CASE REPORT

A case of lepromatous leprosy presenting with punched out infiltrated plaques, a diagnostic enigma

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Summary  Leprosy has been recognised as a great imitator as it has a wide range of clinical presentations. It is important to recognise its atypical presentation for better management of the patient. Here, we present an interesting case of lepromatous leprosy with multiple punched out, infiltrated plaques which was confirmed by histopathology.

Introduction

Leprosy is a chronic, progressive infectious disease caused by Mycobacterium leprae. It can present as two major polar forms (tuberculoid and lepromatous) and other intermediate variants. Mid-borderline leprosy represents the midpoint in the leprosy spectrum. The most astonishing part of leprosy is that it can present in ample ways making clinical diagnosis an enigma. Sometimes, an unusual presentation delays the diagnosis and management. In that case, histopathology comes to rescue in confirming the diagnosis.

Case summary

A 33 years old male belonging to low socio-economic class presented to our dermatology OPD with loss of sensation on both the extremities for last 15 years. Initially it started on left hand which gradually involved the right hand and feet. There were multiple asymptomatic annular red lesions with loss of sensation for 5 years. He recurrently developed epistaxis, edema over hands and feet along with loosening of footwears on walking.

Patient gave history of recurrent trauma by hammer in his occupation which was associated with recurrent blister and ulcer formation. He had difficulty in motor activities due
to deformities in his hands and feet. For this, the patient took treatment from a local doctor but did not improve.

Before 15 years, he had developed some non-specific itchy light-colored skin lesions on trunk which disappeared after some local treatment (documents unavailable).

Patient did not give any history of fever with transient, red elevated lesions (suggestive of Erythema Nodosum Leprosy) on his body, joint pain or any eye abnormality.

A general examination showed characteristic leprosy face with madarosis, infiltrated ear lobules and a depressed nasal bridge. Cutaneous examination revealed multiple (around 20–25), bilaterally symmetrical, discrete to confluent well-defined, erythematos, infiltrated, hypoesthetic plaques and nodules of varying size and shape on face, trunk and extremities. Of them, many lesions (10–12) were annular with sharply punched out inner margins with sloping outer margins and central normal skin. Glove and stocking anaesthesia was demonstrable in both extremities below elbow and knee. Bilateral ulnar, popliteal and supraorbital nerves were thickened, palpable and tender. Single oval deep ulcer with red base and pus discharge was present on the sole of right foot. Motor loss in the form of wasted thenar

**Figure 1, 2 and 3.** Multiple, punched out erythematous plaques and nodules on trunk; Close up view of punched out erythematous plaques on back.

**Figure 4 and 5.** Claw hand deformity with trophic ulcers on both hands; Amputated left big toe.
and hypothenar eminence, claw hand, amputated left big toe, positive card test, book test, pen test, difficulty in extending the wrist as well as feet and everting the feet against resistance were noted (Figures 1–5).

The patient’s routine investigations were within normal range. A skin smear from his ear lobe failed to demonstrate any lepra bacilli. But Fite faraco stain demonstrated acid fast bacilli. Fine needle aspiration cytology (FNAC) from an infiltrated plaque on his back revealed, few epitheloid cells and fibroblast suggestive of borderline leprosy. Dermoscopy of a punched out plaque with Dermlite IV illustrated light pink annular structure with a central clear area. Based on cutaneous morphology of the lesion and cytology report, we suspected the diagnosis of mid-borderline leprosy (Figures 6 and 7).

But, histopathology from the lesion on trunk confirmed the diagnosis as lepromatous leprosy by the presence of atrophic epidermis, sub-epidermal clear grenz zone, foamy histiocytes and macrophage granuloma following neurovascular bundles in deep dermis (Figures 8, 9 and 10).

Thus a final diagnosis of lepromatous leprosy was confirmed and the patient was started on standard multidrug regimen (rifampicin, dapsone and clofazimine), oral corticosteroids and higher antibiotics for trophic ulcers of feet. Following which, patient improved in numbness, edema and ulcers.

Figure 6 and 7. Fine needle aspiration cytology from the nodule exhibited multiple elongated epitheloid cell (slipper shaped); Dermoscopy from the punched out erythematous plaque revealed light pink annular area with central normal skin.

Figure 8, 9 and 10. Histopathology with H & E stain at 40x revealed thin atrophic epidermis, grenz zone and granulomatous infiltrate of foamy histiocytes and sparse lymphocytes; Fite Faraco stain from the plaque at 40x showed thin atrophic epidermis, grenz zone and foamy macrophages in the dermis; Fite Faraco stain at 40x presented foamy macrophages following nerve bundle.
Discussion

Hansen’s disease is a chronic bacterial infection mainly affecting skin and nerves. It presents with diverse manifestations ranging from just an anaesthetic patch to multi-system involvement and deformities. To simplify the diagnosis, till date there are many classifications available based on cutaneous morphology and number of lesions.1,2

Spectrum of leprosy is not just based on clinical examination but it also relies upon histology, bacteriology and immunologic status. But the correlation between histo-bacterio-immune parameters is less than 50%. So, deciding the pole of leprosy spectrum has always been a challenge for dermatologist.3

Mid-borderline leprosy represents the middle of leprosy spectrum with dimorphous skin lesions of both the extreme poles. This explains the presence of both erythematous plaques and nodules in our patient. It is the most unstable form which usually downgrades if left untreated. It is well accepted that erythematous, annular plaques with central punched out appearance and outer sloping margins resembling ‘swiss cheese’ are characteristic of mid-borderline leprosy.1,4

Lepromatous leprosy is the severe and generalised form of leprosy with a wide range of presentations based on the bacillary load. But the most typical presentation is multiple, symmetrical, erythematous, papules, plaques and nodules on trunk and extremities.2,5

Our patient presented with multiple types of skin lesions of all sizes and shapes like plaques, annular plaques and nodules mimicking dimorphous lesions of mid borderline leprosy. Cytology findings from the lesion also favoured BB leprosy. Literature states that deformities occur more with the borderline spectrum of leprosy.5 Glove and stocking anaesthesia as well as symmetrical nerve involvement in our patient again favoured lepromatous leprosy. So, this posed a diagnostic dilemma. But, histopathology and fite faraco stain findings were indicative of LL by the presence of foamy histiocytes and presence of bacilli respectively.

Radhika et al. in her study has mentioned that there is relatively less correspondence between FNAC and histopathology in the case of BB. In our case, FNAC report was suggestive of borderline leprosy while histopathology confirmed the diagnosis of lepromatous leprosy. Singh et al. established the FNAC criteria of borderline leprosy as ill-defined granuloma of epitheloid cells and macrophages while that of lepromatous leprosy by the presence of numerous foamy macrophages.7,8

A possible explanation of this presentation in our case is that as BB leprosy is unstable, it will soon downgrade to LL if untreated. Thus, a diagnostician must be aware of all classic and atypical presentations of leprosy to avoid delay in diagnosis. Skin smear positivity in fite faraco stain, symmetrical cutaneous and peripheral nerve involvement and histopathology are indicative of LL in our case. Here, we report an unusual annular punched out plaque presentation of lepromatous leprosy where histopathology played a key role in confirming the diagnosis.

References


