

Is Aspirin Resistance Dependent on the Clinical Scenario?

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Background: Low response or "resistance" to the anti-platelet effects of aspirin has been associated with adverse clinical outcome. However, a wide range of aspirin resistance rates has been reported - from 5% to 45%. We hypothesized that part of the wide range may be related to the clinical setting in which patients (pts) were tested. For instance, percutaneous coronary intervention (PCI) can be associated with platelet hyper-reactivity and thus affect aspirin response. We, therefore, aimed to test the frequency of aspirin resistance in pts with stable coronary artery disease (CAD) vs. pts undergoing PCI.

Methods: We examined 2 cohorts of pts, both taking aspirin 75-150 mg for at least a week prior to enrollment. The first consisted of 485 pts with stable CAD (at least 6 months from any interventional procedure) and the other 150 pts undergoing non-urgent PCI (for various indications). Pts were tested for aspirin response using the VerifyNow Aspirin assay and platelet aggregation in response to arachidonic acid (AA) and ADP. Aspirin resistance was defined by at least 2 of 3 criteria: VerifyNow score \geq 550, 0.5 mg/ml AA-induced aggregation \geq 20%, and 10 μ M ADP-induced aggregation \geq 70%.

Results: The 2 cohorts had similar clinical characteristics, except for a higher proportion of women in the PCI cohort (31% vs. 17%, $P=0.0001$), a higher rate of hypertension in the PCI cohort (74% vs. 64%, $P=0.03$), but a higher rate of hyperlipidemia in the stable CAD cohort (89% vs. 71%, $P=0.0001$). Among pts undergoing PCI, 19 of the 150 (12.7%) had fulfilled the criteria for aspirin resistance vs. 30 out of 485 pts (6.2%) with stable CAD ($P=0.02$). VerifyNow Aspirin score was higher among the pts undergoing PCI than the pts with stable CAD (457.7 ± 81 vs. 433.3 ± 47 , $P=0.001$).

Conclusions: Pts undergoing PCI had a higher propensity to exhibit resistance to aspirin compared to pts with stable CAD. This difference may be attributed to platelet hyper-reactivity in the peri-PCI period