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Randomized Controlled Trial > Ann Pharmacother. 2008 Oct;42(10):1396-401.

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Comparison of oral aspirin versus topical applied methyl salicylate for platelet inhibition

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Abstract

Background: Oral acetylsalicylic acid (aspirin) is the primary antiplatelet therapy in the treatment of acute myocardial infarction and acute coronary syndrome. Methyl salicylate (MS; oil of wintergreen) is compounded into many over-the-counter antiinflammatory muscle preparations and has been shown to inhibit platelet aggregation locally and to be absorbed systemically.

Objective: To assess the ability of topically applied MS to inhibit systemic platelet aggregation for patients who are unable to tolerate oral drug therapy.

Methods: A randomized, prospective, blinded, crossover study was conducted in 9 healthy men, aged 30-46 years. All subjects ingested 162 mg of aspirin or applied 5 g of 30% MS preparation to their anterior thighs. There was a minimum 2-week washout period between study arms. Blood and urine were collected at baseline and at 6 hours. An aggregometer measured platelet aggregation over time against 5 standard concentrations of epinephrine, and a mean area under the curve (AUC) was calculated. Urinary metabolites of thromboxane B(2) were measured by a standard enzyme immunoassay. Differences in and between groups at baseline and 6 hours were tested by the Wilcoxon signed-rank test.

Results: Baseline platelet aggregation did not differ significantly between the 2 arms of the study (median AUC [% aggregation(*)min]; binominal confidence intervals): aspirin 183; 139 to 292 versus MS 197; 118 to 445 (p = 0.51). Both aspirin and MS produced statistically significant platelet inhibition; aspirin decreased the AUC from 183; 139 to 292 to 85; 48 to 128 (p = 0.008) and MS decreased the AUC from 197; 118 to 445 to 112; 88 to 306 (p = 0.011). No significant difference was detected between baseline and 6-hour thromboxane levels for either aspirin (p = 0.779) or MS (p = 0.327).

Conclusions: Topical MS and oral aspirin both significantly decrease platelet aggregation in healthy human volunteers.

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