

Hemorrhagic gastropathy acute and epistaxis, rural areas clinical markers of SARS-CoV-2 variant B.1617.2 (delta)

Gastropatía aguda hemorrágica y epistaxis, marcadores clínicos en el medio rural de la variante B.1617.2 (delta) del SARS-CoV-2

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To the editors,

The articles by Ramírez-García et al.¹, Carrillo-Esper et al.² and Parra-Romero et al.³ are basic for approaching COVID-19 coagulopathy and vascular disease, specially in the context of the B.1617.2 (delta) variant⁴, due to its probable relationship with hemorrhological disorders. In this regard, cerebrovascular disease (CVD) and thrombocytopenia have been recently reported in patients with COVID-19. We detected three cases with the delta variant, whose ages were 71, 86 and 96 years old, with acute hemorrhagic gastropathy, negative genomic PCR for *Helicobacter pylori*, with thrombocytopenia, low serum values of Von Willebrand factor, D-dimer, ferritin, and interleukin 6, and without thrombophilia genetic factors. In addition, epistaxis without any other etiology was observed in 72 out of 76 new cases in youths (6-25 years old) with the delta variant in Sierra Sur de Oaxaca, from January to July 2021. On the other hand, in the clinical pathology laboratory, of a total of 45 samples

analyzed with multiplex qRT-PCR, from patients positive for the COVID-19 delta variant, 42 corresponded to epistaxis cases. These data could be useful as clinical markers for the early clinical diagnosis.

Based on the foregoing, we consider the assessment of thrombophilia molecular markers to be of utmost importance, given their relevance in anticoagulation. It is suggested that some genotypes of the delta variant produce an acute hemorrhagic macroangiopathy associated with thrombasthenia and thrombocytopenia, while the other SARS-CoV-2 strains produce acute thrombotic inflammatory microangiopathy. These findings must be verified in massive replication studies of genomic surveillance by massive sequencing.

The platelet function disorder associated with the delta variant may be due to a defect in the aggregation or adherence of platelets, both among themselves and to the vascular endothelium, as some of the virus variants may have greater affinity for platelet glycoproteins IIa/IIb and IIb/IIIa⁵.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were carried out on humans or animals for this research.

Data confidentiality. The authors declare that they have followed the protocols of their work center regarding the publication of patient data.

Right to privacy and informed consent. The authors have obtained the informed consent of the patients and/or subjects referred to in the article. This document is in the possession of the corresponding author.

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