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Letter to Editor

Are we Sufficiently Concerned about the Potential Role of Antibiotics as a Stroke Trigger?

Debabrata Chakraborty[1*]
[1]64/4A/9, Beliaghata Main Road, Kolkata, West Bengal, India

***Corresponding Author:** Debabrata Chakraborty
64/4A/9, Beliaghata Main Road, Kolkata, West Bengal, India

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Dear Editor,

We need to use antibiotics commonly in patients having recent strokes. Have we considered the potential impact of these antibiotics on either the current stroke or any future events? Some of our commonly used antibiotics can cause coagulopathy and increase risk of bleeding by a number of ways : piperacillin/tazobactam (coagulopathy through a vitamin K-dependent mechanism and thrombocytopenia), ampicillin/sulbactam (alter the intestinal bacteria, which can lead to vitamin K deficiency and coagulopathy), tigecycline (alter the synthesis of clotting factors), cephalosporins with 1-N-methyl-5-thiotetrazole (NMTT) or 2-methyl-1,3,4-thiadiazole (MTD) side groups (inhibition of vitamin K function).[1] Besides this, any long-term antibiotic therapy can also lead to vitamin K deficiency and coagulation dysfunction secondary to dysbiosis of the intestinal flora and abnormal liver function.[1] When treating hemorrhagic strokes, strokes with hemorrhagic conversion, or cardioembolic strokes on anticoagulants, we must exercise caution when using antibiotics, as they may exacerbate coagulation abnormalities. These antibiotics may precipitate hemorrhage (both intracranial and systemic) especially in elderly patients of Asian origin (increased bleeding risk), those with poor nutrition, malabsorptive conditions, hematological disorders, hepatic/renal dysfunction, or those with underlying malignancy. Therefore, when taking broad-spectrum antibiotics, we should monitor patients with pre-existing vitamin K deficiency or decreased absorption of vitamin K for coagulopathy. Nowadays, most patients take direct oral anticoagulants when indicated, eliminating the need for routine coagulation parameter checks. However, these are situations where patients on direct oral anticoagulants (DOAC) and antibiotics require close monitoring of coagulation parameters. In fact, we may need to provide vitamin K prophylaxis to prevent hypoprothrombinaemia along with antibiotics in life-threatening sepsis when the bleeding risk is high [1].

Antibiotics can precipitate thrombosis by promoting the proliferation and activity of macrophages, thereby leading to the accumulation of lipids in the vessel walls. Broad-spectrum antibiotics impact the gut microbiota's makeup and abundance; the latter may contribute to atherosclerotic vascular disease and hyperactivity of platelets [2]. These antibiotics cause disruption of the microbiota and contribute to increased adiposity by inducing inflammation secondary to modification of metabolic signals [2]. Further studies, such as the use of G89 (Limosilactobacillus reuteri) and G133 (Lacticaseibacillus paracasei) as supplements to counteract gut microbiota disruption caused by antibiotics, are necessary to combat strokes [3]. Thus, certain antibiotics have the potential to trigger thrombotic and hemorrhagic events, necessitating careful selection of antibiotics in susceptible individuals, monitoring coagulation parameters, and intervention at the earliest according to the clinical scenario.

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