HISTOPATHOLOGICAL CORRELATION OF LIPODERMATOSCLEROSIS

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ABSTRACT

Aim-To observe histopathological findings in patients with clinical diagnosis of lipodermatosclerosis. Patients and methods: A total of 5 patients with chronic lipodermatosclerosis were included in the study. All patients were subjected to 1. Complete history taking, 2. General and dermatological examination, 3. Consenting patients were subjected to punch biopsy of the lower limbs. Results: Histopathology of patients with chronic lipodermatosclerosis was studied. Of the 5 cases, 4 showed epidermal atrophy. Increase in pigmentation of basal layer was noted in 3 cases. Dermal changes were observed in 3 cases, showing edematous changes, pseudo-sclerodermatous change and mild septal infiltrates. 2 cases showed extravasation of RBCs. Evidence of lobular panniculitis was noted in 1 case. Conclusion: Histopathological findings of lipodermatosclerosis were consistent with epidermal thinning, increased pigmentation in basal layer, pseudo-scleroderma like changes, extravasation of RBCs and presence of chronic inflammatory infiltrates.

INTRODUCTION

Lipodermatosclerosis (LDS) is a localized chronic inflammation and fibrosis of skin and subcutaneous tissue of lower limbs [1]. The term lipodermatosclerosis was primarily coined to describe effects of chronic venous hypertension in skin. The pathogenesis is multifactorial, involving highly complex interactions occurring in and around the endothelial cells, with involvement of huge variety of cytokines, growth factors and matrix metalloproteinases.

Clinically it is more commonly seen in women with high BMI. Patients present with well defined, indurated plaques in the lower extremities, usually on the medial aspect. LDS in acute stage is characterized by diffuse inflammatory erythema, edema, and tenderness of skin and in chronic stages, indurated areas of pigmentation and fibrotic narrowing of the lower limb occurs. There are different reports on the histopathological findings of LDS. Hence an attempt was made to delineate histopathological findings in LDS.

METHODS

Our study involved a total of 5 patients (males) who presented to our OPD and clinically diagnosed as chronic lipodermatosclerosis. All consenting patients were subjected to detailed history taking, clinical examination and punch biopsy from the skin of lower limbs. All the participated subjects gave their informed consent and ethical clearance was obtained from the local ethical committee.

RESULT

Patients’ age ranged from 33 to 65 years (mean age 51.6 years). All lesions were present on the lower limbs, between the knee and ankle.

Duration of the lesions ranged from 6 months to 2 years (Table 1). Clinically, the lesions were erythematous, tender, indurated plaques or nodules. The characteristic histologic findings were seen exclusively in the
subcutaneous tissue, concerning primarily the lobules but also the septa.

Adipose changes included micropseudocyst and macropseudocyst formation, necrotic adipocytes, lipomembranous change, lipogranulomas with xanthomatous macrophages. The accumulation of chronic inflammatory infiltrates and basophilic elastic fibers located deep in the dermis were present in all the cases.

**Table 1. Observation**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Age/Sex</th>
<th>Duration of LDS</th>
<th>Histopathological findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>53/M</td>
<td>2 years</td>
<td>Epidermal atrophy, lobular panniculitis, extravasation of RBCs</td>
</tr>
<tr>
<td>2</td>
<td>60/M</td>
<td>8 months</td>
<td>Epidermal atrophy, dermal edema</td>
</tr>
<tr>
<td>3</td>
<td>33/M</td>
<td>6 months</td>
<td>Dermal edema with infiltrates</td>
</tr>
<tr>
<td>4</td>
<td>65/M</td>
<td>18 months</td>
<td>Epidermal atrophy with inflammatory infiltrates</td>
</tr>
<tr>
<td>5</td>
<td>47/M</td>
<td>1 years</td>
<td>Epidermal atrophy, extravasation of RBCs</td>
</tr>
</tbody>
</table>

**Figure 1.** Section stained with H&E, showing thinned out epidermis, increased collagen bundles in dermis replacing subcutis (low power magnification).

**Figure 2.** Section stained with H&E, extravasated RBC and fat microlobules (high power magnification).

**DISCUSSION**

Lipodermatosclerosis, synonymous with hypodermitis sclerodermaformis and sclerosing panniculitis, is an inflammatory process of the subcutaneous fat that acutely presents as medial lower leg pain, erythema, tenderness, and warmth [2]. This chronic disease is famous for “bound down, sclerotic” hyperpigmented skin, giving an “inverted champagne bottle” appearance of lower limb [3]. These changes are related to venous insufficiency with resultant extravasation of capillary substances. Various hypotheses associated with the pathogenesis include:

A. Fibrin cuff formation leading to tissue hypoxia
B. Elevated matrix metalloproteinases and fibrinolytic mediator activity causing collagen destruction and ulceration
C. Leukocyte-mediated endothelial damage.

Histology is notable for lobular panniculitis with no evidence of vasculitis. There is mild infiltration of inflammatory cells leading to adipocyte necrosis, fibrosis of the septa with fibrous strands projecting centrally into the fat lobule, lipid filled macrophages and decrease in functional adipocytes. There is eosinophilic pseudomembrane formation on H&E which is thought to be a result of adipocyte breakdown. This sequence of events can be a consequence of any process leading to inflammatory cell infiltrate, including edema and drug response [4].

Lipodermatosclerosis is diagnosed based on the presence of characteristic signs and symptoms. Skin biopsy or blood tests are usually not required to confirm a diagnosis of LDS, but can be performed in rare cases. Ultrasound and magnetic resonance imaging (MRI) may be used to obtain more information regarding the severity of the condition and to determine the best treatment approach [5].

Lipodermatosclerosis is primarily treated with compression therapy to improve venous insufficiency. Other modalities involved in the management of venous insufficiency include limb elevation, and avoidance of prolonged sitting/standing in one place. Patients is advised regular exercise; and weight loss if overweight or obese. Some individuals might require medications to prevent blood clotting; reduce pain and inflammation; and/or to increase blood flow. Depending on the severity of the condition and response to initial treatment, venous surgery may be recommended. Newer modalities include,
ultrasound therapy, the mechanism however is unclear, but might involve immunomodulatory and anti-inflammatory effects of infrared radiation [6], which also upregulates collagen-degrading matrix metalloproteinase [7]. Ultrasound therapy might thus help to reverse both the fibrotic and inflammatory changes of LDS. Advantages include easy portability of equipment and relatively inexpensive. This simple treatment modality is safe and may offer substantial improvement for an otherwise painful and refractory condition.

CONCLUSION
Histopathology of LDS is varied. Clinicopathological correlation may help to exclude the differential diagnosis of erythema nodosum. However, panniculitis is not a hallmark finding in histopathology of lipodermatosclerosis.

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CONFLICT OF INTEREST:
The authors declare that they have no conflict of interest.

REFERENCES: