

Is there an increased incidence of subclinical proximal deep vein thrombosis after mild to moderate course of SARS-CoV-2 infection?

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This commentary refers to ‘Multi-organ assessment in mainly non-hospitalized individuals after SARS-CoV-2 infection: The Hamburg City Health Study COVID programme’ by E.L. Petersen et al., <https://doi.org/10.1093/eurheartj/ehab914> and the discussion piece ‘The everlasting challenge to identify deep vein thrombosis in both clinical practice and research’ by C.-A. Behrendt et al., <https://doi.org/10.1093/eurheartj/ehac164>

Early on in the 2019 coronavirus disease 2019 (COVID-19) pandemic outbreak, thromboembolic events were observed to be implicated in high lethality in hospitalized, critically ill patients.¹ In contrast, there are limited data on the incidence of deep vein thrombosis (DVT) and pulmonary embolism (PE) in the severe acute respiratory syndrome of coronavirus type 2 (SARS-CoV-2) infections following less serious disease courses. Petersen et al.² address the important issue of the potential organ damage, including subclinical thrombotic changes, caused by SARS-CoV-2 infection after a mild or moderate course. This is an important aspect and we are thankful for the focus on this problem. Nevertheless, we have some critical comments.

First, in the methods part, a partially or completely non-compressible vein was postulated, in the results part it says just ‘non-compressible’, which suggests complete obstruction of these veins. A complete obstruction of the common femoral vein (CFV) is usually not subclinal.³ Information on the frequency of partial and complete obstructions would be helpful in this regard. The examinations were moreover confined to the CFV at the level of the saphenofemoral junction. By contrast, the European Society for Vascular Surgery practical guideline on DVT, to which the authors refer, recommends examination of both the CFV and the popliteal vein, ideally including the course of the femoral vein throughout the thigh.³

Second, the observations on the non-compressibility of CFV and their underlying frequencies appear to relate to sub-collectives within both groups that are not clearly apparent from the tables and

supplemental data. Assuming the reported percentages from the text and brackets in Table 3, which are 43.2% in individuals after SARS-CoV-2 infection and 22.2% without prior SARS-CoV-2 infection, these ratios appear surprisingly high. Concerning the data for the uninfected, there is no reasonable relation to the published prevalence of thromboembolism; according to which about 1 out of 12 middle-aged adults will suffer a DVT or PE in the course of their further life.⁴ Also, the rate of 43.2% of non-compressible CFV due to recent thrombosis after COVID-19 would be quite a lot. If one considers that there are 7.5 million ‘cured’ people in Germany at the beginning of 2022, every 10th visitor to the office will be ‘cured’. Of these, over 40%, i.e. almost every second person, would then have experienced a thrombosis, which would correspond to 1 in 25 individuals. This dramatic increase in thrombosis would have been noticed by the phlebological community. Likewise, in a prospective observational study from Spain in which 233 stable COVID-19 patients were followed up by whole-leg compression ultrasonography 1 month after discharge, no DVT was found in any patient.⁵

Conflict of interest: none declared.

References

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