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COVID-19 versus venous thromboembolism: what is known after 2 years of pandemic?

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Coronovirus disease 2019 (COVID-19) is a respiratory disorder caused by the coronavirus designated as SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) [1, 2]. The disease was detected in December 2019, in Wuhan, Hubei Province of the People's Republic of China. It spread rapidly throughout Asia, Europe and other continents. The World Health Organization (WHO) has announced COVID-19 a pandemic.

COVID-19 patients often experience thromboembolic events, mostly venous thromboembolism (VTE), less often arterial thromboembolism (ATE) [3]. These events occur most frequently in patients at intensive care units (ICU), less often in patients hospitalized outside ICU and even less frequently among outpatients. There is general consensus regarding implementation of VTE pharmacological prophylaxis for hospitalized COVID-19 patients, providing there are no contraindications (active bleeding or a high bleeding risk). Recent studies indicate that standard doses of low-molecular weight heparins (LMWH) are recommended for ICU patients while for selected COVID-19 patients of general departments higher (therapeutic) doses of LMWH should be considered [4, 5]. There is no consensus among experts as to the role of primary pharmacological thromboprophylaxis in COVID-19 patients who do not require hospitalization and those discharged as COVID-19 convalescents. An individual approach to anticoagulant therapy is recommended and the most important factors to be considered are: 1) presence/absence of additional (COVID-19 independent) risk factors for VTE and 2) presence/absence of risk factors for bleeding [6].

In April 2021, several research groups described a new disease entity — vaccine-induced immune thrombotic thrombocytopenia (VITT) [7–10]. Diagnostic criteria are: thrombocytopenia and thrombosis within 5–30 days of vector vaccine against SARS-CoV-2, significantly higher concentration of D-dimer and positive ELISA test for antibodies to platelet factor 4 (PF4) with heparin (anti-PF4: H), despite lack of previous exposure to heparin [11]. Worth noting is the marked percentage of VITT cases located in celebral and splanchnic veins. The recently published analysis of 220 VITT cases estimated mortality rate at > 20%, with thrombocytopenia < 30 G/l and intracerebral bleeding as the major fatality risk factors of [12]. VITT management includes: 1) iv immunoglobulins at a dose of 1.0 g/kg/day for 2 days, 2) glucocorticosteroids, 3) non-heparin anticoagulants, i.e. fondaparinux or direct oral anticoagulants (e.g. rivaroxaban, apixaban). Platelet concentrate transfusions should be avoided unless heavy bleeding occurred in the course of severe thrombocytopenia. If other forms of therapy are ineffective, plasmapheresis should be considered [11].

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