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A Rare Case Report of Lupus Erythematosus Profundus

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Abstract **Case Report**

Lupus Erythematosus Profundus is a rare presentation and subset of erythematosus. Lupus erythematosus (LE) is termed as an autoimmune chronic condition which involves a spectrum of symptoms. It is a part of the connective tissue diseases. Its cutaneous form is termed as cutaneous lupus erythematosus (CLE). Prevalence of CLE is about 70 cases per 100,000 persons. The least common variety of CLE is lupus profundus (LP) only 5% of cases. Lupus profundus, although rare, must be kept in the differential diagnoses of ulcerated lesions. It may present as a localized entity or in association with systemic lupus erythematosus (SLE) or it may lead to SLE later in life. Early diagnosis based on histopathology and aggressive treatment is essential to prevent significant physical morbidity and progression to systemic involvement. We report a case of biopsy-proven lupus profundus in a 40-year-old female who presented with lesions on forearms. The lesions were appreciated on the both forearms. They had an erythematous base and edematous necrotizing centers. Patient was treated with methotrexate, hydroxychloroquine and steroids. It may present as a localized entity or in association with SLE or it may lead to SLE later in life.

Keywords: Panniculitis, Lupus Erythematosus.

INTRODUCTION

Panniculitis is inflammation of fat tissue. Lupus Erythematosus Profundus is a rare subset of chronic cutaneous lupus erythematosus. (CCLE)with a reported incidence of 1 to 3% in all lupus erythematosus cases. The most common clinical cutaneous clinical presentation includes indurated plaques or subcutaneous nodules with an overlying normal skin. Lupus erythematosus (LE) is termed as an autoimmune and chronic condition which involves a spectrum of symptoms. It is a part of the connective tissue diseases. The systemic form of LE is called systemic lupus erythematosus (SLE) whereas the cutaneous form is termed as cutaneous lupus erythematosus (CLE). SLE and CLE can coexist as well as exist as separate entities. CLE can also be the precursor of SLE in some cases.

CASE REPORT

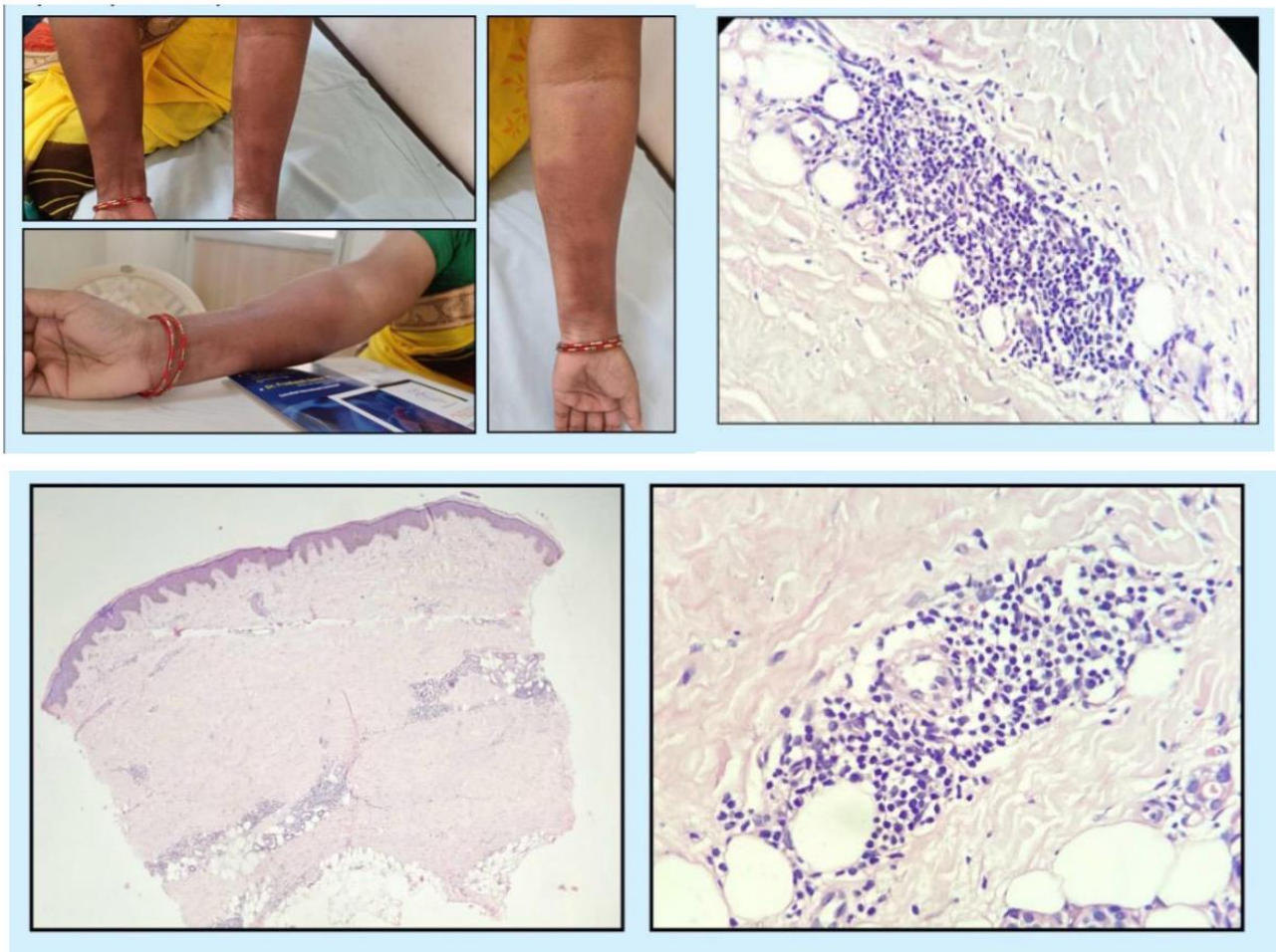
A 35 years old female patient presented with thickening and tightness of skin over left forearm region since last 2 years, which was painful. It later progressed to discoloration of skin. For above complains she took over the counter medicines. After 3 months of initial symptoms, she developed lesions over the right forearm. Over the time the lesions increased in size. Swelling,redness, itching and burning sensations started on the lesions. Later a depressed scar developed. There were no associated systemic symptoms.

With above symptoms we suspected panniculitis or Morphea. On evaluation ANA test was positive. Skin biopsy was done which showed hyperkeratosis and vacuolar degeneration. There was edema in papillary dermis. Mixed inflammatory cell infiltrates were noted in region predominantly comprised of lymphocytes, plasmacytes and few neutrophils. Dermis showed collagenization. Deeper dermis and dermal subcutaneous junction showed moderate chronic inflammation around fat lobules and adnexia.
Patient was treated with methotrexate, hydroxychloroquine and steroids.

CONCLUSION

Lupus profundus is the least common subtype of CLE. It is frequent in middle-aged women [3]. The lesions are common on the face, proximal extremities, buttocks, breasts, and trunk [1, 3]. However, according to Batrani et al., there are only 15 cases in the internet-based literature with LP presenting at a non-acral site [2]. Our case is also a non-acral presentation of lupus profundus.

IMAGES /PICTURES



DISCUSSION

Clinically, LP presents with ulcerated, indurated, subcutaneous, painful or tender nodules [3]. Histological diagnosis is based on the presence of hyaline necrosis of the subcutaneous fat lobules along with lymphocytic infiltration of fat lobules. Plasma cells, lymphoid follicles, and eosinophils may also be present [4]. Presence of mucin deposition along with epidermal changes such as degeneration indicates coexistence of DLE and LP, which is seen in as many as 70% cases [5]. There were no epidermal changes in our case, hence, our diagnosis remains isolated for LP.

CLE is taken as the precursor of SLE in as many as 25% of cases. However, the risk is higher in patients with SCLE than in localized DLE [1]. Grönhagen *et al.,* in an epidemiological study reported the incidence of CLE to be 4/100000; with a male to female ratio of 1:3. It reported 18% of CLE patients to be later diagnosed with SLE, highest diagnosis being for SCLE. On the other hand, it also reported 24% of newly diagnosed CLE patients to already have the diagnosis of SLE [6].

SLE accompanying LP is reported in as few as 2%-5% cases; 15% are able to progress to SLE and 50% have evidence of SLE. Hence, LP can be taken as an initial presenting manifestation of SLE [5]. Zhao *et al.,* have reported only 10 cases of SLE that initially presented with LP from the internet-based medical literature search [7]. Our patient didn't meet the SLE diagnostic criteria of American College of Rheumatology at the time of presentation [8]. However, we are following the patient in view of long-term diagnosis of SLE. Our patient had unremarkable immunological assay. Literature has reported antinuclear antibody to be positive in 27%–95% cases of LP [9-11]. Anti-double-stranded DNA antibodies are identified in fewer cases [12]. Laboratory findings also include lymphopenia, anemia, decreased C4 levels, and positive rheumatoid factor; however, in most cases, serology remains unremarkable as in our case.

The diagnosis of LP is based on clinical, serological and histopathological grounds. The differential diagnosis includes connective tissue panniculitis such as morphea profunda (MP) and inflammatory diseases of subcutaneous fat erythema nodosum, erythema induratum. The most challenging differential diagnosis is subcutaneous panniculitis-like T-cell lymphoma (SPTCL). LP is differentiated from MP by the absence of dermal and subcutal sclerosis. SPTCL histologically presents with atypical CD3+, CD8+ and CD4-T lymphocytes with the expression of clonal a/b T

cell receptors and typical localization of lymphocytes around fat cells in a rim-like manner. Plasma cells are also classically absent in SPTCL [7, 13].

We managed our patient with antimalarial therapy which is the first-line recommendation for LP [7]. For recalcitrant cases, systemic therapies such as thalidomide, dapsone, cyclosporine, and rituximab can be used [14].

Lupus Erythematosus Pro fundus is a rare disease which cause skin disfigurement. It requires high degree clinicopathological suspicion. Early diagnosis and treatment prevent complications.

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