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Acute Coronary Syndromes

FACILITATION OF WHOLE BLOOD ANTI-PLATELET THERAPY MONITORING OVER EXTENDED SPECIMEN STORAGE TIME

ACC Moderated Poster Contributions
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Background: Dual anti-platelet therapy consisting of aspirin (ASA) and a P2Y12 inhibitor is a standard treatment to prevent thrombotic events in patients with acute coronary syndrome or myocardial infarction. However, over 50% of patients may be non-responsive and accurate monitoring using platelet function testing is limited to within 4 hours from sample draw. Here, we evaluated a novel Platelet Stabilizing Formulation (PSF) with the aim of extending the assessment of dual anti-platelet therapy beyond the recommended 4-hour window.

Methods: Whole blood (WB) from 5 subjects was collected into evacuated tubes containing citrate or PSF. WB impedance aggregometry (IA) was performed at 1 (baseline) and 24 hr of ambient storage using ADP high (hi, 20 µM) and low (lo, 1.3 µM) , collagen and TRAP agonists. To model in vivo pharmacologic P2Y12 receptor and cyclooxygenase blockade, blood was incubated with a P2Y12 inhibitor, 2-Methylthioadenosine 5′-monophosphate triethylammonium salt (2MeSAMP), and/or ASA before addition of the agonist.

Results: Aspirin alone inhibited initial aggregation in citrate and PSF-treated blood induced by collagen (58%) and loADP (47%), and showed a weak effect against hiADP (15%). When used alone, 2MeSAMP inhibited initial aggregation in citrate and PSF blood induced by loADP (~60%) and hiADP (~60%) but did not alter TRAP response (<10%). This in vitro antagonism of ADP and collagen response remained measurable in PSF for 24 hr, whereas aspirin or 2MeSAMP efficacy in citrate was undetectable due to reduced platelet aggregation (30% of baseline activity as compared to ~70% in PSF). At baseline, the additive effect of ASA and 2MeSAMP in response to ADP was 65% and 75% in citrate and PSF, respectively, but was preserved for 24 hr only in PSF-treated blood. Synergistic effects of ASA and 2MeSAMP on hiADP-induced aggregation were detected in PSF but not citrate blood.

Conclusion: Extended storage enabled by PSF may permit routine monitoring of anti-platelet therapies, thereby reducing the risk of thrombotic vascular events. Evaluation in patients undergoing anti-platelet therapy is needed to ascertain the clinical benefits of PSF for anti-platelet therapy functional analyses.