Q-Switched Laser-Induced Chrysiasis Treated With Long-Pulsed Laser

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The Cutting Edge: Challenges in Medical and Surgical Therapeutics

**REPORT OF A CASE**

A 70-year-old woman presented for elective treatment of lentigines on her face. This was her first treatment with any laser. She was treated with a Q-switched alexandrite laser (755 nm, 50 nanoseconds, 3.5 J/cm², 15 pulses of 4-mm spot diameter; Candela, Wayland, Mass), causing what appeared to be usual purpura immediately following laser exposure of 8 tan macules. Several weeks later, blue-black discoloration was present, and was attributed to postinflammatory hyperpigmentation, but failed to lighten over the next 4 months.

Her medical history was significant for rheumatoid arthritis, treated with methotrexate and prednisone. Further questioning revealed that she had received a 3-year course of oral gold therapy 20 years prior. The total dosage taken could not be determined.

Four months following Q-switched alexandrite laser therapy, her examination revealed 8 blue macules without texture changes located at the laser treatment sites on her cheeks and forehead ranging from 2 to 6 mm (Figure 1). The lesions did not enhance on Wood's light examination, suggesting dermal pigmentation. In some areas, portions of the original lentigo were still visible in the center of the blue macule. There was no discoloration of the sclera, nails, or hair. A subtle blue-gray hue in the patient's general skin color was noted at the time of reexamination.

A punch biopsy specimen of a representative area demonstrated numerous black particles within macrophages in the dermis. A diagnosis of Q-switched laser-induced chrysiasis was made.

Initially, an attempt was made to clear the hyperpigmentation using the same Q-switched laser, a technique that can often lighten or clear laser-induced cosmetic tattoo darkening. A test spot was created on the inner aspect of the right arm with the original Q-switched alexandrite laser, and the hyperpigmented macule was pulsed again with the same laser. While the center of the lesion did show some clearing, a new rim of blue hyperpigmentation was induced around the treated spot. Further treatment with a Q-switched alexandrite laser was therefore not pursued, because blue pigmentation would always be induced at the border of the treated area. Surgical excision was considered but not pursued, given the number and location of the lesions.

**THERAPEUTIC CHALLENGE**

Chrysiasis refers to the effects of gold in tissues, in particular the skin. There is no known effective treatment for either generalized or local chrysiasis. Gold salts are widely used for the treatment of rheumatoid arthritis and are administered in both oral and intramuscular formulations. All show a propensity to accumulate in tissues and are particularly concentrated in the reticuloendothelial system, but they are also present in the skin, cornea, and lens. Typically, patients present with an irreversible blue to slate-gray discoloration affecting sun-exposed areas on the face, neck, and dorsal aspect of the hands. The cutaneous features often present insidiously, months to years after the ingestion of gold so that the association is not always obvious. In the skin, the deposition of gold occurs in the re-
ticular and papillary dermis in a perivascular pattern. Electron microscopy demonstrates electron-dense areas called aurosomes within macrophage lysosomes.3,5

The mechanism for the hyperpigmentation is not fully understood, but there is a clear association of disease severity with total cumulative dosage of gold. There is also a strong association with ultraviolet exposure.6,7 While the gold is deposited in both sun-exposed and non–sun-exposed skin,7–9 the hyperpigmentation occurs in areas exposed to sunlight. Hyperpigmentation in an area normally protected from sun has been induced with experimental ultraviolet exposure.7 It is thought that gold-associated melanogenesis and physiochemical changes in gold structure within the skin account for these changes.6–8

Localized forms of chrysiasis have also been reported in association with implanted gold-plated needles,10 and more recently after laser treatment with a Q-switched ruby laser in a patient receiving parenteral gold therapy.8 The deposits were examined with transmission electron microscopy and confirmed to be gold. There is no effective treatment for chrysiasis. Our challenge was to find a treatment that could remove the chrysiasis pigmentation, without inducing further pigmentation at the margins of the treatment field.

**SOLUTION**

**SPECTRAL ANALYSIS**

We hypothesized that the laser-induced chrysiasis pig- ment might be removed by a combination of wavelength and pulse duration, which did not induce pig- mentation in surrounding skin. As a first step, we characterized the pigment absorption in her skin using reflectance spectrophotometry, to define the wave- length region in which the darkened skin absorbed substantially more light than adjacent normal skin. An integrating sphere reflectance spectrophotometer (model 5270; Beckman Instruments, Fullerton, Calif) was used to measure the diffuse reflectance spectrum from 400- to 1200-nm of a darkened chrysiasis macule on the face and of an adjacent area of normal-appearing skin. Subtraction of the 2 reflectance curves yielded a difference spectrum (Figure 2). The difference spectrum has a broad band between about 550 nm and 850 nm, consistent with causing a bluish skin hue. The wavelength region near 700 nm was chosen for subsequent testing.

As a second step, we tested how laser-induced chrysiasis depends on pulse duration. This was done by exposing normal-appearing skin on her right inner, up- per arm to lasers with widely different pulse durations and irradiances, at nearby wavelengths within the chrysiasis pigment absorption band. A long-pulsed ruby laser (694 nm, 3 milliseconds; Epilaser, Palomar Medical Products, Burlington, Mass), a coaxial flashlamp-pumped dye laser (680 nm, 0.3 microsecond; Candela), and a Q-switched ruby laser (694 nm, nominally 30 nanosec- onds; Spectrum, Lexington, Mass) were used. Immediate bluish hyperpigmentation was induced at low fluences with all but the normal mode ruby laser (Table). Even at the highest fluence of 50 J/cm², skin response to the 3-mili- second ruby laser consisted of mild erythema appear- ing a few minutes after exposure, with no skin darken- ing. Next, the test sites on her arm with laser-induced chrysiasis were treated with the long-pulsed ruby laser, which resulted in complete or substantial clearing.

**TREATMENT**

The original chrysiasis lesions on her face were then treated using the long-pulsed ruby laser at a fluence of 35 J/cm² with a 10-mm spot size and chilled tip (4°C). Two treatments were performed given about 1 month apart. Two months after the second treatment, the blue macules had resolved almost completely without induction of any new pigmentation (Figure 3).

**COMMENT**

We present the first successful treatment of chrysiasis of the skin. Previous reports have established that development of localized chrysiasis is a risk for any patient with a history of gold intake in sun-exposed skin or af- ter Q-switched laser therapy.8 Surgical excision was previously the only option for removing discrete lesions. We have shown that a normal mode ruby laser can effect- ively clear the hyperpigmentation and, more important, does so without inducing new hyperpigmentation at the periphery of the treated area.

Based on our observations, laser-induced chrysiasis in patients treated with gold is primarily an irradiance- dependent rather than fluence-dependent phenomenon (Table). The irradiance (power delivered per unit area,
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