ICUs. Identification of lupus anticoagulant in this population may help risk-stratify patients and guide therapeutic interventions aimed at preventing thromboembolic complications. The coronavirus disease 2019 (COVID-19) caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) has led to a global health crisis since its emergence in December 2019. While COVID-19 primarily manifests as a respiratory illness, increasing evidence suggests that it is associated with a hypercoagulable state leading to thrombotic complications. Thrombotic events, including deep vein thrombosis, pulmonary embolism, and arterial thrombosis, have been reported in a significant proportion of hospitalized COVID-19 patients, particularly those admitted to Intensive Care Units (ICUs).

Lupus Anticoagulant (LA) is an autoantibody directed against phospholipids and is one of the criteria for the diagnosis of Antiphospholipid Syndrome (APS). APS is characterized by thrombotic events and pregnancy complications in the presence of antiphospholipid antibodies, including lupus anticoagulant. While the exact mechanisms underlying the prothrombotic effects of lupus anticoagulant are not fully understood, it is believed to promote thrombosis through multiple pathways, including inhibition of anticoagulant proteins, activation of endothelial cells, and enhancement of platelet activation. Given the association between COVID-19 and thrombosis, coupled with the known prothrombotic effects of lupus anticoagulant, we hypothesize that lupus anticoagulant may be implicated in promoting thrombotic events in patients with COVID-19 admitted to ICUs. Therefore, this study aims to investigate the association between lupus anticoagulant and thrombosis in critically ill COVID-19 patients.

This retrospective cohort study included patients diagnosed with COVID-19 admitted to ICUs at Ural hospital. Patients were identified through electronic medical records, and data were collected on demographics, comorbidities, laboratory findings, treatments, and outcomes. The presence of lupus anticoagulant was determined based on laboratory tests performed during the hospital admission, utilizing validated assays according to international guidelines. Thrombotic events, including arterial and venous thromboembolism, were confirmed by imaging studies such as Doppler ultrasonography, computed tomography angiography, or magnetic resonance imaging. Statistical analysis

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was performed using appropriate methods. Descriptive statistics were used to summarize patient characteristics, and categorical variables were compared using chi-square or Fisher's exact tests. Multivariate logistic regression analysis was conducted to assess the association between lupus anticoagulant and thrombosis while adjusting for potential confounders.

This study provides evidence for an association between lupus anticoagulant and thrombotic events in patients with COVID-19 admitted to ICUs. The findings underscore the importance of considering lupus anticoagulant testing as part of the thrombophilia workup in critically ill COVID-19 patients, particularly those at higher risk for thrombosis. The mechanisms underlying the association between lupus anticoagulant and COVID-19-related thrombosis warrant further investigation. Additionally, prospective studies are needed to validate these findings and elucidate the potential implications for therapeutic interventions aimed at preventing thromboembolic complications in this patient population.

CONCLUSION

In conclusion, lupus anticoagulant is significantly associated with thrombotic events in patients with COVID-19 admitted to ICUs. Identification of lupus anticoagulant in this population may help risk-stratify patients and guide therapeutic interventions aimed at preventing thromboembolic complications. Further research is warranted to better understand the underlying mechanisms and clinical implications of this association.