



Is There a Relationship Between Olfactory Dysfunction and Decreased Thromboembolic Events After the First Wave of the COVID-19 Pandemic?

Ercan Akşit¹ Ahmet Köder²

¹Department of Cardiology, Çanakkale Onsekiz Mart University, Faculty of Medicine, Çanakkale, Turkey

²Department of Otorhinolaryngology, Trakya University, Faculty of Medicine, Edirne, Turkey

To the Editor,

The Coronavirus disease-2019 (COVID-19) has caused a global pandemic.¹ A recent study suggested that the prevalence of cardiomyocyte injury and microvascular thrombogenicity was lower in the second wave of the COVID-19 pandemic compared with the first wave.² The importance of agents such as ticagrelor for reducing thromboembolic events in COVID-19 has also been noted.³ A recent study suggested that the *UGT2A1/UGT2A2* genes are responsible for the olfactory dysfunction (OD) attributed to COVID-19.⁴ Interestingly, *UGT2A1* polymorphisms are known to be associated with platelet reactivity.⁵ Before the onset of the COVID-19 pandemic, the association between OD and cardiovascular diseases (CVDs) was a topic of interest,⁶ and in a previously published article, we speculated that OD could predict cardiocerebral syndrome.⁷ In the early months of the pandemic, COVID-19 was associated with a significantly higher incidence of OD and increased thromboembolic events compared with other flu infections.² We noticed that both OD and thromboembolic events decreased in the later waves of COVID-19. This is a crucial hypothesis that has not been tested. CVDs are still the leading cause of death, even in early stages of life.⁸ Increasing evidence supports the relationship between OD and cardiovascular events (e.g., OD in heart failure patients) and thromboembolic events (e.g., *UGT2A1* polymorphisms as the responsible partner in different studies). Considering this perspective, it may be important that primary and secondary preventive measures against CVDs are implemented for patients with OD to reduce the long-term effects of endothelial damage in the post-COVID-19 syndrome. Additionally, despite the COVID-19 pandemic, conducting studies examining the

relationships between OD and CVDs may help in the development of new diagnostic and treatment methods against CVDs.

Informed Consent: Informed consent was obtained from the patient.

Authorship Contributions: Concept- E.A., A.K.; Design- E.A., A.K.; Analysis or Interpretation- E.A., A.K.; Data Collection and/or Processing- E.A., A.K.; Literature Review- E.A., A.K.; Writing- E.A., A.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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Corresponding author: Ercan Akşit, Department of Cardiology, Çanakkale Onsekiz Mart University, Faculty of Medicine, Çanakkale, Turkey
e-mail: ercanaksit@comu.edu.tr

Received: March 05, 2023 Accepted: March 13, 2023 Available Online Date: May 08, 2023 • DOI: 10.4274/balkanmedj.galenos.2023.2023-3-15

Available at www.balkanmedicaljournal.org

ORCID iDs of the authors: E.A. 0000-0002-4478-4324; A.K. 0000-0003-4348-8109.

Cite this article as:

Akşit E, Köder A. Is There a Relationship Between Olfactory Dysfunction and Decreased Thromboembolic Events After the First Wave of the COVID-19 Pandemic?. *Balkan Med J.*; 2023; 40(3):228.

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