www.jmscr.igmpublication.org
Impact Factor 5.84
Index Copernicus Value: 83.27
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v5i8.168

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Case Report

Molluscum Contagiosum–Like Lesions in Histoid Leprosy in a 35-Year-Old Indian male

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ABSTRACT
Histoid leprosy is a rare but well-defined entity with specific clinical, histopathological, and bacteriological features. It is considered an uncommon variant of lepromatous leprosy that usually follows treatment failure. Rarely. it occurs de novo without any history of previous inadequate or irregular treatment. Molluscum contagiosum–like lesions in the setting of histoid leprosy is an exteremely rare presentation and may signify the process of "transepidermal elimination" in lesions with high bacillary load. We, hereby, report a case of de novo histoid leprosy in a 35-year-old man with mollusciform lesions in view of the rarity of this condition.
Key words: *histoid, leprosy, de novo, mollusciform, multidrug therapy.*

First reported by Wade in 1963, histoid leprosy is a very rare form of multibacillary leprosy that may arise de novo or following an inadequate treatment with dapsone monotherapy or multidrug therapy (MDT). It commonly affects buttocks, back, face and extremities [1]. The typical clinical features of histoid leprosy are numerous shiny, succulent nodules overlying normal looking skin, along with papules and plaques. Histoid leprosy is treated as a multibacillary disease.

A 35 year old married patient presented with asymptomatic skin coloured raised lesions on the face, trunk and limbs .The duration of complaint was one month. There were no constitutional symptoms or systemic complaints. Patient denied any numbness or weakness. There was no history

of any previous treatment taken and family history was non-contributory.

Patient's general examination was normal. Cutaneous examination revealed shiny, hemispherical, succulent looking discrete to coalescent skin coloured papules and plaques with few plaques showing central umbilication. The lesions ranged in size from 2 mm to 4 cm and were distributed bilaterally and symmetrically on the face, front and back of the trunk and limbs. The consistency was soft to firm.

Neurological exanimation revealed bilateral symmetrically thickened and nontender nerves. Sensory examination revealed loss of temperature, touch and pain sensation in scattered patches over forearms and legs. Motor examination revealed slight weakness of abduction of little finger.Rest of the motor examination was within normal limits.

Routine hematologic investigations, urine analysis, liver and renal function tests were all normal. Slit skin smear revealed acid-fast bacilli of BI - 6+ and MI - 80%. Histopathologic examination of hematoxylin and eosin-stained section revealed atrophic epidermis with flattened rete ridges and dermal infiltration by nodular granulomata formed of spindle shaped histiocytes with pyknotic nuclei oriented in a storiform pattern. Ziehl Nelsons stain for lepra bacilli showed plenty of acid fast bacilli arranged in classical histoid habitus. So, the diagnosis of histoid leprosy was made. Therefore, multi-drug therapy was started for 2 years.

Histoid leprosy was first described by Wade as discrete, firm lesions and dome-shaped nodules which develop on apparently normal skin in patients with lepromatous leprosy [1]

Its exact etiopathogenesis is not well understood as it may arise de novo or may develop after an inadequate and irregular treatment with dapsone monotherapy or MDT[2]

The average age affected is between 21 and 40 years.[3] Histoid leprosy has characteristic clinical, histopathologic and bacterial morphological features. Clinically, it is characterized by cutaneous and/or subcutaneous nodules and plaques on apparently normal skin [4]

The lesions are usually located on the posterior and lateral aspects of the arms, buttocks, thighs, dorsum of hands, and on the lower part of the back and over the bony prominences, especially over the elbows and knees. Classical histopathologic findings include epidermal atrophy as a result of dermal expansion by the underlying leproma and an acellular band located immediately below the epidermis. The leproma consists of fusiform histiocytes arranged in a tangled or storiform pattern containing acid fast bacilli. There are three histologic variants of histoid hansen: pure fusocellular, fusocellular with epitheloid component, and fusocellular with vacuolated cells. The third pattern is most commonly observed .[5]

Histoid leprosy might represent an enhanced response of the multibacillary disease in localizing the disease process. An increase in both cell-mediated and humoral immunity against Mycobacterium leprae, as in lepromatous leprosy, has been hypothesized[6]. Clinical differential diagnoses include post kala azar leishmaniasis, drug eruption and cutaneous metastasis. Each of them can be differentiated from histoid leprosy on the basis of the characteristic histopathology and absence of mycobacteria in slit skin smear. Histoid leprosy is managed by initially administering ROM therapy once, followed by MDT for 2 years[5]

Umbilicated papules mimicking molluscum contagiosum have been reported very rarely in histoid leprosy.[7]

Our patient had classic clinical features of histoid leprosy with multiple umbilicated lesions arising de novo. It has been hypothesized that it may be the process of "transepidermal elimination" seen during the evolution of cutaneous papules with high bacillary load.

Skin and nasal epithelia of individuals with untreated multibacillary leprosy are a potential shedding ground of Mycobacterium leprae into the environment. This assumes importance in view of

the fact that many undiagnosed histoid cases with a high bacillary index can contribute to transmission in endemic areas through the skin[8]

A high index of suspicion is required in diagnosing such cases for prompt treatment as histoid leprosy could serve as a reservoir of leprosy and a source of new cases especially in the post leprosy elimination era.

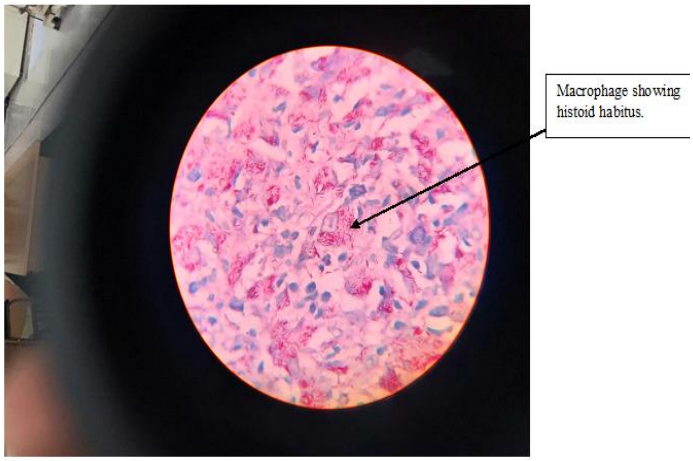

Figure 1. 40x H&E Modified ZN staining or Wade Fite stain-showing lepra Bacilliin globi in foam cells and outside.

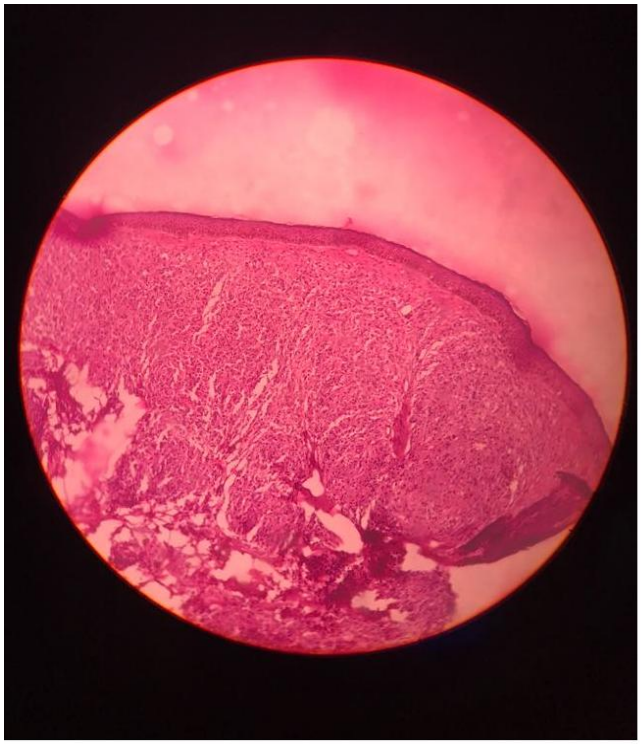

Figure 2. 10x shows thinned out dermis with grenz zone, Foamy cells, and scattered lymphocytes extending deep Into dermis.

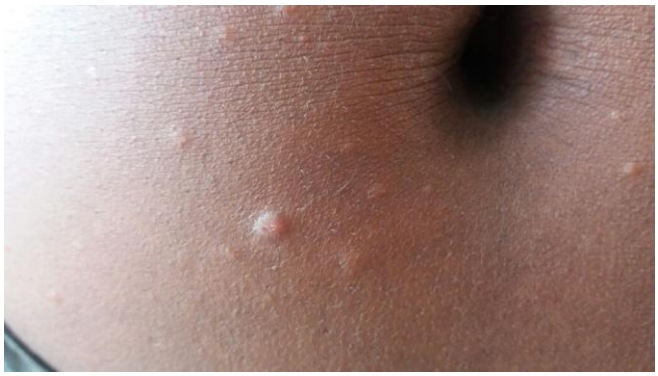

Figure 3. Clinical picture showing multiple shiny papules On abdomen.

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