Correspondence

Letter by Nammas Regarding Article, “Should We Recommend Oral Anticoagulation Therapy in Patients With Atrial Fibrillation Undergoing Coronary Artery Stenting With a High HAS-BLED Bleeding Risk Score?”

To the Editor:

With full interest, we read the article “Should we recommend oral anticoagulation therapy in patients with atrial fibrillation undergoing coronary artery stenting with a high HAS-BLED bleeding risk score?” by Ruiz-Nodar et al.1 In fact, I commend the authors for conducting this highly interesting piece of research using the newly pioneered HAS-BLED risk score for estimation of bleeding risk in patients with atrial fibrillation undergoing percutaneous coronary intervention. Evidently, the late-breaking innovation of the HAS-BLED bleeding risk score has paved the way for a long-awaited new insight to patients who need long-term oral anticoagulation (OAC) therapy.2 No wonder then that, very shortly after its introduction, its assessment has been recommended in patients before prescribing antithrombotic therapy by both the 2010 European Society of Cardiology and 2011 Canadian Cardiovascular Society guidelines for the management of atrial fibrillation.3,4 The authors concluded that “Most patients with atrial fibrillation undergoing percutaneous coronary intervention/stenting have a high risk for major bleeding (HAS-BLED score ≥3). Even in these patients, OAC improves prognosis in these patients (reduced mortality and major adverse cardiac events) with an increase in major bleeding.”1

I am deeply concerned about some methodologic aspects of the current study by Ruiz-Nodar et al. First, the study was conducted by retrospective analysis of patients enrolled during the period from January 2001 to March 2008, well before the first publication of the HAS-BLED bleeding risk score. This makes it very possible that some variables needed for the calculation of the score would be missing from the database at the time of data collection; for instance, liver function and bleeding predisposition. I wonder how labile international normalized ratio, a necessary component of the score, was assessed in all the patients. Second, as already acknowledged by the authors, the adoption of all-cause death, as a primary end point for the inclusion of target vessel failure, an end point not directly related to the benefit nor to the risk of OAC, in the composite end point of major adverse cardiac events, is further unjustified. Third, when studying patients with high bleeding risk (score ≥3), those who were prescribed OAC at discharge had an unexplained lower total mortality at 1-year follow-up as compared with those who were not (9.3 versus 20.1%, P<0.01). Clearly, the lower major adverse cardiac events in the former group (13.0 versus 26.4%, P<0.01) was chiefly driven by such a markedly lower mortality rate. Given that the 2 groups (with and without prescription of OAC) were not systematically matched, and considering the lack of information on the compliance of patients prescribed OAC over a 1-year period, the highly unpredictable response to warfarin OAC, and the many food and drug interactions, the 1-year mortality benefit seen in patients prescribed OAC at discharge cannot be safely attributed to an adequate anticoagulation benefit. Furthermore, the major bleeding rate was remarkably higher (hazard ratio 3.03, P=0.01).

Disclosures

None.

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References

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