

tion, the authors mainly emphasized a switch to thienopyridine derivatives. A switch from clopidogrel to ticlopidine and no relapse in the aforementioned case (4) could be explained by different action mechanisms leading to TTP with clopidogrel and ticlopidine. ADAMTS-13 deficiency is common in ticlopidine-associated cases in contrast to ADAMTS-13 independence in clopidogrel-associated ones (5). Prasugrel-linked TTP cases are few, and the exact mechanism is not clearly identified. Our ticagrelor-linked TTP case was also the first one in literature, and its exact mechanism was also not established. Eventually, P2Y12 inhibition was not re-initiated, and fortunately, no ST or TTP relapse occurred.

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Author's Reply

To the Editor,

We thank the authors for their contribution to our study that was recently published in the *Anatolian Journal of Cardiology* 2017; 17: 73-4 entitled "Ticagrelor-associated thrombotic thrombocytopenic purpura" (1). Initially, we used biolimus-eluting stent during primary percutaneous coronary intervention in the patient. Although DAPT duration was reduced to at least 6 months in patients with stable coronary artery disease, DAPT duration of at least 12 months is still recommended in patients with ST elevation myocardial infarction (2). Numerous studies have indicated that second-generation stents have low stent thrombosis (ST) and major adverse cardiac events. A 3-month usage of these new stents in treatment has shown to be unrelated to increased ST rate (3). Even with these findings, we could not conclude whether ticagrelor cessation at 5 weeks of therapy in our case would not have caused ST. In addi-

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