A Case of Isolated Morphea Secondary to Silicone Breast Implantation

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Figure 1. An indurated, waxy-yellow plaque with a hyperpigmented border overlying a mastectomy scar from 10 years prior.

A 65-year-old female with bilateral silicone breast implants presented with a one-year history of an intermittently pruritic plaque on her left breast. The implants were placed ten years ago following a bilateral mastectomy secondary to breast carcinoma. She denied systemic symptoms, similar lesions prior, or a history of connective tissue or autoimmune diseases. Physical exam revealed an indurated plaque with a yellow-waxy center and a hyperpigmented peripheral border surrounding the mastectomy scar on the left.
breast (Figure 1). No other sclerotic lesions or leaking from the implant were noted. Skin biopsy revealed diffuse dermal sclerosis and absent adnexal structures. It showed a superficial and deep perivascular lymphoplasmacytic infiltrate with a few superficial eosinophils. The overlying epidermis was mildly acanthotic and showed diffuse spongiosis as well as parakeratosis and serum in the horn (Figure 2). This was consistent with morphea. The patient was then treated with high-potency topical steroids without resolution. She is being seen by a breast surgeon for possible removal of the implant.
Morphea is an inflammatory skin disorder with poorly understood pathogenesis characterized by asymmetric sclerotic plaques that can extend into soft tissues. Active lesions present with erythema and induration and are often accompanied by pain and pruritis. Patients typically describe a “bruise-like” lesion that is often mistaken for an infectious process. As lesions become inactive, they develop hyperpigmented borders and a yellow-white color, causing sclerosis and atrophy of the skin.

Morphea has identical histologic findings to systemic sclerosis. This includes a perivascular and periadnexal infiltrate in active lesions, and thinning of the epidermis with loss of adnexal structures and dermal sclerosis occurs in inactive lesions. Systemic sclerosis is differentiated by the clinical findings of Raynaud’s phenomenon, sclerodactyly, digital pitting ulcers, and abnormal nail-fold capillaries. Additionally, antibodies associated with systemic sclerosis include RNA polymerase, topoisomerase, and centromere. Another essential differential to consider in patients with a history of breast cancer is carcinoma en cuirasse, a cause of skin thickening resulting from skin metastasis of various cancers including breast carcinoma.

Breast morphea following breast implants has been described in several case reports. In one case, localized breast morphea was described after a silicone implant ruptured, leading to a transcutaneous flow of the silicone gel. The morphea was around the area of transcutaneous flow, suggesting a relationship between contact with silicone gel and morphea. However, a meta-analysis in 2000 did not show a relationship between connective tissue diseases and silicone breast implants. In this analysis, the authors concluded that eliminating breast implants would likely not reduce the incidence of connective tissue diseases. However, morphea was not exclusively studied, and many cases did not have long-term follow-up. Treatment of morphea typically consists of moderately potent to potent topical steroids with improvement over a few months. However, inactive morphea is often resistant to treatment. This case highlights the importance of recognizing silicone breast implants as a potential cause for isolated and treatment-resistant morphea of the breast.

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**References:**