Letter by Altman and Gonzalez Regarding Article, “Duration of Treatment With Nonsteroidal Anti-Inflammatory Drugs and Impact on Risk of Death and Recurrent Myocardial Infarction in Patients With Prior Myocardial Infarction: A Nationwide Cohort Study”

To the Editor:

We read with great interest the article by Schjerning Olsen et al.¹ reporting the recurrence of myocardial infarction after the use of nonsteroidal anti-inflammatory drugs (NSAIDs) in patients with established cardiovascular disease. Although the presented data are not new,² ³ they are of relevance for clinicians working in the real world. The mechanism underlying the prothrombotic effect of NSAIDs remains poorly understood, but it is feasible that NSAIDs would induce an imbalance between cyclooxygenase (COX)-2–derived prostacyclin and the COX-1–derived thromboxane. These 2 prostanoids provide an important balance in platelet behavior: Prostacyclin counteracts the effect of thromboxane. By decreasing levels of prostacyclin without, at the same time, altering prothrombotic thromboxane levels, selective COX-2 inhibitors⁴ may increase platelet reactivity and could exert prothrombotic effects determining the pathophysiology of the recurrent events. Aspirin decreases both COX-1–derived thromboxane and prostacyclin, preventing this imbalance. In the Schjerning Olsen et al study, 50.9% of the patients in the whole NSAIDs group and between 38.4% and 48.2% of those in the exposed groups also received aspirin. In a clinical trial, we have previously shown a reduction of events in patients with acute coronary syndromes without ST elevation treated with aspirin on top of the preferential COX-2 inhibitor meloxicam 90 days after the coronary episodes.⁵ The concomitant use of aspirin may be of relevance for lowering coronary risk in patients being treated with NSAIDs. As a consequence, it would be of great interest to know whether patients with aspirin on top of any NSAIDs were protected from myocardial infarction. Although not included in the table of the Schjerning Olsen article, it is possible to suppose that some patients were prescribed NSAIDs because of rheumatoid arthritis, a well-known risk factor for myocardial infarction, and for other inflammatory diseases with an impact on cardiovascular morbidity, as well. It would be of relevance to consider the data of groups with and without aspirin presented in a separate manner.

Disclosures

None.

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References

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