Aspirin Resistance Not Tied to Metabolic Syndrome

BY PATRICE WENDLING
Chicago Bureau

The presence of metabolic syndrome did not affect aspirin nonresponsiveness in a study of 104 patients receiving chronic aspirin therapy.

There was no significant difference in aspirin nonresponsiveness, which was defined as platelet aggregation inhibition of less than 80%, among 41 patients with metabolic syndrome and 63 patients without metabolic syndrome (12 patients, or 29%, vs. 14, or 22%, respectively).

Baseline characteristics, including age (65 years vs. 64 years), male gender (30 vs. 40 patients), coronary artery disease (CAD) risk factors, past medical history, past smoking history, and concomitant medications, were similar between patients with metabolic syndrome and those without. All of the patients had documented CAD, Dr. Sotir Polena and colleagues at Lenox Hill Hospital in New York reported at the American Federation for Medical Research Southern Regional meeting in New Orleans.

Metabolic syndrome was defined according to the Adult Treatment Panel III criteria, which require the presence of any three of the following five traits—hyperglycemia, abdominal obesity, hypertension, hypertriglyceridemia, and reduced HDL cholesterol level. Among the metabolic syndrome group, 21 patients had more than four traits.

Most of the patients were on 325 mg/day of aspirin, although some were on 81 mg/day. "We did not find any difference on aggregation studies while comparing different doses," Dr. Polena said in an interview. "We do not evaluate routinely for aspirin nonresponsiveness; [we evaluate] only for research purposes, but in the near future we are planning on starting a routine evaluation for all the patients undergoing a percutaneous evaluation."

The findings are reassuring because metabolic syndrome is a well-established risk factor for CAD, and literature citations indicate aspirin resistance may occur in as little as 5% and as much as 45% of the population.

The discrepancies in prevalence are largely due to differences in the objectives of the tests, and their sensitivity and specificity to the evaluation of platelet function. In addition, the term "aspirin resistance" has been used clinically to describe several different physiological phenomena, Dr. Polena explained.

One definition is the inability of aspirin to protect patients from ischemic vascular events, though the term has also been used to describe aspirin's inability to produce anticipated effects on one or more platelet function tests, such as the inhibition of biosynthesis of thromboxane and the response to an agonist with light transmission aggregation (LTA) testing.

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Investigators at Oxford (England) University have shown that the agreement among the results of the platelet function analyzer (PFA-100), VerifyNow-ASA assays, and LTA testing remained poor among 72 patients still receiving low-dose aspirin therapy 1 year after first being tested, with only one patient identified as a nonresponder by all three tests (Platelets 2008;19:119-24).

A study of 191 patients with stable CAD who received secondary aspirin prophylaxis showed poor agreement among three different tests—Ivy bleeding time, collagen/epinephrine closure time, and urinary 11-dehydrothromboxane B_2 excretion levels, with only 3 patients identified as aspirin-resistant by all three tests (Thromb. Res. 2007;121:413-8).

In Dr. Polena's study, platelet aggregation inhibition was measured prior to elective catheterization by Plateletworks-ICHOR using arachidonic acid as an agonist. Helena Laboratories, Beaumont, Tex., markets the test and supplied the materials for the study.

"This platform was chosen based on the quantitative nature of the test system and also on the fact that it has been closely correlated with LTA, the gold standard of platelet aggregation tests," said Dr. Polena, who received no funding for the study and reported no conflicts of interest. "Correlation between both systems typically demonstrates an *r* value of more than 0.8." ■

Pages 16a—16b