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ASPIRIN SIGNIFICANTLY REDUCES EXCIMER LASER INDUCED PLATELET AGGREGATION

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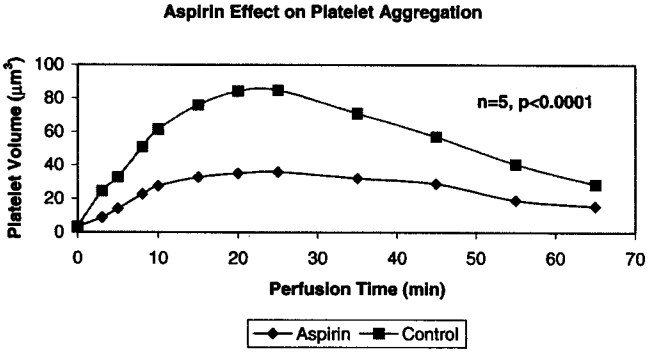
Background and Objective: Prior research demonstrated correlation between high energy excimer laser and platelet aggregation *in-vitro*. We evaluated the effectiveness of aspirin in reducing aggregation with high energy lasing.
Study Design/Materials and Methods: Platelet rich plasma obtained from 5 NZW rabbits was circulated in a dual chamber circuit. Aspirin (0.2 mg/ml) was added to one chamber, the other was control. Excimer laser (308 nm; 35 mJ; 25 Hz; 5 min) was delivered via a 2 mm diameter catheter in both chambers. Platelet aggregation (particle volume) was measured by laser light scattering and Coulter counter. Histology was evaluated by scanning and phase contrast microscopy.
Results: Baseline platelet volume was 3.4 μm^3 for both chambers. Platelet volume peaked at 25 min after lasing and was significantly lower with aspirin (84.8 ± 42.1 vs. 35.9 ± 45.5 μm^3; $p < 0.0001$). Histology demonstrated few small platelet aggregates with aspirin compared with control lased platelets. Aspirin reduced aggregation by 58%.
Conclusions: Aspirin given before lasing significantly reduces platelet aggregation by high energy excimer laser and is important during laser angioplasty.



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Q-SWITCHED Nd:YAG IN THE TREATMENT OF ARGYRIA

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Background and Objective: Argyria is a rare condition which results from prolonged contact with silver or silver ingestion. Cutaneous pigmentation associated with argyria is a clinical dilemma for which no satisfactory treatments have been described. We present a case of argyria successfully treated with the Q-switched Nd-YAG laser.
Study Design/Materials and Methods: We descibe a case of argyria in a young man secondary to colloidal silver ingestion. Though extensive areas of involvement were noted over the body, our initial treatment session was limited to affected areas over the face. The areas were treated with the Q-switched Nd-YAG laser using 4 mm, 6 mm, and 8 mm spot sizes at maximal fluences, and resulted in complete clearing.
Results: The areas treated demonstrated immediate clearing following a single pass with the Q-switched Nd-YAG laser. Side effects associated with treatment included significant pain during treatment, prominent edema, and transient erythema despite pre-treament with intramuscular kenalog, oral anti-histamines, and topical anesthetic.
Conclusions: The cutaneous manifestations of argyria can be effectively cleared with the use of the Q-switched Nd-YAG laser.

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IN VIVO IMAGING OF BIOMARKER EXPRESSION DURING ORAL CARCINOGENESIS

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Background and Objective: Oral carcinogenesis is a multi-step process in which genetic events cause disruption of the normal regulatory pathways controlling basic cellular function, multiple genetic events lead to oral cancer, and various cellular genes are over expressed in oral squamous cell carcinoma. Goal of these studies was to image and quantify expression of specific biomarkers known to play a role in the vascular and extracellular matrix (ECM) changes.
Study Design/Materials and Methods: In the hamster cheek model (10 hamsters) throughout carcinogenesis, in vivo multi-wavelength multi-photon (MPM) and second harmonic generated (SHG) fluorescence techniques were used to image surface and subsurface fluorescence prior to and after the injection of biomarkers: Vascular Endothelial Growth Factor (VEGF); urinase type Plasminogen Activator (uPA) and Inhibitor (PAI-1); Matrix Metalloproteinases 1,2,9 (MMPs). At 4,6,8,10, 12 weeks, one animal was sacrificed. Histopathological sections were prepared and pathology evaluated on a scale of 0-6.
Results: Carcinogenesis-related structural and vascular changes were clearly visible. Biomarker expression was clearly identified, localized and quantified at all time points throughout carcinogenesis, providing important new information on the carcinogenesis process.
Conclusion: Time- and spatially-resolved determination of (1) specific biomarker expression, (2) vascular and ECM changes throughout oral carcinogenesis provide valuable information on mechanisms of dysplastic and malignant change. Supported by: CRFA 30003, NIH (LAMMP) P41 RR01192, AFOSR FA 9550-04-1-0101.