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COVID-19 associated thromboembolism: causing the respiratory failure

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Abstract

Coronavirus disease 2019 (COVID-19) has recently emerged in China and caused a global pandemic. WHO announced that COVID-19 could be characterised as a pandemic due to unprecedented swift global spread and severity of the outbreak. When infected with the virus, patients usually have a fever, dry cough, dyspnoea, myalgia, headache and sometimes diarrhoea. Updates on molecular characteristics of SARS-CoV-2, treatment and epidemiological control are more important to help optimise the disease control measures Thrombotic complication is an essential issue in patients infected with COVID-19. Concomitant venous thromboembolism (VTE) seems to be a potential cause of unexplained deaths in COVID-19 cases. Thrombocytopenia, elevated D-dimer, prolonged prothrombin time, and disseminated intravascular coagulation are the clinical findings related to such condition. In China, anticoagulant therapy in severe COVID-19 was suggested for improving outcome. Studies showed the urgency for VTE diagnostic strategies. Aetiology may be multifactorial, and therefore, we review the available literature relevant to acute venous thromboembolism associated with novel coronavirus infection.

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INTRODUCTION

Since December 2019, the third zoonotic coronavirus breakout causes human to human transmission resulting in novel severe acute respiratory syndrome coronavirus 2. Started from Wuhan, China, this pathogen has become the centre of global attention, due to the rapid spread worldwide (Gorbalenya *et al.*, 2020). Cardiovascular disease, hypertension and diabetes mellitus are the most common underlying diseases in adult patients, with males more severely affected than females (Lai *et al.*, 2020; Giannis *et al.*, 2020). This novel virus is related to the SARS virus and has the potential to develop the severe respiratory syndrome. Initially, the Spike protein(S-protein) of SARS-CoV-2 binds with angiotensin-converting enzyme 2 (ACE2). Furin-like cleavage site in the S- protein causes enhancing viral fusion with host cell membranes. This COVID-19 has a pro-inflammatory and hypercoagulable state with a marked increase in Lactate Dehydrogenase, Ferritin, C-reactive protein, D-Dimer, and Interleukin levels (Han *et al.*, 2020). A thrombotic complication is an essential concern in COVID-19 patients with elevated D-dimer. Acute infections are even associated with a transiently increased risk of venous thromboembolic condition (Danzi *et al.*, 2020). Association between influenza asso-