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Letter to the Editor Regarding "Pulmonary Embolism Prophylaxis in Patients With COVID-19: an Emerging Issue", Heart Lung Circ. 2021;30(10):1435–41.

To the Editor,

I read the article by Sanidas and colleagues [1] with great interest and congratulate the authors on their excellent work. However, I would like to draw attention to several points. Recent findings suggest that SARS-CoV-2 controls the expression of host regulatory molecules such as miRNAs and transcription factors (TFs), often regulating gene expression at transcriptional and post-transcriptional levels and forming highly complex network interactions [2]. Research has identified four miRNAs (hsa-mir-9-5p, hsa-mir-324-3p, hsa-mir-1827, and hsa-mir-1277-5p) and TFs (EP300, MYC, E2F1, and SP1) that co-regulate important target genes responsible for SARS-CoV-2 host invasion [2]. Studies have found evidence supporting common molecular trajectories of SARS-CoV-2 with clotting, pulmonary embolism, pulmonary oedema, and systematic inflammation, showing both fibrinolysis as a major pathway leading to D-dimer increase and cytokines TGF-β and TNF-α as major regulators of fibrinolysis controlling proteins PAI-1 and plasminogen activators also highlighting the role of hsa-mir-9-5p, hsa-mir-324-3p, EP300, and SP1 on pulmonary embolism [2]. It has also been reported that in the circulating exosomes of high D-dimer COVID-19 patients (cut-off 3 μg/mL) there is significant miR-424 upregulation and miR-103a, miR-145, and miR-885 downregulation. Exosomal miR-424 is an independent thromboembolic event predictor in COVID-19 patients [3,4], whereas miR-103a independently regulates D-dimer levels [4]. Furthermore, miR-885 targets von Willebrand factor and miR-145 targets tissue factor [3,4], showing negative correlations with their respective targets [3]. The findings of Sanidas and colleagues [1] add significant information to previously published data; however, also evaluating these aspects would be useful for better understanding of the interplay between pulmonary embolism in patients with COVID-19 and its complex regulatory network.

Conflict of Interest

None declared.

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References

[1] Sanidas E, Grassos C, Papadopoulos D, Velliou M, Barbetseas J. Pulmonary embolism prophylaxis in patients with COVID-19: an emerging issue. Heart Lung Circ. 2021;30(10):1435–41.
[2] Khurana P, Gupta A, Sugadev R, Sharma YK, Varshney R, Ganju L. nSARS-Cov-2, pulmonary edema and thrombosis: possible molecular insights using miRNA-gene circuits in regulatory networks. ExRNA. 2020;2(1):16.
[3] Plowman T, Lagos D. Non-coding RNAs in COVID-19: emerging insights and current questions. Non-coding RNA. 2021;7(3):54.
[4] Gambardella J, Sardu C, Morelli MB, Messina V, Marfella R, Maggi P. Exosomal microRNAs drive thromboembolism in COVID-19. Circulation. 2020;142(Suppl_4):A221.

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