



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

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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.

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