It is well-known that local and systemic side effects due to intralesional corticosteroid injections are common. We report the case of a 28-year-old woman with cutaneous linear atrophy along the abductor pollicis longus tendon, which appeared after an injection of intralesional corticosteroid in the treatment of de Quervain tendonitis.

Case Report
A 28-year-old woman was referred to our dermatology department for disfiguring “white dimples” on her right arm. These white dimples had appeared 4 months earlier and had since increased in size. The patient’s only significant history was a single intralesional corticosteroid injection of triamcinolone 40 mg/mL in the treatment of de Quervain tendonitis. Four months after the injection, the white dimples appeared on her right arm. Findings of the dermatologic examination revealed white and linear atrophic lesions on the flexor aspect of her right forearm.

Results of a biopsy of the affected skin revealed atrophic changes in both the epidermis and the dermis. There was no increase in size, and, within 2 months, had started to heal without specific treatment.

Comment
De Quervain tendonitis is the inflammation of the common tendon sheaths of the extensor pollicis brevis and abductor pollicis longus. The primary focus of the treatment is the reduction of inflammation. The following methods can be used in the treatment of this disease: in severe cases, splinting or nonsteroidal anti-inflammatory drugs and local steroid injection into the tendon sheath can be used; in severe refractory cases, surgical release of the tendon sheaths is considered.

Intralesional corticosteroid injections have been used in rheumatologic disorders. Major complications of intralesional injections in these conditions are septic arthritis or synovitis, atypical mycobacterial infection, skin and periarticular soft tissue linear atrophy, tendon rupture, allergic contact dermatitis, hypersensitivity reactions, granulomatous reactions, and adrenal suppression.
Triamcinolone preparations have been known to produce dermal atrophy. The precise cause of the dermal atrophy produced by corticosteroid is unknown. Several mechanisms have been considered. The most accepted mechanism is the lymphatic spread of the corticosteroid suspension and resultant atrophy of the dermal and epidermal tissues. Another proposed mechanism is the corticosteroid-induced vasoconstriction, especially when administered locally in high concentrations. This enhanced vasoconstriction may facilitate local thrombosis or embolization and capillary closure with resultant local tissue hypoxia. These effects then produce local tissue atrophy, or, in extreme cases, necrosis.

A review of the literature revealed many cases of atrophy following local injections of corticosteroid in the treatment of different lesions. A specific pattern of perilesional, streaklike linear atrophy or hypopigmentation, or both, after intralesional or intra-articular corticosteroid injection is rare. Chodoroff et al., however, reported a case of perilesional atrophy and hypopigmentation after intralesional corticosteroid injection in the treatment of de Quervain tendonitis.

Linear atrophy has been reported after single or multiple injections and can appear several weeks to months later. In our patient, linear atrophy appeared 4 months after a single repository corticosteroid injection. There is a considerably increased risk for producing atrophy when a 40 mg/mL injection of triamcinolone is administered rather than 10 mg/mL. Within 14 months, spontaneous resolution is typical, though some lesions have persisted longer. We learned from the patient's history that a 40 mg/mL injection of triamcinolone had been administered and that the lesions healed spontaneously in 2 months without treatment.

In conclusion, there is a considerably increased risk for producing dermal linear atrophy when injections of corticosteroids in high concentrations are used.

REFERENCES