The Post Kala-azar Dermal Leishmaniasis (PKDL) Atlas
A Manual for Health Workers

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Preface

Post Kala-azar dermal leishmaniasis (PKDL) is a well-recognized complication of visceral leishmaniasis (VL) or kala-azar. It has been described since the beginning of the 20th century both in Asia and Africa, in areas where Leishmania donovani is the causative parasite. Its potential role in the transmission of kala-azar in particular in the interepidemic periods has been suggested many years ago and this was supported by feeding experiments in sandflies. Yet, PKDL has been neglected both from a clinical and an epidemiological point of view. This is partly caused by the difficulty in recognizing PKDL and making a firm diagnosis. The clinical spectrum varies and the list of differential diagnoses is extensive. We believe a reasonable diagnosis of PKDL can be made on clinical grounds only on the basis of a good clinical assessment by which differential diagnoses can be excluded.

This manual aims to be a guide to better and earlier recognition of PKDL by those who work in the field in remote areas. It may also be of use in the teaching of health workers at all levels.

Geneva, August 2012

Note
While the diagnosis of PKDL and the conditions shown in the differential diagnosis was often confirmed, in others it is a clinical diagnosis based on experience. Despite this, we feel that this atlas reflects current clinical practice. There is a great need for further studies to develop and to evaluate a clinical algorithm for PKDL and to develop simple and accurate tools that can be used under field conditions. The same would of course apply to the differential diagnosis.

As PKDL is common in Sudan and has been well described, the differential diagnosis of macular and papular/nodular PKDL is discussed extensively in the chapter on PKDL in Sudan. For other areas only the most common conditions encountered or conditions specific for that area are presented.
1. Introduction
Map. Visceral Leishmaniasis endemic countries and occurrence of PKDL

- Endemic countries with no PKDL or sporadic cases only.
- PKDL rate ≤ 5%
- PKDL rate 6-10%
- PKDL rate 11-20%
- PKDL rate > 20%

PKDL reported in HIV-Leishmania co-infected patients

Design: J. Alvar-Beltrán.
Post-kala-azar dermal leishmaniasis (PKDL) is a complication of visceral leishmaniasis (VL) or kala-azar. It is common in areas endemic for VL caused by *L. donovani*. These include countries in Africa in particular Sudan and in Asia, Bangladesh and to a lesser extent India. PKDL may also sporadically occur in *L. infantum* or *L. chagasi* endemic areas, mainly the Mediterranean countries and Latin America.

The condition is characterized by the occurrence of a skin rash after an episode of VL; the interval varies according to the endemic area. The rash is usually in the face, from which it may or may not spread to other parts of the body. In contrast to VL, the patient is not ill and PKDL is not fatal. In the Sudanese type, self cure is the rule while in Bangladesh and India, all cases are treated.

Risk factors for PKDL are not well known; previous treatment of VL with inadequate dosage of drug and the drug used, malnutrition, HIV infection and young age may play a role.

The importance of PKDL is twofold:

- **Clinical**: patients develop a rash that may last for weeks or months; in particular in small children, the rash may become generalized and severe with mucosal lesions in the mouth, causing general discomfort.

- **Epidemiological**: smears or biopsies taken from the lesions may show *Leishmania* parasites and there is evidence that the sandfly vector may take up these parasites while taking a blood meal and thus PKDL patients may play an important role in transmission (anthroponotic transmission). It is thought that VL occurs in cycles with epidemics of thousands of cases, followed by a period of seemingly low transmission. It is likely that chronic PKDL patients who harbour parasites may play an important role in subsequent upsurges in VL cases.

Diagnosis is usually clinical by the triad of the typical rash, its distribution and the previous episode of VL. There are however, often difficulties and exceptions: many patients do not have a previous episode of VL and the rash may mimic other common skin conditions. In addition, the presentation in Africa and Asia is quite different with the maculopapular form and typical spread being the most common in Sudan and the macular form being much more common in Bangladesh, often with a more atypical distribution.

Parasites may be found in the lesions but this requires a skin smear or biopsy; in papular or nodular PKDL the parasites can usually be demonstrated but in the macular type they are scanty. Serological diagnosis is not very helpful as most patients will have a previous history of VL and antibodies may persist as a result and therefore a positive test may be difficult to interpret.
PKDL occurs when the immune response to *Leishmania* parasites changes from a Th$_2$ dominated response to a mixed Th$_1$/Th$_2$ response under the influence of drug treatment or spontaneously. This change in immune response may be further manipulated by adding an immunomodulator to drug treatment, thus promoting cure.

The management of PKDL differs: in Sudan most cases self heal; the most severe are treated usually with 6-8 weeks of sodium stibogluconate (SSG). In Bangladesh all cases are treated with 6 cycles of monthly SSG (20 days of SSG injections and 10 days drug free period). PKDL in immunocompromised patients is always treated and probably liposomal amphotericin B is the best drug. For all areas there is a need for better identification of who needs treatment and shorter, more effective and cheaper regimens.

Table 1: Differences between PKDL in Sudan and in the Indian subcontinent$^1$

<table>
<thead>
<tr>
<th></th>
<th>Sudan</th>
<th>Indian subcontinent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epidemiology</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest rate reported in field study</td>
<td>4.8/100</td>
<td>4.8/1000</td>
</tr>
<tr>
<td>Maximum reported PKDL rate after VL</td>
<td>60%</td>
<td>10-20%</td>
</tr>
<tr>
<td>Interval after VL</td>
<td>0-6 months</td>
<td>0-3 years</td>
</tr>
<tr>
<td><strong>Clinical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Most common presentation</td>
<td>polymorphic, papular</td>
<td>polymorphic, papulonodular, macular</td>
</tr>
<tr>
<td>Typical distribution</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Face-arms/chest-legs</td>
<td>yes</td>
<td>face and body, face nearly always</td>
</tr>
<tr>
<td>Sun-exposed areas</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Spontaneous cure</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>May occur with visceralized disease</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>May occur without previous VL</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>May occur while on Rx for VL</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>May have other post KA manifestations</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>(uveitis, conjunctivitis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genital lesions</td>
<td>uncommon</td>
<td>common</td>
</tr>
<tr>
<td>Mucosal lesions</td>
<td>rare</td>
<td>not uncommon</td>
</tr>
</tbody>
</table>

$^1$ adapted from ref 1.
2. PKDL in Africa: clinical presentation and differential diagnosis

a. PKDL in Sudan
   • Macular PKDL
   • Macular PKDL and differential diagnosis
   • Papular and nodular PKDL
   • PKDL grading system
   • Severe PKDL
   • Differential diagnosis of papular and nodular PKDL
   • Chronic PKDL
   • Other post-kala-azar manifestations
   • Evolution

b. PKDL in Ethiopia
   • Papular rash
   • Differential diagnosis
In Africa, PKDL by far mostly occurs in Sudan. It is much less common in Ethiopia, Kenya or Uganda. The reason for this is not clear; differences in the parasite or the genetic background of the population may be of importance. Up to 50-60% of VL cases develop PKDL, usually within 0-6 months after treatment. Some patients do not have a previous history of VL and probably had subclinical VL infection.

Clinical presentation
In contrast with VL, the patient is generally well, except in severe cases. The initial presentation is usually with some papules around the mouth; these increase in number and size and spread further to cover most of the face. The most common presentation is a maculopapular rash with papules occurring on a macular background. The papules may be small resembling measles; others increase in size and may be called nodules; these may become confluent. PKDL is often described in 3 grades of density and spread of lesions. Patients may present with a macular rash only, but this is much less common as e.g. in Bangladesh. The macular rash seems not to follow the classical spread as in the papulonodular form. Other more uncommon presentations include a patchy distribution of plaques and the verrucous type. Ulceration is not a feature, but there may be sloughing of heavily affected parts of the skin; in case of mucosal involvement ulcers may form. The skin may become quite dry with scaling.

<table>
<thead>
<tr>
<th>Papular/nodular rash</th>
<th>Macular rash</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cutaneous leishmaniasis</td>
<td>Leprosy</td>
</tr>
<tr>
<td>Leishmaniasis recidivans</td>
<td>Vitiligo</td>
</tr>
<tr>
<td>Diffuse Cutaneous Leishmaniasis (DCL)</td>
<td>Pityriasis versicolor</td>
</tr>
<tr>
<td>Mucosal leishmaniasis</td>
<td>Tinea corporis</td>
</tr>
<tr>
<td>Miliaria rubra (prickly heat)</td>
<td>Tinea barbae</td>
</tr>
<tr>
<td>Leprosy</td>
<td>Pityriasis alba</td>
</tr>
<tr>
<td>Lupus vulgaris</td>
<td>Discoid lupus erythematous</td>
</tr>
<tr>
<td>Measles and other viral infections</td>
<td>Onchocerciasis</td>
</tr>
<tr>
<td>Acne</td>
<td>Burn scars</td>
</tr>
<tr>
<td>Neurofibromatosis</td>
<td>Birth marks</td>
</tr>
<tr>
<td>Urticaria pigmentosa/ mastocytosis</td>
<td>Pellagra</td>
</tr>
<tr>
<td>Darier’ disease</td>
<td>Chloasma</td>
</tr>
<tr>
<td>Scabies</td>
<td></td>
</tr>
<tr>
<td>Discoid lupus erythematous</td>
<td></td>
</tr>
<tr>
<td>Granuloma multiforme</td>
<td></td>
</tr>
<tr>
<td>Granuloma annulare</td>
<td></td>
</tr>
<tr>
<td>Lichen planus</td>
<td></td>
</tr>
<tr>
<td>Mollusca contagiosa</td>
<td></td>
</tr>
<tr>
<td>African histoplasmosis</td>
<td></td>
</tr>
<tr>
<td>Keloids</td>
<td></td>
</tr>
<tr>
<td>Tuberous sclerosis</td>
<td></td>
</tr>
<tr>
<td>Mycosis fungoides</td>
<td></td>
</tr>
<tr>
<td>Infantile eczema</td>
<td></td>
</tr>
<tr>
<td>Psoriasis</td>
<td></td>
</tr>
</tbody>
</table>
As a rule, after treatment or spontaneous cure, the skin fully recovers without scarring. In those who have chronic PKDL, often for many years, depressed scars may develop or the skin may become fibrotic.

PKDL may develop while still on treatment for VL or patients may present with visceralized disease. This is called para-kala-azar dermal leishmaniasis. These cases may be clinically ill, with fever, splenomegaly etc. Similarly, PKDL may coincide with leishmanioma.

There are other post-kala-azar manifestations that may occur concomitantly with PKDL; of these uveitis and conjunctivitis are the most common. These conditions are often not recognized and may lead to blindness. As in PKDL, parasites persist in the eye for unknown reasons and the developing immune response causes inflammation and destruction. Similarly, post-kala-azar mucosal leishmaniasis in the nose has been described.
1-5 Macular PKDL mainly around the mouth and spread to other parts of the face.
PKDL in Sudan

Macular PKDL

THE PKDL ATLAS. A Manual for Health Workers
Macular PKDL

6 Macular rash affecting the “butterfly” area.

7 Symmetrical hypopigmented patches resembling lepromatous leprosy (see also fig. 131).
Macular lesions mainly on the trunk.

Close-up of the abdomen.

Same patient; lesions on the upper legs.
Macular PKDL and differential diagnosis

11 Pityriasis alba.

12 Tinea corporis.
Scattered hypopigmented patches.

13 Pityriasis versicolor.
Usually more common in the trunk than in the face. This patient also had VL and the rash disappeared with stibogluconate treatment for VL only, suggesting an increased susceptibility for this fungal skin infection during VL.
Discoid lupus erythematosus: healed scars on both cheeks and upper lip (14, 15, 16)
Discoid Lupus Erythematous.
Symmetrical depigmented inflammatory lesions in the butterfly area and arms (17, 18).
19 Discoid lupus erythematosus. Hypopigmented lesions.

20 Discoid lupus erythematosus. Hypopigmented and hyperpigmented lesions may co-exist.
Macular PKDL and differential diagnosis

21 Lupus vulgaris. Violaceous infiltrated ulcerating plaque with atrophic scarring in the center.

22 Burn scars. Depigmented irregular scars caused by previous burn injury.
Segmental or zosteriform vitiligo.
Clinical clues: Depigmented lesions, asymmetrical in the distribution of a dermatome(s). This type may occur in younger patients. The dark vertical lines on both cheeks are tribal markings.

Table 3: Differential diagnosis of macular PKDL and vitiligo

<table>
<thead>
<tr>
<th></th>
<th>Macular PKDL</th>
<th>Vitiligo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family members</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td></td>
<td>similar exposure to VL</td>
<td>genetic</td>
</tr>
<tr>
<td>Predilection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>face</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>acra</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>central back</td>
<td>affected</td>
<td>sparing central back</td>
</tr>
<tr>
<td>Appearance</td>
<td>hypopigmented</td>
<td>depigmented</td>
</tr>
<tr>
<td>Bordering skin</td>
<td>normal</td>
<td>sometimes hyperpigmented</td>
</tr>
<tr>
<td>Other skin abnormalities</td>
<td>macules may be erythematous; papules, nodules</td>
<td>none</td>
</tr>
<tr>
<td>Sparing of most pigmented areas (axillae, inguinal area)</td>
<td>yes</td>
<td>no</td>
</tr>
</tbody>
</table>
Vitiligo.

Clinical clues: The macules are depigmented and not hypopigmented as in PKDL (see Table 3). The age of the patient also suggests vitiligo, rather than PKDL. Note that some of hairs in the beard and moustache are white.
Scleroderma.
Clinical clues:
Symmetrical hypopigmented lesions, taut skin, reduced mouth opening, impairment in movement of the fingers.
Onchocerciasis.
Clinical clues: leopard skin with hypopigmented macules (32, 33); look for onchocercomata (34, arrow) and scratch marks (35).
36 Borderline leprosy.
Note the raised edge, the area of hypopigmentation with central repigmentation.
Clinical clues: look for other signs of leprosy: anaesthetic patches, thickened nerves. See Table 4.

**Table 4: Differential diagnosis of PKDL and leprosy**

<table>
<thead>
<tr>
<th></th>
<th>PKDL</th>
<th>Leprosy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epidemiology</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Most important age group</td>
<td>young children</td>
<td>older individuals</td>
</tr>
<tr>
<td>Frequency in endemic areas</td>
<td>common</td>
<td>uncommon</td>
</tr>
<tr>
<td><strong>Clinical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lesions</td>
<td>M, P, N, plaques</td>
<td>M, P, N, plaques</td>
</tr>
<tr>
<td>Symmetrical lesions</td>
<td>yes</td>
<td>indeterminate, tuberculoid: no</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lepromatous: yes</td>
</tr>
<tr>
<td>Uniform in size</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>Single lesion</td>
<td>uncommon</td>
<td>common in undetermined and tuberculoid leprosy</td>
</tr>
<tr>
<td><strong>Neurological features</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- clinical</td>
<td>none</td>
<td>anaesthetic patches, thickened nerves, nerve palsies, loss of sweating</td>
</tr>
<tr>
<td>- pathological</td>
<td>neuritis in cutaneous nerves</td>
<td>id</td>
</tr>
<tr>
<td>Lobulation of ears</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Madarosis</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>Predilection sun exposed parts</td>
<td>yes</td>
<td>no (cooler parts)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin slit smear</td>
<td>Leishmania amastigotes (Giemsa stain)</td>
<td>acid-fast bacilli (modified ZN stain)</td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-cure</td>
<td>yes (the rule in Sudan)</td>
<td>no</td>
</tr>
<tr>
<td></td>
<td>no (Asia)</td>
<td></td>
</tr>
</tbody>
</table>

M macules, P papules, N nodules.
Macular PKDL and differential diagnosis

37 Cutaneous leishmaniasis scars.
Hyperpigmented depressed scars of previous cutaneous leishmaniasis ulcers. The longitudinal scars on the cheek are tribal markings.

38 Prayer marks in a muslim man.
Note the hyperpigmentation on the forehead caused by frequent pressure exerted while praying.
Pseudomelanosis. Although kala-azar means the “black disease”, which refers to hyperpigmentation of the skin found in Indian kala-azar, this hyperpigmentation is not found in Sudanese patients. In these 2 confirmed kala-azar cases, the black discoloration was caused by dirt and could be removed with water and soap.
PKDL in Sudan: Papular and nodular PKDL

Papules in various stages of development and density and of various sizes; the initial localization around the mouth is typical.
Micropapular rash.
Papular and nodular PKDL

45 Macropapular rash.
Further spread of the lesions to the nose, around the eyes and the forehead.
Further spread of the lesions to the nose, around the eyes and the forehead.
Papular rash with increasing density.

Papular and nodular PKDL
Papular and nodular PKDL

Maculopapular rash.
57 Maculopapular rash.
Papular PKDL, covering the whole face.
Papular PKDL, covering the whole face. Note verrucous plaques over eyebrows (60).

Nuer tribesman from South Sudan. Note absence of lesions on forehead: the horizontal lines are tribal markings and fibrotic changes may prevent PKDL papules to develop.
2. PKDL in Africa: clinical presentation and differential diagnosis

Micropapular rash, measles-like.
Hyperpigmented papules.
Papular and nodular PKDL

66 Nodular lesions.
PKDL in Sudan

Papular and nodular PKDL

Nodular lesions.
Papular and nodular PKDL

Papules and nodules become confluent to form plaques.
Patient with three solitary plaques on forehead (73), chin (74) and earlobe (121).
Papular and nodular PKDL

75 Plaques in the face (Figures 75-79).
Papular and nodular PKDL
PKDL in Sudan

Papular and nodular PKDL

78
Papular and nodular PKDL
Grade 1.1 Lesions only in the face and restricted to area around the nose and mouth with normal skin in between.

Table 5: Grading system of PKDL in Sudan

<table>
<thead>
<tr>
<th>Grade</th>
<th>Distribution</th>
<th>Density</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>face mainly with some lesions on trunk and arms</td>
<td>scattered lesions</td>
</tr>
<tr>
<td>2</td>
<td>face, upper parts of trunk, arms and legs affected, gradually becoming less distally; hands and feet free</td>
<td>moderate density with normal skin in between</td>
</tr>
<tr>
<td>3</td>
<td>all over body; including hands and feet</td>
<td></td>
</tr>
</tbody>
</table>
Grade 1.1.
Transition into Grade 1.2 (Figures 81-82).

Grade 1.3.
Dense maculopapular rash, but mainly on the face.
Grade 1.3 The whole face is affected with a dense papular rash (84) and macular rash (85) with few papules in other areas.
PKDL grading system

Grade 2.1
Papular rash and leishmaniommas (arrows). In this patient there was no previous history of VL.

Grade 2.2.
Papular rash. Most parts of the body are affected; the rash is dense but still normal skin can be seen.
**Grade 2.2.** Most of the trunk is also involved.
PKDL grading system

Grade 3.2.
Macular rash.

Grade 3.2.
Combined macular and papular rash.
92 Grade 3.2 Maculopapular rash.
PKDL grading system

Grade 3.3.
Mainly hyperpigmented papules.
Grade 3 macular PKDL.
In macular PKDL, the rash does not follow the normal distribution as in papular/nodular PKDL. The face and lower arms may be affected with little involvement of the trunk (95, 96).
Grade 3 macular rash
Alternatively, the trunk may be affected with few or no lesions on the face and arms (97, 98). Grading is therefore difficult.
Grade 3.3
Maculopapular rash.
PKDL grading system

**100 Grade 3.3.** Papulonodular rash.
101 Grade 3.3 Micropapular rash.
2. PKDL in Africa: clinical presentation and differential diagnosis

**PKDL grading system**

102

**Grade 3.3**
Close-up: dense maculopapular rash.

103

**Grade 3.3**
Papulonodular rash.
All parts of the body are involved with plaques around the mouth and on eyebrows.
Severe PKDL grade 3 in the face with crusts and sloughing of the skin.
Severe PKDL

105

Severe PKDL grade 3 in the face with crusts and sloughing of the skin.

106
Severe PKDL grade 3. (Figures 107-112).
Severe PKDL
Severe PKDL
Severe PKDL
Grade 3.
Desquamation of the skin.
PKDL also affects the hands (back and palm).
PKDL affecting the earlobe.
PKDL in Sudan

120

PKDL affecting the earlobe; see also fig. 85.

121

122

123
PKDL in the genital area. The ulcerative lesions may be leishmaniomias.
**Miliaria rubra.**
Clinical clues: young child, wrapped in many layers of cloth, despite hot weather conditions; typically tiny papules on the forehead and not around the mouth as in PKDL; may be itchy.

**Acne.**
Clinical clues: adolescent age, different stages of development, greasy skin, comedones (papules with white head).

*Note that some photographs included in this chapter are from other countries than Sudan for comparison*
Differential diagnosis of papular and nodular PKDL

Clinical clues: anaesthetic skin patches, thickened nerves, e.g. greater auricular nerve, peripheral nerve palsies, such as claw hand (ulnar nerve) and wrist drop (radial nerve) with destruction of phalanges.

Top left: the variation in size of papules, nodules and plaques is not seen in PKDL.
Top middle: lepromatous leprosy (cf. Fig 7).
Top right: tuberculoid leprosy: the great auricular nerve is clearly visible and palpable.
Middle left: wrist drop and destruction of phalanges. Middle right: wrist drop and claw hand. Bottom: collapse of the nose; not seen in PKDL.

See also Table 4.
Lepromatous leprosy.

Clinical clues:
Elderly person, madarosis, lesions in different stages of development, preference for cooler body parts: upper arms rather than lower arms that are exposed to sunlight.
Differential diagnosis of papular and nodular PKDL

Lepromatous leprosy.
Details of Fig 136.
Lues stage II.
Clinical clues:
History of genital ulcer; as in PKDL, palms and soles may be involved.
Differential diagnosis of papular and nodular PKDL

**Keloids.**
Clinical clues:
There is thickening of the skin due to fibrosis, typically in a scar. Surgical removal usually results in a (more severe) relapse.
Herpes zoster. Clinical clues: This is typical in a dermatome and may present with vesicles on an erythematous background in a white skin; in a black skin, mainly vesicles are seen that may break down. It is common in Africa in young people as an early sign of HIV infection.

143 Herpes zoster in a kala-azar patient.

144 Herpes zoster scar in ophthalmic branch of the trigeminal nerve.
Differential diagnosis of papular and nodular PKDL

Herpes zoster in white skin; vesicles on erythematous background.

Herpes zoster in the black skin; because of the different skin structure, the vesicles are less likely to break down. (147, 148).

Varicella (chickenpox).
Clinical clues:
Vesicles in various stages of development; evolves over days.
Multiple tricho-epitheliomas
(presumed diagnosis; differential diagnosis: cylindroma, syringoma, adenoma sebaceum).
Predilection for central part of face; may run in families: the patient's brother had the same condition.

Dermatosis papulosa nigra.
Common in the black skin, mostly in upper part of face, cheeks and temples. Numbers of papules increase with age; starts during adolescence.

Keloid after varicella in childhood; now presents with herpes zoster scar.
Differential diagnosis of papular and nodular PKDL

Mollusca contagiosa in an HIV-positive patient.
Noma or cancrum oris in a VL patient who had relapsed after treatment; note the splenomegaly.
Differential diagnosis of papular and nodular PKDL

Measles.
The micropapular rash is difficult to distinguish from PKDL.

Measles-like PKDL.
Measles.

In measles, the rash can be virtually indistinguishable from the micropapular rash in PKDL. The rash may also desquamate (159) (see also 113-115).

Clinical clues: look for other signs of measles such as fever, cough, conjunctivitis (158), Koplik’s spots (160) and otitis media (discharge of pus probably as a result of a perforated eardrum, 161).
Differential diagnosis of papular and nodular PKDL

[Image: Close-up of a hand with lesions]

162 **Scabies** Typical interdigital lesions (162); may also be more widespread (163).
PKDL in Sudan

Differential diagnosis of papular and nodular PKDL

African histoplasmosis.
Differential diagnosis of papular and nodular PKDL

**Neurofibromatosis.**

Clinical clues:
- Family history: in this case, his grandmother, father and his 2 siblings also had the disease compatible with autosomal dominant inheritance.
- Chronic slowly progressing. Nodules in different sizes, also affecting the scalp.
- The nose and earlobes remain free. Note the multiple and giant nodules on the abdomen; this is not seen in PKDL.
168 Endemic syphilis. macular lesions with raised edge.

169, 170 Yaws. nodular (from the Democratic Republic of Congo). Note atypical distribution on face and extremities, leaving chest clear.

171 Late yaws: gangosa.
Kaposi’s sarcoma
in the face and on
the chest in HIV
positive patients.
They usually begin
as macular lesions
that become
elevated as plaques;
typically purple
in color.
Sporotrichosis.
Spread along the lymphatics. While this is also seen in cutaneous leishmaniasis, it is not a feature of PKDL.
Differential diagnosis of papular and nodular PKDL

176
Shilluk tribe.

Tribal markings.

Nuer tribe.

177
Nuer tribal markings, with PKDL papules occurring between the markings.
Differential diagnosis of papular and nodular PKDL

Nuer tribe. The papules do not appear on the forehead, perhaps because of fibrosis caused by scarring.

Nuer tribe. Here the papules are also found on the forehead despite the scarring, but with less density than on the cheeks and nose.
Differential diagnosis of papular and nodular PKDL

183, 184 Psoriasis.
Swelling of the face is not a feature of PKDL; no diagnosis was made in this case, but the appearance and the patient’s age may suggest Burkitt’s lymphoma.
Köbner's phenomenon.
In this child, previous scars, caused by traditional scarification for an unknown illness, became visible as PKDL lesions appeared in them; after treatment of PKDL, the lesions disappeared and the scars became invisible again.
2. PKDL in Africa: clinical presentation and differential diagnosis

Differential diagnosis of papular and nodular PKDL

189 Köbner’s phenomenon.
Note the preferential localization of the papules in the scars causing an asymmetrical distribution which is otherwise unusual in PKDL.
Depressed scars developing after longstanding PKDL.
Chronic PKDL

191
Depressed scars developing after longstanding PKDL.

192
Fibrosis of the skin in longstanding PKDL.
Post kala-azar manifestations

Post-kala-azar conjunctivitis and uveitis.
The patient was treated for VL in the recent past but developed increasing swelling of the eyelids and loss of vision. These were not diagnosed as related to persistent Leishmanial infection in the eyes and led to complete blindness.
Concomitant PKDL and post-kala-azar conjunctivitis (top) and blepharitis (bottom).
Post kala-azar manifestations

202 Post-kala-azar uveitis. Note the irregular pupil and nodules.

Differential diagnosis:

203 Onchocerciasis (middle).
Left: beginning of overgrowth of cornea (pannus).
Right: uveitis; irregular pupil.

204 205

Trachoma (below).
Left: inversion of eyelids. Right: after eyelid surgery; the cornea is opaque.
206 Mucosal lesions in severe PKDL.
Other post kala-azar manifestations (conjunctivitis, uveitis, mucosal)

207 Para-kala-azar dermal leishmaniasis.
VL (confirmed in a lymph node aspirate) and concomitant micropapular PKDL.
Nuer tribesman before (208, 209) and after 30 days of stibogluconate treatment (210, 211). In spite of the difference in brightness of the figures, his skin indeed became lighter after treatment.
Evolution of PKDL

Before treatment (212, 213), after 30 days of treatment with SSG (214) and after 6 weeks (215).
Initial presentation (216), 2 weeks later (217) and after treatment (218).
PKDL in Ethiopia. Various examples

Various examples of papular rash, mainly around the mouth.

219

220

221
222 Papular rash, covering most of the face.
PKDL in Ethiopia. Various examples

Papular rash.
PKDL in Ethiopia. Various examples

Papular rash.
2. PKDL in Ethiopia. Various examples

Papular rash with plaques (228).
Papules confluent to form nodules (229); nodules confluent to form plaques (230, 231)
Subcutaneous nodular lesions in an HIV co-infected VL patient: para-kala-azar dermal leishmaniasis.
The same strain was isolated from the spleen and from the skin.
The lesions resemble those of Kaposi's sarcoma.

Kaposi's sarcoma in an HIV+ve patient.
Diffuse Cutaneous Leishmaniasis; the lesions had been there for 3 years and were never treated.
Differential diagnosis

**Discoid Lupus Erythematosus.** The patient was initially diagnosed as PKDL and treated with zinc ointment (not an accepted treatment for PKDL).
3. PKDL in Asia: clinical presentation and differential diagnosis

a. PKDL in India: hospital-based experience

b. PKDL in Bangladesh: community-based experience
PKDL in Asia is much different from Africa. There are few longitudinal studies with active follow-up of VL cases and most reports are on cases that present with chronic PKDL. PKDL is increasingly reported from Bangladesh as more field studies are conducted.

The interval between VL and PKDL is usually 2-3 years; however, patients may report a shorter interval. In one study with active follow-up 40% of cases developed PKDL within 12 months of VL.

**Clinical presentation**

The currently available data do not permit a general description that is applicable to all areas (India, Bangladesh, Nepal); data from field studies are different from hospital-based studies; whether these differences are the result of patient delay, reporting bias or true differences related to parasite involved, genetic background, treatment received etc. is unknown.

Hospital-based studies in India: The polymorphic form showing hypopigmented or erythematous macules with papules and/or nodules is the commonest. A monomorphic presentation with only papulonodules may be seen, the monomorphic form macular form being uncommon. Both of these monomorphic forms can mimic leprosy. In addition vitiligo is also an important differential diagnosis for macular PKDL. Verrucous lesions may be seen, though uncommon. A generalized redness of the face and body with scattered papules and plaques indicates the rare erythematous form of PKDL.

Field studies in Bangladesh: The macular form is by far the most common presentation. While the face is usually involved the spread to other parts of the body does not always follow the classical pattern described for Sudan. Most cases in Bangladesh present with longstanding lesions that seem to spread and remain macular; sometimes concomitant papules may be found in the face. Up to 10% of cases present without a previous history of VL.

In the Indian subcontinent all cases are treated as they are considered chronic cases also given the long interval after VL; while the skin returns to normal, repigmentation may take time and cannot be taken as a parameter for cure.

Other post-kala-azar manifestations have been described such as post-kala-azar uveitis.

The main differential diagnosis for the macular form is vitiligo (Table 3) and for all forms leprosy (Table 4).
Macular lesions on the legs.
PKDL in Asia: clinical presentation and differential diagnosis

Macules and small papules.
Macules on arms and legs, but face and trunk relatively spared.
PKDL in India

Macules on limbs and face with some small papules on the nose.
Nodules on the face; macules on the rest of the body with virtually total hypopigmentation sparing axillae and inguinal areas.
Hypopigmentation in the face with few papules/nodules; nearly total hypopigmentation of the back sparing the central area where some normal skin can be seen as irregular macules; lesions on the tongue and hypopigmentation of the hand palms.
Hypopigmented patches in the face, typically around the mouth.
Hypopigmented lesions involving the whole face sparing the neck; this is only appreciated in a lateral view. The lesions on the back spare the mid back and elbows (the arrow indicates the site of a biopsy).
Macular lesions in inguinal area, penis shaft and glans.

Hypopigmentation of the thighs with nodular plaques on the scrotum, penis and papules on tip of glans penis.
PKDL in India

PKDL: papules and nodules on inner thighs, scrotum and penis.
Erythrodermic PKDL. This is uncommon; there is facial erythema and sparing of the axillae. The rest of the body is also faintly erythematos.

Papules and nodules on the tongue and buccal mucosa.
Papules and nodules on the tongue.
Discrete papules on chin (258, 259); crops of nodules on chin and nose (260)
PKDL in India

Verrucous or hypertrophic form.
Tumor-like nodules on face (note sparing of eyebrows).

Tumor-like; there is a spontaneous furrow.
Differential diagnosis

**Vitiligo.** Note the total loss of pigment (depigmentation) and the involvement of the central part of the trunk.

**Lepromatous leprosy.** Lesions are prominent on the forehead and the cheeks while the central part of the face is spared. The patient on the right has madarosis. All these are not features of PKDL.
Borderline leprosy.

Red, raised plaques on the face and limbs, note sparing areas around the nose and the mouth.
Borderline tuberculoid leprosy: the nose and chin are free; note the madarosis.
Macular lesions.
PKDL in Bangladesh

3. PKDL in Asia: clinical presentation and differential diagnosis

271 Macular lesions.

272
PKDL: macular lesions, some are confluent.

Large areas of confluent macules leaving some islands of normal skin.
Combined confluent macular lesions on the arms (275) and papulonodular lesions on the face (276).
Extensive confluent macules on the back and face leaving little normal skin.
PKDL in Bangladesh

Extensive confluent macules on the back.
Mostly macular rash with papular rash on the shin.
PKDL in Bangladesh

Maculopapular rash.
Nodular rash with infiltration of the skin in particular on the nose.
PKDL in Bangladesh

Macular lesions on chest and nodular lesions on arms and fingers.
Figures 290-318: In this section, a number of figures are shown of patients who were treated with liposomal amphotericin B 5 mg/kg twice weekly for three weeks. In some patients this leads to cure within 12 months; in others the rash is slow to disappear. It is difficult to assess if these lesions will further heal, leave residual hypopigmentation or indicate treatment failure; for the latter another diagnosis should also be considered.

290 291

Macular rash before and after 6 weeks; no response can be noted.

292 293
Response to treatment

Macular rash before and after 6 weeks, 4 months and 7 months; note the slow recovery (e.g. around the mouth).
Residual lesions can be seen 10-11 months after treatment (301).
Response to treatment

Almost complete disappearance of lesions 4 months after treatment
PKDL in Bangladesh  
Response to treatment

Good response 7 months after treatment.
Good response of nodular lesions 6 weeks after start of treatment.
Clear residual lesions 5 months after start of treatment.
Response to treatment

Poor response to treatment; lesions are virtually unchanged.
Gradual disappearance of the macular lesions 9 months after start of treatment.
Chronic arsenicosis.
Hypopigmented and hyperpigmented lesions resembling PKDL. Common in Bangladesh; although epidemiologically the endemic areas do not overlap, this condition may be confused with PKDL.
Complications of chronic arsenicosis: solar elastosis (320), squamous cell carcinoma (321,322) and lentigo simplex (323). These complications are not seen in chronic PKDL.
**Differential diagnosis**

**Chronic arsenicosis**

Papulonodular lesions on lower limbs and warty hyperkeratotic lesions on the soles (324); psoriatic plaques on the right shin and palmoplantar warty hyperkeratosis (325). The presence of these lesions are useful to differentiate from PKDL.
4. PKDL in other areas

a. PKDL in China

b. PKDL in Brazil
PKDL in China

Advanced nodular PKDL.
The patients are from Taiwan but they contracted VL on mainland China.
PKDL in Brazil

Presented with skin lesions after second episode of VL; *L. infantum* was isolated from the bone marrow. Amastigotes were seen in a biopsy from the skin lesions. There were no facial lesions; the patient was HIV negative.
5. PKDL in immunocompromised patients and other skin manifestations of *Leishmania* in HIV-positive patients
PKDL may occur in immunocompromised patients; most cases have been described among HIV infected patients. Other conditions include bone marrow transplant patients and patients with immunosuppressive therapy. In HIV infected patients with VL, a variety of skin lesions has been described and these may occur before, during or after VL. They are sometimes referred to as atypical (disseminated) cutaneous lesions or diffuse cutaneous leishmaniasis in the course of VL, but these may basically be the result of the same pathophysiological mechanism that underlies PKDL: an immune reaction to *Leishmania* parasites in the skin with subsequent clinical manifestations.

Although most evidence is anecdotal, one study found PKDL to be more common and more severe in patients who were HIV positive than in HIV negative patients.

The clinical presentation and characteristics may differ from those found in immunocompetent patients; the most important differences are the atypical distribution and evolution and the abundance of parasites in mainly nodular lesions (Table 7).

**Table 7: Differences between PKDL in immunocompetent and immunocompromised patients**

<table>
<thead>
<tr>
<th></th>
<th>Immunocompetent</th>
<th>Immunocompromised</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parasite</strong></td>
<td><em>L. donovani</em> mainly</td>
<td>Also <em>L. chagasi</em>/ <em>L. infantum</em></td>
</tr>
<tr>
<td><strong>Frequency</strong></td>
<td>(reference)</td>
<td>more frequent, more severe</td>
</tr>
<tr>
<td><strong>Main clinical presentation</strong></td>
<td>macular or maculopapular</td>
<td>nodular</td>
</tr>
<tr>
<td><strong>Other post KA manifestations</strong></td>
<td>yes, uveitis</td>
<td>yes, uveitis</td>
</tr>
<tr>
<td><strong>Post or para KDL</strong></td>
<td>post &gt;&gt; para</td>
<td>para &gt;&gt; post</td>
</tr>
<tr>
<td><strong>Parasites numbers</strong></td>
<td>scanty</td>
<td>abundant</td>
</tr>
<tr>
<td><strong>Parasites found in skin</strong></td>
<td>&lt;60%</td>
<td>90%</td>
</tr>
<tr>
<td><strong>Ulcerating</strong></td>
<td>no</td>
<td>genital ulcers described</td>
</tr>
<tr>
<td><strong>Face affected</strong></td>
<td>the rule</td>
<td>not always</td>
</tr>
<tr>
<td><strong>Acra involved</strong></td>
<td>no</td>
<td>often; symmetrical</td>
</tr>
<tr>
<td><strong>Evolution</strong></td>
<td>typical</td>
<td>atypical</td>
</tr>
</tbody>
</table>

**Table 8: Clinical entities in HIV infection with disseminated cutaneous *Leishmania* lesions, parasites isolated and areas from where reported**

**Disseminated cutaneous leishmaniasis without VL (history or present):**

- *L. tropica* (India)
- *L. chagasi* / *L. infantum* (Nicaragua)

**Disseminated cutaneous lesions preceding VL:**

- *L. infantum* (France)

**Disseminated cutaneous lesions with concomitant VL**

- *L. donovani* (Ethiopia)
- *L. infantum* + *L. donovani* (Brazil)
- *L. infantum* (France)

**Disseminated cutaneous lesions after VL:**

- *L. donovani* (Ethiopia)
- *L. infantum* (France, Italy, Greece)
- *L. chagasi* / *L. infantum* (Brazil)
- *L. major* (Burkina Faso)
PKDL in immunocompromised patients

**331**
Mild papular rash in an HIV-positive patient from Spain, who had multiple relapses. *L. infantum* was isolated from the skin.

**332**
Nodules and plaques in an HIV-positive patient.
PKDL in an Italian HIV patient

a. At presentation; *Leishmania* PCR from a skin biopsy was positive.
b. After 3 cycles of miltefosine.
c. After liposomal amphotericine.
Cutaneous dissemination of visceral leishmaniasis in an HIV-positive patient from Brazil. *Leishmania* parasites were isolated in aspirates from bone marrow and skin. There is a compact inflammatory infiltrate on deep dermis under a normal epidermis (338; arrows); numerous amastigotes can be seen (339, arrow).
PKDL in immunocompromised patients

PKDL in 2 HIV positive patients from Spain. Dermatomyositis-like presentation showing erythematous plaques with periungual erythema on dorsum of hands (340) and in the face particularly on the upper eyelids (341).

342
Diffuse infiltration around the nose.
Three patients from Ethiopia.
HIV-positive with papules and nodules in the face, abdomen and extremities. Amastigotes were demonstrated in spleen aspirate and slit skin smear.

HIV-positive with amastigotes demonstrated from bone marrow and skin scrapings.

HIV-positive and PKDL with Kaposi’s sarcoma-like lesions on the lower legs; amastigotes were found in a skin scraping.
Cutaneous and mucosal lesions in an HIV-positive patient from Bolivia; *L. (V) braziliensis* was isolated from the skin. The ulceration shown (352, 353) is not a feature of PKDL.
Two HIV-positive patients from India with low CD4 counts and no previous history of VL. A smear from the lesions in each patient showed *Leishmania* amastigotes. There are nodular lesions on the hands (354) and infiltrated plaques on the nose, dorsum of the left wrist and on the index finger of the right hand (355).
Diffuse cutaneous leishmaniasis in a patient who was HIV-positive patient for 8 years and on antiretroviral therapy. This patient was from Kerala, South India. There was no previous history of VL. *Leishmania* amastigotes were demonstrated in a smear.
PKDL in immunocompromised patients

Disseminated cutaneous lesions and mucosal lesions in HIV infection without VL

Three HIV positive patients from India with multiple CL lesions due to *L. tropica*. 
6. Other forms of leishmaniasis that resemble PKDL or that may be found in the same endemic area

Note
The conditions shown do not necessarily overlap in epidemiology with PKDL.
Leishmanioma in Sudanese patients

The presence of leishmania parasites was confirmed by PCR (L. donovani) (367). The lesion cured spontaneously and a scar was seen 6 months later (368).
Leishmanioma in Sudanese patients

Leishmanioma in a patient with PKDL (367); close-up (368). A smear from the lesion was positive in leishmanial PCR.

Healed leishmanioma.
Other forms of leishmaniasis that resemble PKDL or that may be found in the same endemic area

370 Erysipeloid infiltrative lesion of the nose and cheeks.

371 Note swelling, crusts and scales.

372 Facial lesion with cheilitis.
Other forms  

Cutaneous leishmaniasis (*L. major*) from Sudan

373

Cutaneous leishmaniasis on the pinna of the ear, showing crusting.

374

Multiple disseminated micro-nodular lesions. Note the face is free of lesions, which is unusual in PKDL.
Cutaneous leishmaniasis (*L. tropica*) from Afghanistan

Clinical manifestations of CL showing various degrees of ulceration.
Various degrees of ulceration in cutaneous leishmaniasis (*L. tropica*) from Afghanistan.

Lupoid form.
6. Other forms of leishmaniasis that resemble PKDL or that may be found in the same endemic area

Cutaneous leishmaniasis (L. aethiopica) from Ethiopia

CL due to L. aethiopica: diffuse infiltration of the skin
Other forms  Leishmaniasis recidivans

Leishmaniasis recidivans due to *L. aethiopica* in Ethiopian patients.
Leishmaniasis recidivans

Leishmaniasis recidivans due to (L. tropica) from Morocco. Note the healed scar from which new lesions develop.
Leishmaniasis recidivans due to (L. tropica) from Afghanistan. Note the healed scars from which new lesions develop.
Leishmaniasis recidivans

Leishmaniasis recidivans due to *L. tropica* from Morocco (390,) and Afghanistan (391, 392).
Diffuse Cutaneous Leishmaniasis (DCL) from Ethiopia. The patient originating from the Highlands where CL and not VL is endemic; there is no previous history of VL. *Leishmania* parasites were found in a skin scraping.
Other forms of leishmaniasis that resemble PKDL or that may be found in the same endemic area.
Diffuse cutaneous leishmaniasis (DCL) in Venezuela is normally caused by *L. amazonensis*. 
Diffuse cutaneous leishmaniasis

Venezuela (see also Figs 357, 358 and 351 for comparison)
Mucosal leishmaniasis (Sudan). Fungating or tumour-like form. May be primarily mucosal \((L.\ major)\) or post-kala-azar mucosal leishmaniasis \((L.\ donovani)\). Clinical clues:
Chronic ulceration of the nasal mucosa; previous history of VL. Lesions on the palate and the gums should be looked for.
Leishmaniasis in tattoos

Cutaneous leishmaniasis in a tattoo due to *L. infantum* from Spain in an HIV-infected patient who had no previous history of CL or VL. The predilection for previous lesions or damaged skin is also a feature of PKDL (see Kőbner’s phenomenon, Figs 186-189).
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39. ICDDR,B Health and Science Bulletin Vol. 5 No. 4 December 2007
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- Indian Journal of Sexually Transmitted Diseases 2010;31:42-4. Figs 233
- Transactions of the Royal Society of Tropical Medicine and Hygiene 1995;89:647-52. Fig 405.

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