Letter by Meves and Neubauer Regarding Article “Clinical Outcomes Using a Platelet Function-Guided Approach for Secondary Prevention in Patients With Ischemic Stroke or Transient Ischemic Attack”

To the Editor:

Depta et al.1 reported that platelet function-guided modification of antiplatelet therapy in patients with stroke may be associated with higher rates of adverse clinical outcomes. Platelet function testing was performed by using optical platelet aggregometry. The definition of antiplatelet modification was any increase of the current antiplatelet dosage, the addition of another antiplatelet agent, or the switch to another antiplatelet agent.

There are some remarks concerning the methodology and data of the presented study. At first, aspirin low response was defined as altered aggregation after stimulation with arachidonic acid and with adenosine diphosphate. Unfortunately, adenosine diphosphate is unspecific in regard to the aspirin pathway and is not recommended for measuring platelet inhibitory effects of aspirin.2 Therefore, a distinction should have been drawn between these nonresponders due to the unspecific response to adenosine diphosphate stimulation in contrast to the low response to arachidonic acid stimulation, which is the recommended test.

Second, compliance is a major issue for the response to antiplatelet therapy. Because patients with antiplatelet therapy modification were older and significantly more often treated with various antihypertensives than those without antiplatelet therapy modification, measurements of serum thromboxane B2 would have been necessary to rule out bias due to compliance problems.

Third, much important demographic information on the study cohort is missing such as laboratory values and the aspirin dosages before and after therapy modification. Additionally, one third of their patients (n=129) were on dual therapy with aspirin and clopidogrel, which is not comprised in standard guideline recommendations. Several questions arise such as why dual antiplatelet therapy was prescribed, how many of the dually treated patients were single nonresponders, which drug was then modified, and what were the event rates in this specific group. Another drawback was that there were no time intervals between event and measurement. Probably low response to antiplatelet therapy was only temporary in nature in the acute clinical setting of an ischemia due to platelet hyperreactivity.3 Furthermore, only 24 of all patients were retested after therapy modification. Without control testing after therapy modification, it is unclear from the data provided whether the patients being modified were only transferred from one “ineffective” antiplatelet regimen to another without actually being optimized.

One last concern is the inclusion of patients with transient ischemic attack. The correct diagnosis of transient ischemic attack can be difficult even in a clinical setting for a number of reasons such as the advanced age of the patients or being treated by a non specialist for stroke medicine.4 The diagnosis of transient ischemic attack in this study has to be considered carefully because the data were only provided by the electronic medical record system and/or paper charts, which are prone to misdiagnoses.

Thus, we would handle the authors’ results and conclusion with caution. Evaluation of the effectiveness and safety of modification of antiplatelets on the basis of platelet function testing requires a detailed approach including such aspects as the method, time point of measurements, control measurement results as well as detailed patient demographic characteristics. We welcome the authors’ recommendation to determine the clinical value of platelet function-guided optimization of antiplatelet therapy in a randomized trial.

Disclosures

None.


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