How has the need to measure antiplatelet activity arisen?

First, we do routinely measure hematologic or physiological surrogates for other effective secondary prevention strategies. Specifically, international normalized ratio monitoring is used to determine warfarin dosage; blood pressure is the yardstick for titration of antihypertensive agents and glycemic control using HbA1c in diabetes. In these cases, clinical efficacy is usually related to these surrogates. Furthermore, a significant proportion of patients on antiplatelet therapy have clinical events, suggesting a variability of therapeutic effect. Hence, the concept of biological measurement of antiplatelet function is attractive to determine likely treatment responders.

Although the concept of measuring antiplatelet activity is appealing, there appears to be a disconnect between theory and reality. There are 2 issues of importance. These include the lack of a gold standard measure of antiplatelet functioning together with the paucity of evidence linking antiplatelet resistance and recurrent vascular events in a stroke population. A major concern with a number of these measures is poor agreement between different techniques for antiplatelet resistance and poor reproducibility over time. Indeed, these confounding factors are a major impediment in the use of these tests in prediction of future vascular events. Despite this, there is some evidence linking aspirin resistance to composite vascular outcomes of stroke, myocardial infarction, and vascular death. In addition, aspirin resistance is common, 30% prevalence in a stroke population.

So, is this avenue of research worth pursuing? We think it is. This would include better techniques with better evidence of reproducibility and correlations between the measures and stroke risk in large population cohorts. However, to address our original question, should antiplatelet activity be measured routinely, our answer at this stage is an unequivocal no.

Disclosures

None.

References


Key Words: antiplatelet RX antiplatelet secondary prevention ischemic stroke
Antiplatelet Activity
Stephen M. Davis and Geoffrey A. Donnan

Stroke. 2009;40:2275; originally published online April 30, 2009;
doi: 10.1161/STROKEAHA.108.537654
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/40/6/2275

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