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【 LETTERS TO THE EDITOR 】

Detailed Pathophysiology of Ischemic Colitis Following Plasma Donation

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To the Editor We have read with great interest a case report written by Mizumura et al. (1), showing that the lack of a transient colonic blood supply might be a key factor in the onset of ischemic colitis. Two concerns should improve the quality of their report.

First concern is whether such a transient (only 10 seconds) colonic hypo-perfusion would cause ischemic colitis. In *in vivo* animal models of experimental transient clamping of the colonic artery, a 10-second hypo-perfusion had little impact on the morphofunctional derangement (2). We do not have any data of the accurate duration and magnitude of the hypo-perfusion required to cause such an onset of ischemic colitis.

Instead, thrombophilia (i.e., a deficiency of protein C, protein S, and anti-thrombin III) might be associated with ischemic colitis. During the plasma donation, blood is drawn from a body and then is processed through a machine that separates the plasma, followed by the return of residual blood component. Concentrations of the anticoagulant factors including protein C, protein S, and anti-thrombin III might be reduced following such plasma donation (3). The ensuing reduced body blood might induce further hypercoagulability.

In this case, the patient may have demonstrated an anticoagulant factor deficiency at baseline, and therefore ischemic colitis may have been triggered by both plasma donation and transient colonic hypo-perfusion.

In younger patients without any systemic atherosclerosis, which is a major cause of ischemic colitis in the elderly cohort, it might therefore be recommended to make a detailed investigation of the patient's coagulation state as well as carefully evaluate the influence of any concurrent medications in order to identify the etiology of ischemic colitis.

The authors state that they have no Conflict of Interest (COI).

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