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Lipodermatosclerosis: A Case Report and Overview from Dermatopathologist Perspective

Kavita Chauhan[*1], Kabir Singal[2], Asmara Syed[3], Syed Sajid Hussain Shah[3], Anshoo Agarwal[3], Fariha Kauser[4], S. Geetha Subramaniam[5]

[*1]Department of Pathology, Faculty of Medicine, Swami Vivekananda Subharti University, Meerut, India

[2]JSS Medical College, Mysore, Karnataka, India

[3]Department of Pathology, Faculty of Medicine, Northern Border University, Arar, Kingdom of Saudi Arabia

[4]Health practitioner & Ex Medical Educationist from University of Dundee, Scotland

[5]Faculty of Health and Life Sciences & Centre for Health, Well-being, and Sustainability, INTI International University, Persiaran Perdana BBN, Putra Nilai, 71800 Nilai, Negeri Sembilan, Malaysia

***Corresponding Author:**

Email ID: AAKC2024@outlook.com

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ABSTRACT

Background: A chronic skin disorder called lipodermatosclerosis is characterized by thickening and hardening of the skin, frequently with alterations in the layers of connective tissue and underlying fat. It is essential to comprehend the pathological characteristics of lipodermatosclerosis in order to make an accurate diagnosis and plan customized treatment plans. The patient's comprehensive pathological findings, who was diagnosed with lipodermatosclerosis, are presented in this case study.

Case report: Fifty-eight years old man consulted the dermatologist with the complaints of progressive skin thickening, discoloration and reduced mobility in his lower leg. Biopsy findings were consistent with the diagnosis of lipodermatosclerosis.

Conclusions: This case report highlights the detailed pathological findings in a patient with lipodermatosclerosis. The observed changes in the epidermis, dermis, and subcutaneous tissue layers provide a better understanding of the complex pathogenesis of this condition.

Keywords: *Pathological Findings, Lipodermatosclerosis. Skin, Lower Extremities*

1. INTRODUCTION

Lipodermatosclerosis, also called hypodermitis sclerodermaformis or sclerosing panniculitis, is a chronic inflammatory disease marked by induration of the lower limb skin and the development of subcutaneous fibrosis in humans [1]. Several pathogenic pathways have been hypothesized about this lesion but there is no clear comprehension of the pathophysiologic mechanisms [2]. Fibrinolysis abnormalities have also been linked to lipodermatosclerosis and venous insufficiency, despite the overwhelming data supporting this connection [2,3]. It is believed that elevated pressure in the lower limb venous circulation causes the movement of fibrinogen and some chemical substances from the capillary to the dermis & subcutis [4].

Gradual fibrosing panniculitis eventually results in the characteristic presentation of lipodermatosclerosis, in which the lower extremity is shaped like an upside-down champagne bottle [5]. This traditional description is typical of advanced-stage lipodermatosclerosis; the clinical manifestation of the illness varies greatly according on the disease's stage [6]. Typically, the diagnosis is established clinically, based only on distinguishing characteristics. When the diagnosis is unclear, a histopathological study helps to confirm the diagnosis. [3,7]

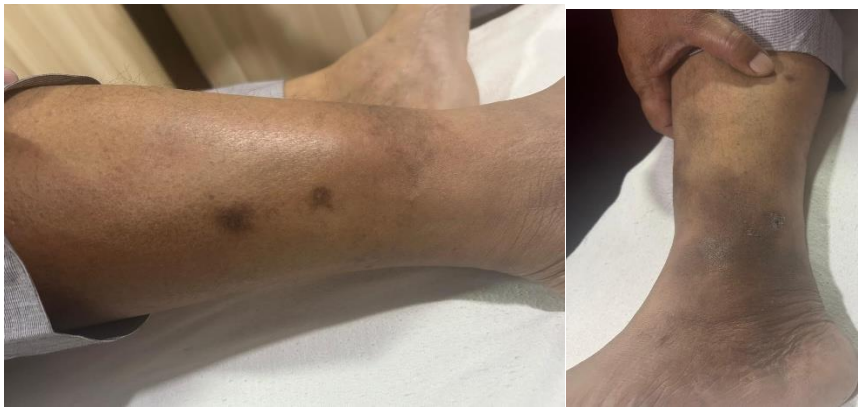
Chronic venous insufficiency, which can be caused by a reduction in the efficacy of the calf muscle pump, incompetent venous valve or obstruction to venous flow, is typically the situation in which lipodermatosclerosis develops [8]. Increased weight, old age, deep vein thrombosis, presence of venous insufficiency among the family member, and cigarette smoking

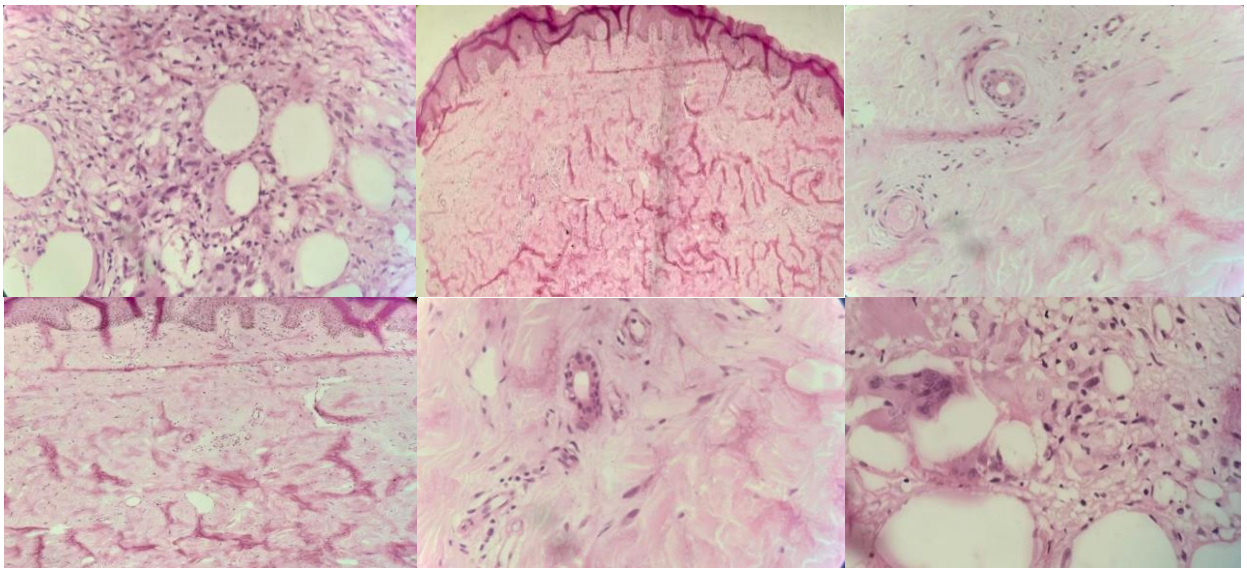
are some of the risk factors that are most frequently linked to the development of lipodermatosclerosis [4,9]. The main signs of lipodermatosclerosis are: 1. Discoloration of the affected skin, which can range from reddish-brown to purple. 2. Thickening and hardening of the skin, which is sometimes referred to as having a "woody" texture. 3. Reduced range of motion or movement in the affected limb; tissue alterations; pain, soreness, or a burning feeling in the affected area [10].

2. CASE PRESENTATION

Fifty- eight years old man consulted the dermatologist with the symptoms of progressive skin thickening, discoloration, and reduced mobility in his left lower leg. Physical examination revealed a woody, indurated texture and a reddish-brown discoloration of the affected skin (Figure 1-2). Based on clinical features, the differential diagnosis of erythema nodosum leprosum, Lipodermatosclerosis, morphea, vasculities and panniculitis were considered. For the confirmation of the diagnosis, a skin biopsy from dorsal aspect of ankle was performed. Greyish white to greyish-brown, soft tissue piece measuring 1.1x0.4x0.3 cm was received from Dermatology OPD which was processed by using histopathological techniques.

Pathological Findings: Microscopic examination of the skin biopsy revealed compact orthokeratin covered by epidermis with mild lymphocytic exocystosis and focal acanthosis. Superficial and deep dermis showed sclerosis. Sweat glands were surrounded by collagenised tissue. Subcutaneous tissue revealed septa; and lobular panniculitis, fibrosis focal vascular proliferation and occasional granuloma with multinucleate giant cells. Adipocytes were cystically dilated. Inflammation was predominantly lymphocytic (Figure 3-6). There was no evidence of vasculitis. Acid fast bacilli could not be demonstrated on fite stain. These pathological findings were characteristic of lipodermatosclerosis and reflected the progressive changes in the skin and underlying tissues due to chronic venous insufficiency and tissue ischemia.





Figures 1 and 2: Skin thickening and reddish-brown discoloration of the left lower

Figure 3: Compact orthokeratin covered epidermis with mild lymphocytic exocystosis and focal acanthosis. Superficial and deep dernis show sclerosis. Figure 4-6: Sweat glands were surrounded by collagenised tissue. Figure 7: Subcutaneous tissue

reveal septa; and lobular panniculitis, Figure 8: Fibrosis and focal vascular proliferation and occasional granuloma with multinucleate giant cells. Adipocytes were cystically dilated. Inflammation was of predominantly lymphocytic lymphocytes (H& E stained sections, Magnifications Fig 3 : 4x10, Fig 4-6: 10 x 10, Fig 7: 20 x 10, Fig 8; 40x10)

3. DISCUSSION

Hypodermitis sclerodermiformis, sclerosing panniculitis, liposclerosis, pseudoscleroderma, indurated cellulitis, stasis panniculitis, and chronic leg cellulitis are other names for lipodermatosclerosis [11]. In patients with chronic venous insufficiency, hyperpigmentation, small white-scarred spots ("atrophie blanche"), localized skin induration, minor ulcerations, and erythematous plaques are common clinical findings in chronic lipodermatosclerosis [12]. Lipodermatosclerosis's distinctive histological characteristics can be seen in the septa, lobules and subcutaneous tissue. The fibrosis process that characterizes the condition is explained by the existence of elastic fibers deep within the septa [13].

The pathological changes observed in this case report provide valuable insights into the underlying mechanisms of lipodermatosclerosis. The epidermal thickening, increased pigmentation, and dermal sclerosis are likely the result of chronic inflammation and altered cellular signalling pathways. The decreased adipose tissue and increased fibrosis in the subcutaneous layer contribute to the characteristic woody and indurated texture of the affected skin. The histopathological manifestation of lipodermatosclerosis exhibits significant variability contingent on the disease's evolutionary stage [13]. During the acute phase, hemosiderin deposition, capillary bleeding, and cystic fat necrosis are frequently accompanied by infiltration of lymphocytes in the septa which surround the fat lobule [11,14] (Figure 3-6). More advanced lipodermatosclerosis is characterized by specific histopathological findings such as lobular panniculitis with a mixed cellular infiltration (Figure 7-8), adipocyte breakdown, and lipomembranous alteration resulting in the creation of "pseudocysts" inside the subcutaneous tissue [7,15]. Deteriorating adipocytes with thick, eosinophilic margins characterize the lipomembranous change, a histopathologic characteristic strongly suggestive of lipodermatosclerosis [16]. The substantial fibrosis in the septa and hyaline sclerosis overlaid on a backdrop of chronic stasis alterations are linked to chronic lipodermatosclerosis [17]. A variety of staining methods are used to emphasize the existence of fibrin around the vessels, that could help to diagnose lipodermatosclerosis [18]. Usually, Vasculitis is not noticeable in any stage of the course of the disease, despite varied degrees of panniculitis and necrosis [19]. These pathological findings underscore the importance of a comprehensive evaluation, including skin biopsy, in the diagnosis and management of lipodermatosclerosis [18].

Depending on the disease's stage, a variety of illnesses will be included in the differential diagnosis for lipodermatosclerosis. Lipodermatosclerosis' acute stage is frequently confused with various panniculitides, erythema nodosum, cellulitis, and trauma-induced fat necrosis [20-24]. Clinicians should immediately rule out lipodermatosclerosis when they see well-defined, firm, and highly sensitive plaques that resemble cellulitis in the lower legs.

Perivascular inflammation containing polymorphonuclear leukocytes is a feature of cellulitis. If an infection is suspected, a complete blood count may be carried out [25]. On the other hand, erythema nodosum is typified by a significant granulomatous infiltration with giant cells and manifests with a mixed cellular infiltrate without vasculitis in addition to septal panniculitis [26]. Isolated lesions are the hallmark of fat necrosis, which is frequently linked to a traumatic past [27].

Morphea is characterized by thick collagen bundles without lipomembranous dystrophy and ivory-colored, indurated plaques with a distinctive purple ring under the microscope [28]. On a histological examination, necrobisis lipoidica appears as an alternating, layered inflammatory process [29]. In patients with reduced renal function, gadolinium exposure is linked to nephrogenic systemic fibrosis [30].

The detailed understanding of the underlying pathological processes can guide the development of targeted therapies and inform the clinical decision-making process for healthcare providers [20,31]. Reducing inflammation, improving skin and tissue health, and managing the underlying venous insufficiency are the main objectives of treatment for lipodermatosclerosis [21]. Exercise and physical therapy to improve circulation and mobility; Antibiotics or anti-inflammatory medications; Compression therapy, such as compression stockings or bandages; Topical creams or ointments to improve skin hydration and reduce inflammation; In severe cases, surgical interventions to address the underlying vascular issues Preventive measures and lifestyle adjustments: People who are susceptible to lipodermatosclerosis can prevent the condition by doing the following: staying in a healthy weight range and exercising frequently; wearing supportive clothing, such as compression stockings; elevating the legs whenever possible to improve venous return; avoiding extended periods of standing or sitting; and maintaining healthy skin through moisturizing and gentle cleansing [32].

Although lipodermatosclerosis can be treated to decrease symptoms and slow the disease's progression, the condition usually has a protracted and progressive course [33-34].

Because of the ongoing inflammation and fibrosis, the chronic stage of lipodermatosclerosis is sometimes considered a separate stage that precedes ulceration. It is marked by poor wound healing. A number of studies have indicated a clear relationship between the degree of skin induration, the likelihood of ulceration later on, and the ability to heal. Ulcers can result from mild stresses like as scratching in people who have significant induration [35]. Lower extremity pain is another possible side effect of lipodermatosclerosis [36]. People with severe lipodermatosclerosis

could experience continuous, throbbing pain in their legs. Severe, burning pain is typically experienced during the acute phase of the disease [1].

The lipodermatosclerosis seems to be complex skin condition that is usually associated with chronic venous insufficiency. By understanding the causes, symptoms, and management strategies, healthcare professionals can provide more effective care for individuals affected by this condition. Continued research and patient education are essential to improving the quality of life for those living with lipodermatosclerosis.

4. CONCLUSIONS

This case report highlights the detailed pathological findings in a patient with lipodermatosclerosis. The observed changes in the epidermis, dermis, and subcutaneous tissue layers provide a better understanding of the complex pathogenesis of this condition. Incorporating pathological evaluation into the diagnostic workup can enhance the diagnostic accuracy and help in the appropriate management of these cases of lipodermatosclerosis.

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