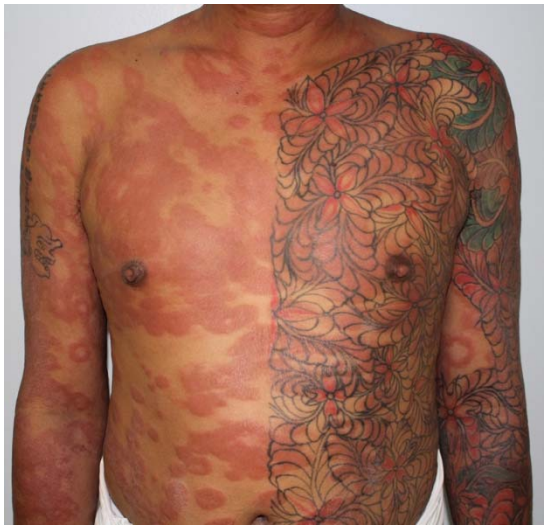
Case 23.1
A 28 year-old Thai man from Khonkean

Chief complaint: Generalized erythematous edematous plaques on the trunk, face, and extremities for 2 weeks


(Fig. 23.1.1)

Present illness:
1 year ago, he initially developed the skin lesions on trunk, face and all extremities, and had been treated with unknown intravenous medication. The eruption partially resolved with oral antihistamine.
2 weeks ago, the lesions became more erythema and edema, then he visit our clinic.

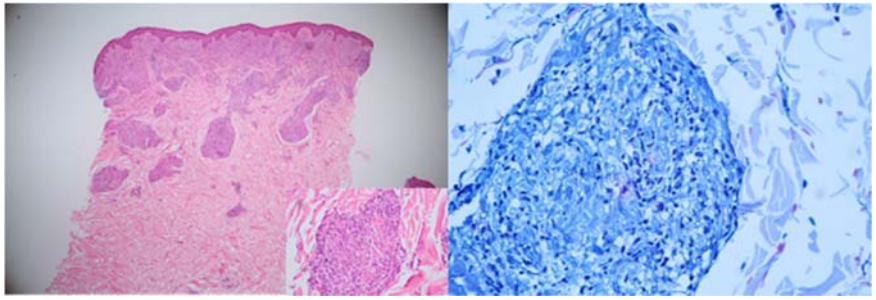
Past history: No underlying disease

Physical examination:
A healthy man
Vital signs: Normal
HEENT: No facial palsy, no lagophthalmos, no lymphadenopathy
Extremities: No joint deformity, no foot drop
Neurological system: Pinprick sensation impairment at lesional skin, no peripheral nerve enlargement, no muscle atrophy

Dermatological examination: (Fig. 23.1.1)
Generalized well-defined erythematous edematous plaques, some lesions arrange in annular pattern with decrease pinprick sensation, on the trunk, face, and extremities

Investigations:
- Slit skin smear: Right ear 4+, left ear 4+, right arm 4+
- CBC, liver and renal functions were normal.

Histopathology: (S16 - 32090, Abdomen) **(**Fig**.** 23**.**1**.**2**)**


(Fig. 23.1.2)

- Dense nodular infiltration of epithelioid and foamy histiocytes, intermingle with lymphocyte, plasma cells, forming tuberculoid

and sarcoidal granuloma along adnexal structure, including nerve bundle and arrector pili muscle
- Focally positive Fite staining

Diagnosis: Borderline lepromatous leprosy with type 1 reversal reaction

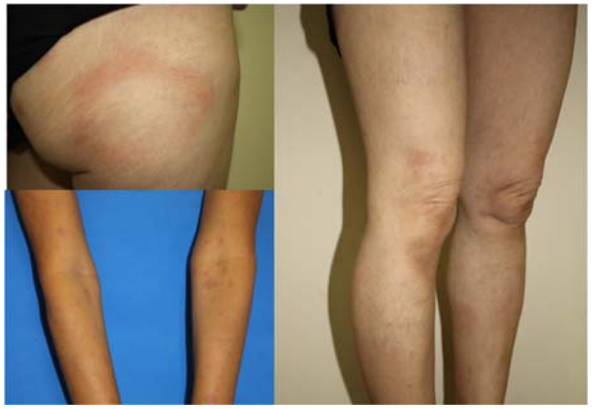
Treatment:
- Multibacillary regimen: Rifampin 600 mg monthly, clofazimine 300 mg monthly and 50 mg daily, and dapsone 100 mg daily
- Oral prednisolone 60 mg daily (1 mg/kg/day)

Presenter: Kulsupa Nimmannitya, MD

Consultant: Ruedee Phasukthaworn, MD

Case 23.2
A 51 year-old Thai female from Phayao

Chief complaint: Multiple erythematous nodules and plaques on the buttock, and extremities for 5 weeks


(Fig. 23.2.1)

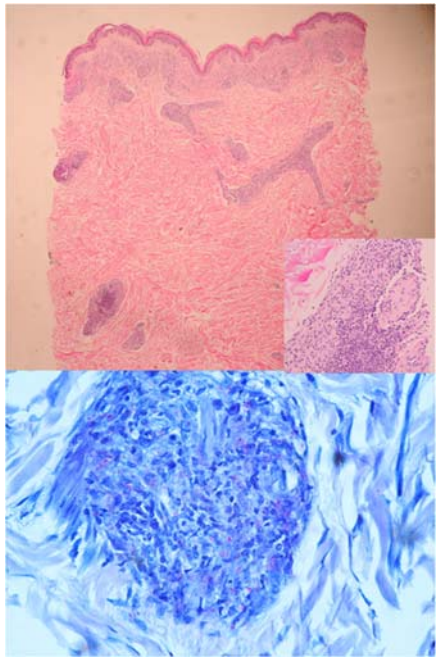
Present illness: The erythematous rash initially appeared on the buttock and both legs without any symptoms for 5 weeks ago, then extended to the trunk. She has never been treated before and denies history of contact with leprosy patient.

Past history: No underlying disease

Physical examination:
A healthy female
Vital signs: Normal
HEENT: No facial palsy, no lagophthalmos, no lymphadenopathy
Extremities: No joint deformity, no foot drop
Neurological system: Intact sensation, no peripheral nerve enlargement, no muscle atrophy

Dermatological examination: (Fig**.** 23**.**2**.**1**)**
Multiple scattered ill-defined erythematous nodules on trunk and all extremities, with an annular erythematous plaque on the right buttock

Histopathology: (S17 - 26208, Right buttock) (Fig. 23.2.2)


(Fig. 23.2.2)

- Dense nodular infiltration of epithelioid and foamy histiocytes, intermingle with lymphocyte, plasma cells, forming tuberculoid

and sarcoidal granuloma along adnexal structure, including nerve bundle and arrector pili muscle
- Strongly positive Fite staining

Investigations:
- Slit skin smear: Right ear 4+, left ear 2+, right elbow 5+, left arm -ve
- CBC: Normal

Diagnosis: Borderline lepromatous leprosy

Treatment
- Multibacillary regimen: Rifampin 600 mg monthly, clofazimine 300 mg monthly and 50 mg daily, and dapsone 100 mg daily

Presenter: Kulsupa Nimmannitya, MD

Consultant: Ruedee Phasukthaworn, MD

Discussion:

Leprosy is a chronic granulomatous bacterial infection caused by *Mycobacterium leprae* with primarily affect the skin and peripheral nerves.[1] Based on the immunological response of the host to *M.leprae*, leprosy is classified into 5 major types: tuberculoid (TT), borderline tuberculoid (BT), mid-borderline (BB), borderline lepromatous (BL), and lepromatous (LL) forms.[2] A high cellular immune response is seen in cases with TT type, resulting in a low bacillary load and only a few skin lesions. Patients with the LL type have a high bacillary load and many diffuse skin lesions. The border line types are immunologically unstable and can be complicated by reversal reaction. Patients with borderline states have symptoms of both the TT and LL forms. Diagnosis is based on clinical signs, slit-skin smear test, and skin biopsy. Currently, *M.leprae* can be detected by polymerase chain reaction and serologic assays for anti-PGL antibodies by means of the enzyme-linked immunosorbent assay (ELISA), which is useful in only multibacillary cases[1]

Leprosy reactions, including type 1 reversal reaction (T1R) and type 2 reaction (erythema nodosum leprosum), are induced by the host immune responses to *M.leprae*, and may occur before, during or after treatment.[3] T1R is a result of delayed hypersensitivity. It occurs in borderline patients and presents as erythema and edema of pre-existing skin lesions or the development of new lesions, and neuritis. Type 2 reaction is due to the formation of immune complexes in association with an excessive humoral reaction. It typically occurs when patients with lepromatous forms undergo treatment. It represents a small vessel vasculitis (cutaneous and systemic), of which the most common clinical manifestation is erythema nodosum leprosum (ENL). There are general symptoms, such as fever, malaise, myalgia, edema, arthralgia, and lymphadenitis. Internal involvement, such as liver or kidney damage, may also occur.[2] Neuritis may occur as part of ENL, but may be less dramatic than in T1R.[4]

Our patients were diagnosed as borderline lepromatous leprosy owing to the presence of compatible skin lesions which are multiple, asymmetry, infiltrative and punch-out lesions (with hypoesthesia occurred in the first patient), positive slit skin smear, and histopathological result. Additionally, the first patient had acute onset of erythema and edema of pre-existing skin lesions that made the suspicion of T1R, although the neuritis was absent at the time of diagnosis.

In terms of the treatment, the WHO recommends multidrug

therapy (MDT) with rifampicin, clofazimine, and dapsone for first-line treatment of leprosy.[5] Multibacillary (MB) cases, as our patients, are treated with rifampin 600 mg monthly, clofazimine 300 mg monthly and 50 mg daily, and dapsone 100 mg daily for 2 years.[6] In cases of T1R, MDT should be maintained and added by oral prednisolone (1-1.5 mg/kg/day). Treatment of T1R aims to control the acute inflammation and avoid possible nerve damage. The dose and treatment duration for the steroids should be adjusted according to clinical response. General improvement can occur within three months or take over six months.[7] Patients with recent neural lesions (less than 6 months in duration) respond better than those who undergo therapy at later stage.[7]

References:

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