Purpose/Relevance:

TML is widely used to reduce IOP but the mechanism of action, and the optimal treatment parameters, remain unclear. This study attempts to identify ways that the micropulse laser can affect IOP.

Methods:

Non-human Primate Eyes (NPE) eyes were obtained immediately after removal, 2 mm thick radial sections of the limbus prepared, maintained in a saline bath & oriented to allow TML delivery through the sclera to the CB. TML Joules were 0.75 (n=2), 1.50 (2), & 2.5 (1) joules (simulating clinically relevant energies). Microscope videography (MVG) was real time, HROCT both before and after, & histology after TML. Distance from scleral spur to probe was measured and analyzed by paired t-tests.

Results:

The TML focused at ~700 µm, near the interface of sclera and CB. The scleral surface facing the CB underwent shrinkage resulting in local SCS enlargement. (Fig.)
The TML focused at ~700 µm, near the interface of sclera and CB. The longitudinal muscle of the CB near the interface immediately shortened/contracted. At 0.75, 1.5 & 2.25 Joules, the scleral spur moved posteriorly by 27, 107 & 92 µm respectively; each differed significantly from baseline (p < 0.0001) TM moved inward & posteriorly with Schlemm’s canal (SC) enlargement. At the interface of the sclera and TM, microscopy & histology revealed both sclera and CB shrinkage further confirmed by newly developed OCT ability to image the entire CB thickness. Secretory epithelium (CBSE) damage was absent.

Discussion:

Absence of CBSE damage and CB changes confined to a localized area suggests that direct damage to the CBSE is an unlikely mechanism of action. Enlargement of the suprachoroidal space perhaps induces a uveoscleral flow increase. The scleral spur & TM motion similar to pilocarpine effects may also enhance outflow. New micropulse laser iterations are possible with different probe focal lengths, wavelengths, and duty cycles. Some combination may provide persistent improvement in aqueous outflow pathway dimensions. Systematic lab & clinical studies would be necessary to determine optimal parameters and limit risks.

Conclusion:

TML effects caused a Δ in sclera thickness, shrinkage of CB longitudinal muscle fibers, enlargement of SCS, posterior movement of SS, TM movement inward & posteriorly with SC enlargement. Absence of CBSE & limited CB damage suggest suprachoroidal space enlargement, uveoscleral flow increase and possible outflow pathway changes rather than CBSE effects as a primary mechanism of action.

References:


Category:
Surgery