Post-Stripping Sclerodermiform Dermatitis

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Background: Cutaneous sclerosis, a process that results in hardening of the skin, is the hallmark of scleroderma and sclerodermoid disorders. Cutaneous sclerosis is usually classified as secondary or primary, depending on the presence or absence of underlying diseases. Primary cutaneous sclerosis is a feature of idiopathic inflammatory processes that are often associated with autoimmune disorders, whereas secondary cutaneous sclerosis arises in the context of many pathological processes of varying causes, including chronic graft-vs-host disease, defined metabolic or genetic disorders, and exposure to certain infectious organisms, drugs, or chemicals.

Observations: Three patients had localized cutaneous sclerosis overlying the site of a surgically removed (stripped) great saphenous vein. In all 3 patients, lesions were clinically characterized by multiple hypopigmented and indurated plaques distributed linearly along the path of the preexisting vein. Extensive history, physical examination, and diagnostic tests did not reveal known predisposing factors for cutaneous sclerosis.

Conclusions: Although the observed association of sclerodermiform dermatitis and venous stripping in these 3 patients does not imply a causal relationship, the absence of other identifiable predisposing factors and the striking linear distribution of the cutaneous lesions along the path of the preexisting vein are suggestive. This post-stripping sclerodermiform dermatitis may be a rare late complication of saphenous vein stripping.

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CTANEOUS sclerosis, often referred to as scleroderma, is a process that results in hardening of the skin. It is characterized clinically by induration, a loss of the skin’s capacity to wrinkle, a certain degree of epidermal atrophy, decreased mobility above the underlying tissue, and frequently a combination of hypopigmented and hyperpigmented patches. The atrophic and indurated phases are usually preceded by an inflammatory (erythematous and edematous) phase.

Cutaneous sclerosis can be primary (systemic sclerosis, localized sclerosis, or sclerosis as part of an overlap syndrome or eosinophilic fasciitis) or secondary. Secondary cutaneous sclerosis is known to arise in the context of many disease processes. These include chronic graft-vs-host disease, certain infectious processes (borreliosis), specific chemical (eg, toxic oil or vinyl chloride) or drug (eg, bleomycin sulfate, isoniazid, and sodium valproate) exposure, metabolic (eg, amyloidosis or diabetes mellitus) or genetic (eg, Rothmund-Thomson syndrome or progeria) disorders, mucinoses, certain malignant neoplasms such as breast carcinoma, radiation exposure, and repetitive trauma.

We saw 3 patients affected by cutaneous sclerosis that was curatorously distributed along the site of the great saphenous veins and associated with a history of prior surgical removal (stripping) of the great saphenous vein(s). To our knowledge, the association between venous stripping and cutaneous sclerosis overlying the site of the preexisting saphenous vein has not been reported to date. Venous stripping may, therefore, be a rare, previously unidentified, cause of secondary cutaneous sclerosis.

REPORT OF CASES

PATIENT 1

A 42-year-old woman with a history of varicose veins and long-standing vulvar lichen sclerosus et atrophicus (LSA) was seen 6 years after bilateral stripping of the great saphenous veins because of the appearance of a hypopigmented plaque in the left inguinal region. On examination, the plaque was well circumscribed, slightly in-
durated, ivory colored with an erythematous border, and associated with previously unidentified vulvar lesions. The last were asymptomatic but clinically and histologically typical of LSA. A complete clinical and laboratory workup revealed only an IgG titer (1:1024) positive for Borrelia burgdorferi. No IgM antibodies against B burgdorferi were detectable, and the results of both the culture and polymerase chain reaction analysis of a skin biopsy specimen were negative. Nevertheless, the patient was treated with ceftriaxone sodium (1 g/d intramuscularly for 14 days); despite this, the hypopigmented lesions became progressively indurated and extended linearly along the distribution of the left great saphenous vein down to the ankle (Figure 1). A biopsy specimen of 1 of the ivory-colored, well-circumscribed, and indurated plaques of the leg showed slight epidermal atrophy, superficial telangiectasias, and diffuse dermal sclerosis extending to the dermo-hypodermal junction. A perivascular and interstitial lymphocytic infiltrate was observed in the lower dermis. These histological features were suggestive of localized scleroderma or morphea.

Clinically and histologically, the poststripping linear sclerodermiform lesions of the leg associated with longstanding vulvar LSA strongly suggest linear LSA. The extensiveness of the cutaneous lesions, their linear distribution on the site of the stripped vein, and their appearance following venous stripping nevertheless suggest a causal relationship between the venous stripping and the sclerodermiform dermatitis.

PATIENT 2

A 62-year-old woman with a history of varicose veins was seen 2 years after bilateral stripping of the great saphenous veins, followed by the administration of 6 sessions of polidocanol (Aetoxysclerol) to sclerose residual small varicose veins. She presented with multiple hypopigmented and indurated plaques on the inner side of both thighs, with a strikingly linear distribution along the path of the great saphenous vein from the inguinal region to the knee, extending largely beyond the site of polidocanol small varicose vein sclerosis (Figure 2, A and B). The findings of a clinical and laboratory workup were completely normal, and on histological examination, the skin lesions were characterized by epidermal atrophy with vacuolar degeneration of the basal layer, diffuse sclerosis of the deep dermis, superficial lymphangiectasias, and the absence of adnexal structures (Figure 2, C).

These findings were consistent with a late stage of morphea. Although the sclerotherapy may have contributed to the development of these lesions, their linear distribution along the path of the stripped saphenous veins and beyond the site of polidocanol injections suggests an association with the process of stripping. Furthermore, morphea-like localized cutaneous sclerosis has not been reported to date as a complication of sclerotherapy.3

PATIENT 3

A 63-year-old man had a history of stripping of the left great saphenous vein 2 years before consultation, followed by a second stripping on the same leg 20 months later because of recurrence. On examination, he had several erythematous and tender panniculitis-like plaques in a retiform and linear pattern along the path of the left great saphenous vein from the knee downward. Within 3 months, the plaques became progressively indurated and slightly hypopigmented, and the erythema disappeared (Figure 3, A [arrows]). Investigations revealed...
only an isolated homogeneous antinuclear antibody titer of 1:320 in the absence of clinical signs or anomalous laboratory test results compatible with collagen vascular disease. Histological examination of a lesional skin specimen showed a deep perivascular lymphocytic infiltrate localized in the septa and the adipose lobules (Figure 3, B). The septa of the lower hypodermis were thickened by dense sclerosis extending to the muscular fascia. These features were consistent with morphea profunda.

Taken together, the skin lesions of this patient were most reminiscent of sclerodermiform panniculitis at the site of the stripped saphenous vein.

**COMMENT**

These 3 patients are unique in that they all presented clinically with linear sclerodermiform dermatitis overlying the path of a prior saphenous venectomy. Significantly, none
of these patients had any known identifiable predisposing factors, underlying collagen vascular disease, or active infectious disease (ie, *B burgdorferi* infection) (Table). Varicose veins are common in people older than 40 years, and surgical management is frequently required for severe or symptomatic varicosities. Complications related to this procedure are rare and seldom serious, the most frequent being ecchymoses and hematomas, telangiectasias, recurrent varices, hypesthesia or numbness, localized infections, cutaneous ulceration, and thromboembolic disease. In addition, several cases of erectile dysfunction and 1 of neuroma as a consequence of saphenous vein stripping have also been reported. Recently, Hruza and Hruza reported 2 cases of subacute spongiotic dermatitis associated with sensory peripheral neuropathy at the site of saphenous vein sampling for coronary bypass surgery (saphenous vein graft donor site dermatitis). This complication was clinically distinct from the one reported here, however, and in addition to being an early complication (within 2-5 months of coronary bypass surgery) of venous sampling, completely resolved. No report of cutaneous sclerosis or sclerodermiform dermatosis at the site of and subsequent to saphenous vein stripping has been published to date.

Although the observed association of sclerodermiform dermatitis and prior venous stripping in these 3 patients does not necessarily imply a causal relationship, the absence of another identifiable predisposing factor or cause and the strikingly linear distribution of the cutaneous lesions along the path of the stripped saphenous vein are suggestive. Furthermore, with follow-up (3 years), no collagen vascular disease was discovered, no lesions appeared in other locations, and all lesions remained localized to the site of venous stripping.

The mechanism of localized cutaneous sclerosis in our patients is unclear. By analogy, among the suggested causes of localized scleroderma, physical factors such as trauma and x-ray irradiation have been reported. Given the low frequency of sclerodermiform dermatitis following saphenous vein stripping, trauma cannot be considered a unique or major predisposing factor; other factors such as a personal or genetic predisposition, borreliosis, a predisposition to inappropriate wound remodeling, and inflammatory reactions following wounding must also be considered. In this context, patient 1 had bilateral saphenous vein stripping, and localized scleroderma developed only on the left leg, supporting the suggestion that physical factors such as trauma are not the unique predisposing factor. Furthermore, patient 1 did have preexisting vulvar LSA, which may have been a predisposing factor and in which trauma could

### Clinical and Histological Characteristics of 3 Patients With Sclerodermiform Dermatitis After Great Saphenous Vein Stripping

<table>
<thead>
<tr>
<th>Patient No./Age, y</th>
<th>Time From Stripping to Skin Lesions, y</th>
<th>Lesion Type/Lesion Location</th>
<th>Histological Diagnosis</th>
<th>Coexistent Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/42</td>
<td>6</td>
<td>LSA-like/both GSV</td>
<td>Morphea</td>
<td>Vulvar LSA</td>
</tr>
<tr>
<td>2/F/62</td>
<td>2</td>
<td>Morphea-like/both GSV</td>
<td>Morphea</td>
<td>None</td>
</tr>
<tr>
<td>3/M/63</td>
<td>2</td>
<td>Sclerodermiform panniculitis/left GSV</td>
<td>Morphea profunda</td>
<td>None</td>
</tr>
</tbody>
</table>

*LSA indicates *lichen sclerosus et atrophicus; GSV, great saphenous vein.
have contributed to the development of linear lesions along the path of the great saphenous vein (Koebner phenomenon). The lesions on the leg, however, were histologically diagnosed as localized scleroderma or morphea and not cutaneous LSA. The Koebner phenomenon has also been reported for morphea, but its occurrence is exceptional, and the long delay (6 years) between the stripping and the appearance of the skin lesions suggests that they are unlikely to be due solely to the Koebner phenomenon. Although controversial, morphea and LSA have in some patients been related to European genotypes of *Borrelia* species, and although appropriate antibiotic therapy in the context of serologic tests positive for *Borrelia* IgG did not modify the cutaneous lesions in patient 1, the contribution of this pathogen to the lesional process cannot be excluded.

Because venous stripping damages nerves and components of the local microvasculature, in addition to the skin and subcutaneous tissue, the cutaneous sclerosis observed may be somehow related to these damages. Among the currently proposed mechanisms for the development of fibrosis, recent evidence supports the role of low tissue oxygen tension as a stimulus for transforming growth factor β and collagen synthesis. Transforming growth factor β is a potent mediator of connective tissue synthesis and has been detected in the lesional tissue of localized scleroderma, suggesting that under certain conditions, decreased oxygenation may favor molecular processes that lead to localized cutaneous sclerosis.

The dermatosis in these 3 patients was unique because it developed linearly at the site of venous stripping, which suggests that it is somehow related to the surgical procedure. This poststripping sclerodermiform dermatitis may be a rare, previously unidentified, late complication of saphenous vein stripping.

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REFERENCES