Section: Dermatology

Case Series

WADE'S HISTOID HANSEN DISEASE REVISITED: A CASE SERIES

***Nivedha Priyanka A*[1]**

[1]*Assistant Professor, Department of Dermatology, Venereology and Leprosy, Kanyakumari Medical Mission Research Centre, Medical College and Hospital, Muttom, Tamil Nadu, India*

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Corresponding Author:
Dr. Nivedha Priyanka A,
Assistant Professor, Department of Dermatology, Venereology and Leprosy, Kanyakumari Medical Mission Research Centre, Medical College and Hospital, Muttom, Tamil Nadu, India.
Email: ashoknivedha08@gmail.com

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ABSTRACT

Background: Histoid Hansen disease is a rare clinicopathological variant of lepromatous leprosy, classically described by Wade, and is often associated with relapse, dapsone resistance, or irregular antileprosy therapy. Because its papulonodular lesions may mimic several benign and inflammatory dermatoses, delayed diagnosis can contribute to ongoing transmission.

Case Series: We report three male patients aged 25, 45, and 59 years who presented with multiple, gradually progressive, firm papules and nodules over apparently normal skin. The lesions involved the trunk, back, abdomen, chest, thigh, extremities, face, ear, and forearm. All patients had a history of irregular dapsone monotherapy for 2–4 months. Peripheral nerve examination revealed bilateral ulnar nerve thickening with tenderness in all three cases. Sensation was preserved in two patients, while one patient had sensory impairment over both forearms; no motor deficit was observed. Histopathological examination of representative lesions showed mild hyperkeratosis, epidermal atrophy, a clear Grenz zone, and dermal infiltration by macrophages, elongated histiocytes, and scattered lymphocytes, consistent with histoid Hansen disease. The clinicopathological correlation supported the diagnosis in all three patients.

Conclusion: Histoid Hansen disease remains an important diagnostic and public health concern in endemic regions, particularly among patients with irregular or inadequate antileprosy treatment. A high index of suspicion is important particularly when multiple firm papulonodular lesions are seen over normal-appearing skin, even in the absence of sensory or motor impairment. Prompt recognition, histopathological confirmation and starting multidrug therapy early are important in preventing disability as well as in reducing community transmission.

Keywords: Antileprosy Agents, Dapsone, Diagnosis, Differential, Leprosy, Lepromatous, Mycobacterium leprae.

INTRODUCTION

Leprosy (also known as Hansen disease) is a chronic granulomatous infectious disease which is caused by Mycobacterium leprae.[1] M Leprae is an acid-fast bacillus having a strong predilection for skin, peripheral nerves and mucosa of the upper respiratory tract. The clinical spectrum of leprosy is wide and depends largely on the host immune response, ranging from tuberculoid disease with strong cell-mediated immunity to lepromatous disease with poor cellular immunity and high bacillary load. Among these forms, histoid leprosy represents a rare, distinctive, and diagnostically important variant of multibacillary leprosy.[2]

Histoid leprosy was first described by H. W. Wade in 1963 as a special clinicopathological variant of lepromatous leprosy.[3] It is characterized clinically by well-defined, smooth, shiny, dome-shaped papules, nodules, or plaques that may appear on apparently normal-looking skin. These lesions are usually firm, skin-coloured to erythematous or hyperpigmented, and may be discrete or multiple. Commonly involved sites include the trunk, back, buttocks, face, and extremities, while palms and soles are usually spared. Because the lesions may resemble several other dermatological disorders, including dermatofibroma, neurofibromatosis, molluscum contagiosum, sarcoidosis, cutaneous metastasis, and

post-kala-azar dermal leishmaniasis, histoid leprosy can easily be missed unless it is specifically considered in the differential diagnosis.[4]

Histoid leprosy usually occurs in patients with lepromatous leprosy who relapse after inadequate or irregular treatment, particularly following dapsone monotherapy, where dapsone resistance has been considered an important contributing factor.[2] However, cases may also occur de novo, without any previous history of antileprosy treatment. This makes the condition more significant from both clinical and epidemiological perspectives. The incidence of histoid leprosy in India has been reported to vary from 2.79% to 3.60% among total leprosy patients.[3] Although uncommon, its recognition is important because patients often harbour a very high bacillary load and may serve as a reservoir for continued transmission of M. leprae in the community.[5]

The histopathological features of histoid leprosy are peculiar and often help in confirmation of the diagnosis. On histopathological examination epidermis may show flattening or atrophy. Additionally, a clear Grenz zone is typically present beneath it. The dermis shows presence of circumscribed collections of spindle-shaped histiocytes which are seen arranged in interlacing bundles, whorled patterns or storiform architecture. Slit-skin smear examination usually shows a high bacteriological index which further indicates the infective potential of these cases.[6]

From a clinical point of view, histoid leprosy poses a diagnostic challenge because cutaneous lesions may be the dominant manifestation, while sensory impairment and motor deficits may be minimal or absent in the early stages. Peripheral nerve thickening, particularly of the ulnar, common peroneal, posterior tibial, or great auricular nerves, may be present and should be carefully looked for during examination. Early diagnosis is essential not only to prevent nerve damage and deformity in the affected patient but also to reduce the risk of community spread. In the era of leprosy elimination, failure to recognize such atypical and highly bacilliferous cases may hinder control efforts.[7]

The present case series describes three cases of histoid Hansen disease which were diagnosed and managed in the Department of Dermatology, Venereology and Leprosy, Kanyakumari Medical Mission Research Centre, Medical College and Hospital. All three patients presented with multiple papulonodular skin lesions with peripheral nerve involvement. All patients had history of irregular dapsone monotherapy. Histopathological examination showed features consistent with histoid Hansen disease in all 3 cases. Through this case series, we aim to highlight the continuing occurrence of this rare variant, its clinical mimicry, and the importance of maintaining a high index of suspicion for early diagnosis and appropriate treatment. Written informed consent was obtained from all patients for publication of their clinical details and images.

CASE SERIES

Case 1: Patient Details and Clinical Presentation

A 45-year-old male presented to the dermatology outpatient department with complaints of multiple small raised lesions over the chest, abdomen, upper trunk, back, and thigh for the past 3 months. The lesions were gradually progressive in number and size. There was no history of ulceration, discharge, spontaneous regression, or systemic symptoms.

Past Treatment History: The patient gave a history of irregular dapsone monotherapy for a duration of 3 months. This history was clinically significant, as histoid leprosy is known to occur in association with inadequate or irregular antileprosy treatment, especially in patients previously exposed to dapsone.

Cutaneous Examination: On dermatological examination, multiple well-defined hyperpigmented papules and infiltrated nodules were present over the chest, abdomen, right thigh, and back. The lesions measured approximately 0.5–2 cm in size. They were discrete, firm, and distributed over apparently normal surrounding skin. The clinical morphology of papulonodular lesions raised the possibility of histoid Hansen disease, along with other differentials such as dermatofibroma, neurofibromatosis, molluscum contagiosum, sarcoidosis, and lepromatous leprosy.

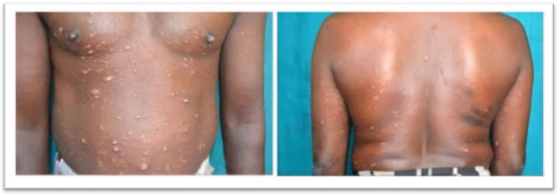

Figure 1: Clinical photographs of Case 1 showing multiple discrete, well-defined, hyperpigmented papules and infiltrated nodules over the anterior trunk, abdomen, thigh, and back, distributed over apparently normal surrounding skin, consistent with histoid Hansen disease.

Peripheral Nerve Examination: Bilateral ulnar nerve thickening with tenderness was present. Sensory examination was intact. There was no evidence of motor function deficit in the upper or lower limbs.

Histopathological Examination: Skin biopsy from a representative lesion showed mild hyperkeratosis and epidermal atrophy. A clear Grenz zone was noted beneath the epidermis. Deeper to the Grenz zone, the dermis showed dense infiltration by macrophages, elongated histiocytes, and scattered lymphocytes. The presence of elongated histiocytes in the dermis and a Grenz zone strongly supported the diagnosis of histoid Hansen disease.

Final Diagnosis: Based on the clinical presentation, irregular dapsone therapy, peripheral nerve involvement and characteristic histopathological features a final diagnosis of histoid Hansen disease was made.

Case 2: Patient Details and Clinical Presentation

A 59-year-old male presented with complaints of multiple raised solid skin lesions for the past 2-3 months. The lesions were gradually progressive and involved multiple body sites. There was no history of ulceration, pain, or constitutional symptoms.

Past Treatment History: The patient had a history of irregular dapsone monotherapy for 2 months. This treatment history was relevant because histoid leprosy may develop in patients with previous inadequate antileprosy therapy and may represent a highly bacilliferous form of multibacillary Hansen disease.

Cutaneous Examination: On examination, multiple discrete, non-tender, skin-coloured firm nodules measuring approximately 1–2 cm were present over the extremities, trunk, and back. The lesions were firm and papulonodular, occurring over apparently normal skin. The morphology and distribution of lesions were suggestive of histoid leprosy.

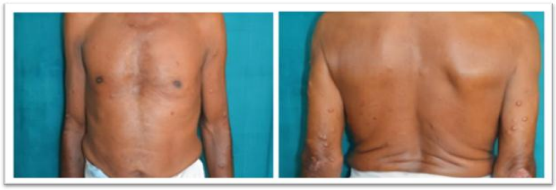

Figure 2: Clinical photographs of Case 2 showing multiple discrete, skin-coloured to hyperpigmented firm nodules over the trunk, back, and upper limbs, with papulonodular morphology suggestive of histoid Hansen disease.

Peripheral Nerve Examination: Bilateral ulnar nerve thickening and tenderness were present. Sensation was intact. No motor function deficit was detected.

Histopathological Examination: Histopathological examination of a representative skin lesion showed mild hyperkeratosis and epidermal atrophy. A Grenz zone was seen beneath the epidermis. The dermis showed infiltration by macrophages, elongated histiocytes, and scattered lymphocytes. These histopathological findings were consistent with histoid Hansen disease.

Final Diagnosis: In view of the multiple firm papulonodular lesions, bilateral ulnar nerve thickening, previous irregular dapsone monotherapy, and characteristic histopathological findings, the patient was diagnosed as a case of histoid Hansen disease

Case 3: Patient Details and Clinical Presentation

A 25-year-old male presented with complaints of multiple raised skin-coloured lesions over the face, ear, and forearm for the past 2 months. The lesions were gradually progressive. There was no history of ulceration, discharge, or systemic complaints.

Past Treatment History: The patient gave a history of irregular dapsone monotherapy for 4 months. This history was important because histoid leprosy has been classically associated with relapse or persistence of disease following inadequate dapsone therapy, although de novo cases may also occur.

Cutaneous Examination: On cutaneous examination, multiple discrete papulonodular lesions measuring approximately 0.5–2 cm were present over the ear, face, and forearm. The lesions were skin-coloured and firm. The papulonodular morphology, involvement of exposed sites, and background history of irregular antileprosy treatment suggested histoid Hansen disease.

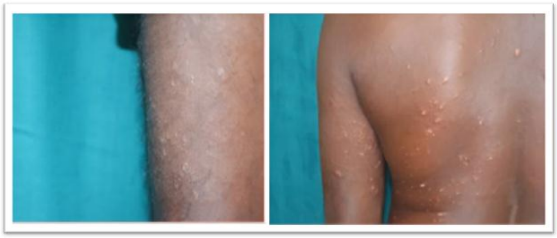

Figure 3: Clinical photographs of Case 3 showing multiple firm, skin-coloured papulonodular lesions over the forearm and trunk, occurring over apparently normal skin in a patient with peripheral nerve involvement.

Peripheral Nerve and Sensory Examination: Bilateral ulnar nerve thickening with tenderness was present. Sensory impairment was noted over both forearms. There was no motor function deficit.

Histopathological Examination: Skin biopsy from a representative lesion showed mild hyperkeratosis and epidermal atrophy. A clear Grenz zone was identified beneath the epidermis. The dermis beneath the Grenz zone showed infiltration by macrophages, elongated histiocytes, and scattered lymphocytes. These findings confirmed the diagnosis of histoid Hansen disease.

Final Diagnosis: Based on the clinical morphology of multiple papulonodular lesions, peripheral nerve involvement along with sensory impairment and history of irregular dapsone monotherapy a diagnosis of histoid Hansen disease was made. characteristic histopathological features confirmed the diagnosis.

Histopathological Examination: Epidermis exhibited mild hyperkeratosis and atrophy. Grenz zone was seen. Deeper to Grenz zone the entire dermis was infiltrated with macrophage, elongated histiocyte and scattered lymphocytes. All the three patients showed the feature of histoid Hansen disease.

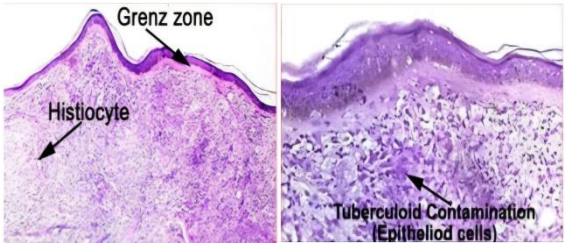

Figure 4: Histopathological photomicrographs of skin biopsy from a representative lesion showing epidermal atrophy with a clear Grenz zone and dermal infiltration by macrophages and elongated histiocytes, consistent with histoid Hansen disease. Focal tuberculoid contamination with epithelioid cell granuloma is also noted.

DISCUSSION

Histoid Hansen disease is a distinctive clinicopathological variant of multibacillary leprosy, originally defined by Wade in 1963 as the "histoid variety" of lepromatous leprosy, and later reviewed in detail by Sehgal VN et al as a form with characteristic clinical, bacteriological, and histopathological features.[8] In the present series, all three patients were males aged 25, 45, and 59 years, and all presented with multiple firm papulonodular lesions over apparently normal skin, which is in agreement with the classic description of histoid lesions as cutaneous or subcutaneous papules, nodules, and less frequently plaques. The morphology in our patients—discrete, firm, skin-coloured to hyperpigmented nodules and papules—closely resembled the descriptions by Gupta, who emphasized skin-coloured succulent nodules and plaques on normal skin, and Karthikeyan K who highlighted the unique clinical, histopathological, and microbiological profile of histoid leprosy.[9] The present cases therefore reinforce the continuing relevance of Wade's entity, particularly in endemic regions where atypical papulonodular dermatoses must be evaluated carefully for Hansen disease.

The distribution of lesions in our cases involved the trunk, back, abdomen, chest, thigh, extremities, face, ear, and forearm. This pattern correlates with the observations of Kaur I et al who reported frequent involvement of thighs/buttocks, arms, back, face, forearms, and legs in their retrospective series of 40 cases from India.[10] Similarly, Nair SP et al described histoid leprosy as presenting with cutaneous lesions over apparently normal skin, with clinical and histopathological findings that support diagnosis.[11] In classical teaching, the lower back and buttocks are among the most common sites, while palms and soles are usually spared; this was consistent with our series, as none of the three patients had palmar or plantar involvement. Earlobe and facial lesions in the third patient were also noteworthy, because exposed-site involvement may lead clinicians toward differential diagnoses such as molluscum contagiosum, sarcoidosis, dermatofibroma, neurofibromatosis, post-kala-azar dermal leishmaniasis, and conventional lepromatous leprosy. Recognition of this distribution is important because histoid leprosy may clinically mimic noninfective nodular dermatoses and may be missed unless specifically considered.

A significant finding in the present series was the history of irregular dapsone monotherapy in all three patients. Murthy SV et al observed that histoid leprosy may emerge after dapsone monotherapy, irregular or inadequate therapy, and possible dapsone resistance, although de novo cases are also increasingly recognized.[12] In contrast, Pandey P et al reported de novo histoid leprosy, showing that prior antileprosy therapy is not mandatory for the development of this variant.[13] Our three cases therefore fall into the classical post-treatment or inadequately treated category rather than the de novo category. This distinction has epidemiological importance because histoid leprosy is highly bacilliferous and may act as a hidden reservoir of infection, a concern emphasized by Palit A et al in the post-elimination era.[14] None of our patients had erythema nodosum leprosum at presentation; this supports the traditional view that histoid lesions are less commonly associated with ENL, although reactions have been documented in larger series.[15] Thus, absence of lepra reaction should not reduce clinical suspicion when morphology and histology are suggestive.

Peripheral nerve involvement was present in all three patients, with bilateral ulnar nerve thickening and tenderness. Sensation was preserved in two patients, while the third had sensory impairment over both forearms; none had motor deficit. This pattern is comparable with the findings of Mathur M et al who reported ulnar nerve enlargement as a frequent neurological finding in histoid leprosy from central Nepal.[16] Mendiratta V et al also stressed that histoid Hansen disease may present with variable clinical expression and must be diagnosed by combining clinical, bacteriological, and histopathological criteria.[17] The relatively preserved sensory and motor function in our patients may reflect early presentation, limited duration of symptoms, or the predominance of cutaneous manifestations. However, the presence of nerve thickening in all cases emphasizes that histoid leprosy should not be regarded as a purely cutaneous disease. Careful peripheral nerve examination remains essential, even when lesions resemble benign nodular dermatoses.[18]

Histopathology was decisive in all three cases. The biopsies showed epidermal atrophy, a clear Grenz zone, dermal infiltration by macrophages and elongated histiocytes, and scattered lymphocytes, consistent with histoid Hansen disease. Canuto MJM et al described spindle cells as the principal histopathological feature of histoid leprosy.[19] Mathur M et al similarly reported epidermal atrophy, well-circumscribed dermal infiltrates, spindle-shaped histiocytes, and positive Fite-Faraco staining in confirmed cases.[20] The term "histoid habitus" refers to spindle-shaped histiocytes containing bacilli arranged in groups or parallel bundles along the long axis of cells. Slit-skin smear typically shows numerous acid-fast bacilli that are longer, uniformly stained, and less often arranged in globi than in conventional lepromatous leprosy. "Tuberculoid contamination," when present, denotes epithelioid cell granulomas, giant cells, and lymphocytic infiltrate resembling tuberculoid leprosy. In the present series, the consistent Grenz zone and elongated histiocytic infiltrate support the diagnosis and distinguish histoid leprosy from lepromatous leprosy and other nodular mimickers. Early Diagnosis and complete multidrug therapy are essential to prevent disability and reduce transmission.

CONCLUSION

Histoid leprosy is a unique type found in Indian subcontinent signifies that it is highly active variant of lepromatous leprosy. As a result of success of Multidrug therapy in place of dapsone monotherapy the incidence reduced drastically. However, the emergence of histoid form shall become a public health challenge in this eradication era.

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