

Histoid Leprosy Presenting as Fibroma on Initial Histopathology: A Rare Case Report

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ABSTRACT

Leprosy, caused by *Mycobacterium leprae*, persists in many developing nations. Histoid leprosy, an uncommon variant of lepromatous leprosy, may occur even in untreated individuals, thereby challenging its earlier association with drug resistance. This is a case of a 45-year-old female with a previous history of infiltrating ductal carcinoma, who made a full recovery with lumpectomy and chemotherapy six years ago, and presented with multiple multinodular lesions over her trunk and limbs. The clinical presentation and initial histopathological findings suggested a soft-tissue tumour, raising concern about the recurrence of malignancy. However, further investigation revealed fusiform histiocytes and Acid-Fast Bacilli (AFB), confirming a diagnosis of histoid leprosy. Through this case, we aim to highlight the challenges in diagnosing histoid leprosy due to its clinical resemblance to soft-tissue tumours, particularly in patients with a history of malignancy. The case illustrates the potential for diagnostic confusion in endemic regions where both infectious and neoplastic conditions may coexist.

Keywords: Hansen's disease, Histoid leprosy, Manifestations of leprosy, *Mycobacterium leprae*, Solitary fibroma

CASE REPORT

A 45-year-old woman presented to the outpatient department of a district clinic in Central India with complaints of multiple multinodular lesions on her trunk, upper limbs, and lower limbs for the past two months. She had a prior history of infiltrating ductal carcinoma of the right breast, for which she underwent lumpectomy followed by seven cycles of chemotherapy with cyclophosphamide, methotrexate, and 5-fluorouracil six years earlier.

After a disease-free interval of five years, she developed a solitary nodule in the same breast, which was biopsied and diagnosed as a fibroma.

The current multinodular lesions initially on her left breast and gradually progressed to the lower limbs—first the left and then the right limb. The patient reported no systemic symptoms and no history of sensory changes or disability. There were no clinical signs suggestive of cancer recurrence.

On physical examination, the nodules were normochromic with mild erythema, well-defined, firm, and had a smooth, shiny surface, measuring approximately 1-2 cm in diameter [Table/Fig-1,2]. The



[Table/Fig-2]: Well-demarcated nodular lesions over the forearms.



[Table/Fig-1]: Shiny, skin coloured papulo-nodular lesions on the lower limbs.

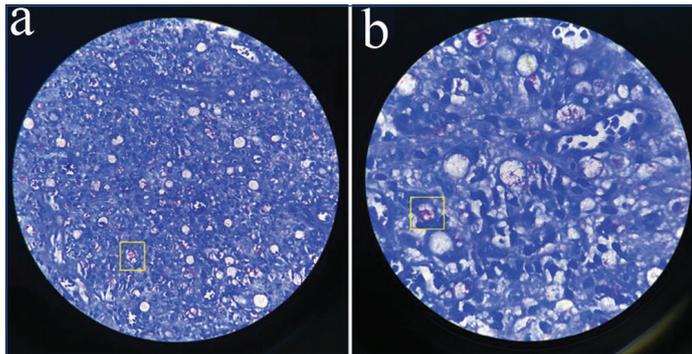
surrounding skin was infiltrated but there was no evidence of leonine facies or madarosis. Liver and renal function tests were normal limits, and other laboratory profile has been mentioned in [Table/Fig-3].

Investigation	Results	Reference Range
Haemoglobin (g/dL)	10.7	10-14
Leucocytes (cells/cumm)	6000	4000-11000
Lymphocytic count	29.1%	10-30%
Haematocrit	32.8%	35-54%
Serum blood urea (mg/dL)	9.7	12.8-42.8
Blood urea nitrogen (mg/dL)	4.35	4-20
Serum creatinine (mg/dL)	0.66	0.6-1.1
Serum uric acid (mg/dL)	1.78	1.5-6.0
Serum bilirubin (total) (mg/dL)	0.28	0.2-1.2
Serum bilirubin (indirect) (mg/dL)	0.17	0.2-0.5
Serum bilirubin (direct) (mg/dL)	0.11	0-0.3
Serum SGOT (U/L)	20.21	0-31
Serum SGPT (U/L)	9.84	0-34

[Table/Fig-3]: Laboratory profile of liver function test and kidney function test.

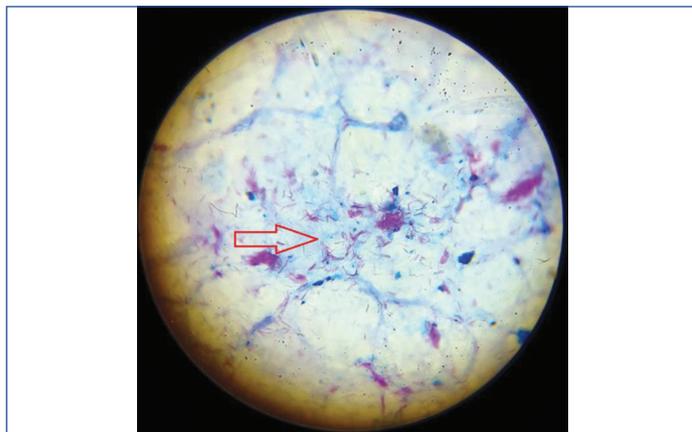
Histopathological report from the initial lesion on the right breast showed an infiltrative lesion composed of round to oval cells with

moderate pleomorphic vesicular nuclei, prominent nucleoli, and a moderate amount of pale cytoplasm [Table/Fig-4]. The cells were arranged in nests, and numerous lipoblasts were observed, indicating a diagnosis of a soft-tissue tumour with moderate atypia.



[Table/Fig-4]: Histopathology slide of the initial lesion on the right breast showed an infiltrative lesion composed of round to oval cells with moderate pleomorphic vesicular nuclei; a) 40x magnification; b) 100x magnification.

Multiple lesions on the right arm warranted excision, and subsequent biopsy of the skin lesion revealed fusiform histiocytes arranged in short bundles within the dermis. Numerous Acid-Fast Bacilli (AFB) were demonstrated on Fite–Faraco staining, confirming the diagnosis of histoid leprosy [Table/Fig-5].

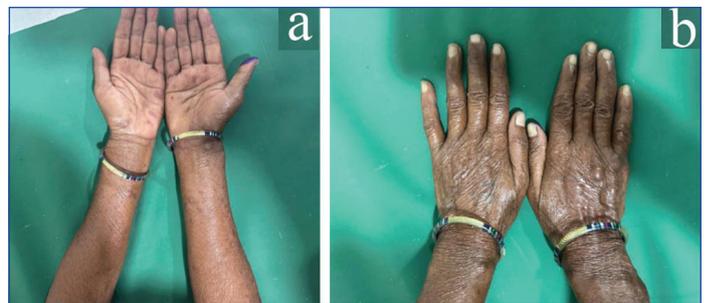


[Table/Fig-5]: Microscopic image showcasing Acid-Fast Bacilli (AFB) at 40x magnification.

The patient was started on a multi-drug therapy of 100 mg Dapsone, 600 mg Rifampicin, and Clofazimine 50 mg daily for 24 months. The postoperative recovery was uneventful. At one-month follow-up, most nodules had subsided, showcasing the importance of early diagnosis and appropriate treatment [Table/Fig-6,7].



[Table/Fig-6]: Resolution of nodules confirming remission during post-treatment follow-up after one month in lower limbs.



[Table/Fig-7]: Resolution of nodules confirming remission during post-treatment follow-up after one month in arms.

DISCUSSION

Leprosy, or Hansen's disease, is a chronic granulomatous condition caused by *Mycobacterium leprae* that primarily affects the skin and peripheral nerves [1]. The global target for eliminating leprosy was achieved in 2000, with worldwide prevalence of less than 600,000 cases [2].

A rarer form of lepromatous leprosy, first described by Wade [3]. Histoid leprosy was initially thought to be linked to drug resistance in multibacillary patients treated with dapsone monotherapy [3,4]. However, this form can also occur in individuals without prior treatment, indicating that it is not limited to a resistance pattern.

The clinical manifestation ranges from small papules to subcutaneous nodules to cutaneous plaques, commonly involving the body prominences, the buttocks, back, extremities, and face [5,6].

Murthy SV et al., reported that histoid leprosy constituted 8.7% of cases among patients with lepromatous leprosy and 1.2% of all leprosy cases [7]. The palms and soles remain unaffected, while histoid lesions have frequently been observed along peripheral nerve trunks, particularly the ulnar nerve [2]. In the present case, lesions were also noted on the palms, which is an uncommon finding.

In contrast to its usual presentation, the unusual presentations of histoid leprosy have been considered differentials of systemic lupus erythematosus, mycosis fungoides, neurofibromatosis, and cutaneous leishmaniasis [8-11].

Histopathological examination remains the gold standard for diagnosing leprosy, typically identifying an infiltrate composed primarily of fusiform histiocytes, which bear a resemblance to fibroblasts and may sometimes mimic a fibro histiocytic neoplasm. This infiltrate is usually accompanied by a few foamy macrophages and a significant presence of AFB [7]. The presence of nodules, a hallmark of both soft-tissue tumours and leprosy, can create a significant diagnostic challenge, especially in patients with a history of malignancy. In endemic regions, this overlap can lead to leprosy lesions being misdiagnosed, delaying appropriate treatment and affecting patient outcomes. While there have been reports of histoid leprosy presenting as a large tumour or being confused with neurofibromatosis, to the best of the authors knowledge, this is the first documented case where histoid leprosy initially presented as a soft-tissue tumour based on histopathological assessment.

CONCLUSION(S)

While histopathological examination is the gold standard method for diagnosing histoid leprosy, in endemic zones, leprosy should be included in the differential diagnosis for patients with relevant clinical signs and symptoms, regardless of the initial histopathological outcomes.

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