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REPLY: Collapse of the Aspirin Empire

Is it Diabetic Gastroparesis or Cardioprotective Paresis?

Drs. Cerit and Duygu raise an interesting question of whether diabetic gastroparesis may play a role in apparent "aspirin resistance" in diabetic patients (1). It is possible that in some patients, diabetic gastroparesis impairs absorption of a number of enteric-coated (EC) drugs, including EC aspirin. Indeed, the incidence of gastroparesis in patients with diabetes may be as high as 30% to 40% (2). Because blood glucose is important in regulating gastric emptying, as well as other factors, it is possible that patients treated with EC aspirin have variable gastric emptying and a higher frequency of prolonged tablet retention in the stomach (3). With a greater gastric residence time, the amount of aspirin available to be dissolved and absorbed in the upper gastrointestinal tract may be lower after EC aspirin than after immediate-release forms. In patients with diabetic gastroparesis, the lower rate and extent of aspirin absorption could conceivably be magnified. It is also possible that patients with diabetic gastroparesis have more advanced diabetes, and are therefore more likely to have heightened platelet activity. This potential effect on platelets may then appear to manifest as "aspirin resistance" as well (4). However, our study did not explicitly evaluate the presence or absence of diabetic gastroparesis, though the 2 non-EC aspirin formulations studied did not appear to have any issues with impaired absorption (5). As such, more work is certainly needed on defining the optimal antiplatelet therapy in diabetic patients.

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