The Post Kala-azar Dermal Leishmaniasis (PKDL) Atlas

A Manual for Health Workers

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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

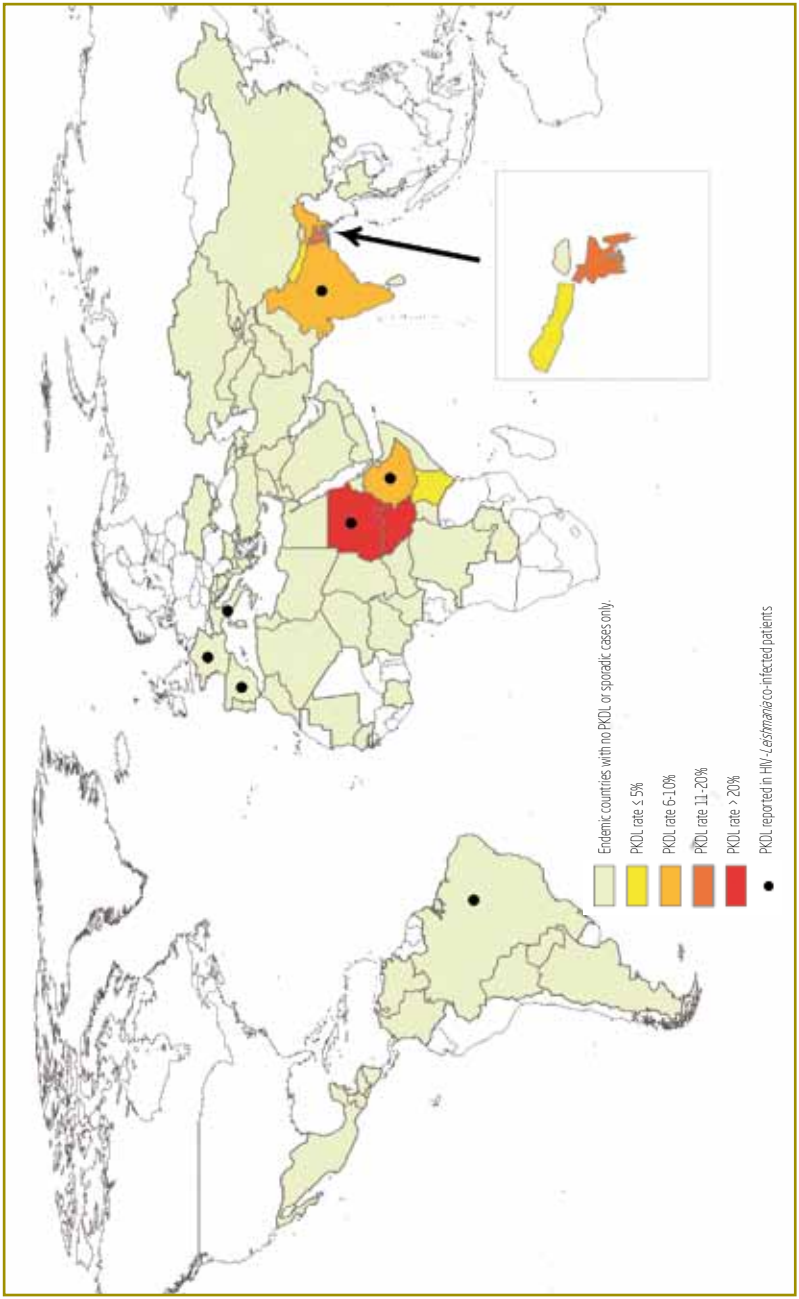
Note

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**



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]

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

[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 2: Differential diagnosis of PKDL in Africa

Papular/nodular rash	Macular rash
Cutaneous leishmaniasis	Leprosy
Leishmaniasis recidivans	Vitiligo
Diffuse Cutaneous Leishmaniasis (DCL)	Pityriasis versicolor
Mucosal leishmaniasis	Tinea corporis
Miliaria rubra (prickly heat)	Tinea barbae
Leprosy	Pityriasis alba
Lupus vulgaris	Discoid lupus erythematosus
Measles and other viral infections	Onchocerciasis
Acne	Burn scars
Neurofibromatosis	Birth marks
Urticaria pigmentosa/ mastocytosis	Pellagra
Darier' disease	Chloasma
Scabies	
Discoid lupus erythematosus	
Granuloma multiforme	
Granuloma annulare	
Lichen planus	
Mollusca contagiosa	
African histoplasmosis	
Keloids	
Tuberous sclerosis	
Mycosis fungoides	
Infantile eczema	
Psoriasis	

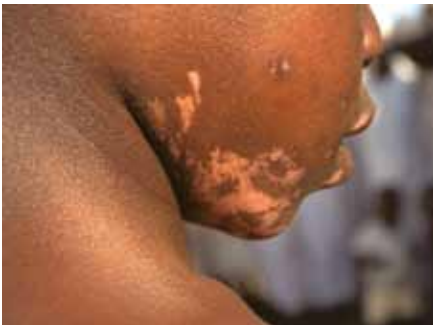
As a rule, after treatment or spontaneous cure, the skin fully recovers without scarring. In those who have chronic PKDL, often for many years, depressed scars may develop or the skin may become fibrotic .

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.

PKDL in Sudan

1-5 Macular PKDL mainly around the mouth and spread to other parts of the face.



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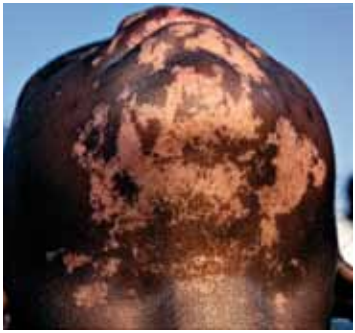

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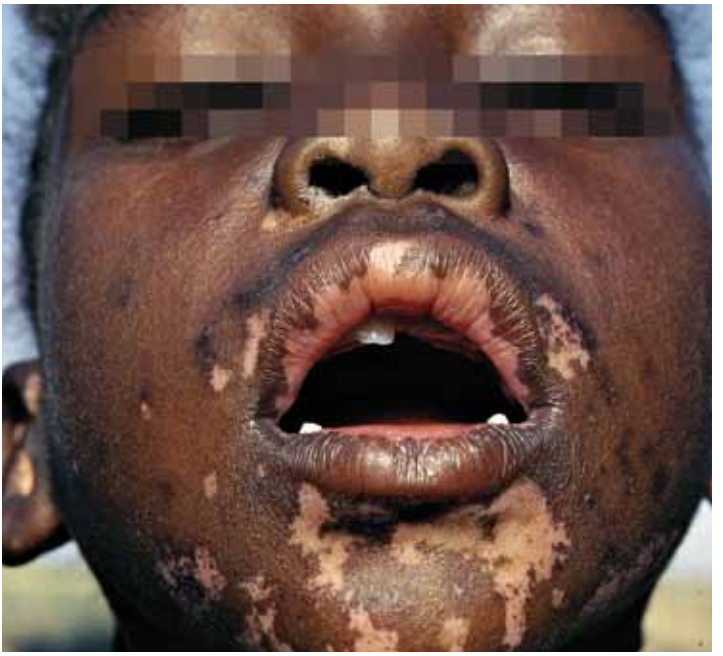

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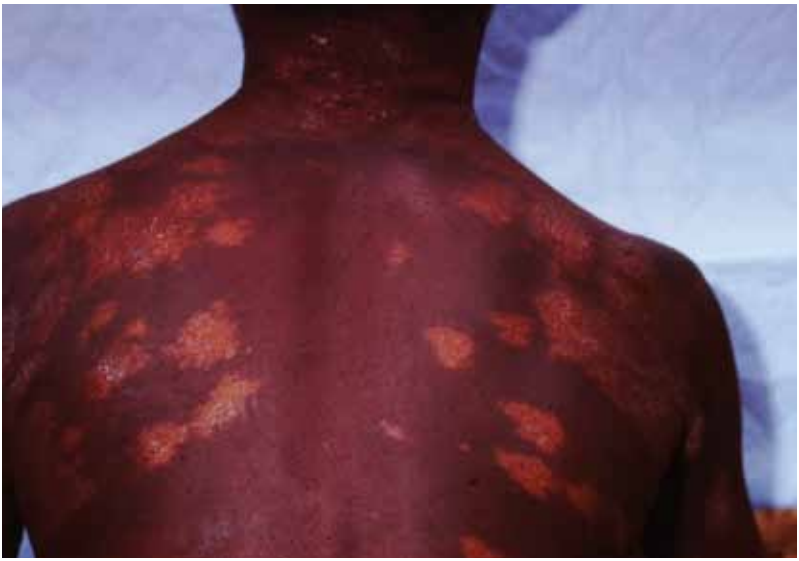
5

Macular PKDL

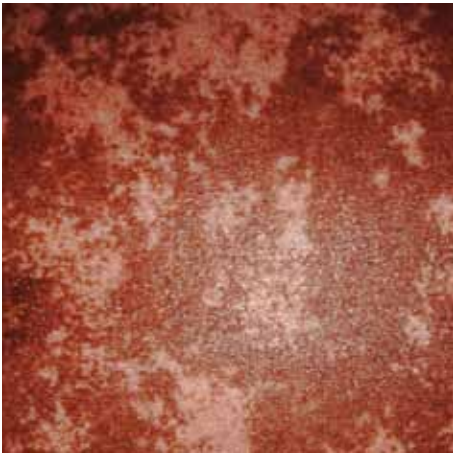
6 Macular rash affecting the "butterfly" area.

7 Symmetrical hypopigmented patches resembling lepromatous leprosy (see also fig. 131).

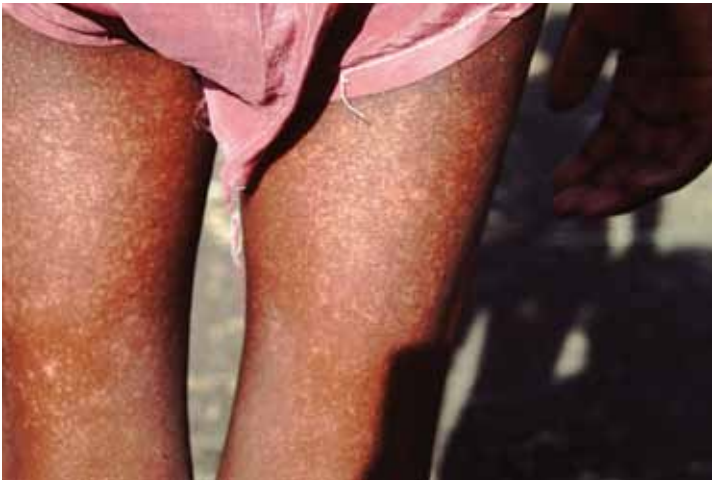


8 Macular lesions mainly on the trunk.

9 Close-up of the abdomen.

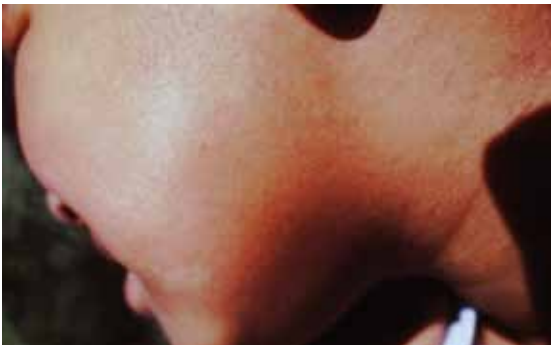


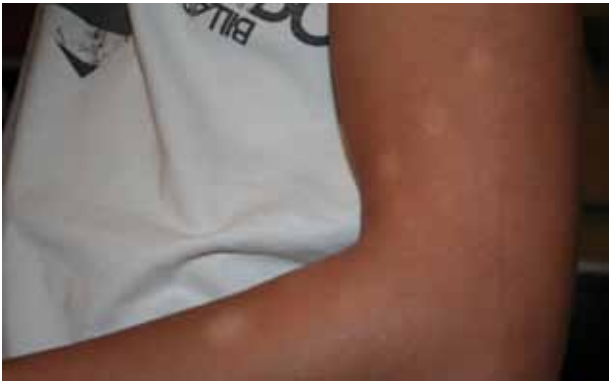
10 Same patient; lesions on the upper legs.



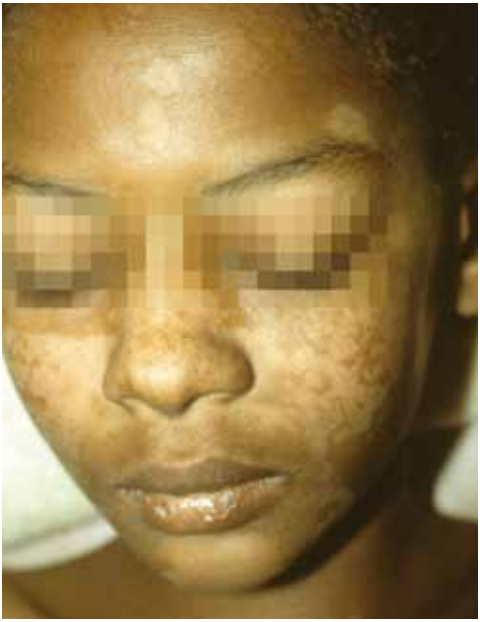
Macular PKDL and differential diagnosis

11 Pityriasis alba.

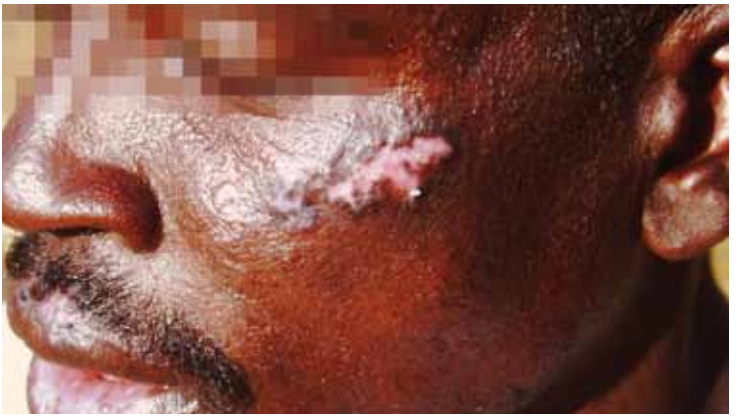




12 Tinea corporis. Scattered hypopigmented patches.



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.



14

15

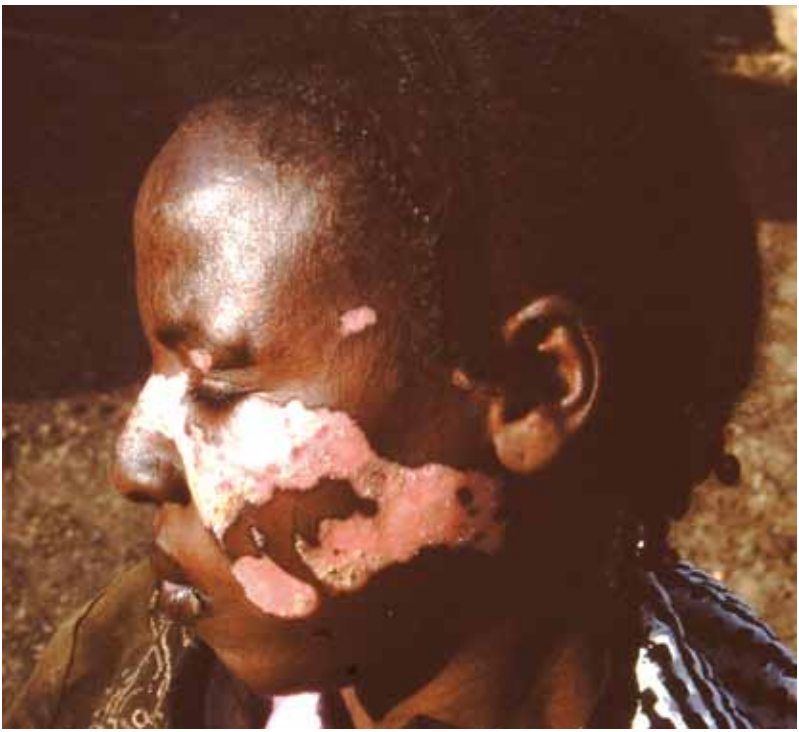
Discoid lupus erythematosus; healed scars on both cheeks and upper lip (14, 15, 16)

16

17

Discoid Lupus Erythematosus.
Symmetrical depigmented inflammatory lesions in the butterfly area and arms (17, 18).

18

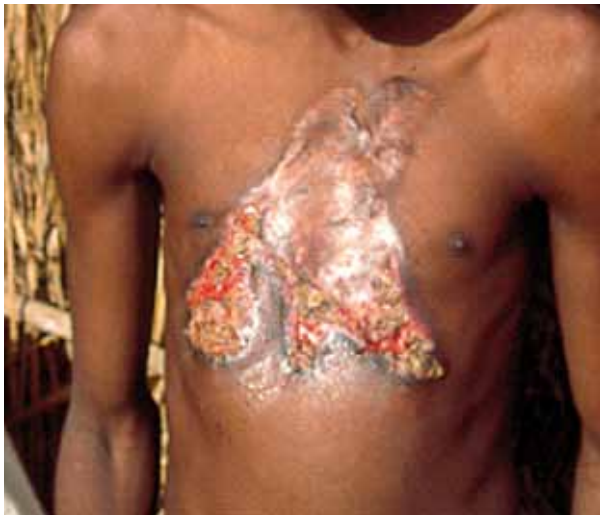


19 Discoid lupus erythematosus. Hypopigmented lesions.

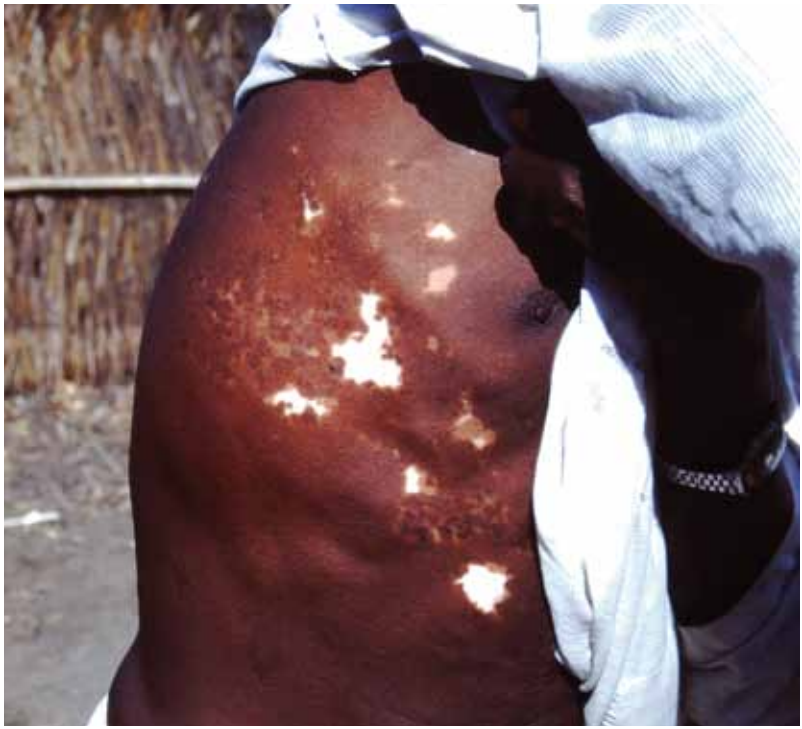
20 Discoid lupus erythematosus. Hypopigmented and hyperpigmented lesions may co-exist.

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.



23

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.

24

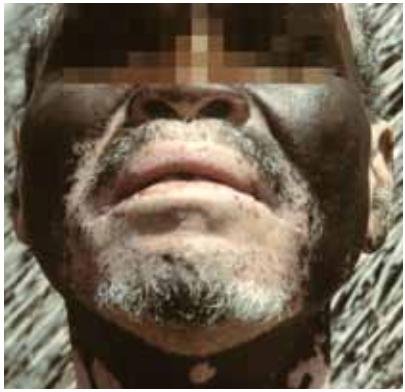
Table 3: Differential diagnosis of macular PKDL and vitiligo

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

25

26

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.



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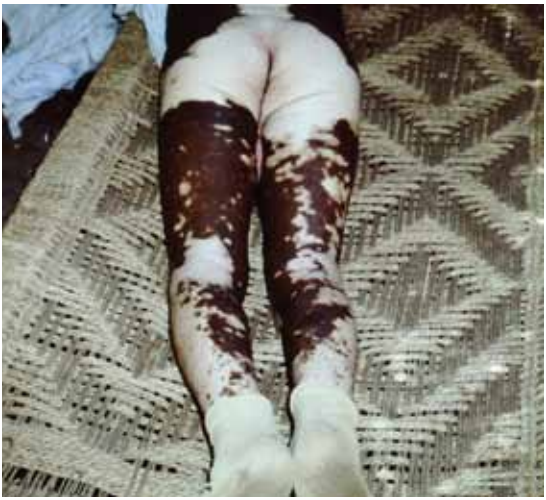

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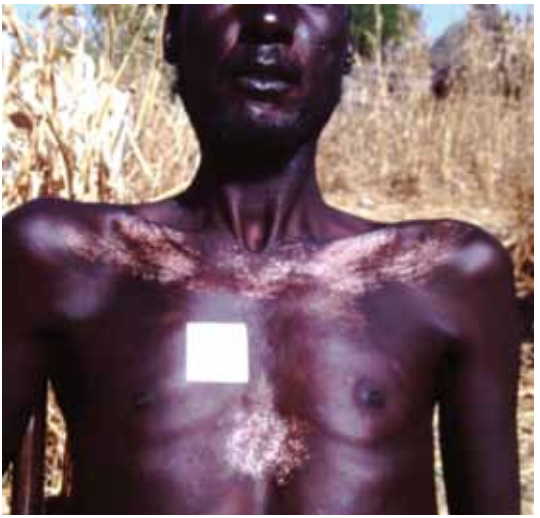

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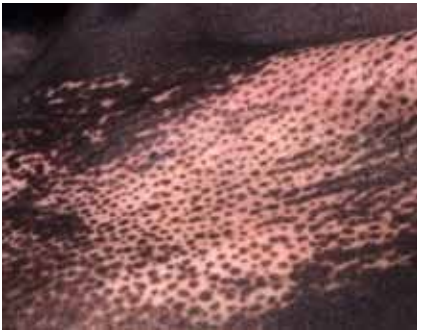
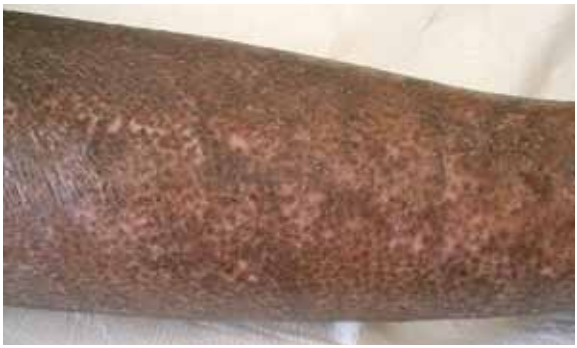


Scleroderma.
Clinical clues:
Symmetrical hypopigmented lesions, taut skin, reduced mouth opening, impairment in movement of the fingers.

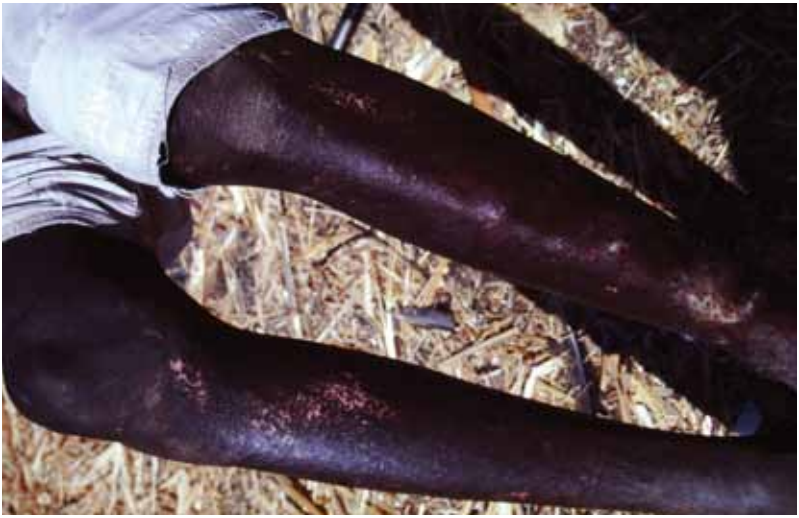
31

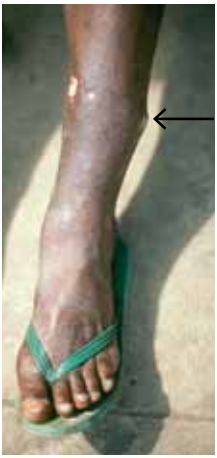
32



Onchocerciasis. Clinical clues: leopard skin with hypopigmented macules (32, 33); look for onchocercomata (34, arrow) and scratch marks (35).

33





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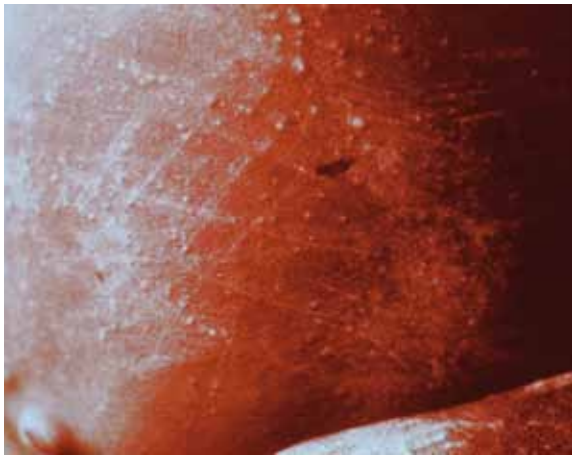

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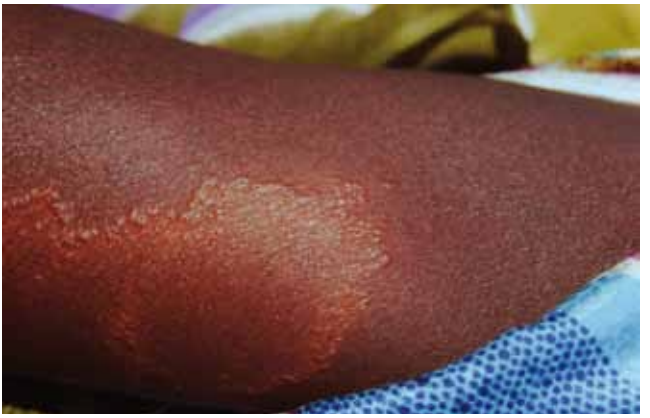


36 Borderline leprosy.
Note the raised edge, the area of hypopigmentation with central repigmentation.
Clinical clues: look for other signs of leprosy: anaesthetic patches, thickened nerves. See Table 4.

Table 4: Differential diagnosis of PKDL and leprosy

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

M macules, P papules, N nodules.

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.

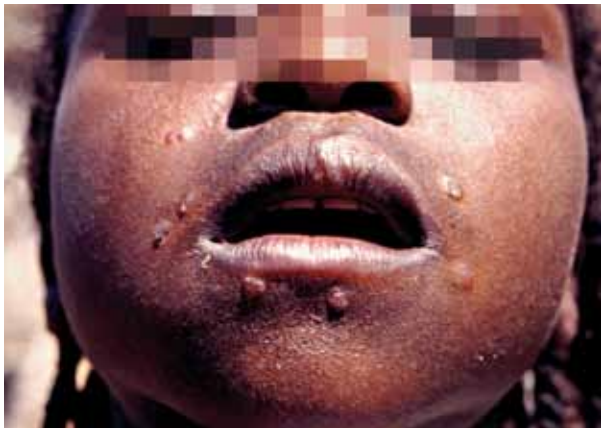
39

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.

40

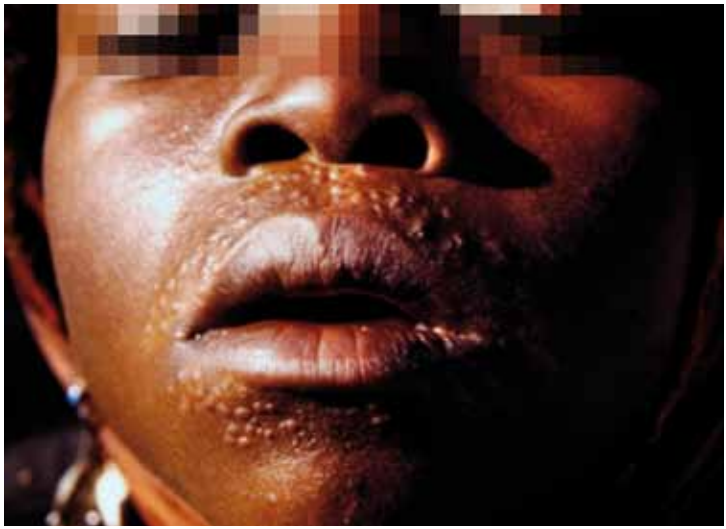
PKDL in Sudan: Papular and nodular PKDL

41



Papules in various stages of development and density and of various sizes; the initial localization around the mouth is typical.

42



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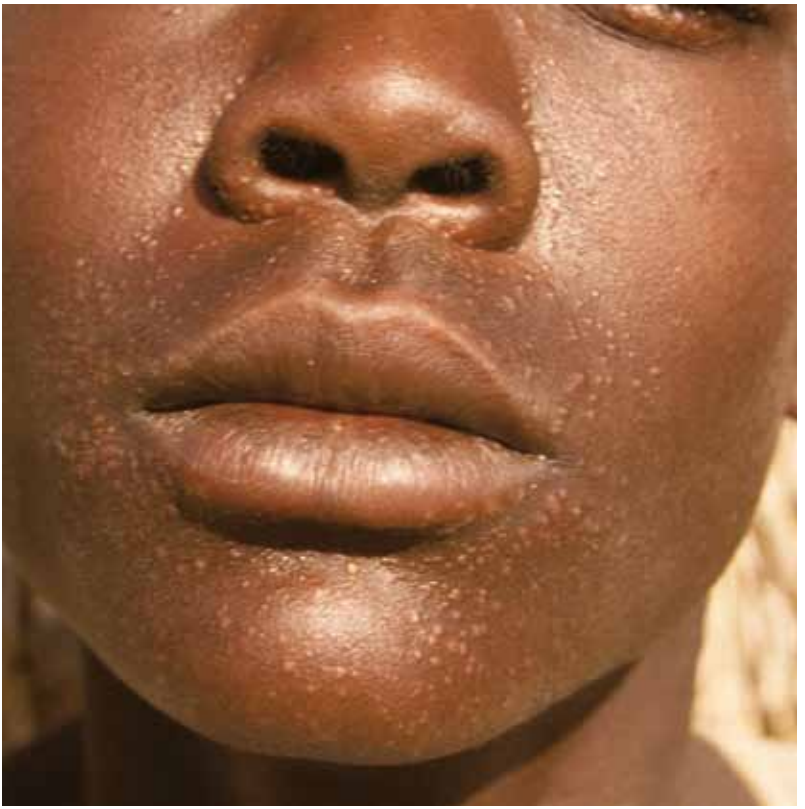
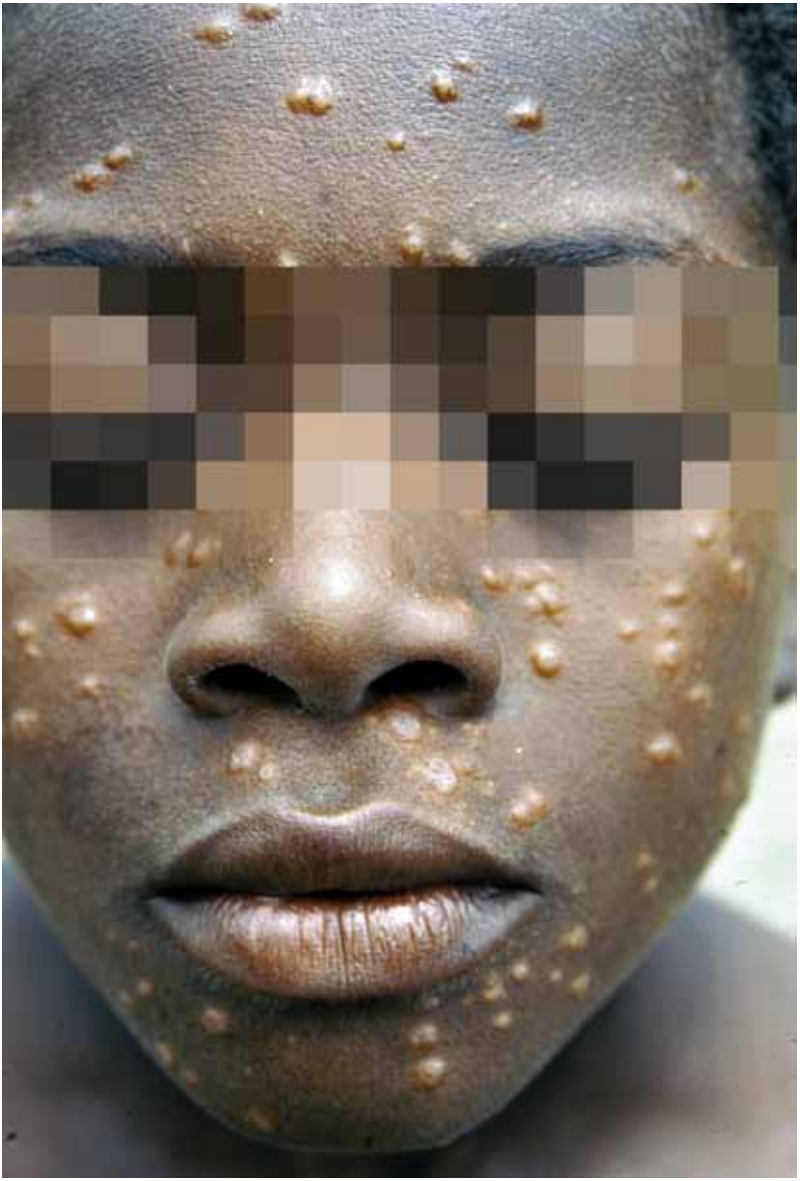
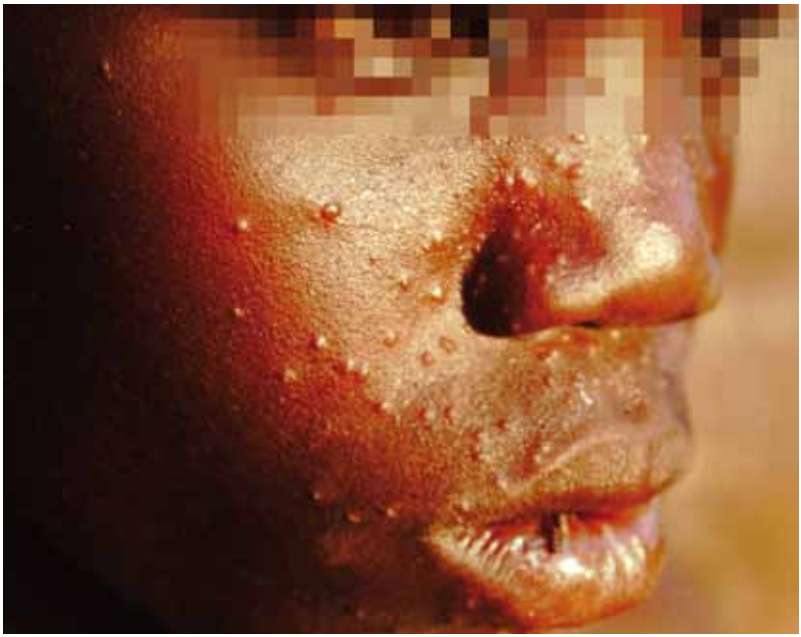


44 Micropapular rash.

Papular and nodular PKDL



45 Macropapular rash.

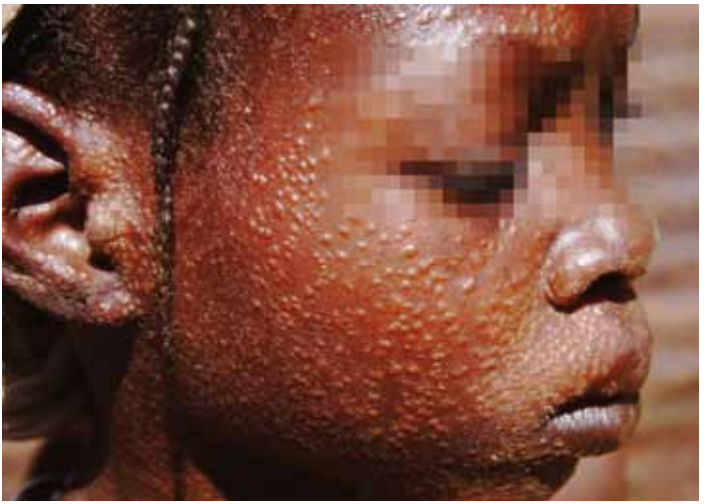


46

Further spread of the lesions to the nose, around the eyes and the forehead.

47

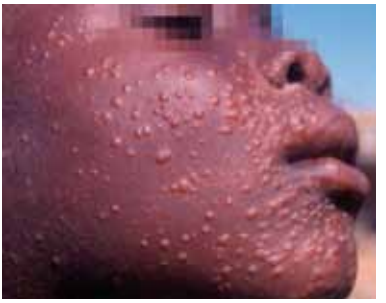
48 Further spread of the lesions to the nose, around the eyes and the forehead.



49

50

51 Papular rash with increasing density.

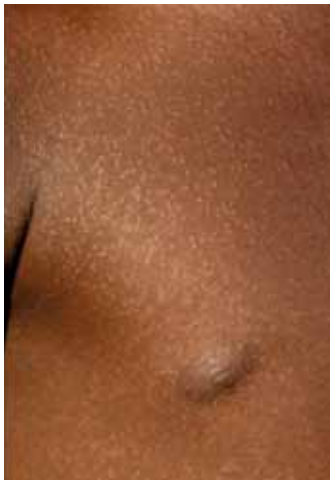


52

Papular and nodular PKDL

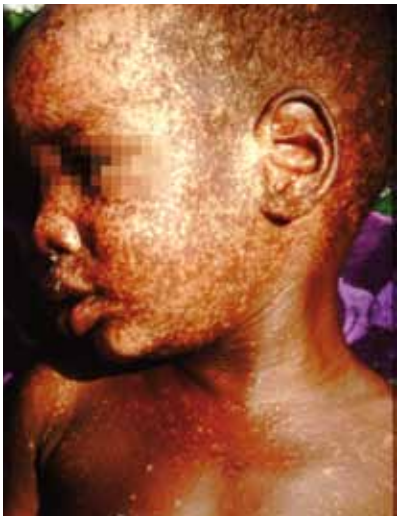
53

Maculopapular rash.

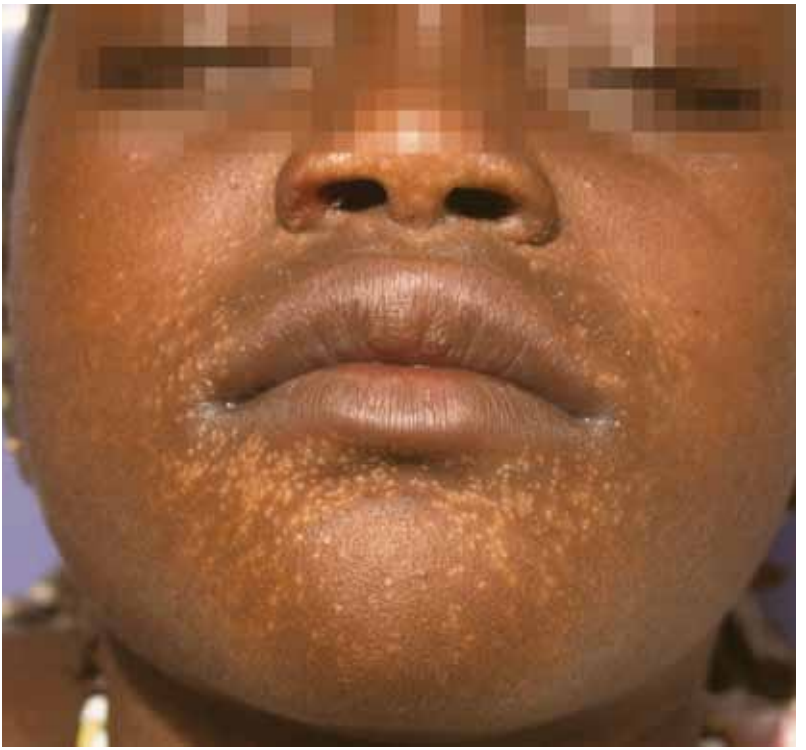


54

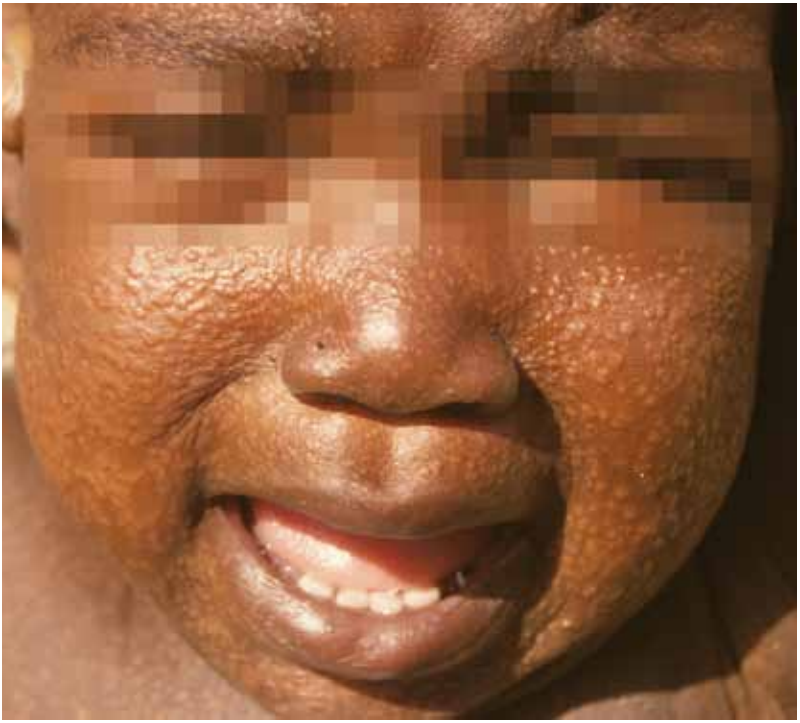
55



56



57 Maculopapular rash.

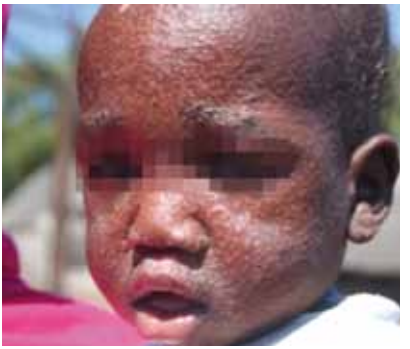


58 Papular PKDL, covering the whole face.

59

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

60



61

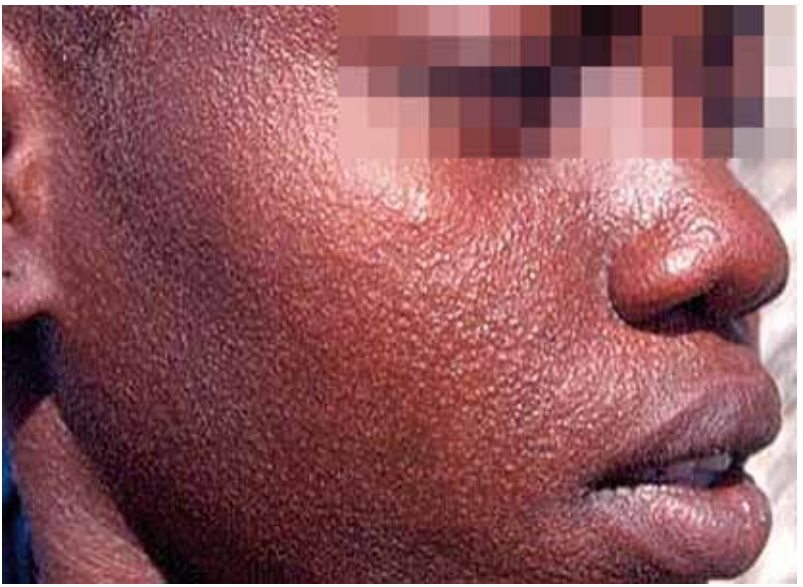
Nuer tribesman from South Sudan. Note absence of lesions on forehead: the horizontal lines are tribal markings and fibrotic changes may prevent PKDL papules to develop.

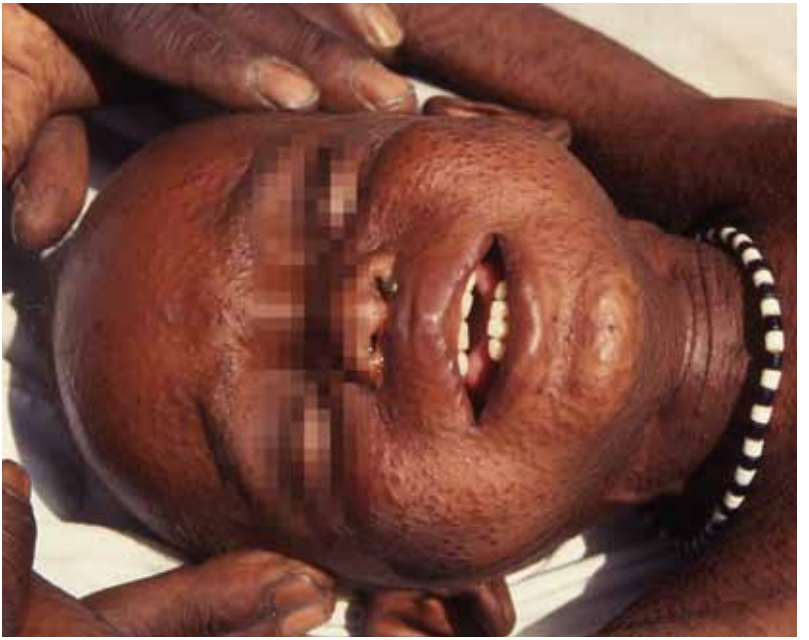
Papular and nodular PKDL

62

Micropapular rash, measles-like.

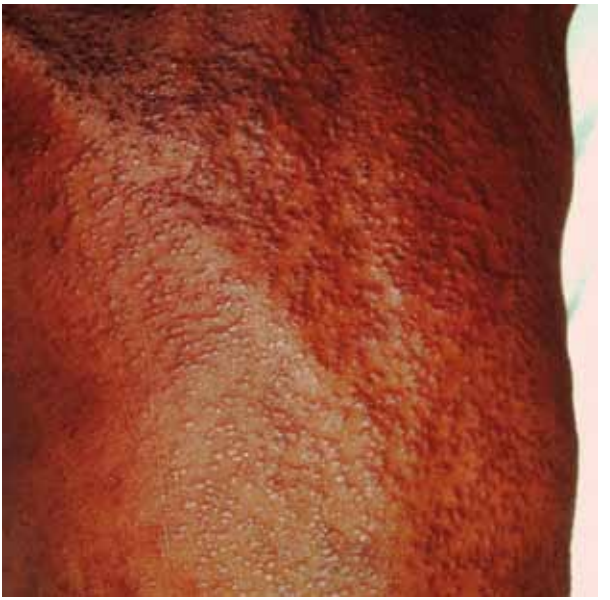
63



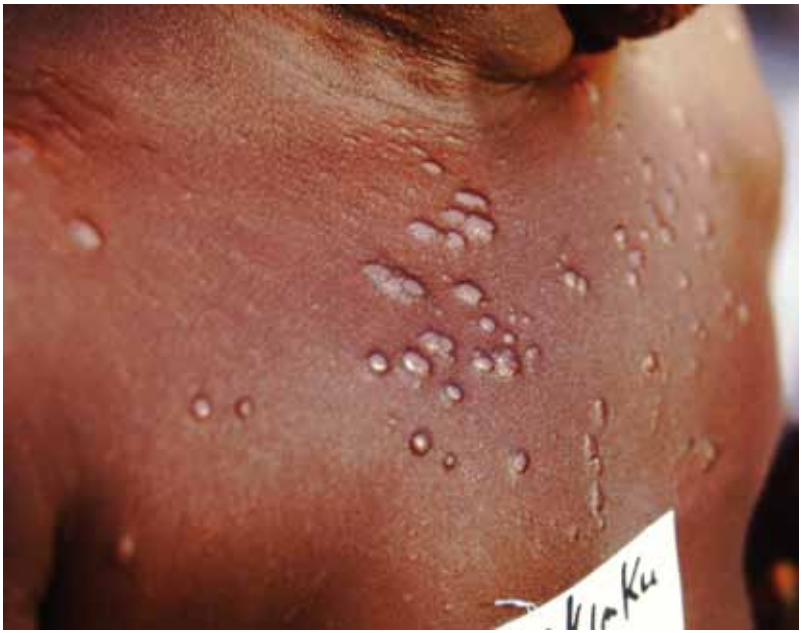


64

Hyperpigmented papules.



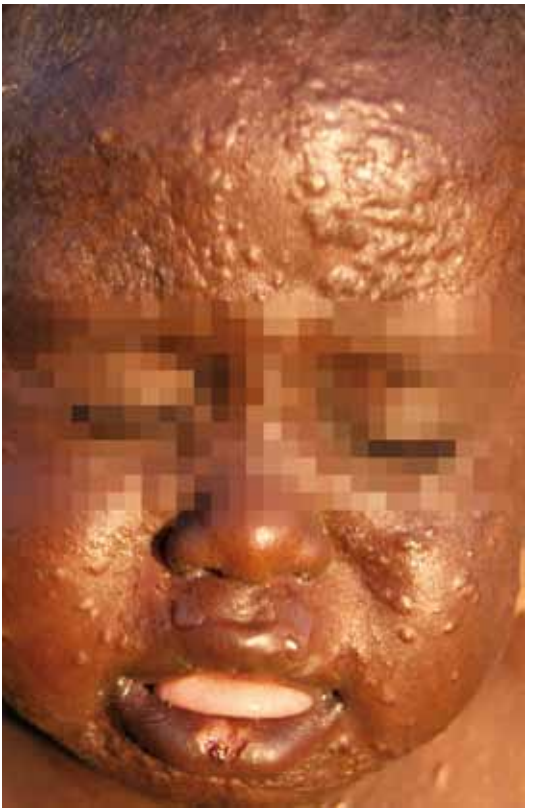
65



66 Nodular lesions.

67

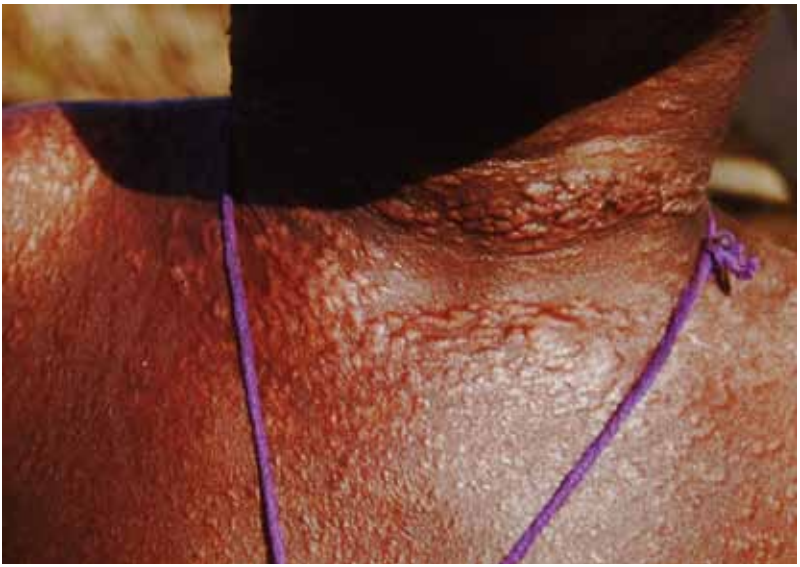
68



Nodular lesions.

69

Papular and nodular PKDL

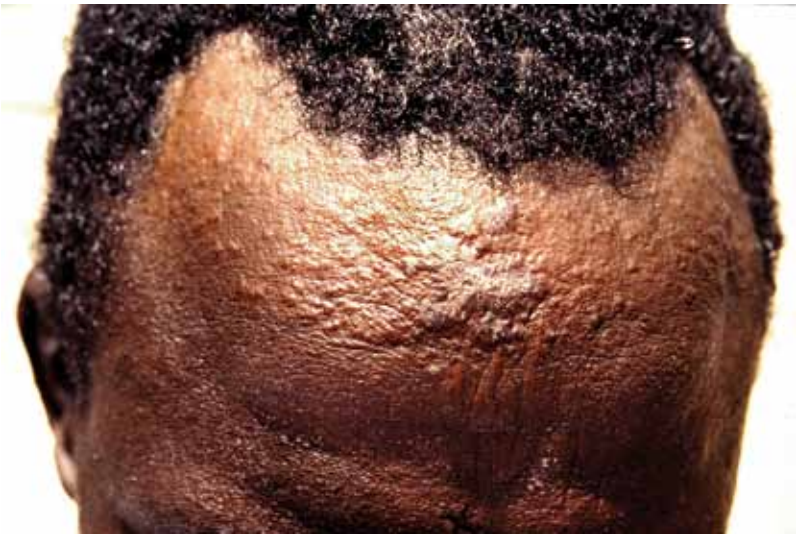


70

71

Papules and nodules become confluent to form plaques.

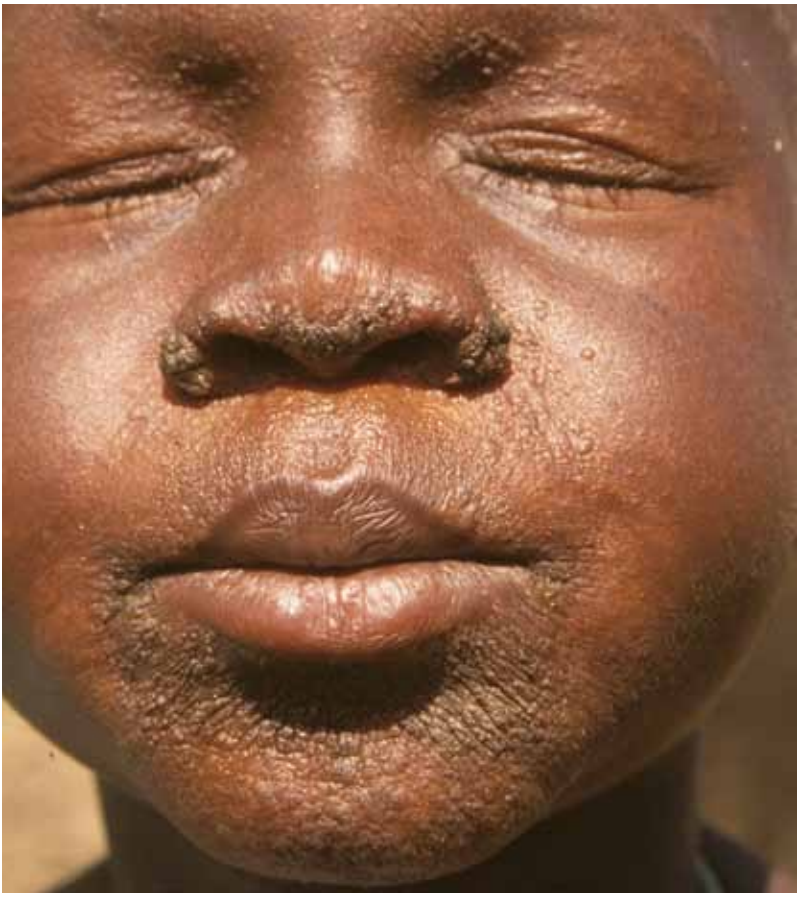
72



73

Patient with three solitary plaques on forehead (73), chin (74) and earlobe (121) .

74



75 Plaques in the face (Figures 75-79).

76

Papular and nodular PKDL

77

78

Papular and nodular PKDL

79

80
Grade 1.1 Lesions only in the face and restricted to area around the nose and mouth with normal skin in between.

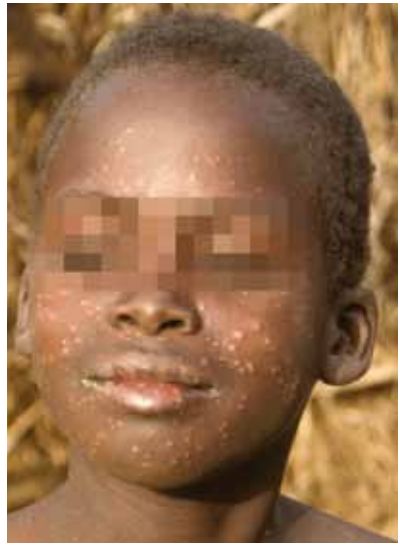
Table 5: Grading system of PKDL in Sudan

	Distribution	Density
Grade 1	face mainly with some lesions on trunk and arms	scattered lesions
Grade 2	face, upper parts of trunk, arms and legs affected, gradually becoming less distally; hands and feet free	moderate density with normal skin in between
Grade 3	all over body; including hands and feet	

81

Grade 1.1.
Transition into Grade 1.2 (Figures 81-82).

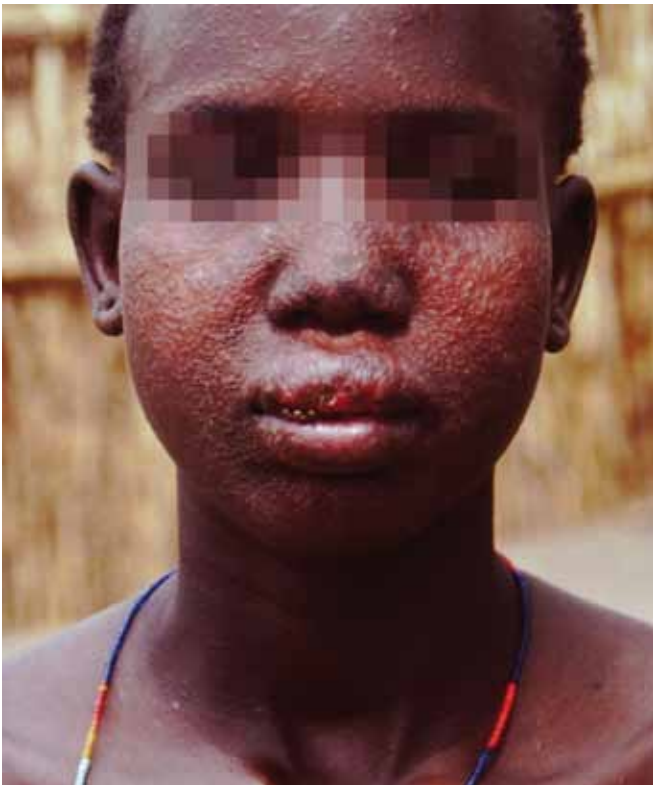
82



Grade 1.3.
Dense maculopapular rash, but mainly on the face.

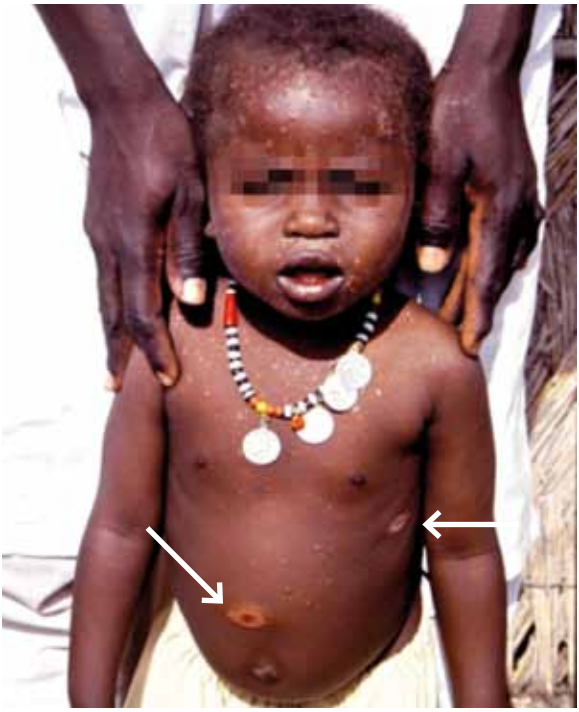
83

84



Grade 1.3 The whole face is affected with a dense papular rash (84) and macular rash (85) with few papules in other areas

85



86

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

87

Grade 2.2.
Papular rash. Most parts of the body are affected; the rash is dense but still normal skin can be seen.

88

Grade 2.2. Most of the trunk is also involved.

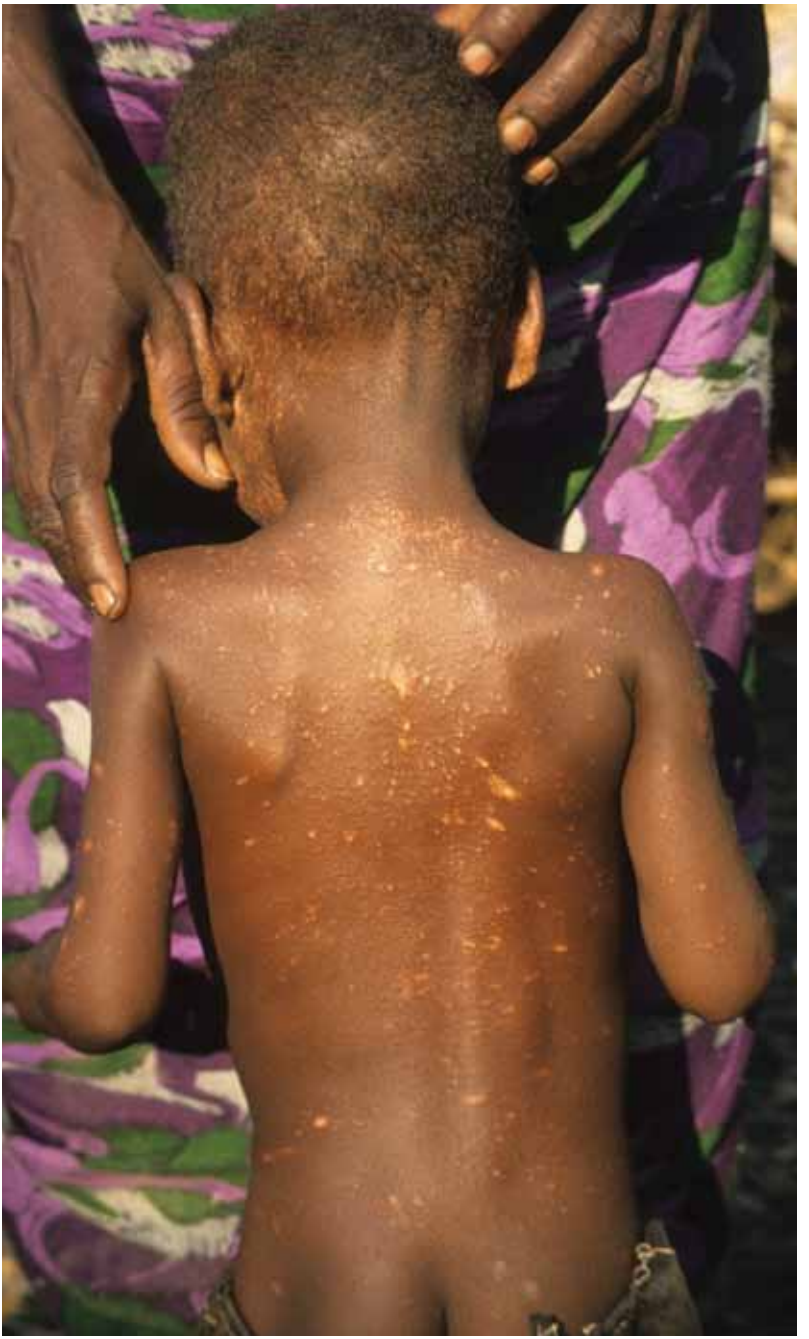
89

90

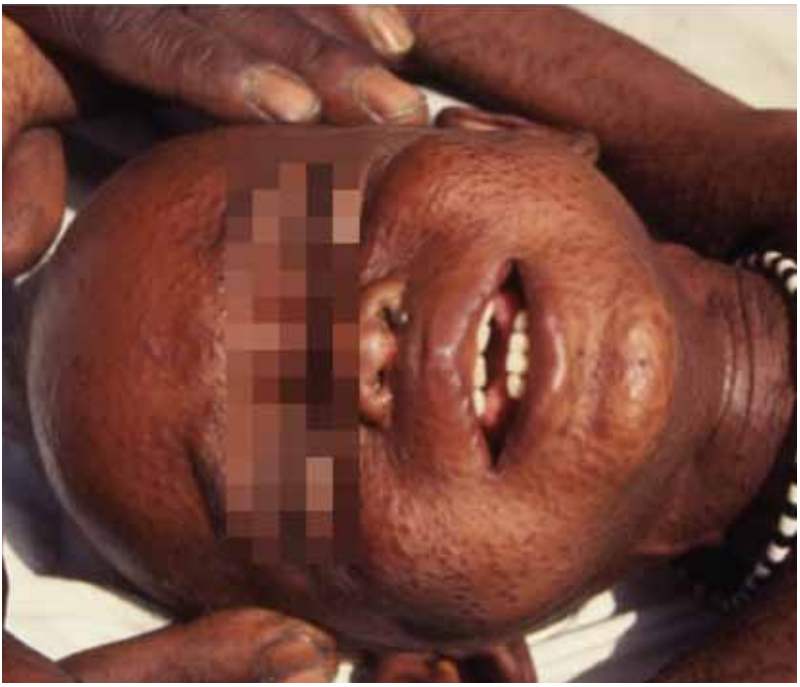
Grade 3.2.
Macular rash.

91

Grade 3.2.
Combined macular and papular rash.



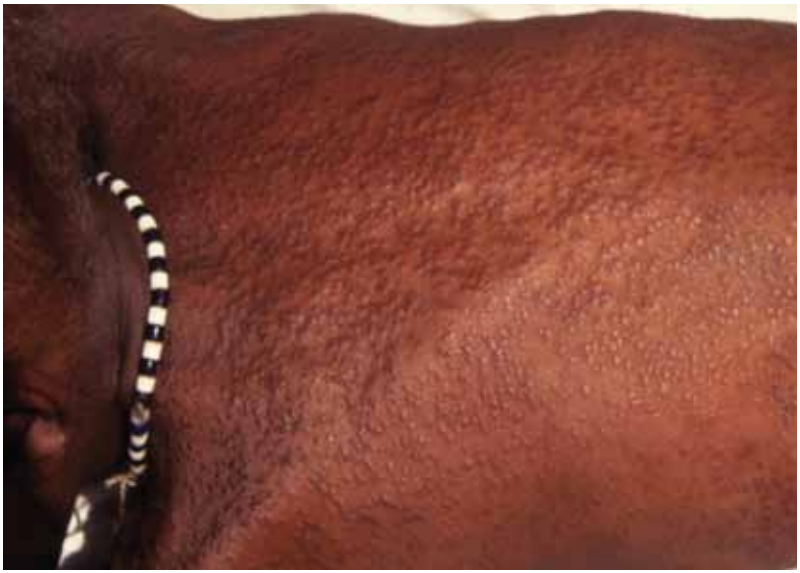
92 Grade 3.2 Maculopapular rash .

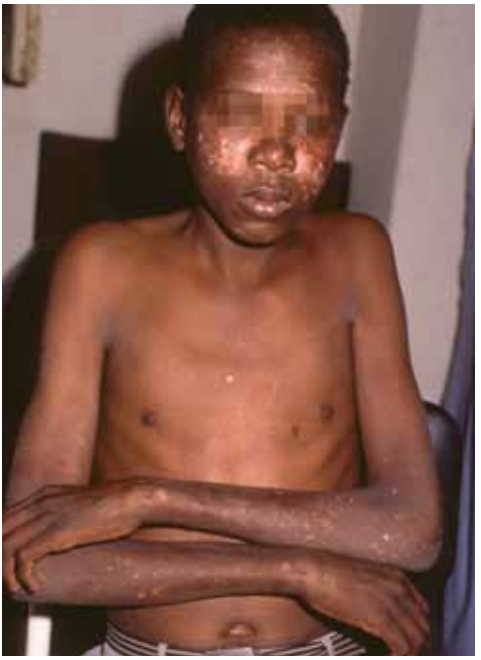


93

Grade 3.3.
Mainly hyperpigmented papules.

94

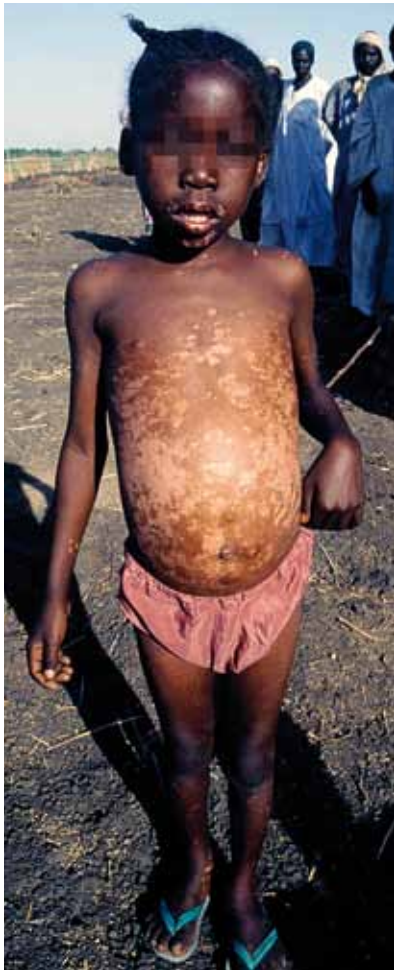




95

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).

96



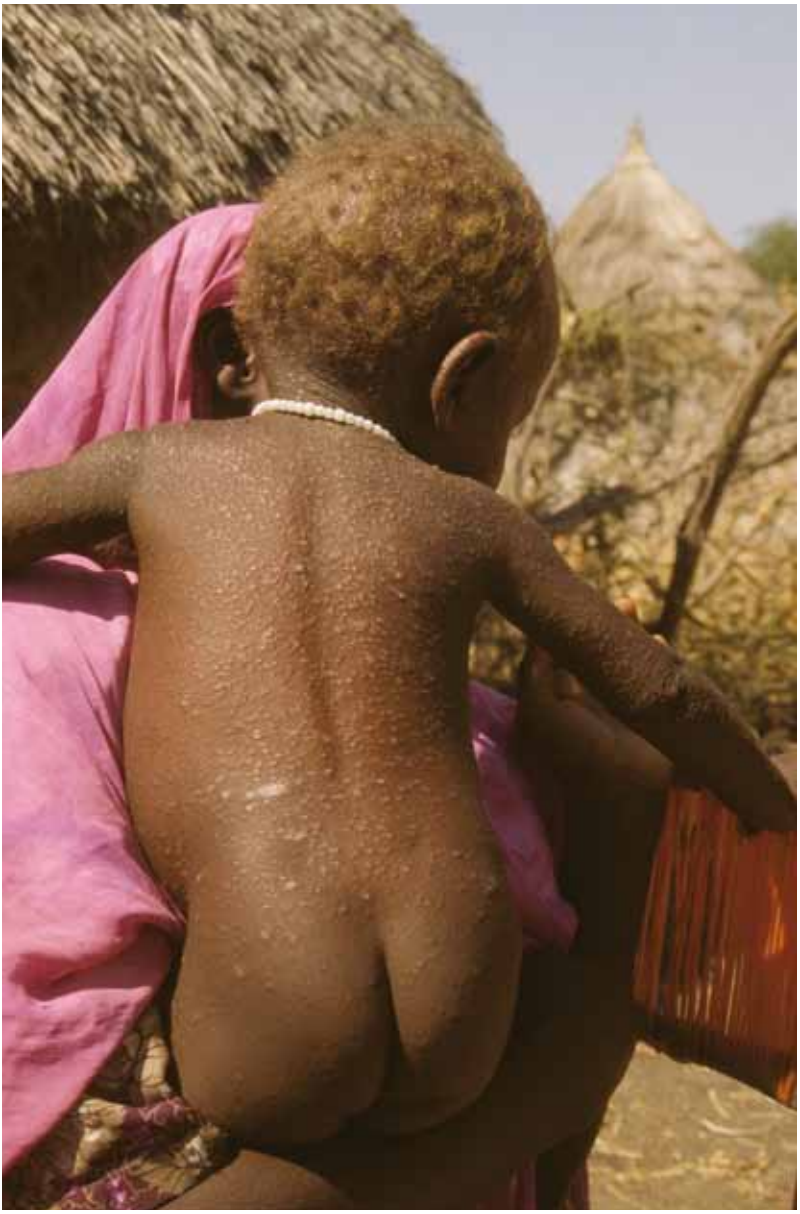
97

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.

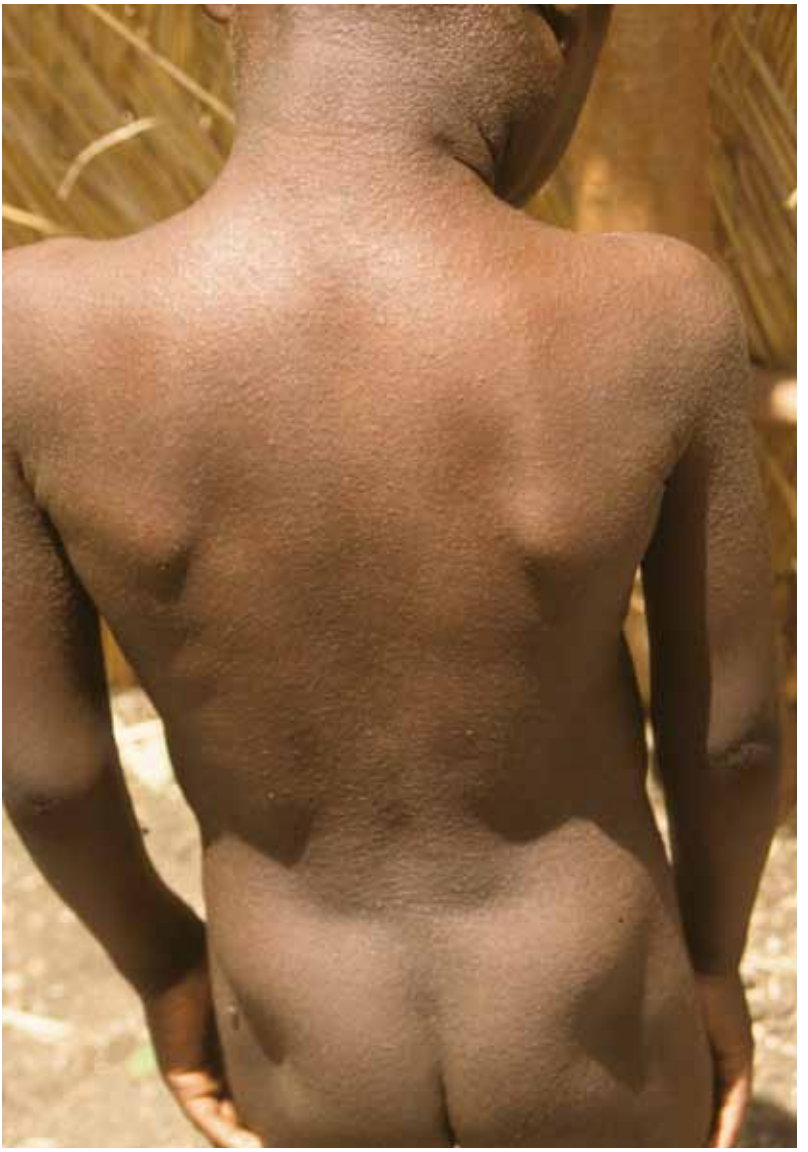
98

99

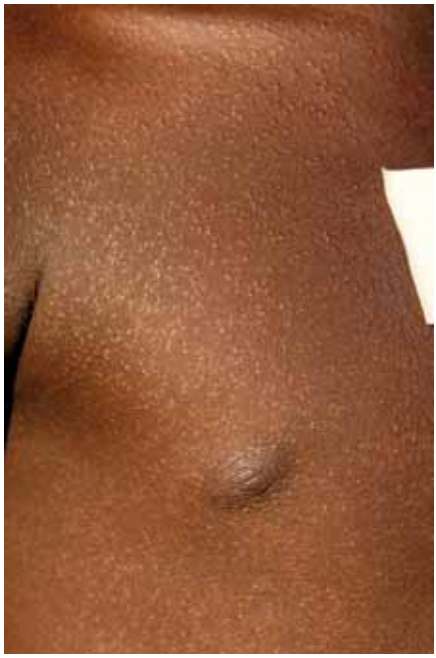
Grade 3.3
Maculopapular rash.



100 Grade 3.3. Papulonodular rash.



101 Grade 3.3 Micropapular rash.

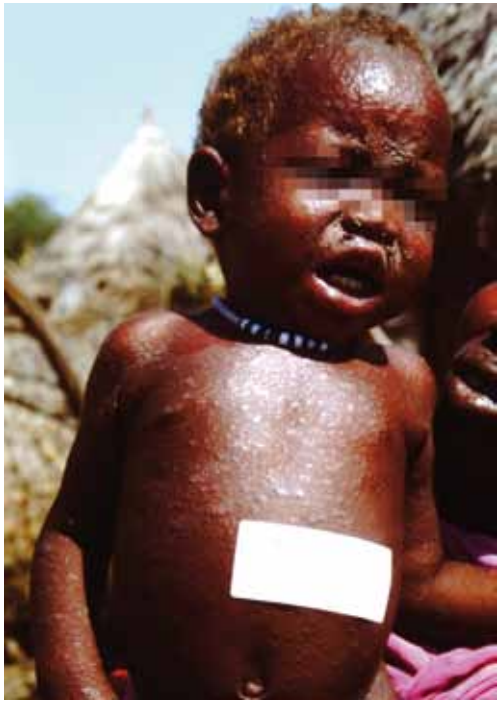


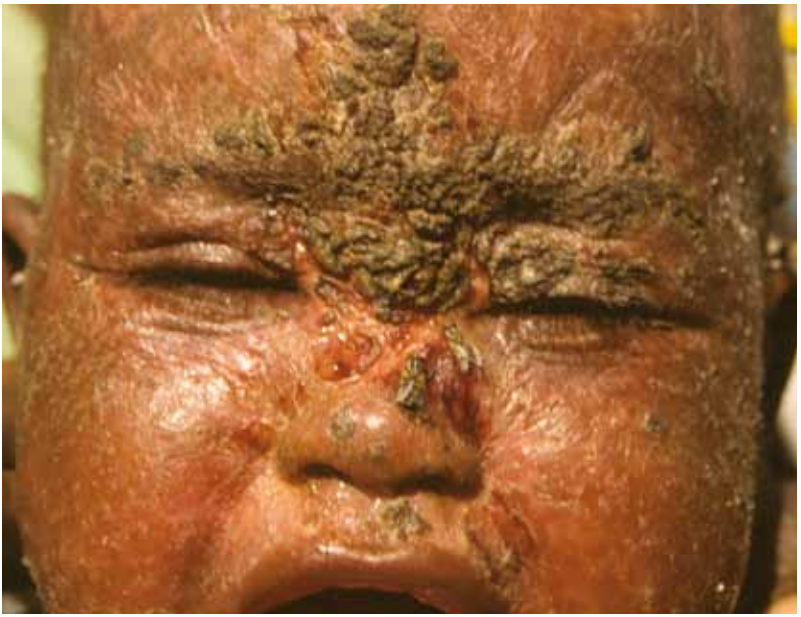
102

Grade 3.3
Close-up: dense maculopapular rash.

103

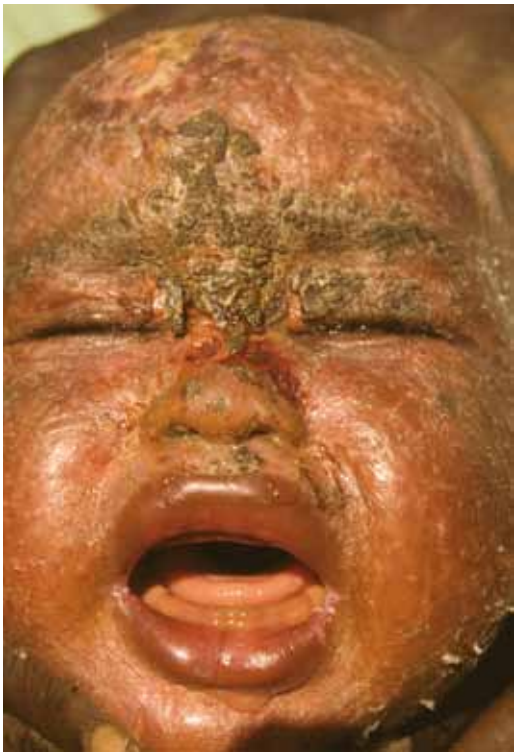
Grade 3.3
Papulonodular rash. All parts of the body are involved with plaques around the mouth and on eyebrows.





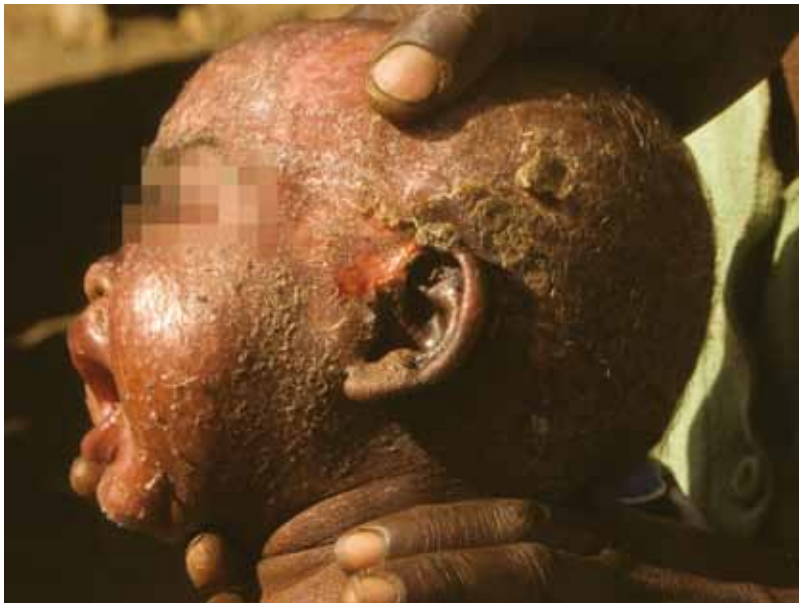
104 Severe PKDL grade 3 in the face with crusts and sloughing of the skin.

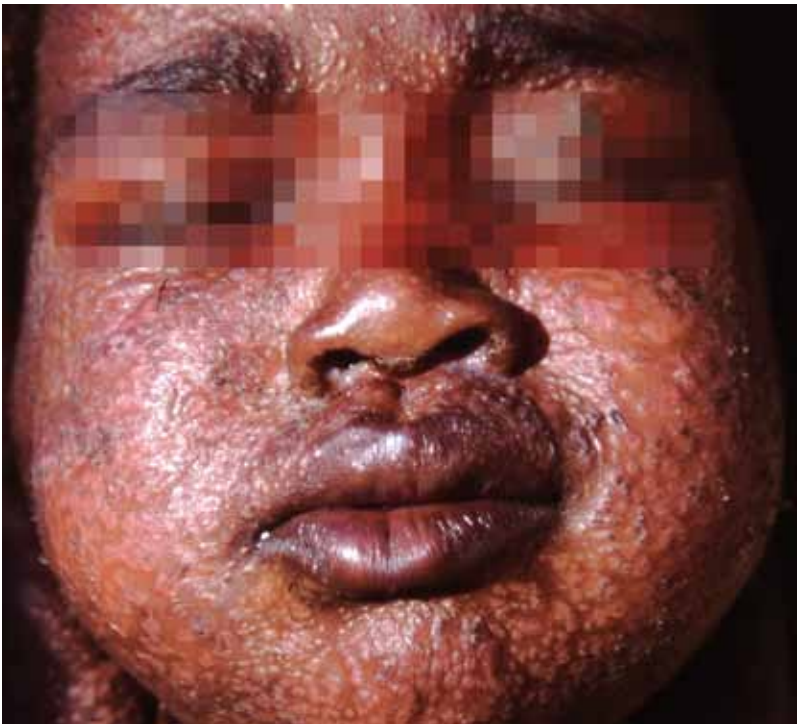
105



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

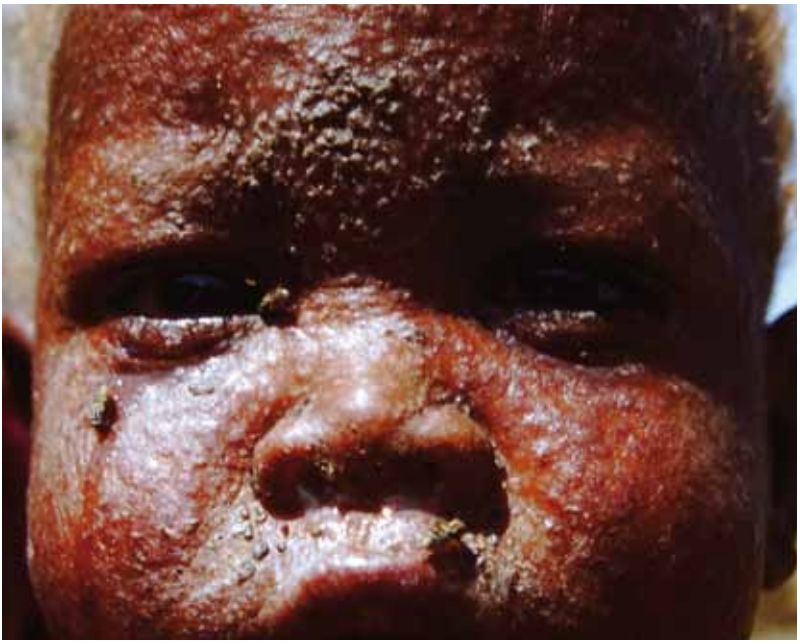
106





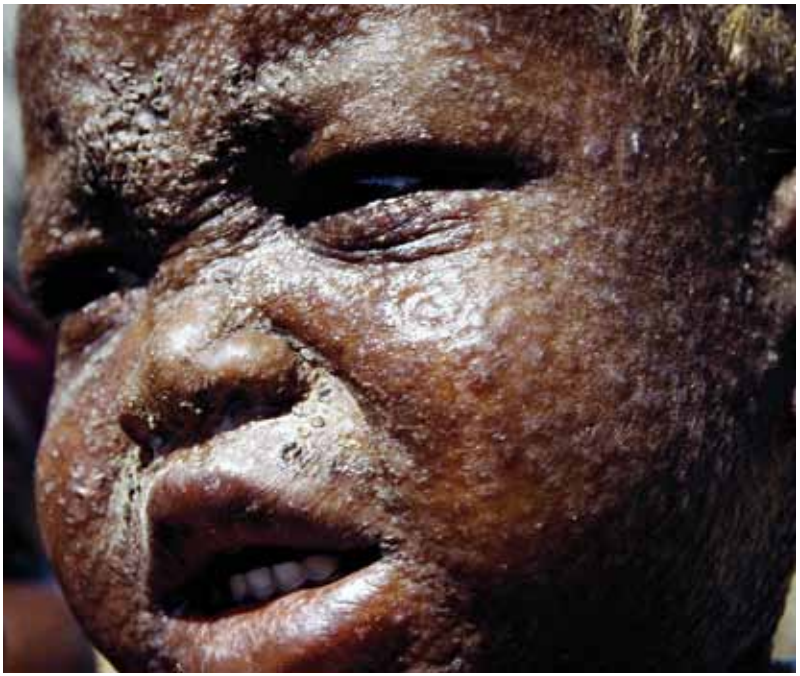
107 Severe PKDL grade 3. (Figures 107-112).

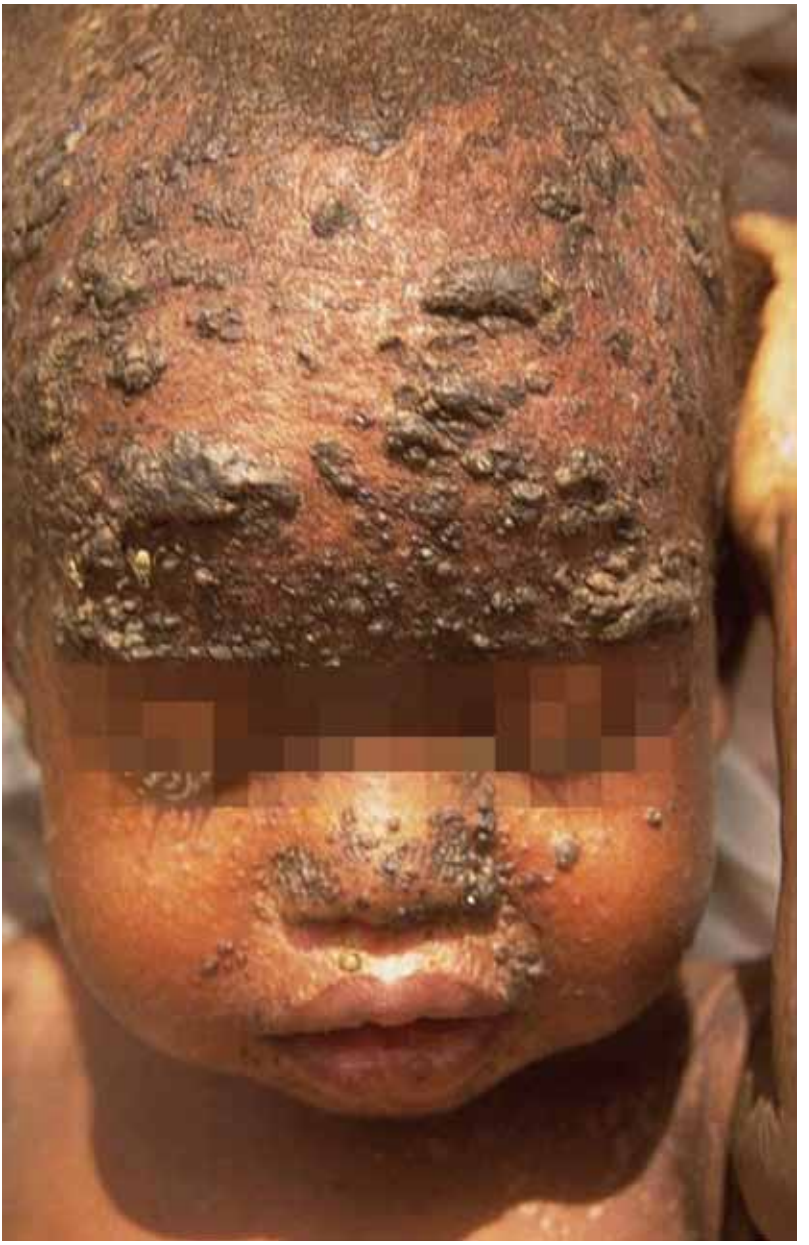
Severe PKDL



108

109





110

Severe PKDL

111

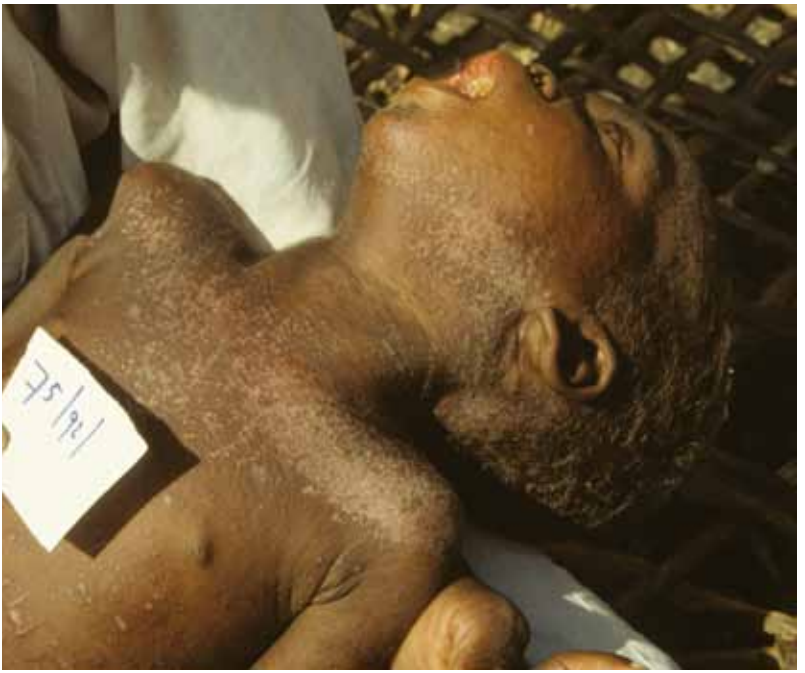
112

113

114

Grade 3.
Desquamation of the skin.

115

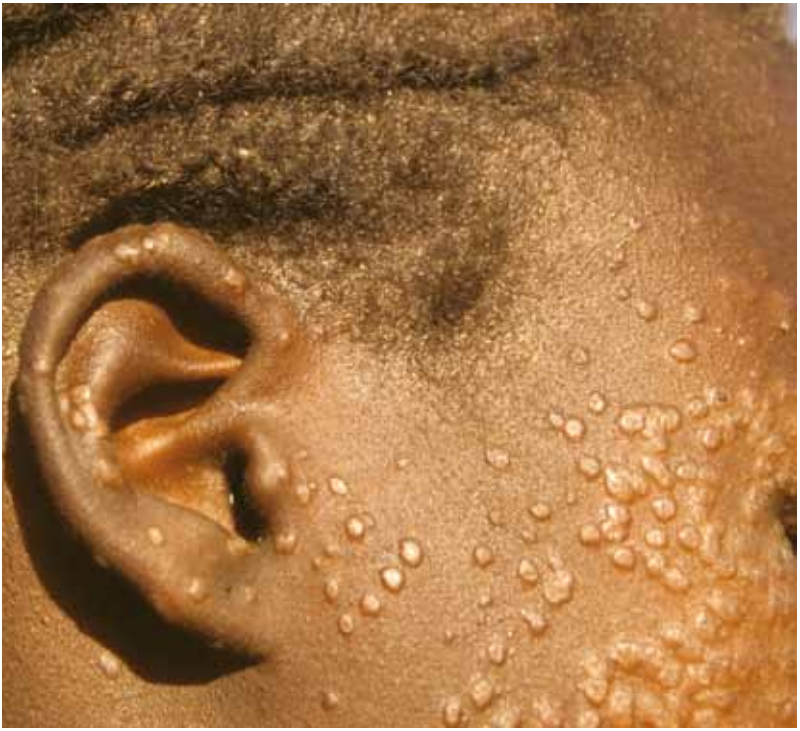


116

Grade 3. PKDL also affects the hands (back and palm).

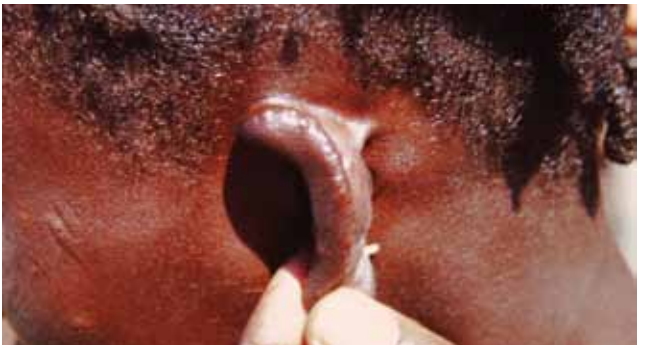
117

118

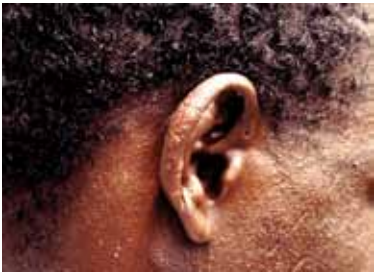


PKDL affecting the earlobe. **119**

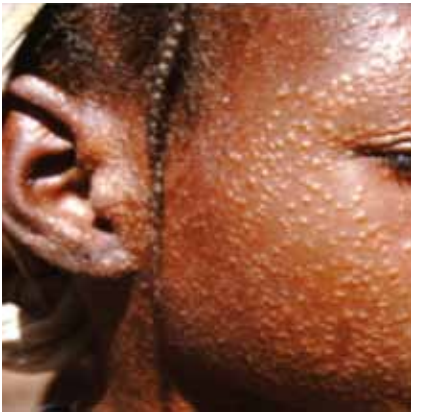
120



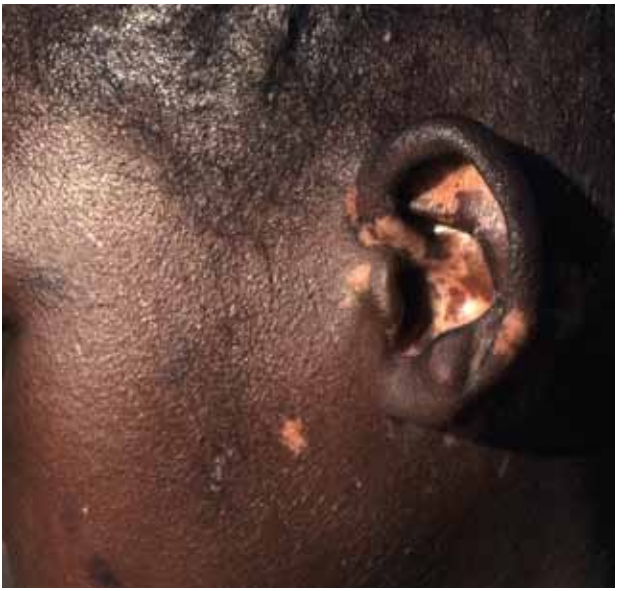
121



PKDL affecting the earlobe; see also fig. 85.

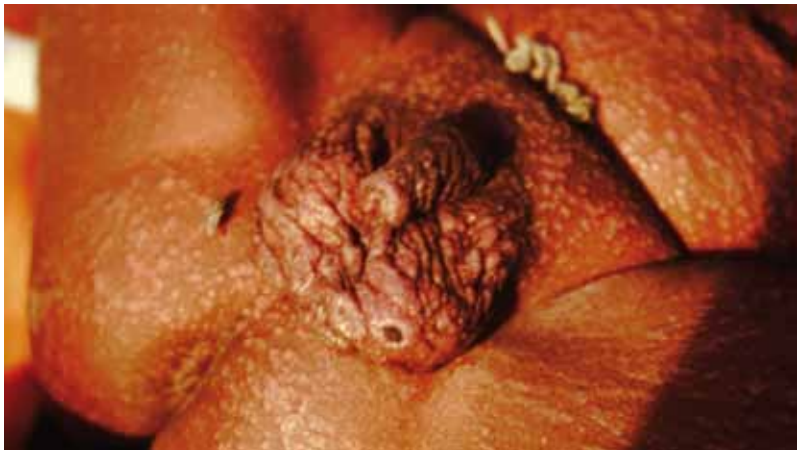


122



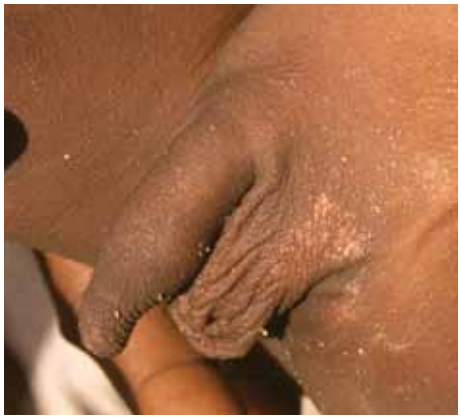
123

Severe PKDL



124

125



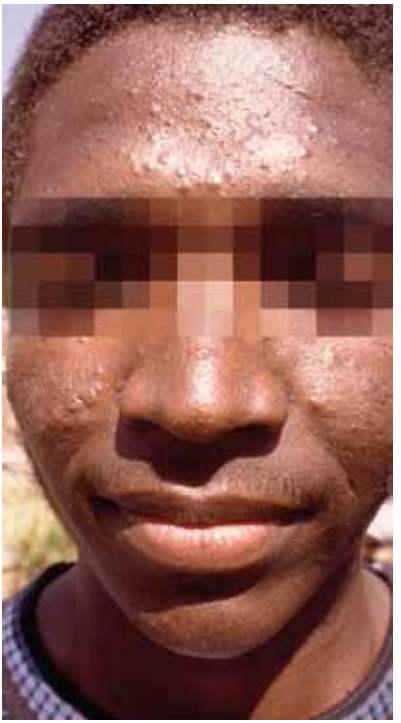
PKDL in the genital area. The ulcerative lesions may be leishmaniomas.

126

127

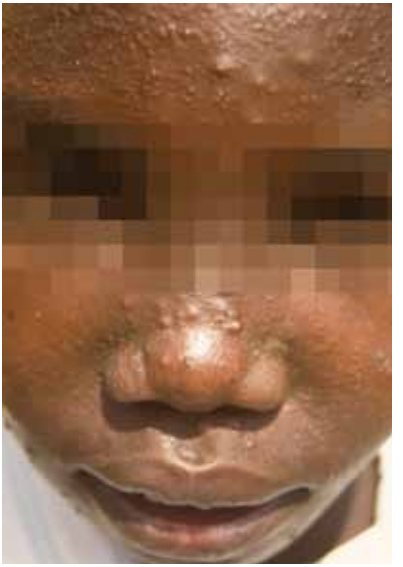
Miliaria rubra.
Clinical clues: young child, wrapped in many layers of cloth, despite hot weather conditions; typically tiny papules on the forehead and not around the mouth as in PKDL; may be itchy.

128



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

129



*Note that some photographs included in this chapter are from other countries than Sudan for comparison

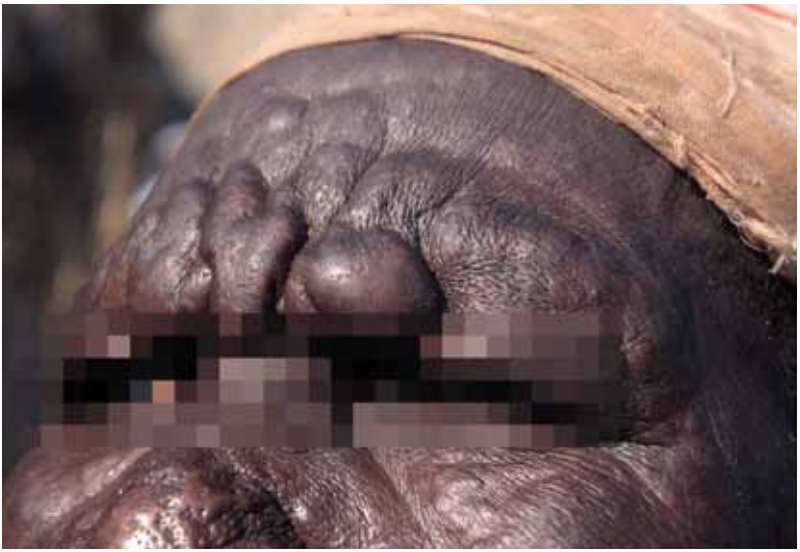
130, 131, 132, 133, 134, 135. Leprosy.
Clinical clues: anaesthetic skin patches, thickened nerves, e.g. greater auricular nerve, peripheral nerve palsies, such as claw hand (ulnar nerve) and wrist drop (radial nerve) with destruction of phalanges.
Top left: the variation in size of papules, nodules and 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.

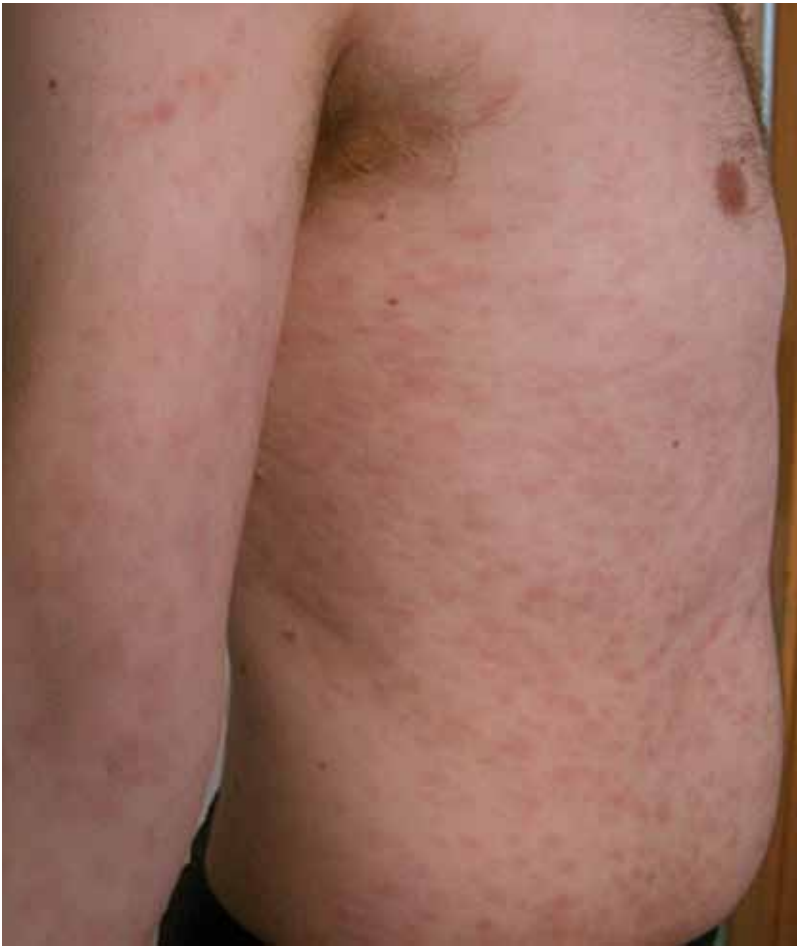
136 Lepromatous leprosy.
Clinical clues:
Elderly person, madarosis, lesions in different stages of development, preference for cooler body parts: upper arms rather than lower arms that are exposed to sunlight.

137

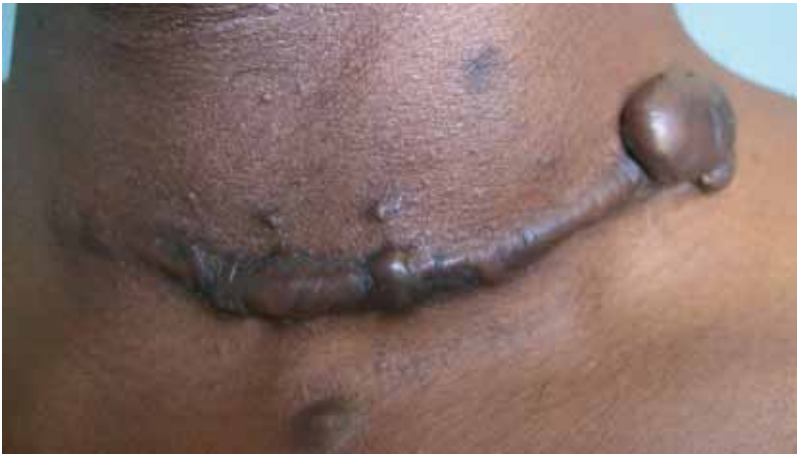
Lepromatous leprosy.
Details of Fig 136.

138





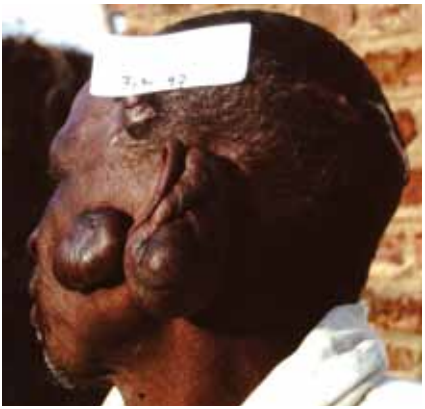
139
Lues stage II.
Clinical clues:
History of genital ulcer; as in PKDL, palms and soles may be involved.



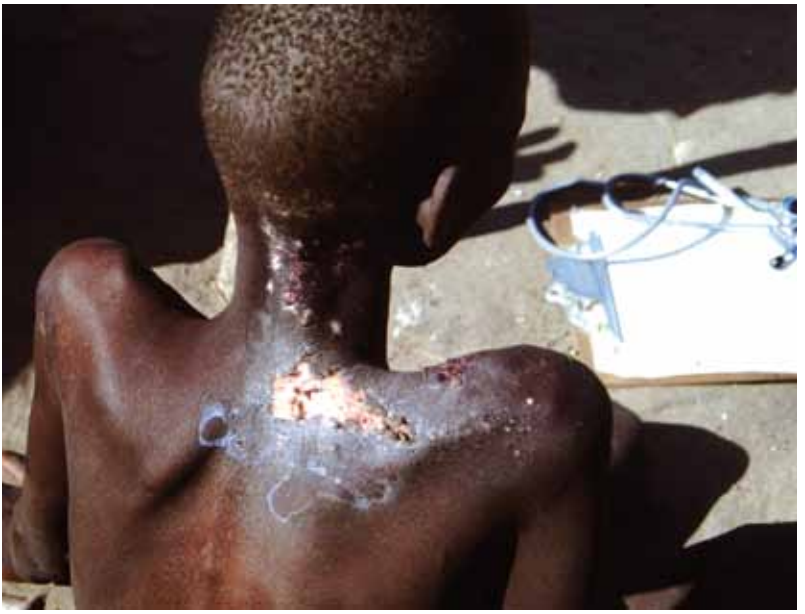
140

141

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.



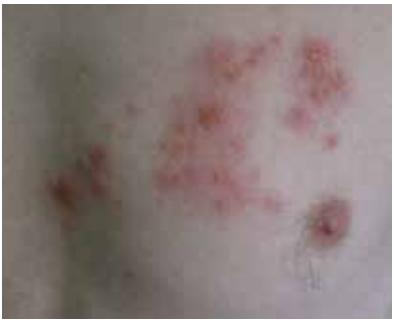
142



143 Herpes zoster in a kala-azar patient.

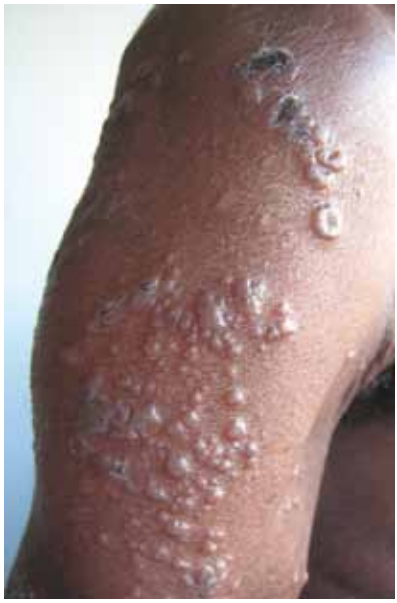
Herpes zoster. Clinical clues: This is typical in a dermatome and may present with vesicles on an erythematous background in a white skin; in a black skin, mainly vesicles are seen that may break down. It is common in Africa in young people as an early sign of HIV infection

144 Herpes zoster scar in ophthalmic branch of the trigeminal nerve.



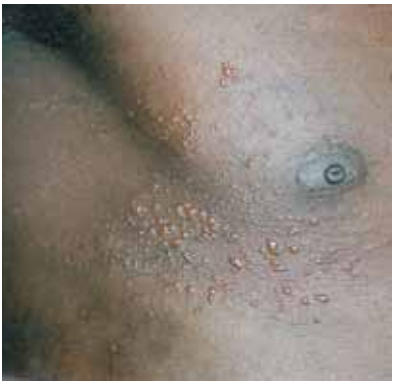
145
Herpes zoster in white skin; vesicles on erythematous background.

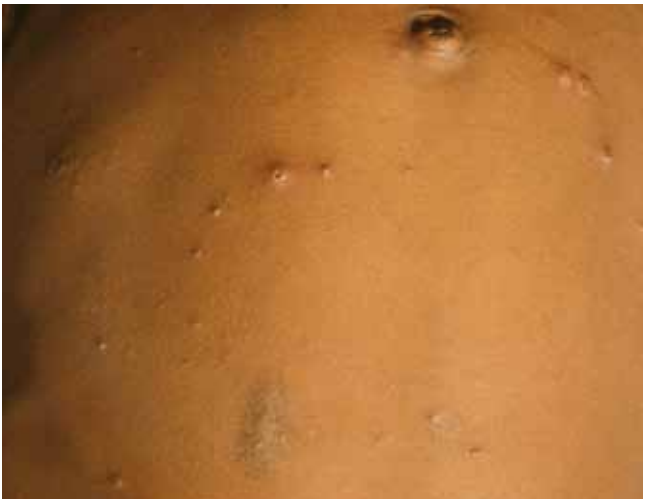
147



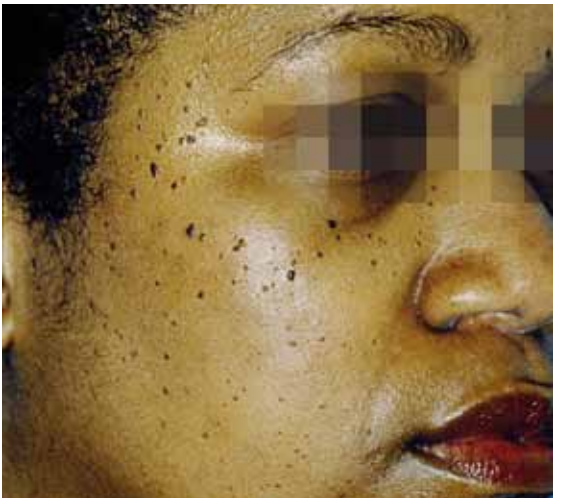
Herpes zoster in the black skin; because of the different skin structure, the vesicles are less likely to break down. (147, 148).

146



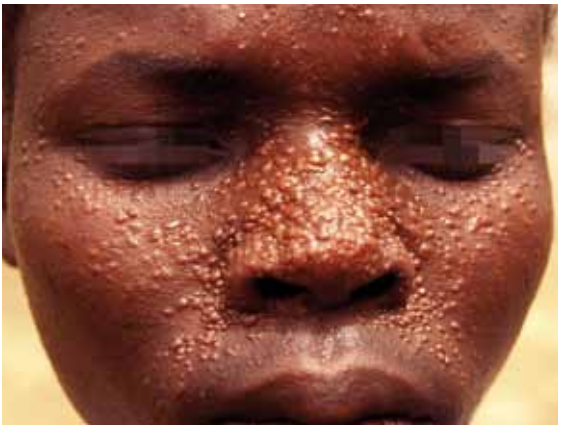


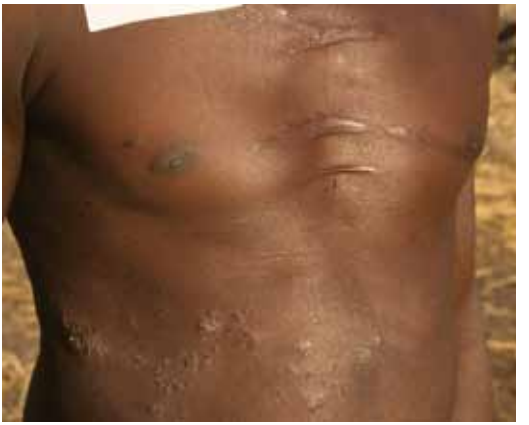
148 Varicella (chickenpox). Clinical clues: Vesicles in various stages of development; evolves over days.



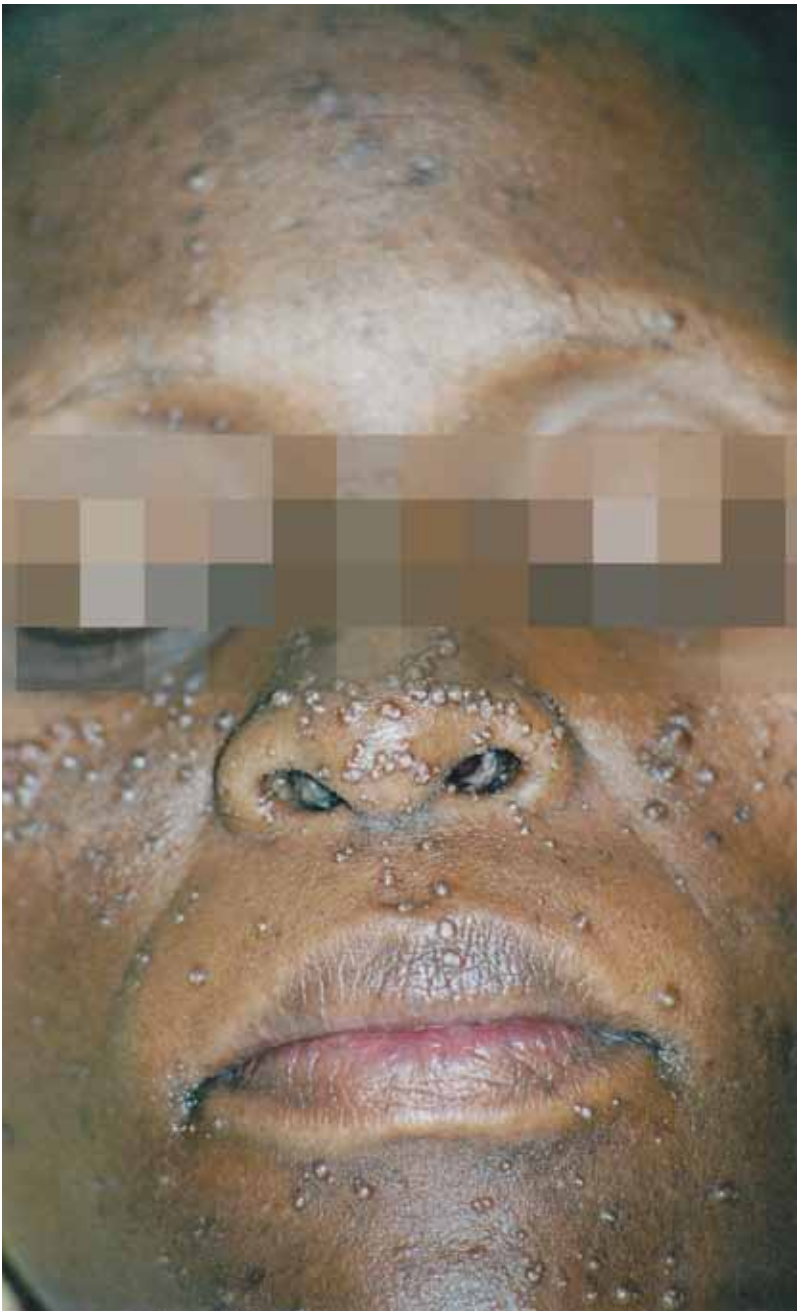
149
Dermatosis papulosa nigra.
Common in the black skin, mostly in upper part of face, cheeks and temples. Numbers of papules increase with age; starts during adolescence.

150
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.





151
Keloid after varicella in childhood; now presents with herpes zoster scar.

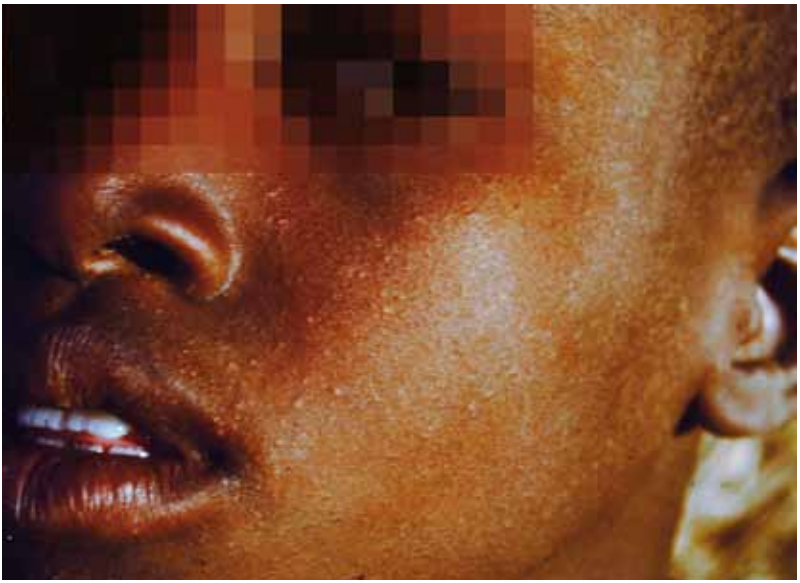


152
Mollusca contagiosa in an HIV-positive patient.

153

Noma or cancrum oris in a VL patient who had relapsed after treatment; note the splenomegaly.

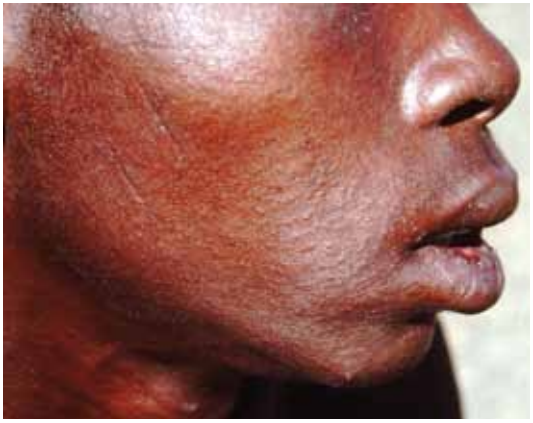
154



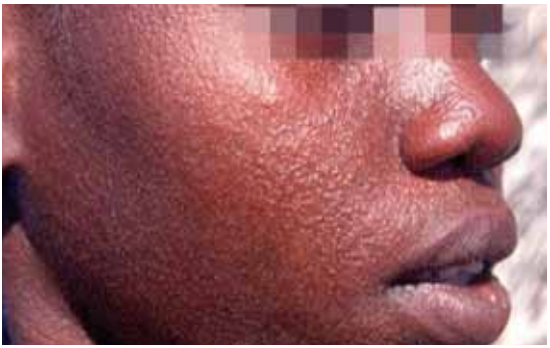
155

156

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

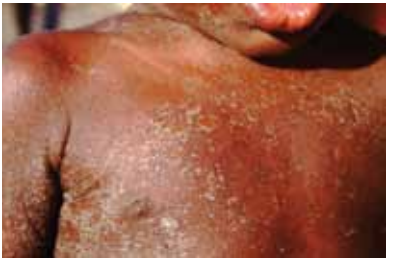


157 Measles-like PKDL.



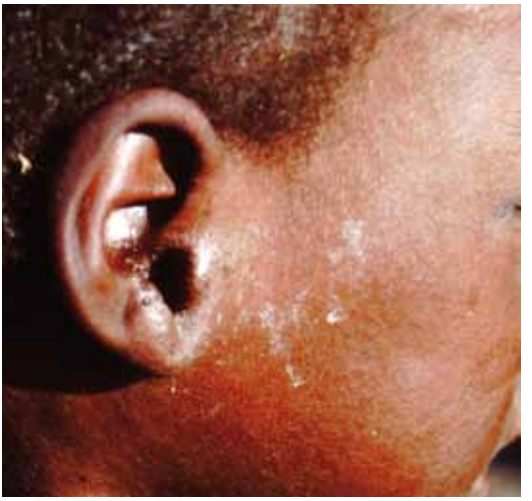
158

159

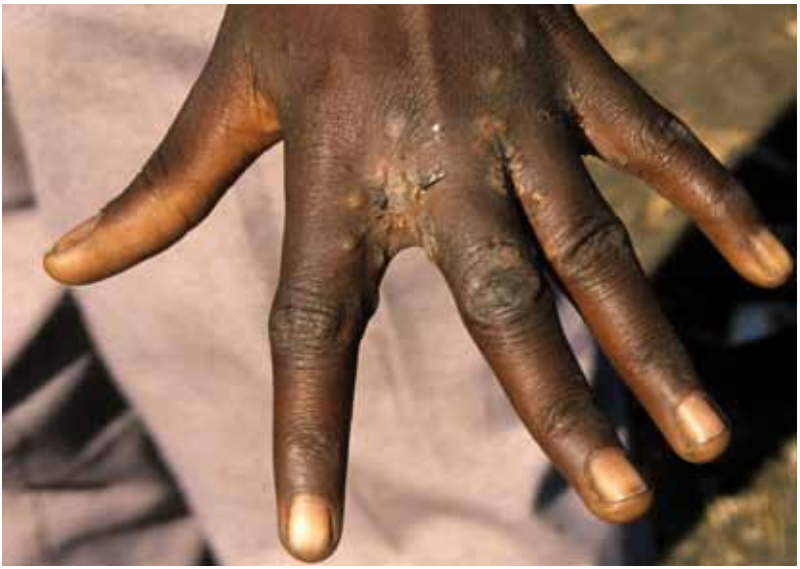


160

161

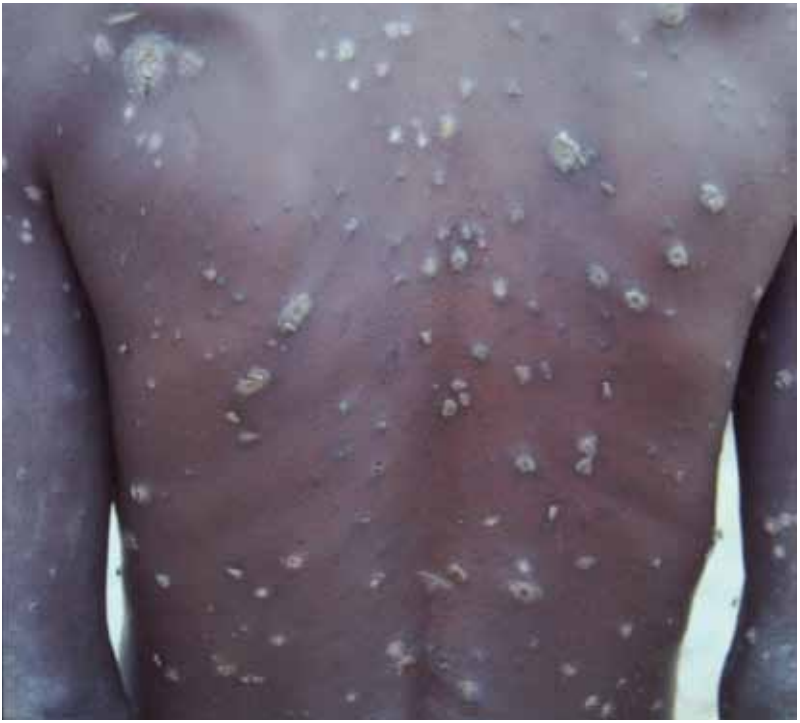


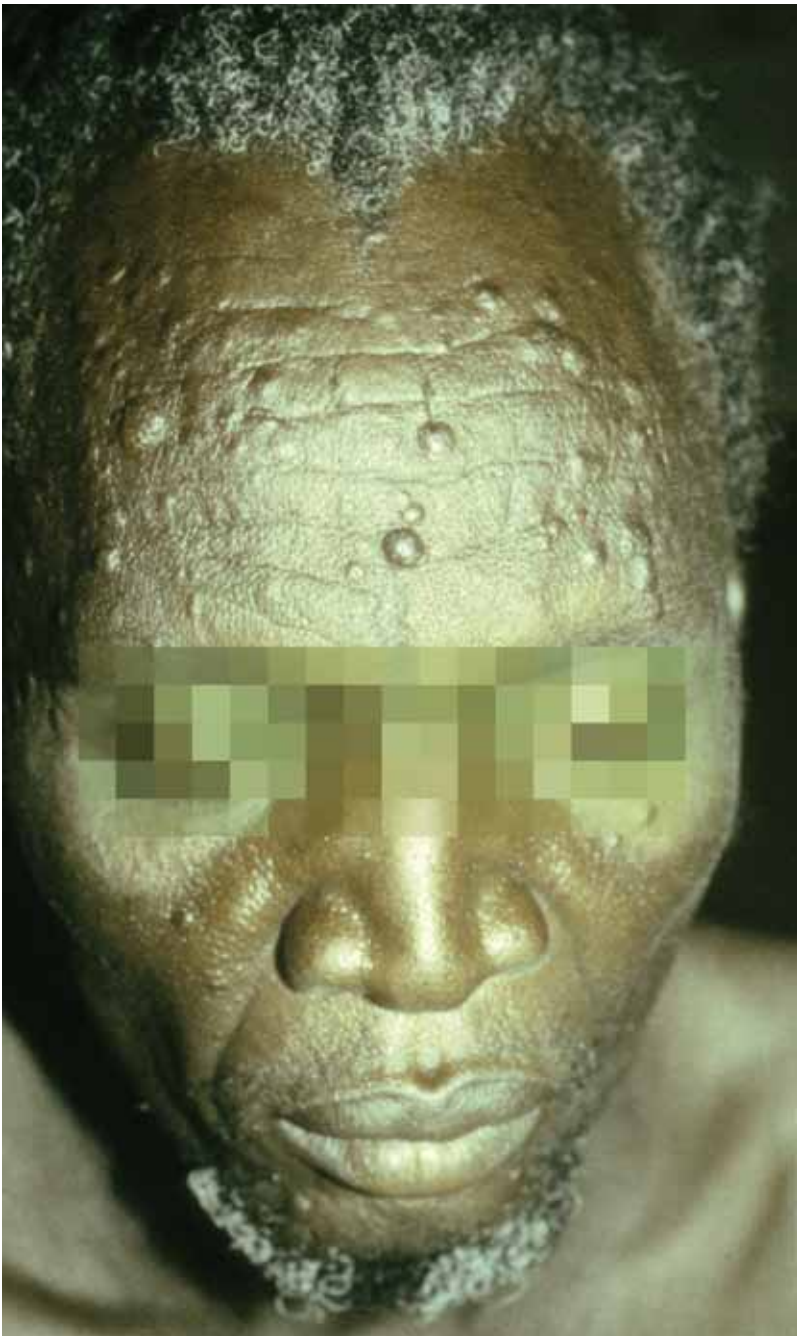
Measles.
In measles, the rash can be virtually indistinguishable from the micropapular rash in PKDL. The rash may also desquamate (159) (see also 113-115).
Clinical clues: look for other signs of measles such as fever, cough, conjunctivitis (158), Koplik's spots (160) and otitis media (discharge of pus probably as a result of a perforated eardrum, 161).



162

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

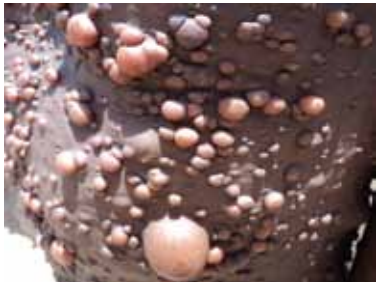




164 **African histoplasmosis.**

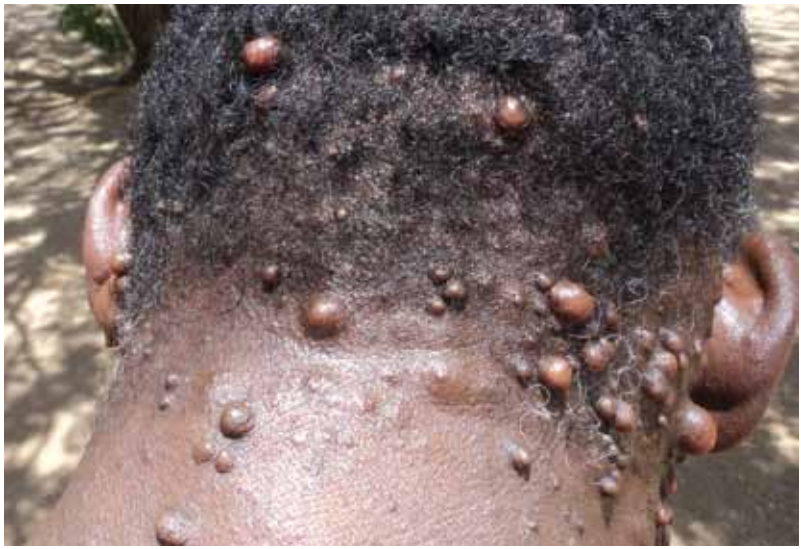
165

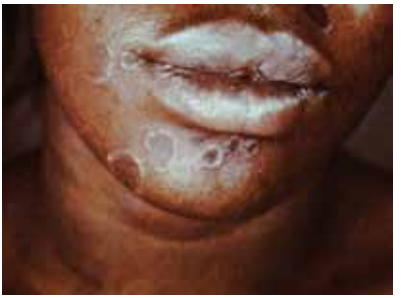
Neurofibromatosis.
Clinical clues:
Family history: in this case, his grandmother, father and his 2 siblings also had the disease compatible with autosomal dominant inheritance. Chronic slowly progressing. Nodules in different sizes, also affecting the scalp. The nose and earlobes remain free. Note the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



166

167





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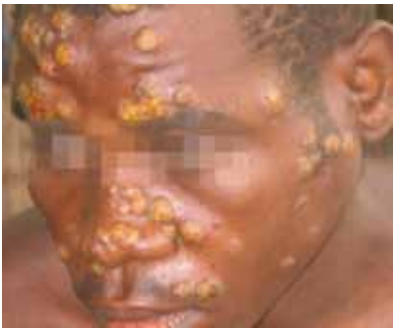
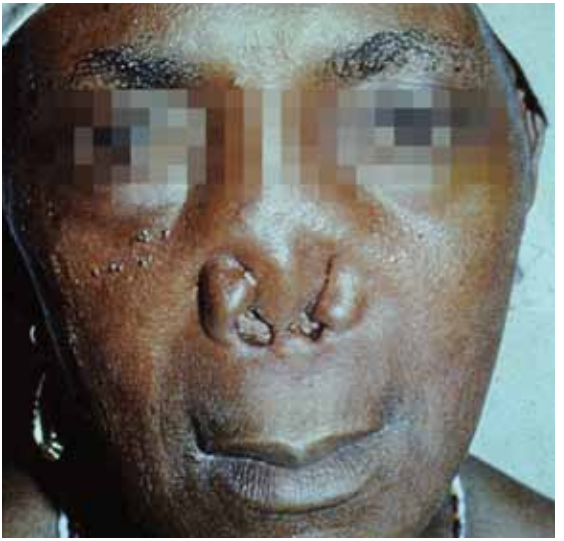


169

170



171

168 Endemic syphilis, macular lesions with raised edge.

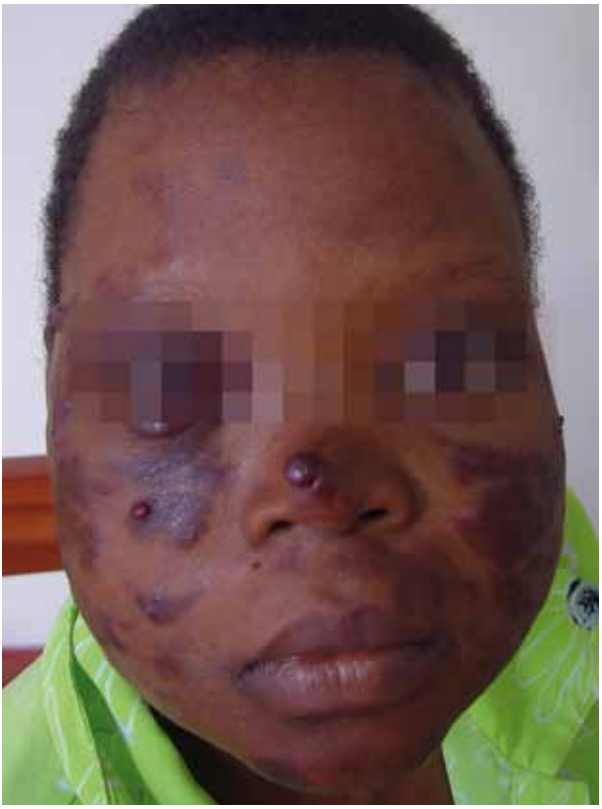
169, 170 Yaws, nodular (from the Democratic Republic of Congo) Note atypical distribution on face and extremities, leaving chest clear.

171 Late **yaws**: gangosa.

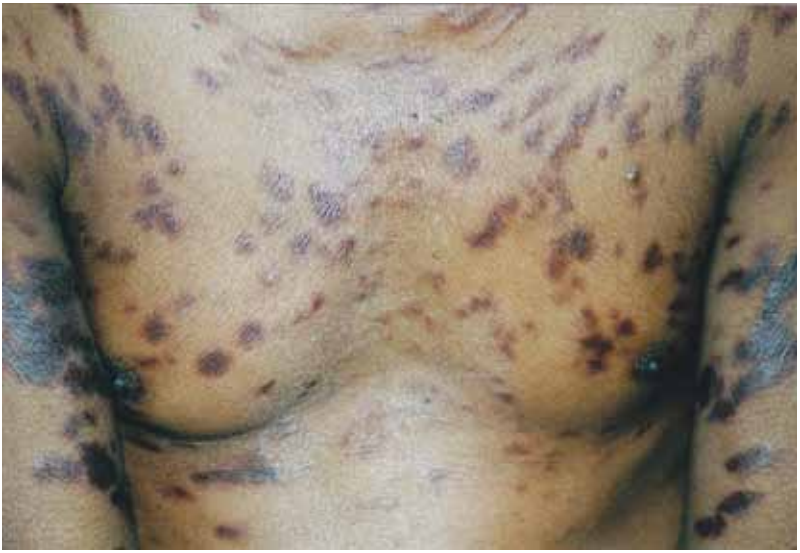
Differential diagnosis of papular and nodular PKDL

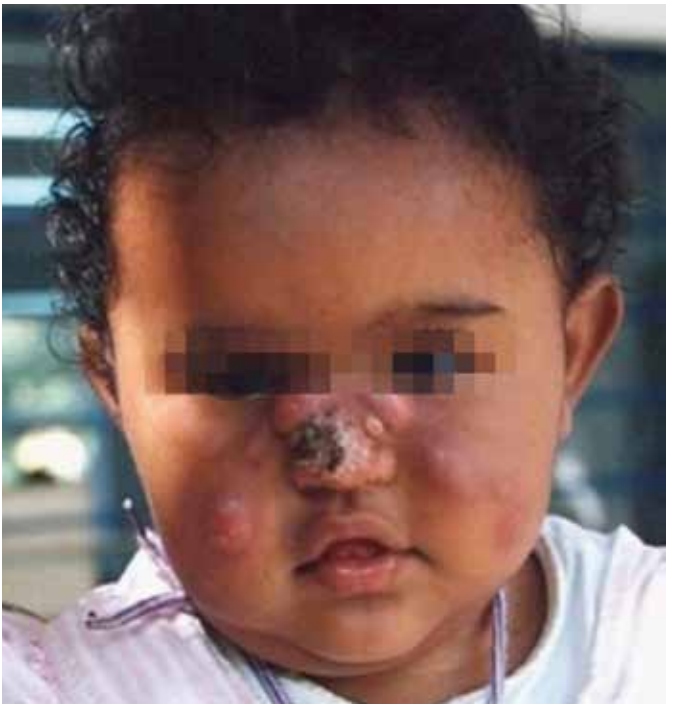
172

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.



173

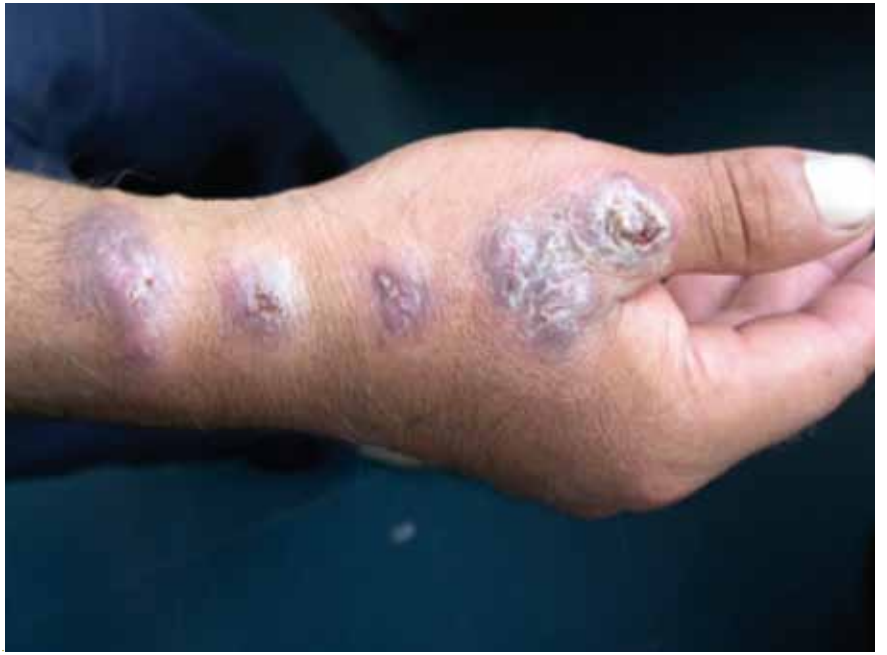




174

Sporotrichosis. Spread along the lymphatics. While this is also seen in cutaneous leishmaniasis, it is not a feature of PKDL.

175



176

Shilluk tribe.

Tribal markings.

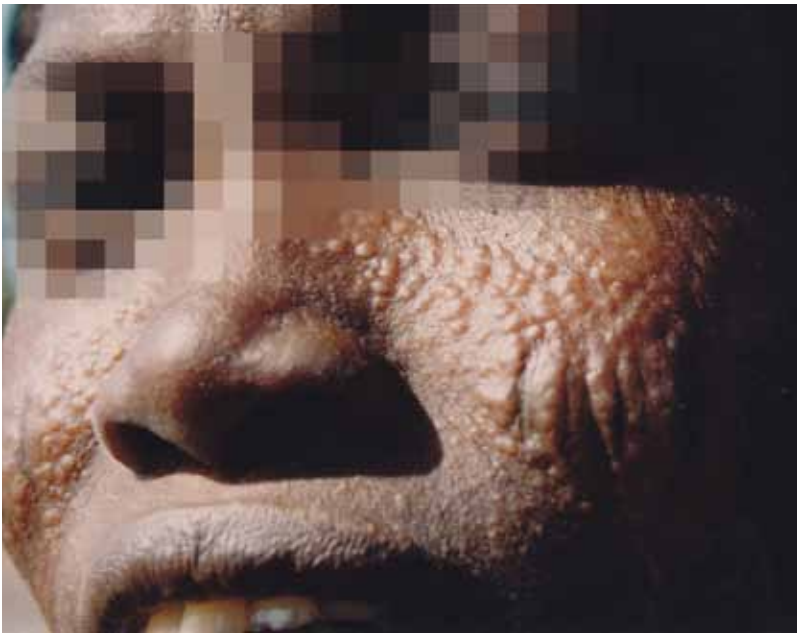
Nuer tribe.

177

178

Nuer tribal markings, with PKDL papules occurring between the markings.

179

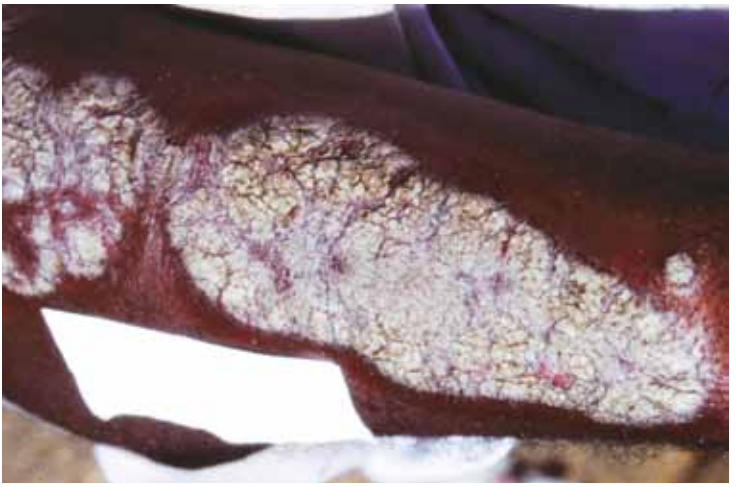


180
Nuer tribe. The papules do not appear on the forehead, perhaps because of fibrosis caused by scarring.

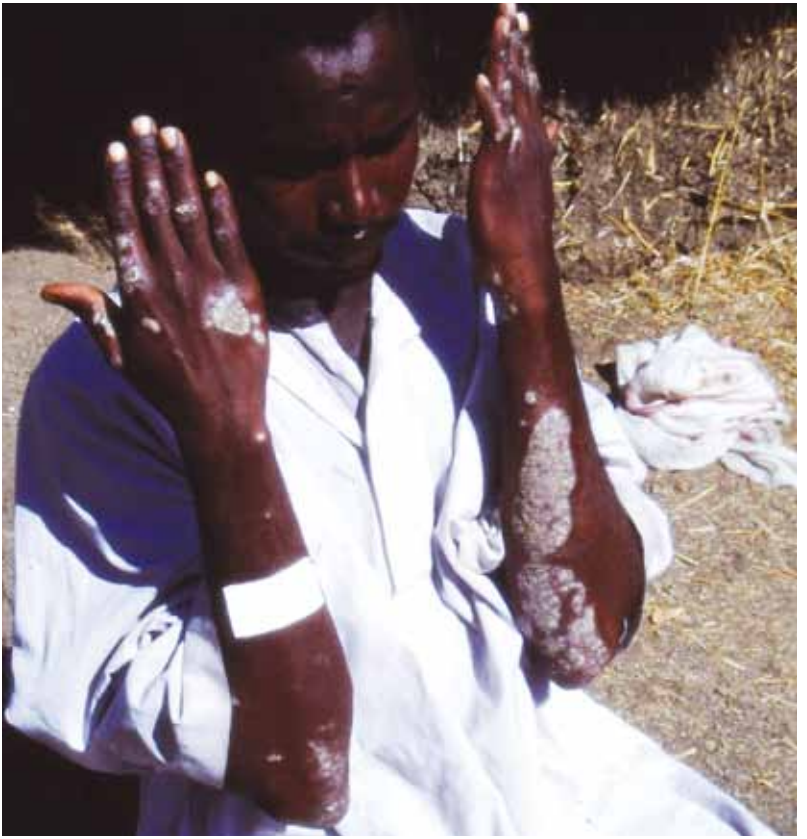
Nuer tribe. Here the papules are also found on the forehead despite the scarring, but with less density than on the cheeks and nose.

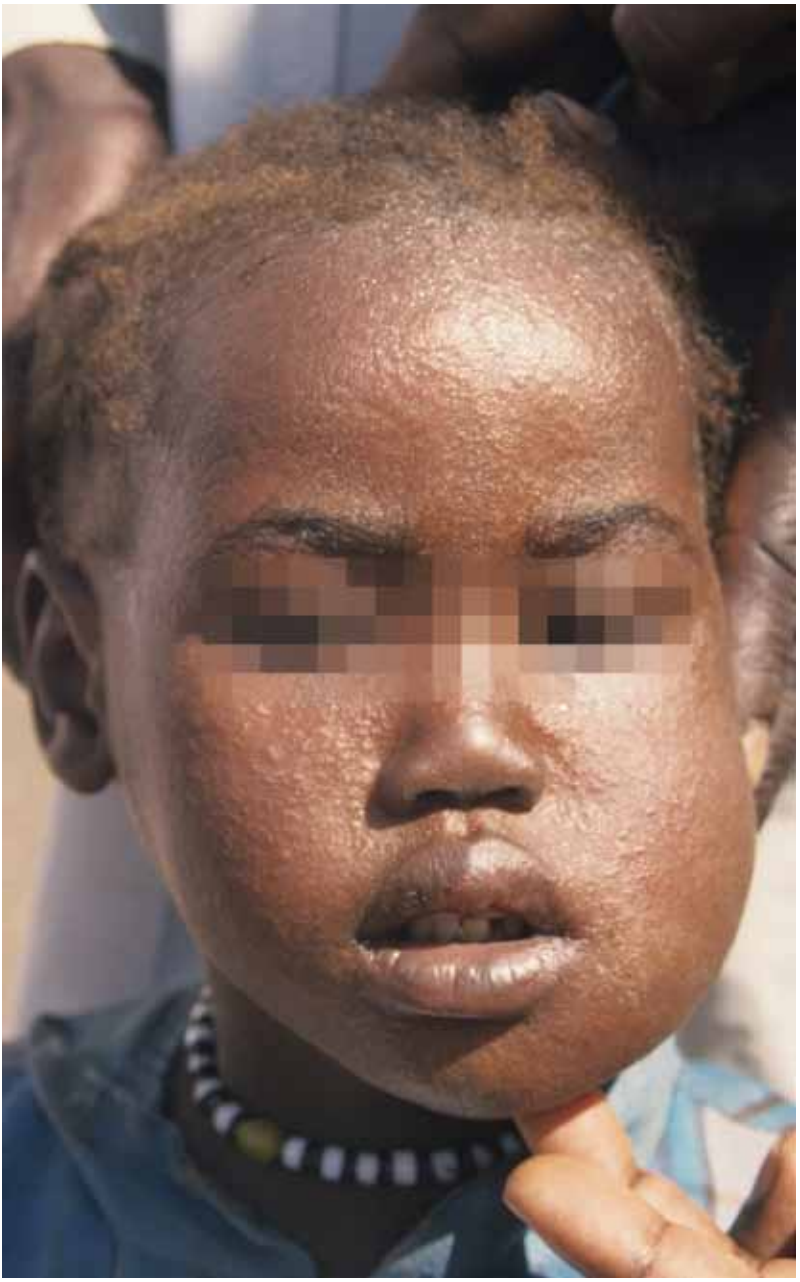
181

182

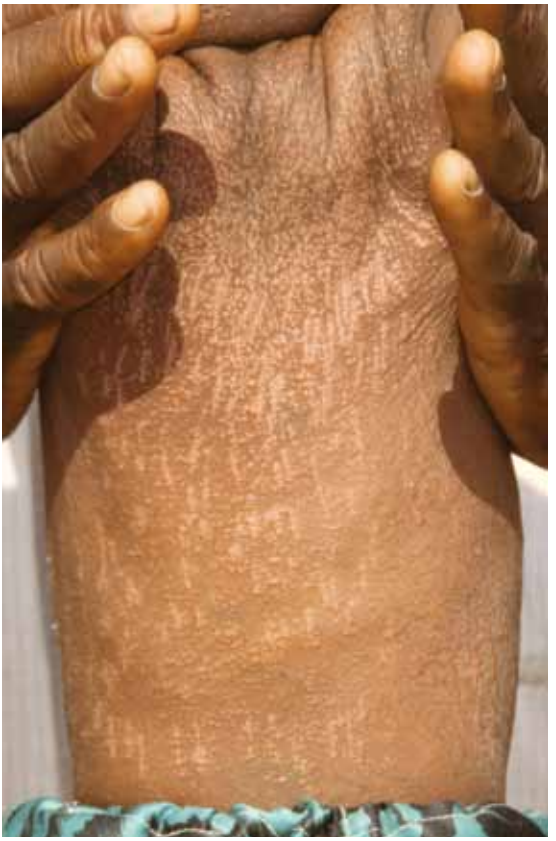


183, 184 Psoriasis.





185 **PKDL.**
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.

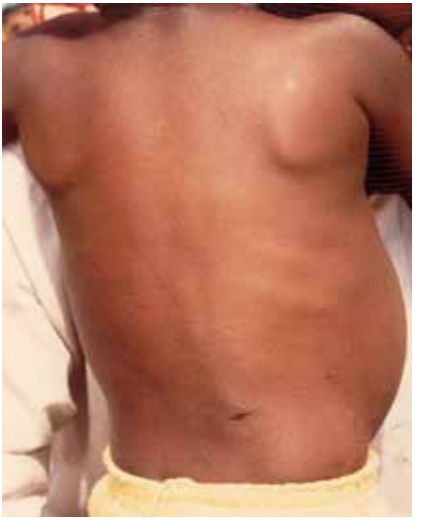


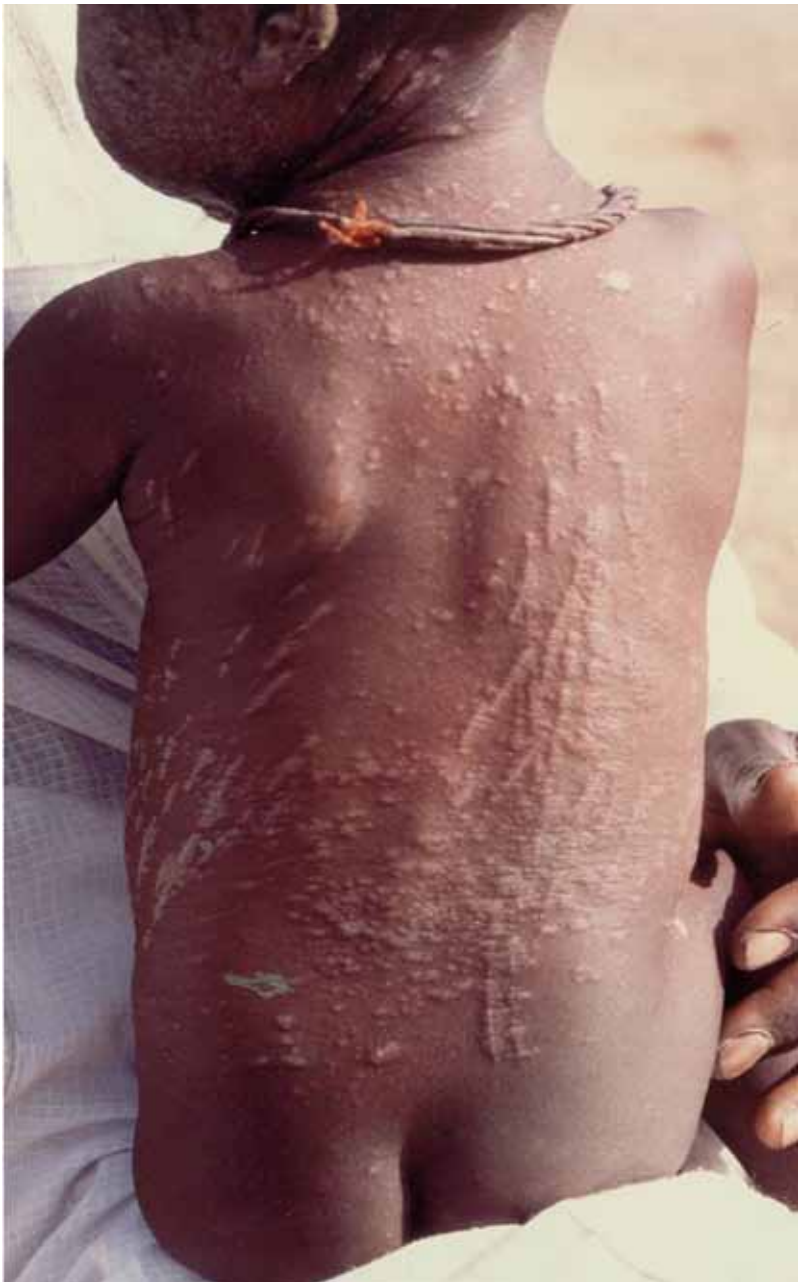
186

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.

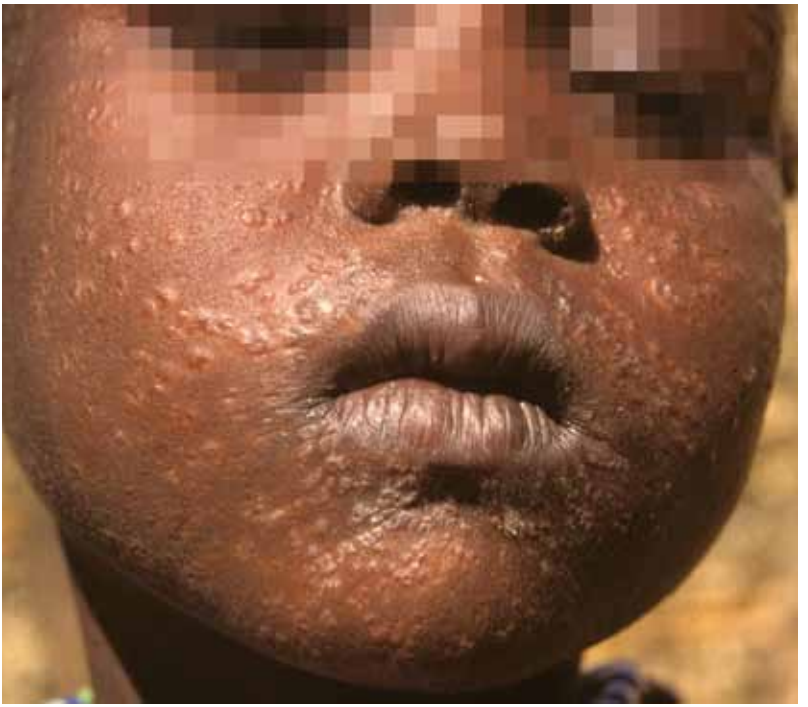
187

188

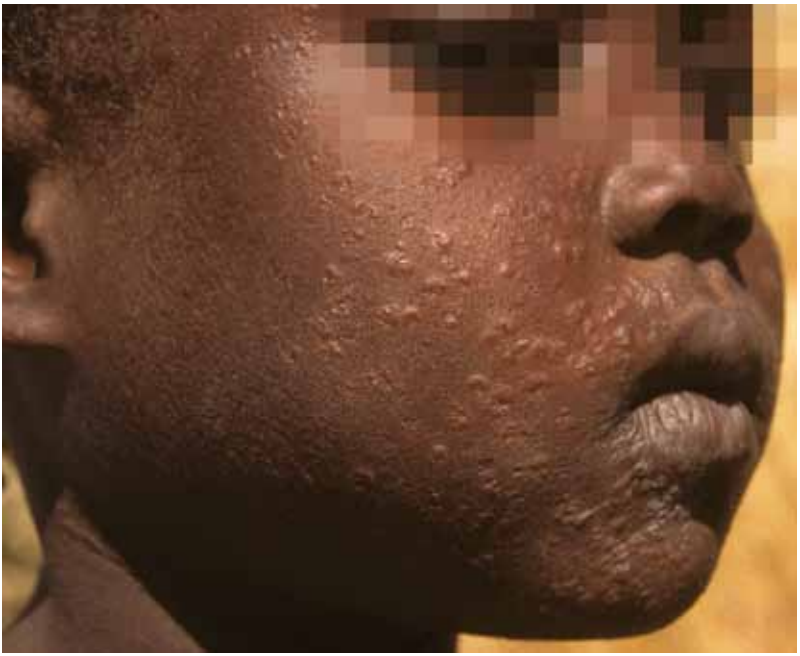




189 Köbner's phenomenon.
Note the preferential localization of the papules in the scars causing an asymmetrical distribution which is otherwise unusual in PKDL.



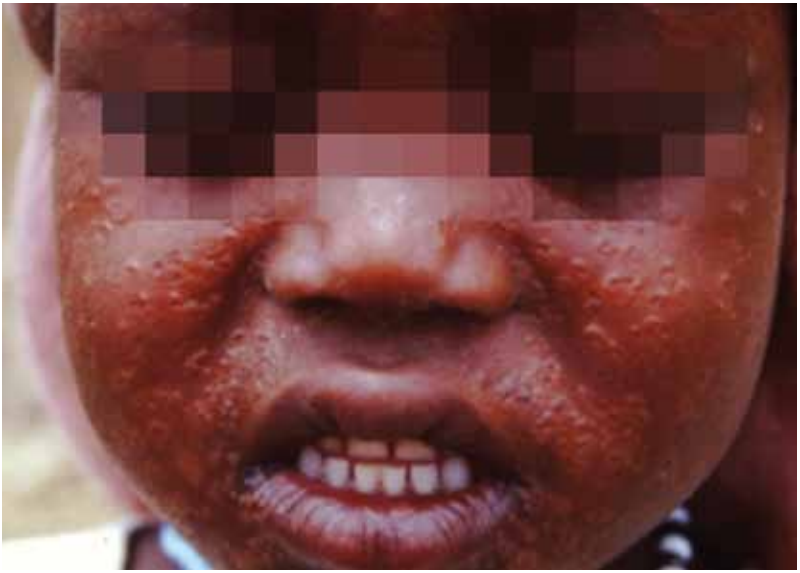
190 Depressed scars developing after longstanding PKDL.



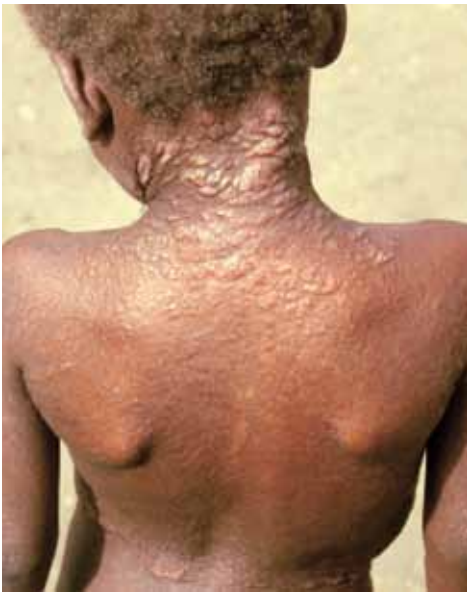
191

Depressed scars developing after longstanding PKDL.

192



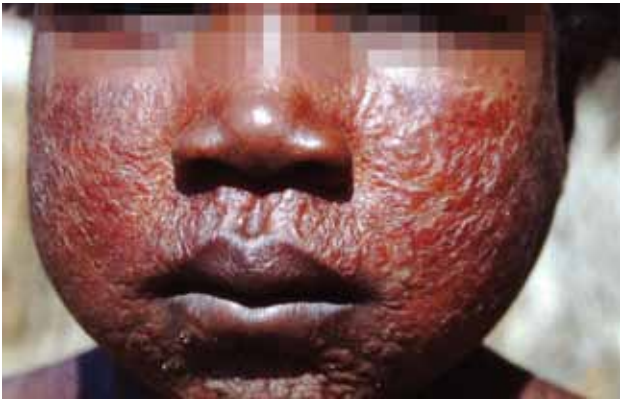
193



194

195

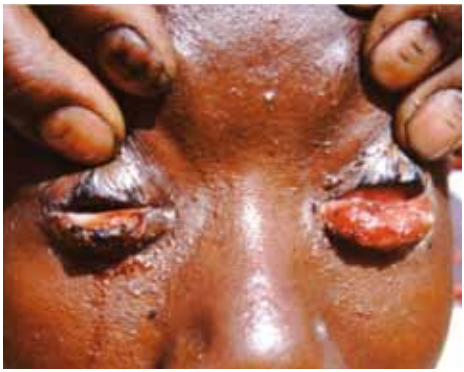
Fibrosis of the skin in longstanding PKDL.



196

Post kala-azar manifestations

197



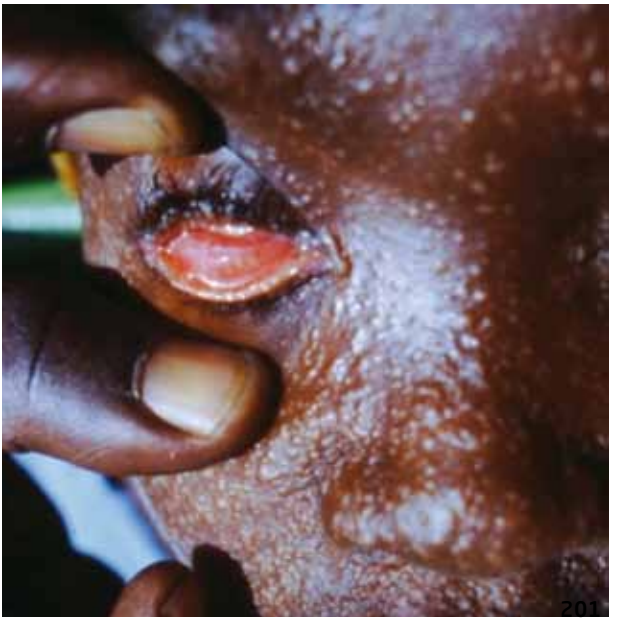
198

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.

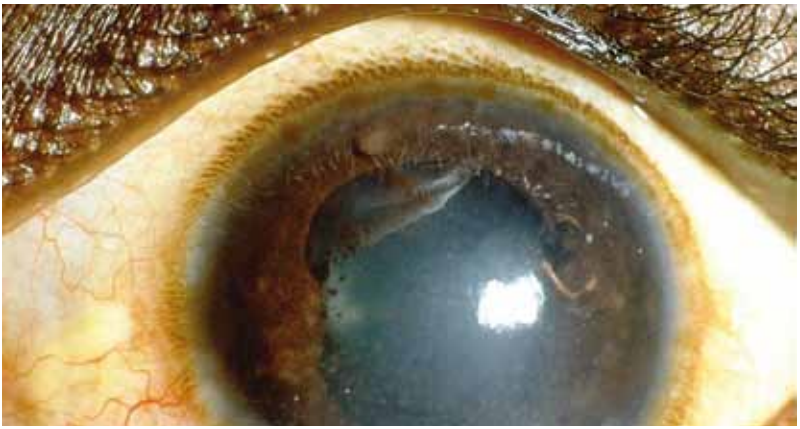
199

200

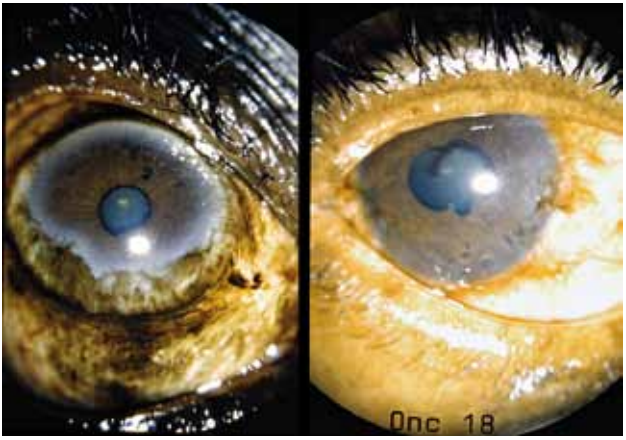
Concomitant PKDL and post-kala-azar conjunctivitis (top) and blepharitis (bottom).



201



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



Differential diagnosis:

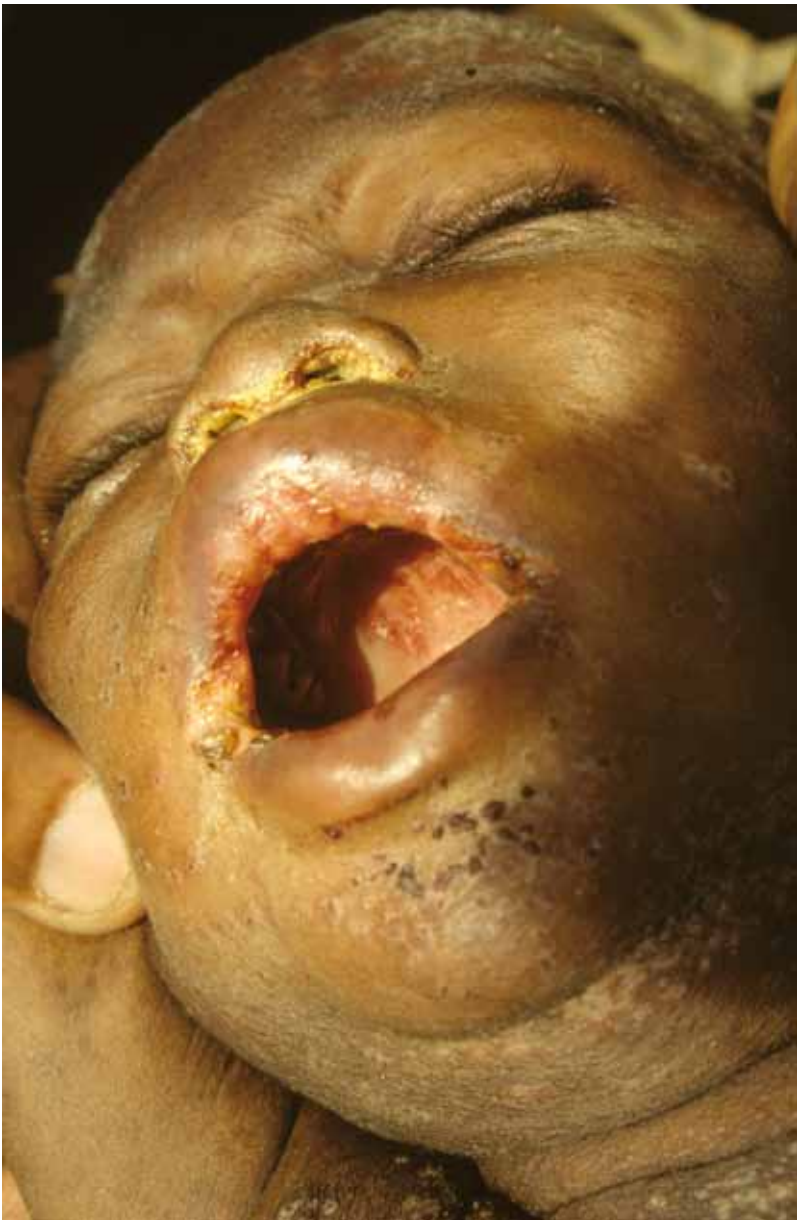
203

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.



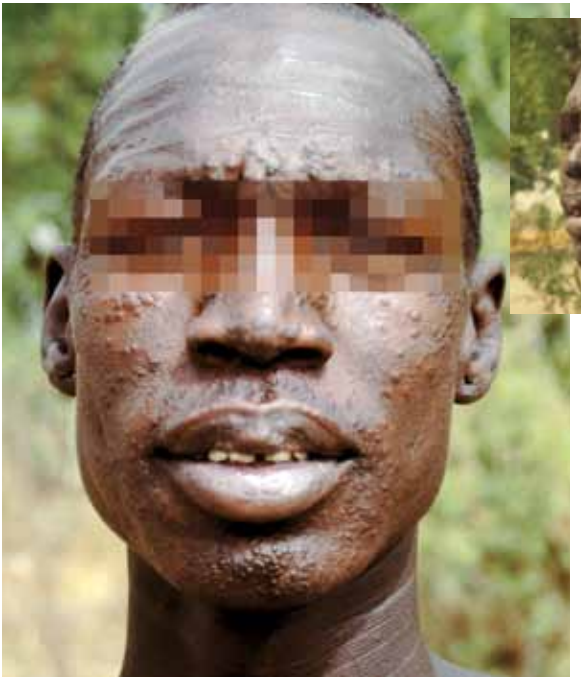
206 Mucosal lesions in severe PKDL.

207 Para-kala-azar dermal leishmaniasis.
VL (confirmed in a lymph node aspirate) and concomitant micropapular PKDL.

208

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.

209



210

211

Evolution of PKDL

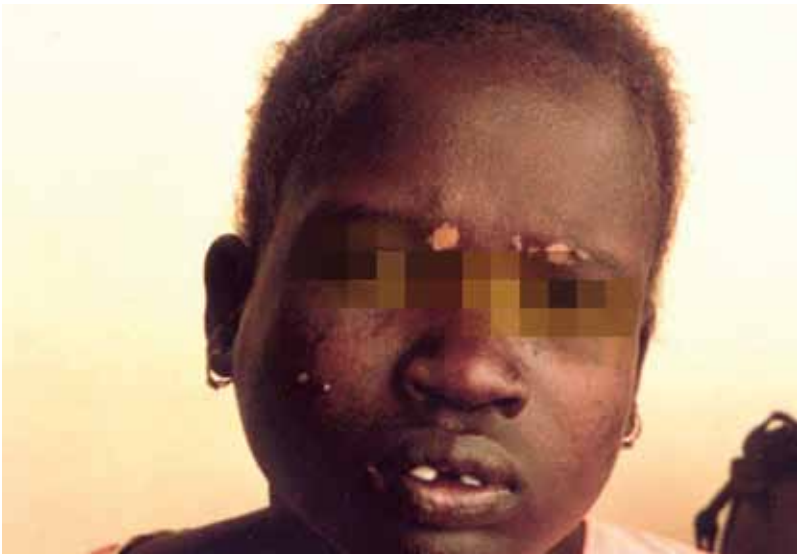
212

213

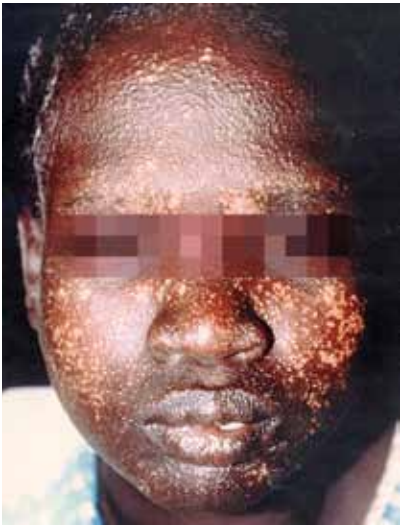
Before treatment (212, 213), after 30 days of treatment with SSG (214) and after 6 weeks (215).

214

215



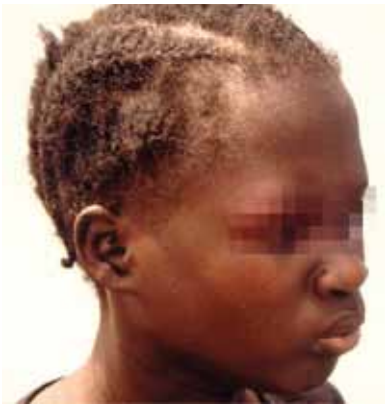
216



217

Initial presentation (216), 2 weeks later (217) and after treatment (218).

218



PKDL in Ethiopia

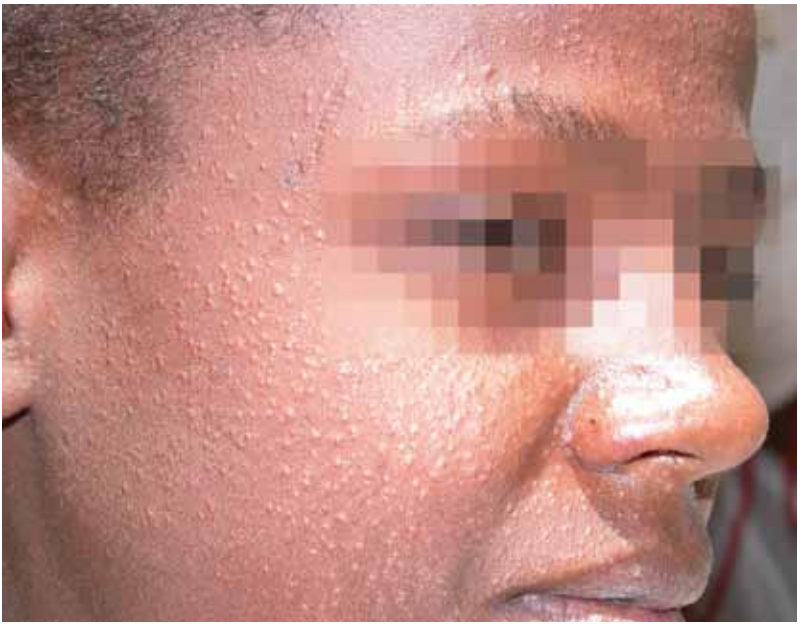
219 Various examples of papular rash, mainly around the mouth.

220

221

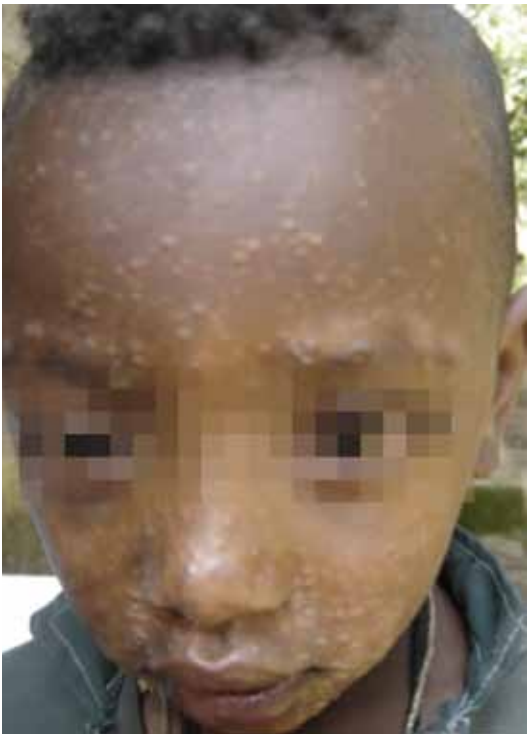
222 Papular rash, covering most of the face.

PKDL in Ethiopia. Various examples



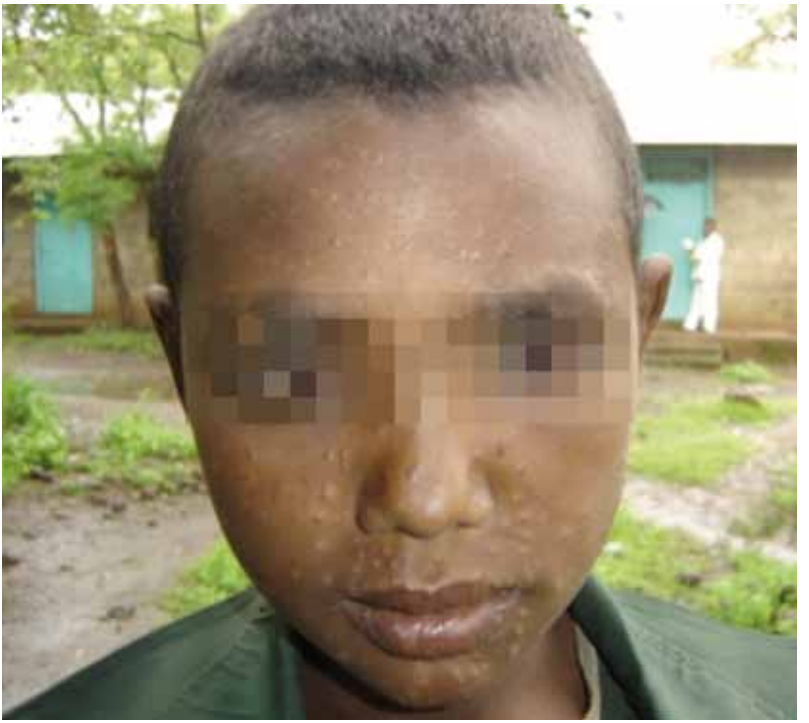
223

Papular rash.



224

PKDL in Ethiopia. Various examples

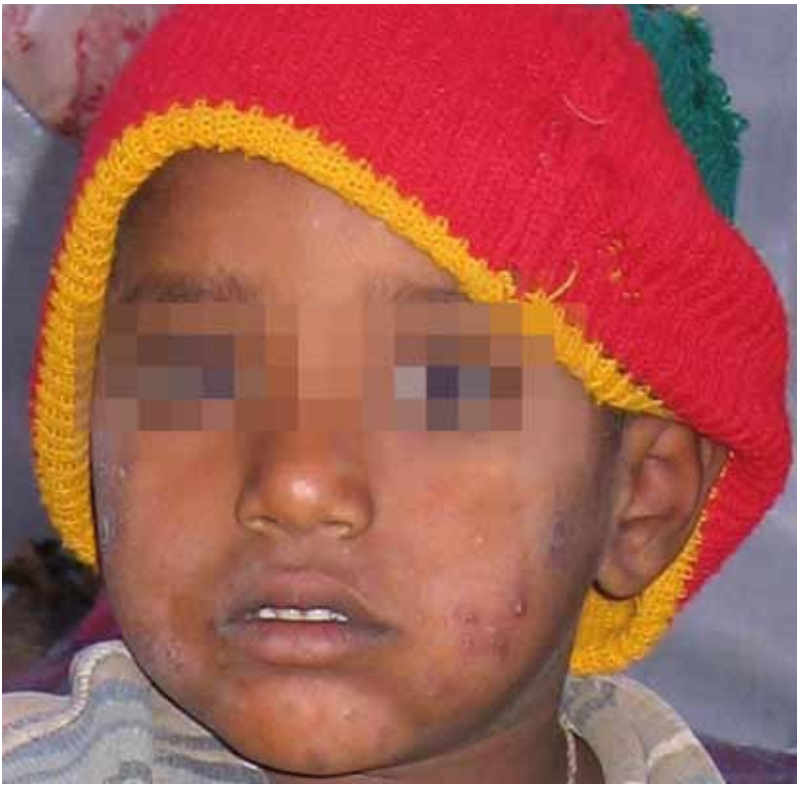


225

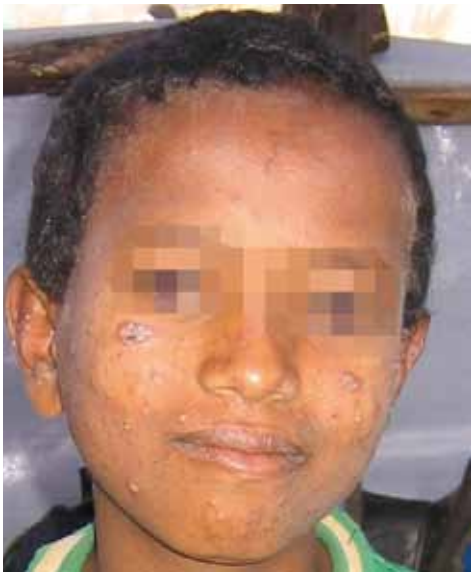
Papular rash.

226

PKDL in Ethiopia. Various examples

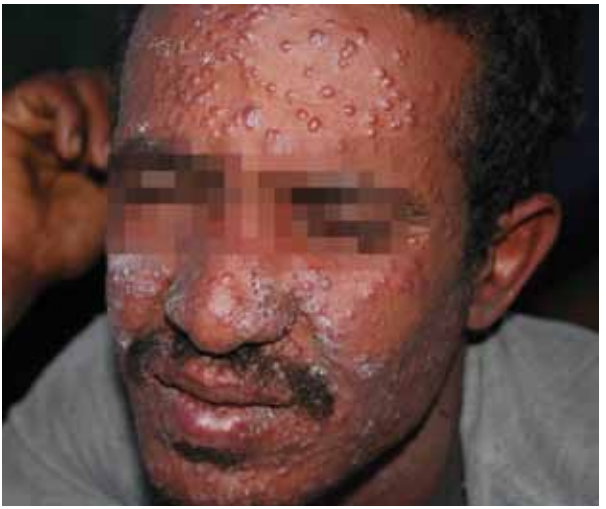


227 Papular rash with plaques (228).



228

PKDL in Ethiopia. Various examples

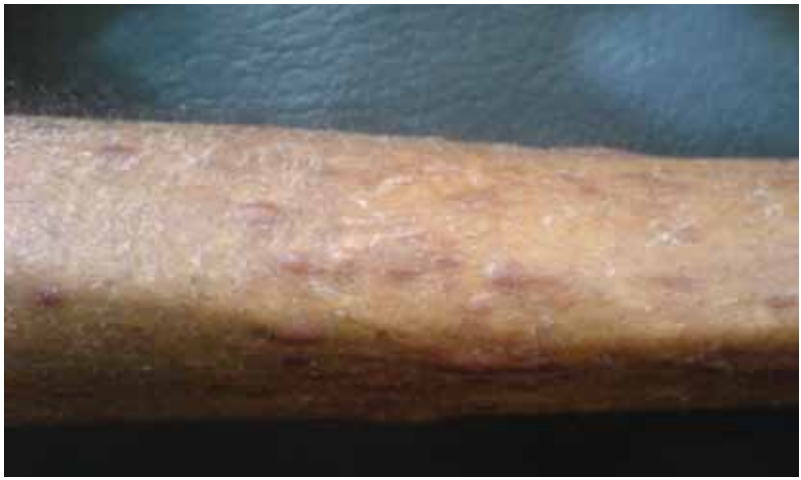


229

230

Papules confluent to form nodules (229); nodules confluent to form plaques (230, 231)

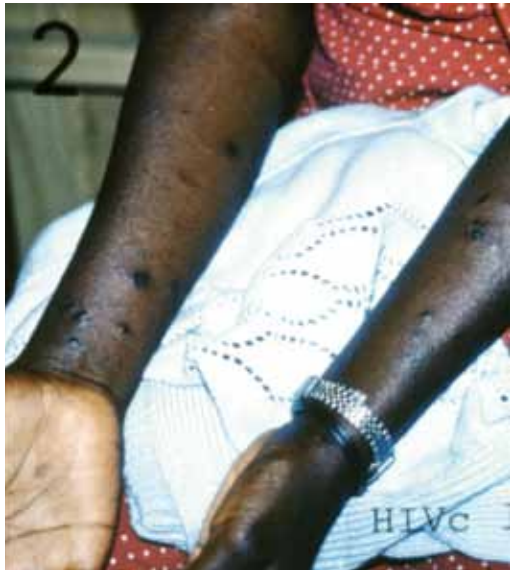
231

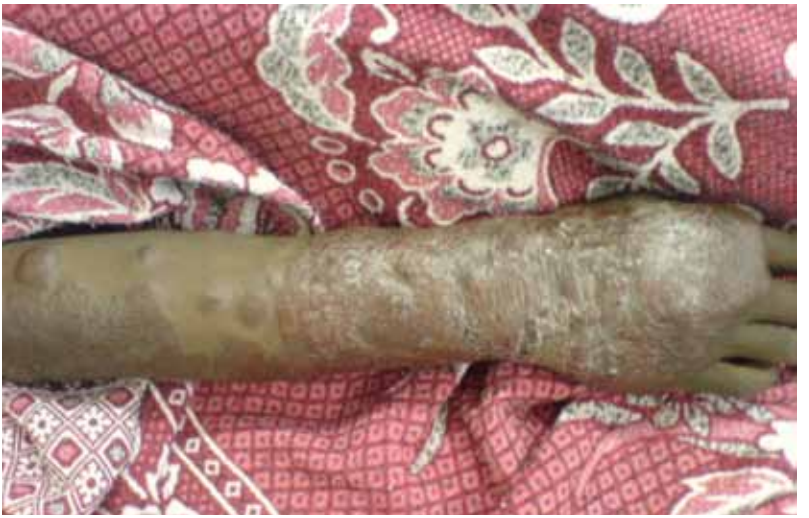


Subcutaneous nodular lesions in an HIV co-infected VL patient: para-kala-azar dermal leishmaniasis.
The same strain was isolated from the spleen and from the skin.
The lesions resemble those of Kaposi's sarcoma. **232**

233

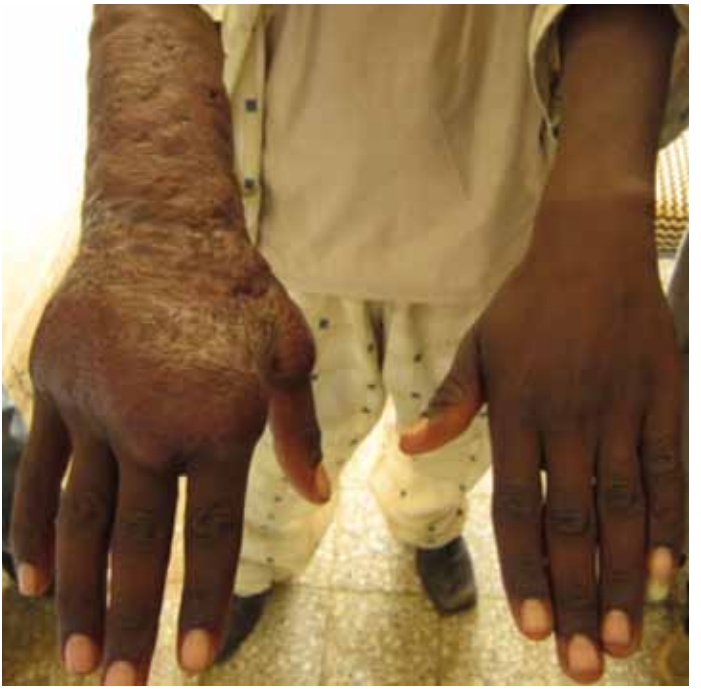
Kaposi's sarcoma in an HIV+ve patient. **234**





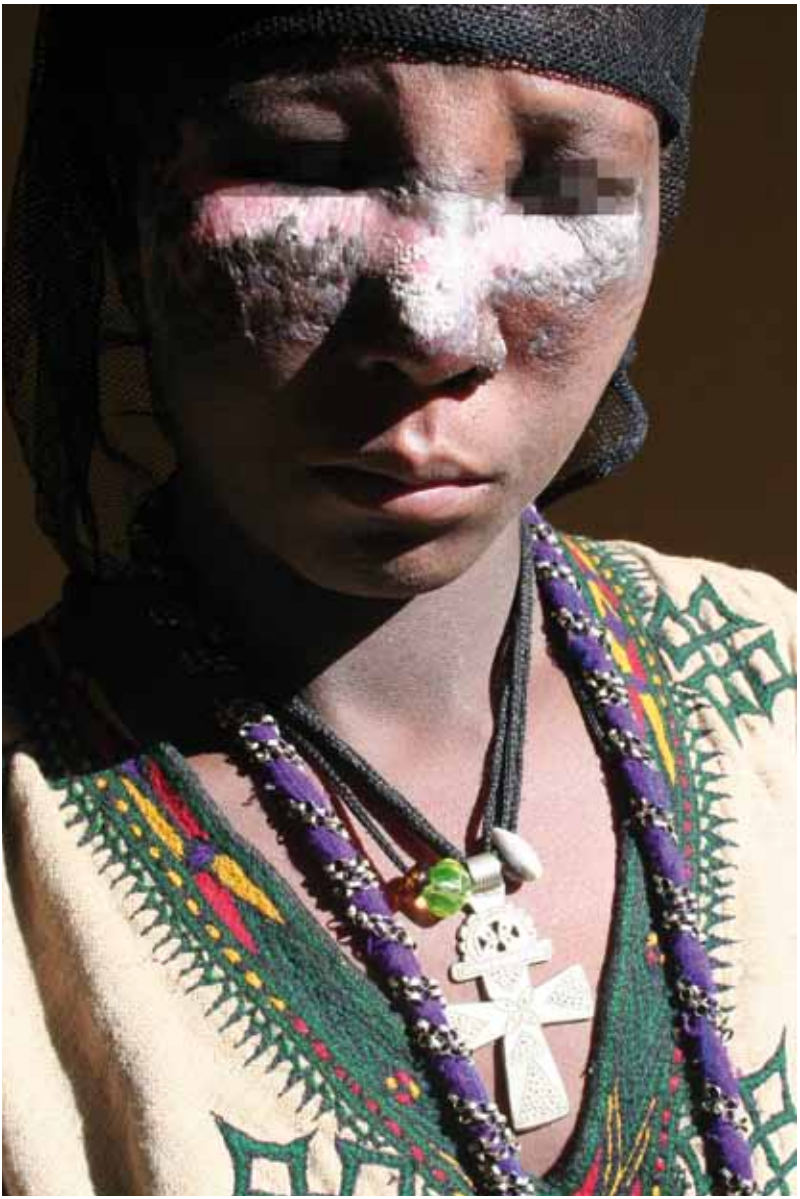
235

Diffuse Cutaneous Leishmaniasis; the lesions had been there for 3 years and were never treated.



236

Discoid Lupus Erythematosus. The patient was initially diagnosed as PKDL and treated with zinc ointment (not an accepted treatment for PKDL). **237**



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.

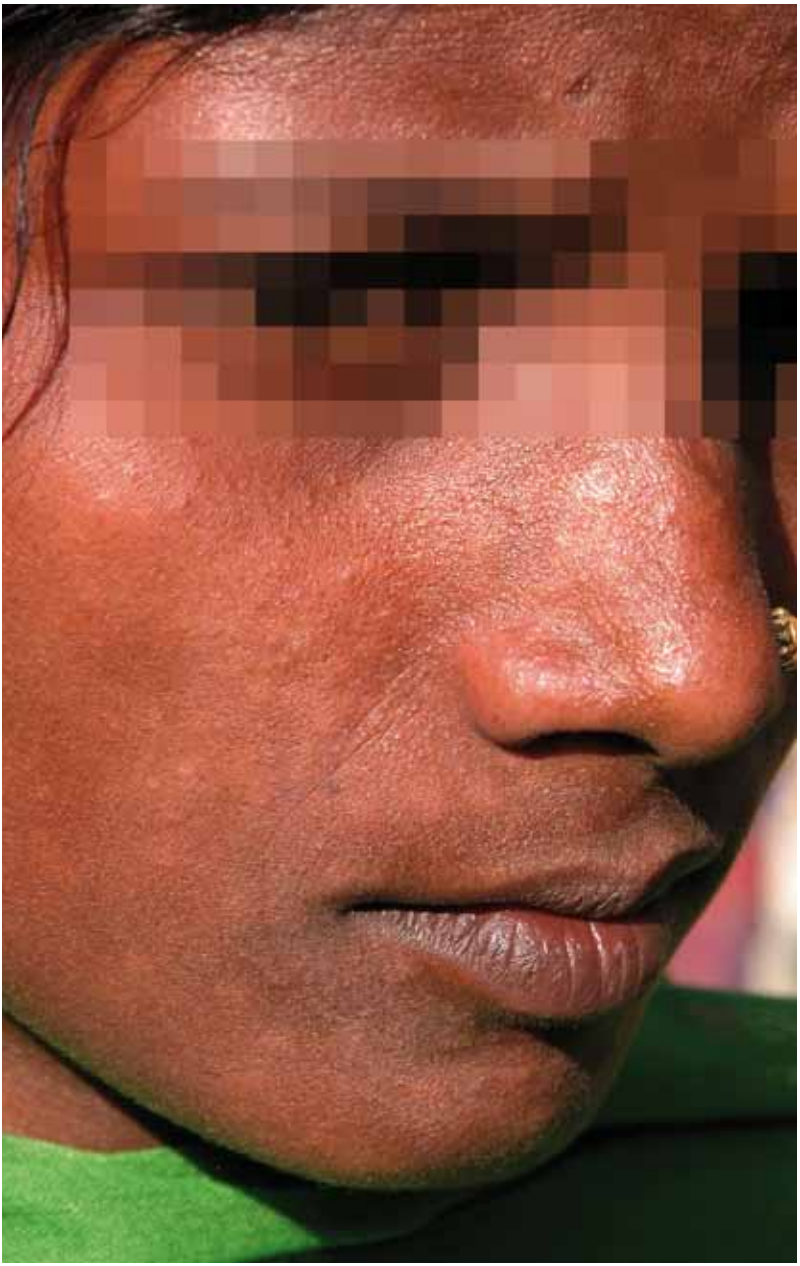
In the Indian subcontinent all cases are treated as they are considered chronic cases also given the long interval after VL; while the skin returns to normal, repigmentation may take time and cannot be taken as a parameter for cure.

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

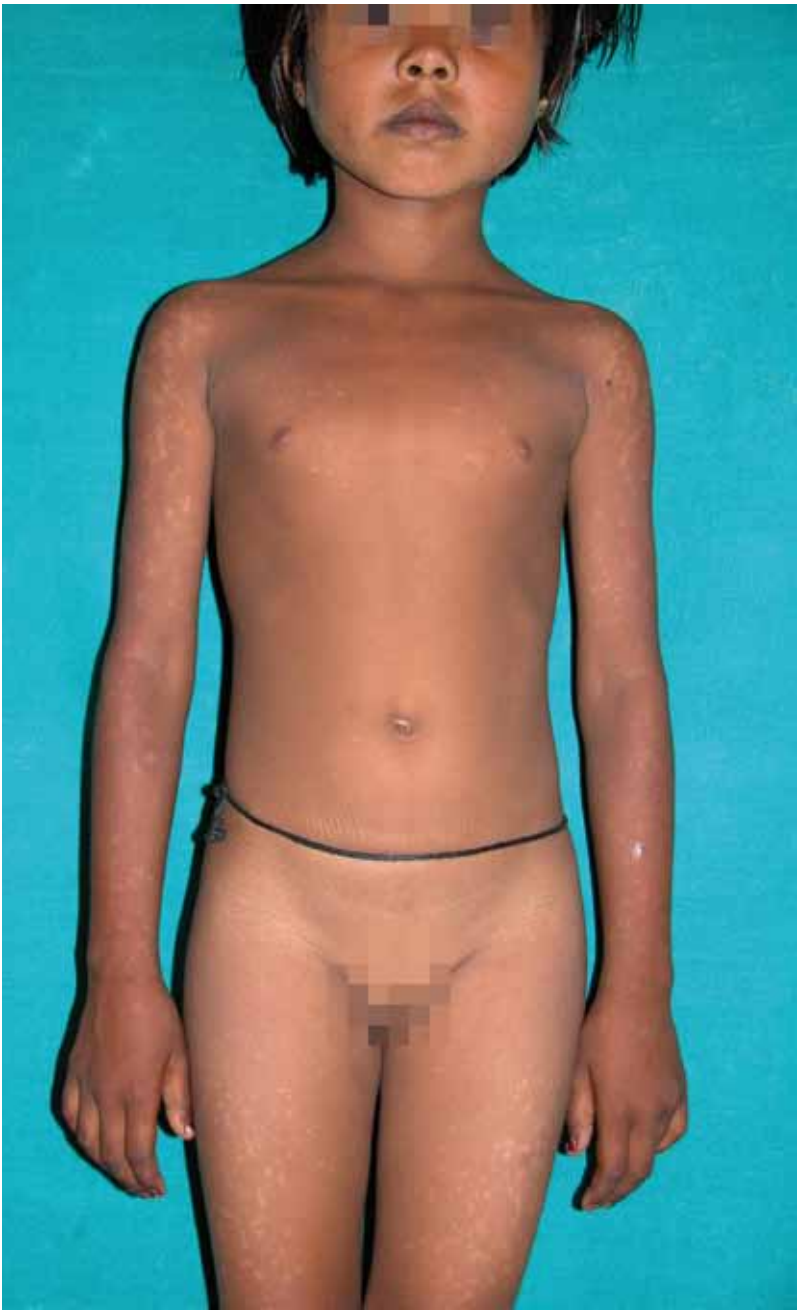
The main differential diagnosis for the macular form is vitiligo (Table 3) and for all forms leprosy (Table 4).

PKDL in India

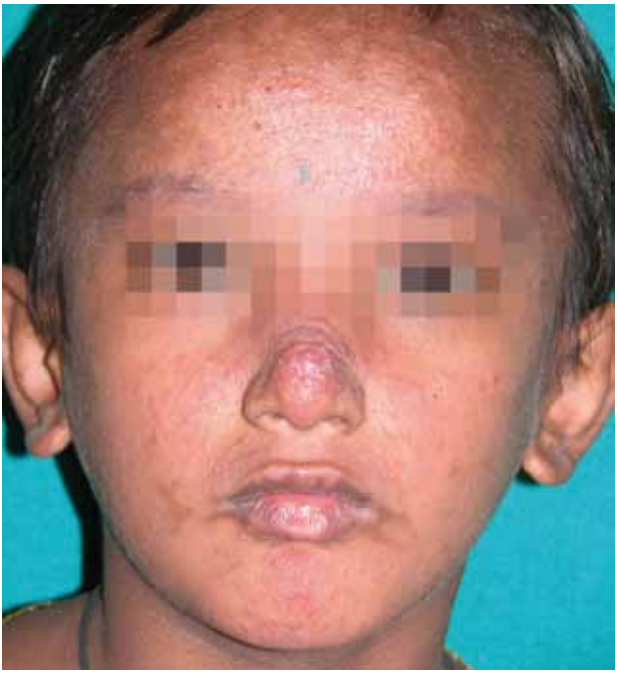
238 Macular lesions on the legs.



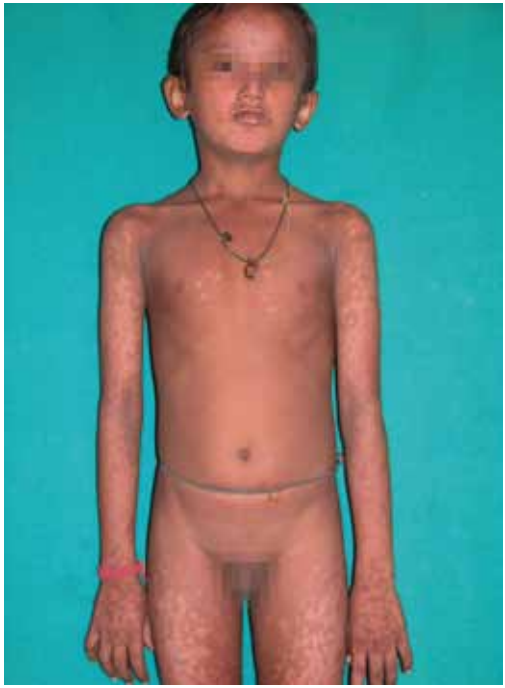
239 Macules and small papules.



240
Macules on arms and legs, but face and trunk relatively spared.

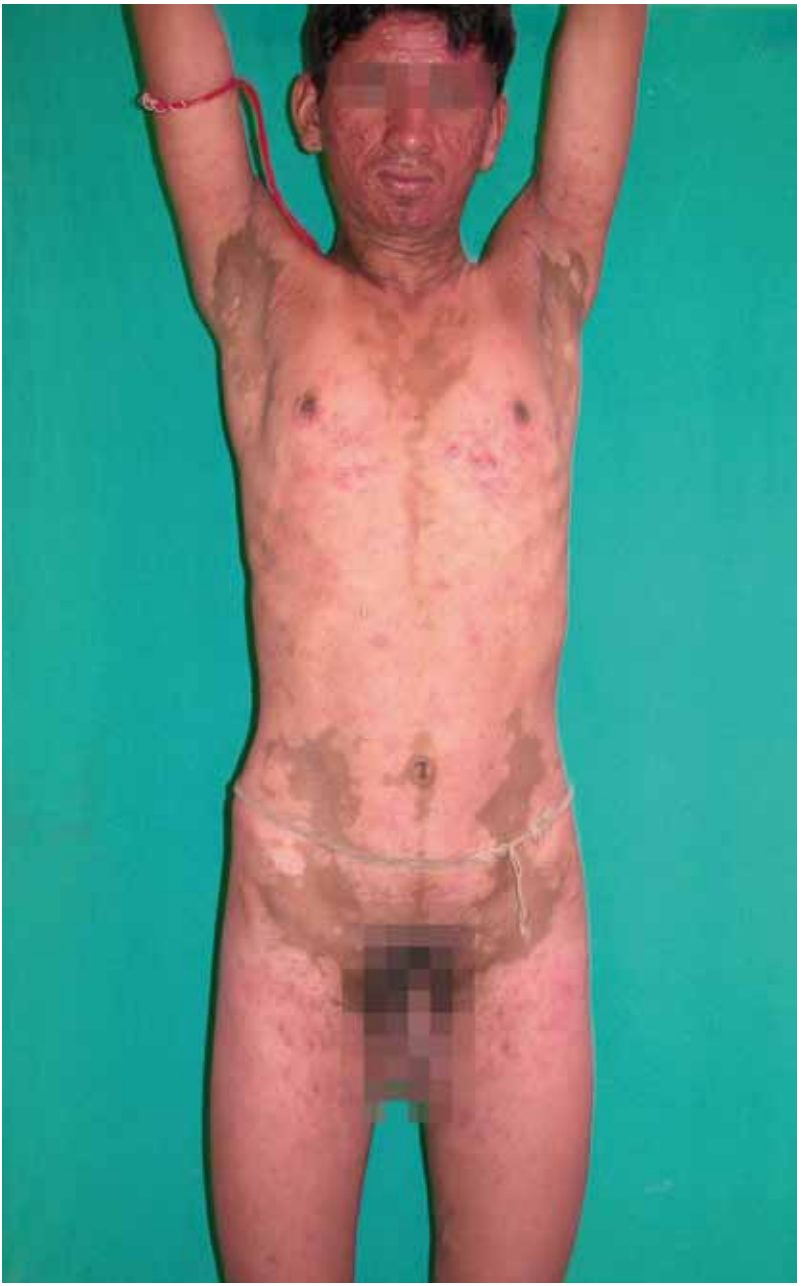


241



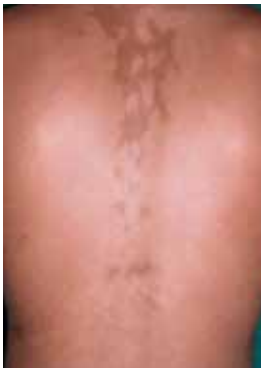
Macules on limbs and face with some small papules on the nose.

242

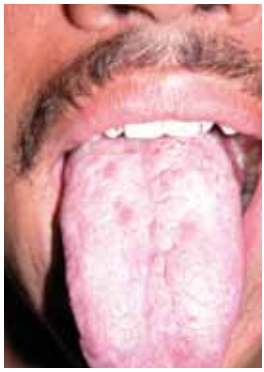


243
Nodules on the face; macules on the rest of the body with virtually total hypopigmentation sparing axillae and inguinal areas.

244



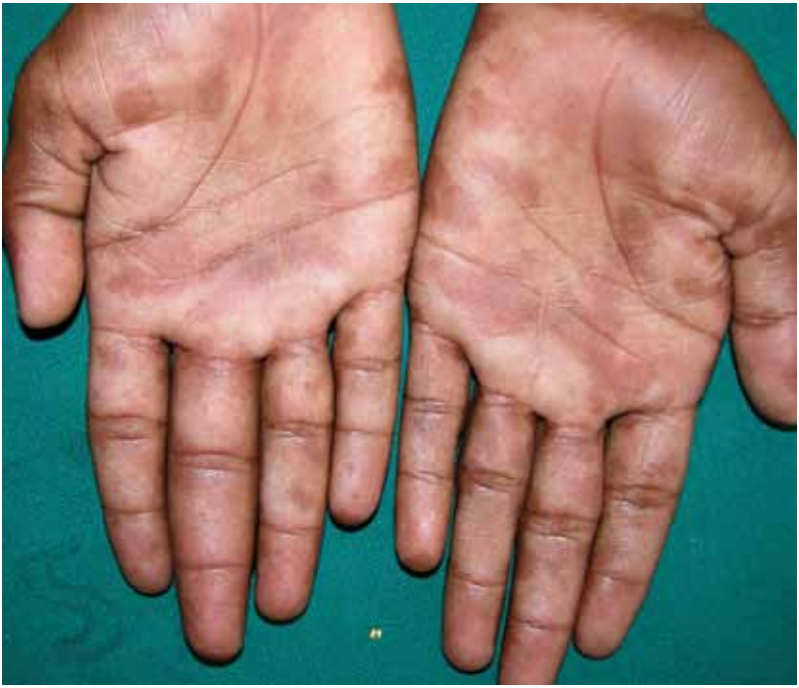
245



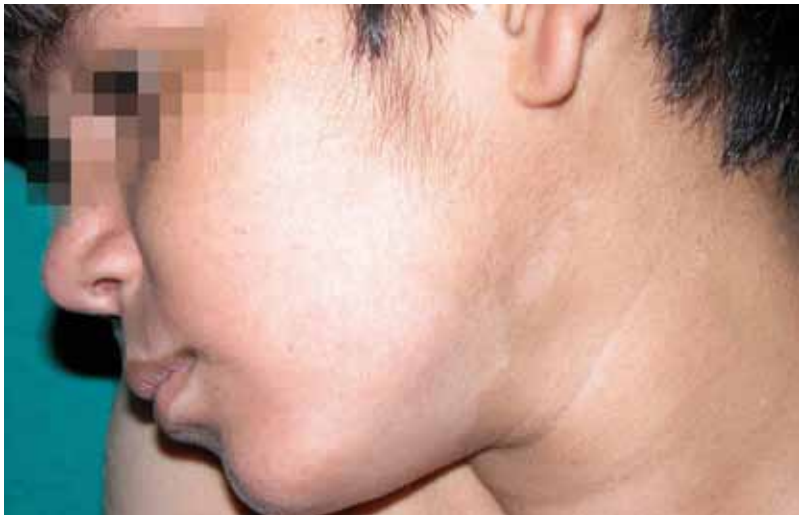
246

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.

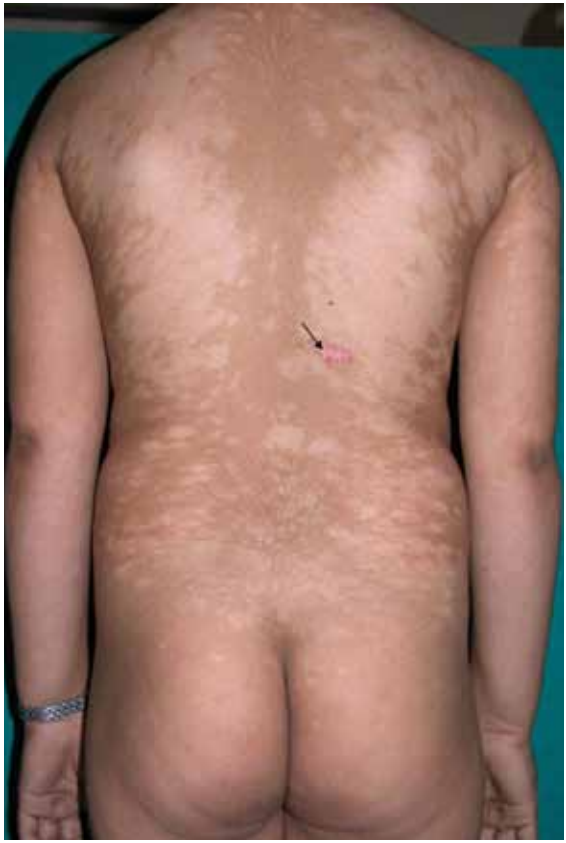
247



248
Hypopigmented patches in the face, typically around the mouth.



249

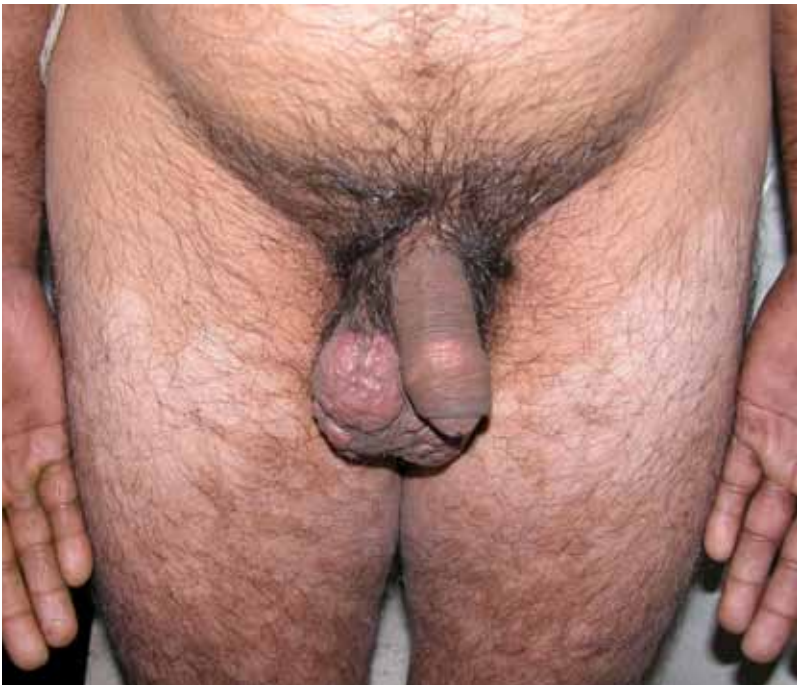


Hypopigmented lesions involving the whole face sparing the neck; this is only appreciated in a lateral view. The lesions on the back spare the mid back and elbows (the arrow indicates the site of a biopsy).

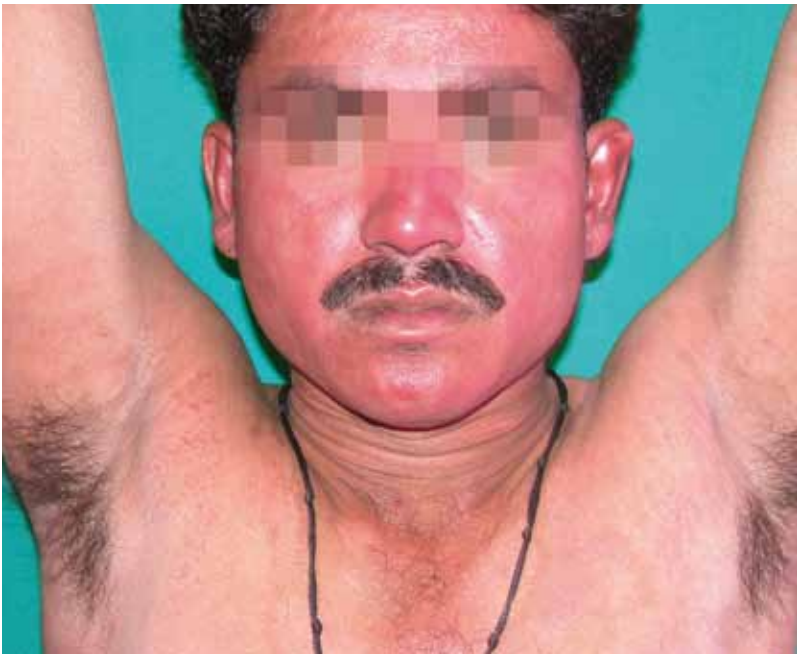
250

251 Macular lesions in inguinal area, penis shaft and glans. **252**

Hypopigmentation of the thighs with nodular plaques on the scrotum, penis and papules on tip of glans penis. **253**

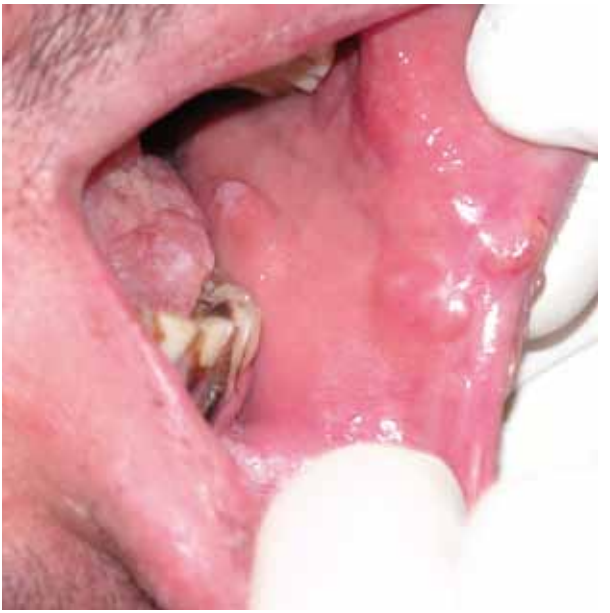


254 PKDL: papules and nodules on inner thighs, scrotum and penis.

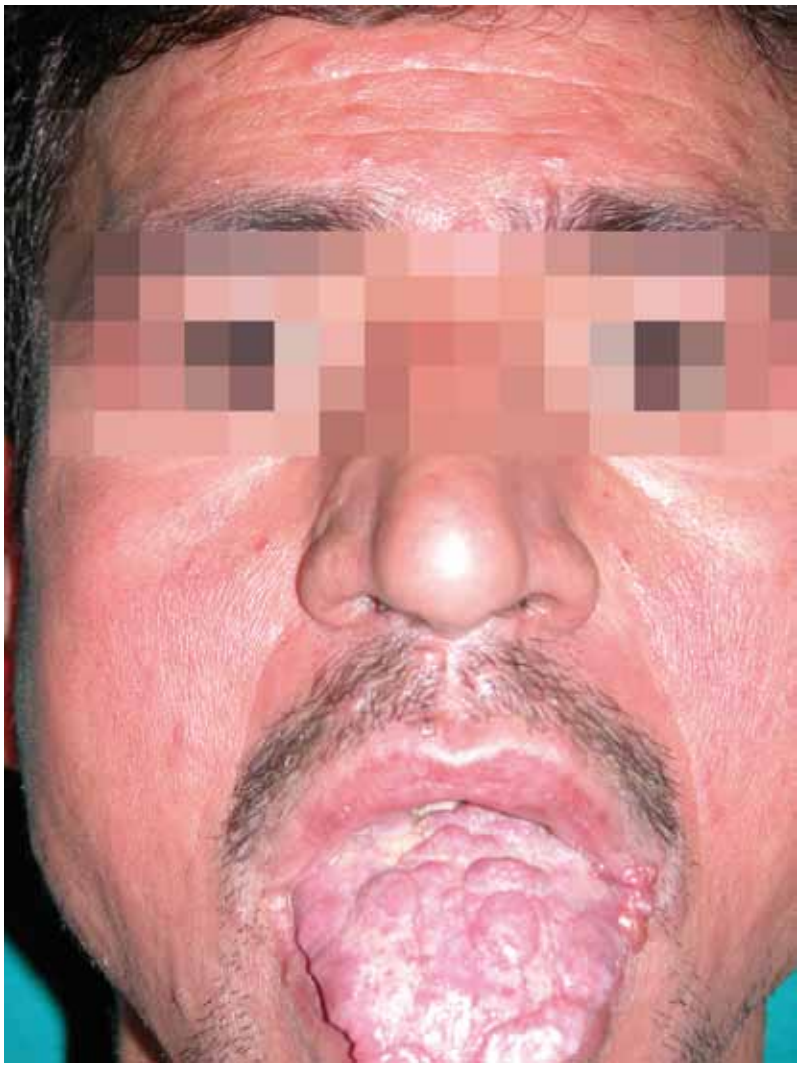


255 Erythrodermic PKDL. This is uncommon; there is facial erythema and sparing of the axillae. The rest of the body is also faintly erythematous.

256

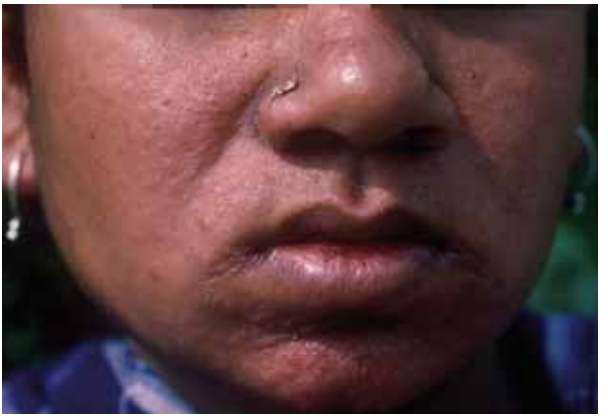


Papules and nodules on the tongue and buccal mucosa.

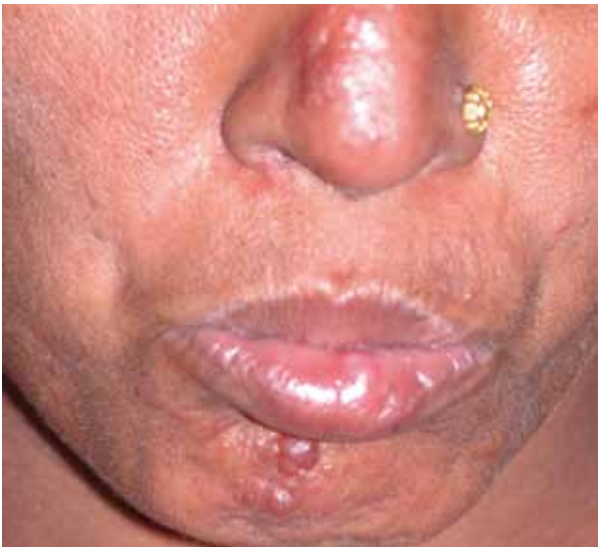


257 Papules and nodules on the tongue.

258

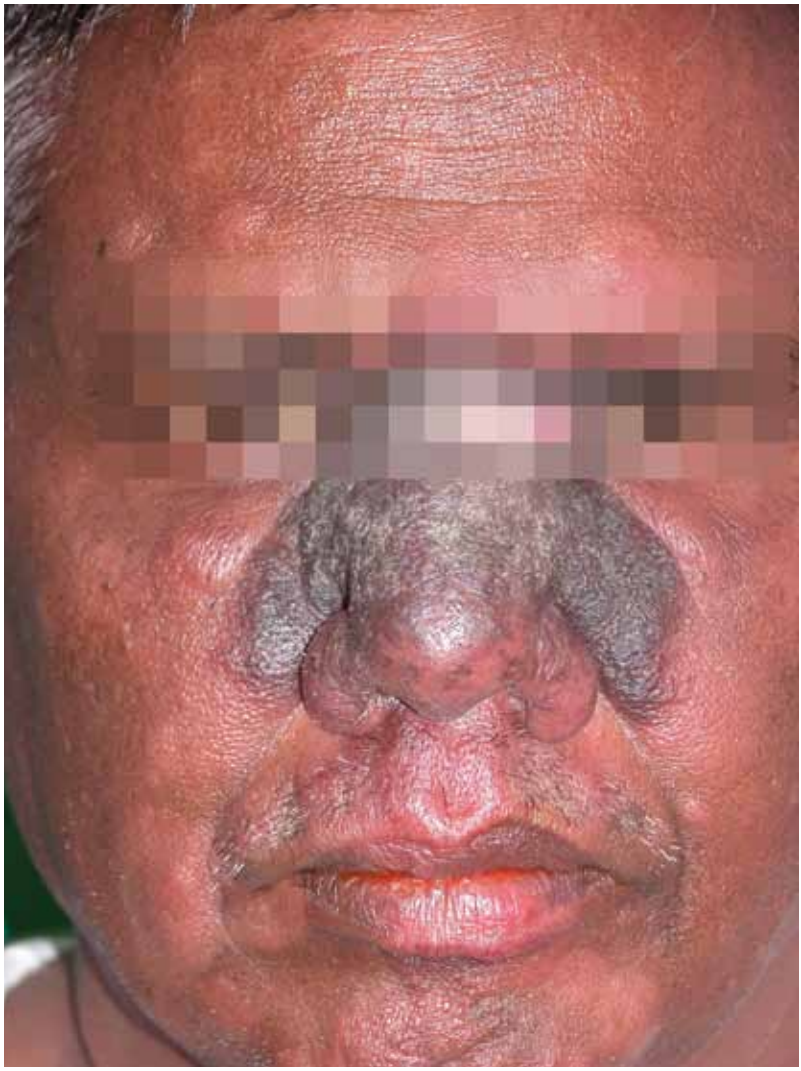


259

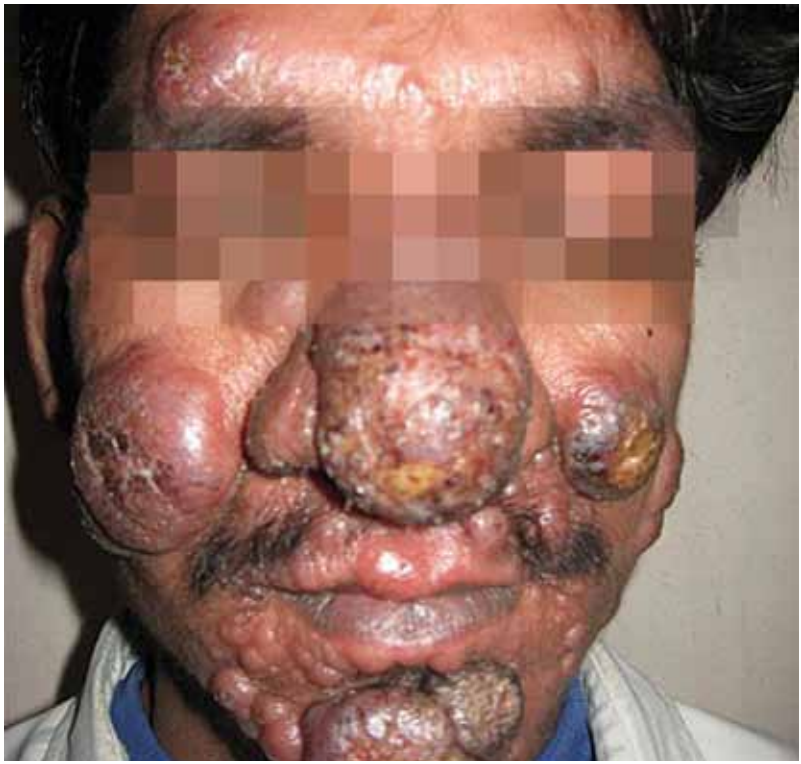


Discrete papules on chin (258, 259); crops of nodules on chin and nose (260)

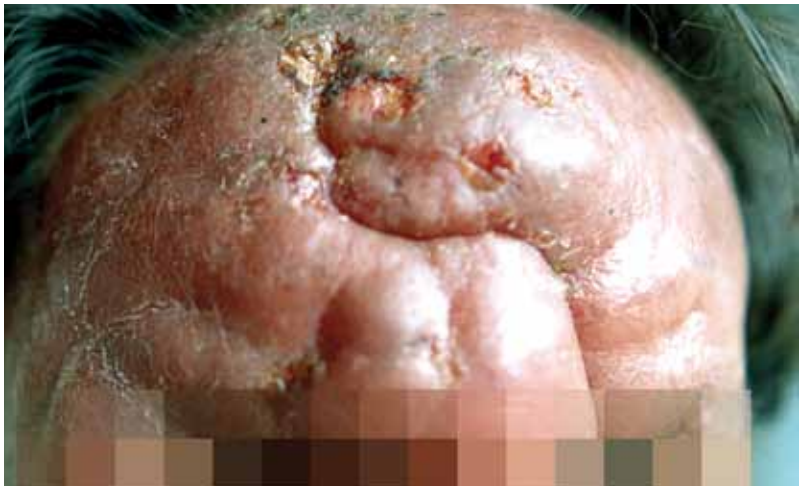
260



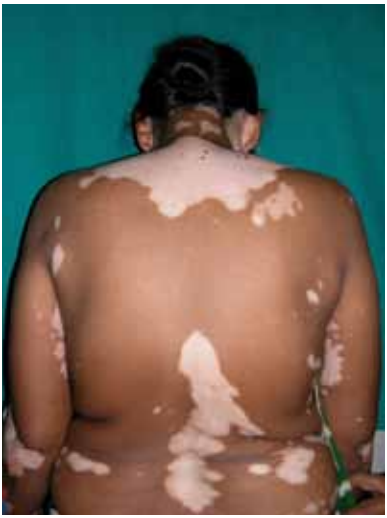
261 Verrucous or hypertrophic form.

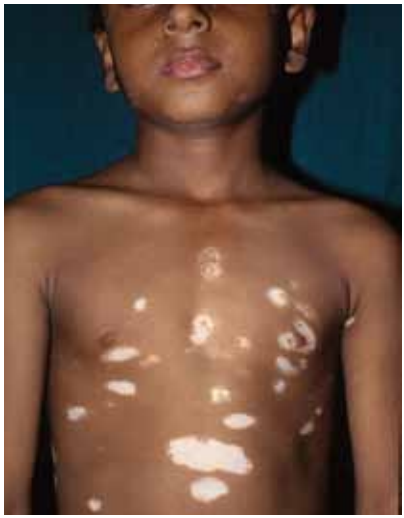


262 Tumor-like nodules on face (note sparing of eyebrows).



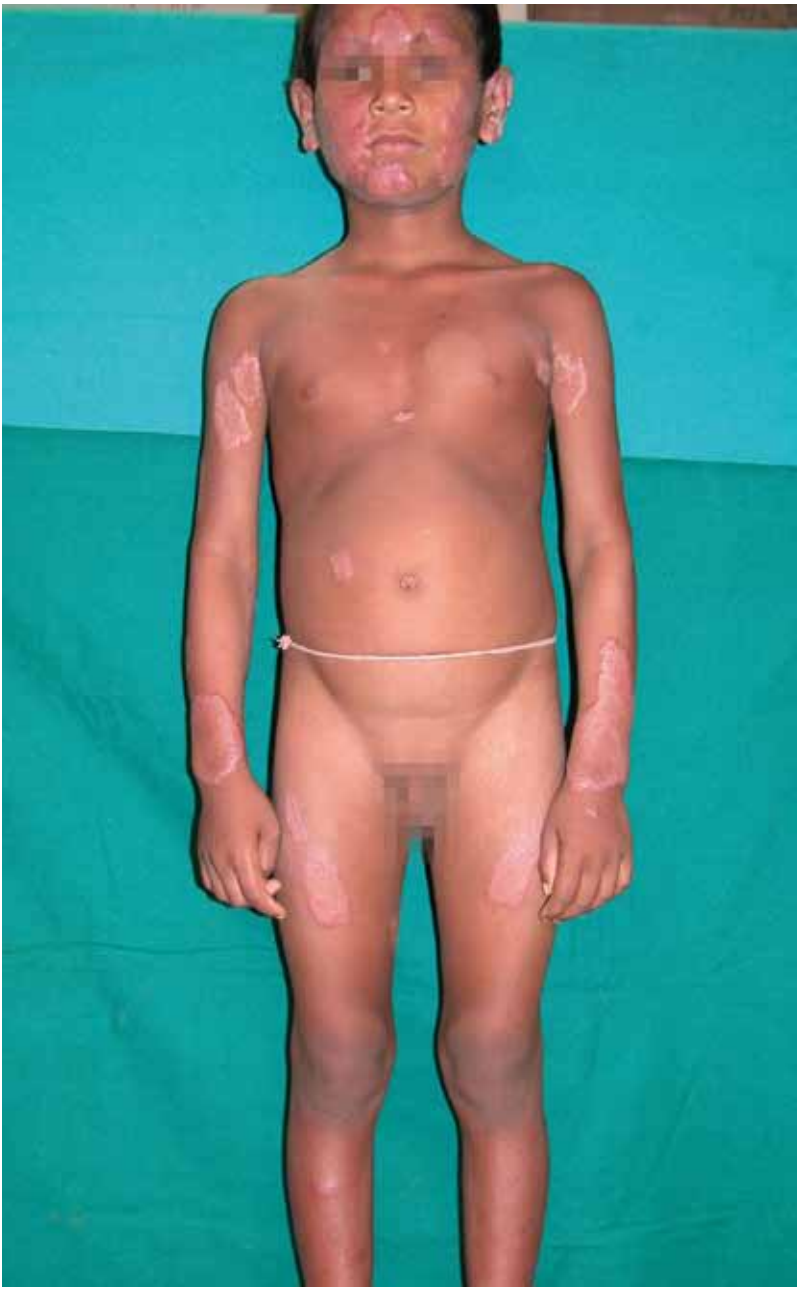
263 Tumor-like; there is a spontaneous furrow.





264 **Vitiligo.** Note the total loss of pigment (depigmentation) and the involvement of the central part of the trunk. **265**

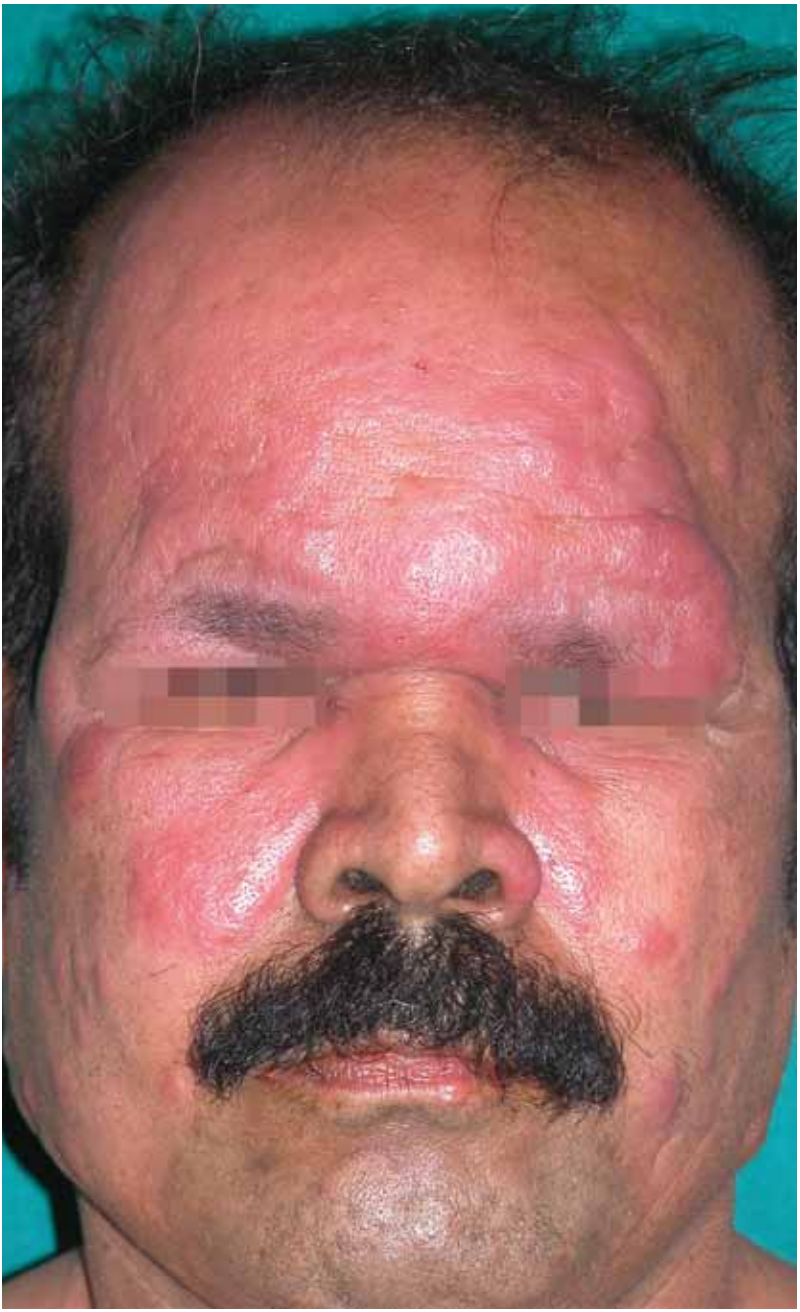
266 **Lepromatous leprosy.** Lesions are prominent on the forehead and the cheeks while the central part of the face is spared. The patient on the right has madarosis. All these are not features of PKDL. **267**



268
Borderline leprosy.
Red, raised plaques on the face and limbs, note sparing areas around the nose and the mouth.

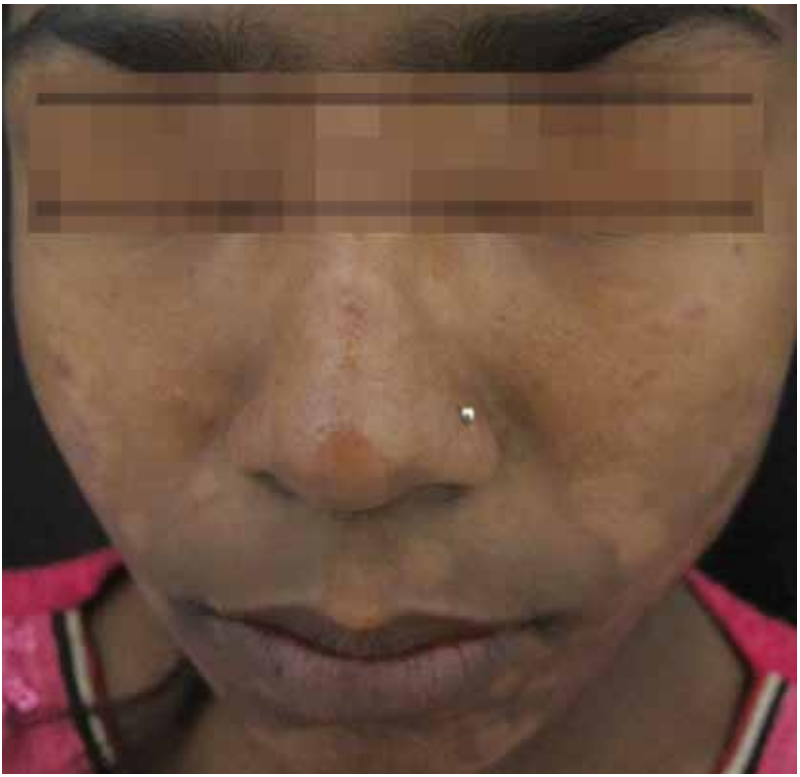
269

Borderline tuberculoid leprosy; the nose and chin are free; note the madarosis.



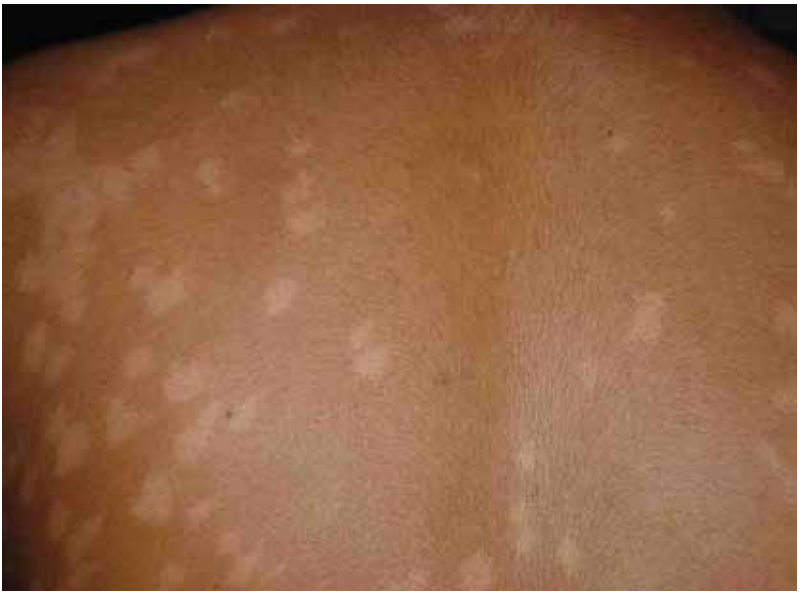
PKDL in Bangladesh

270
Macular lesions.



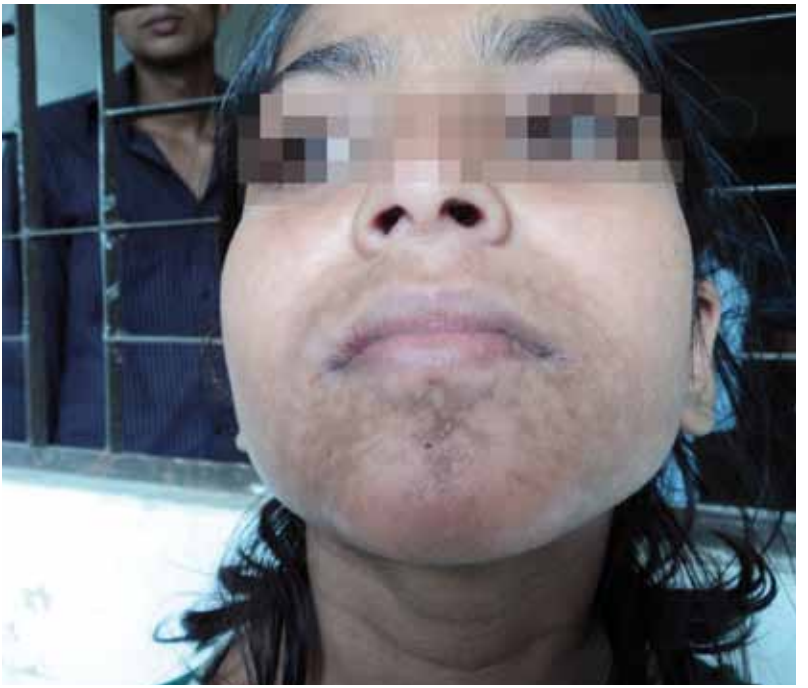
271 Macular lesions.

272

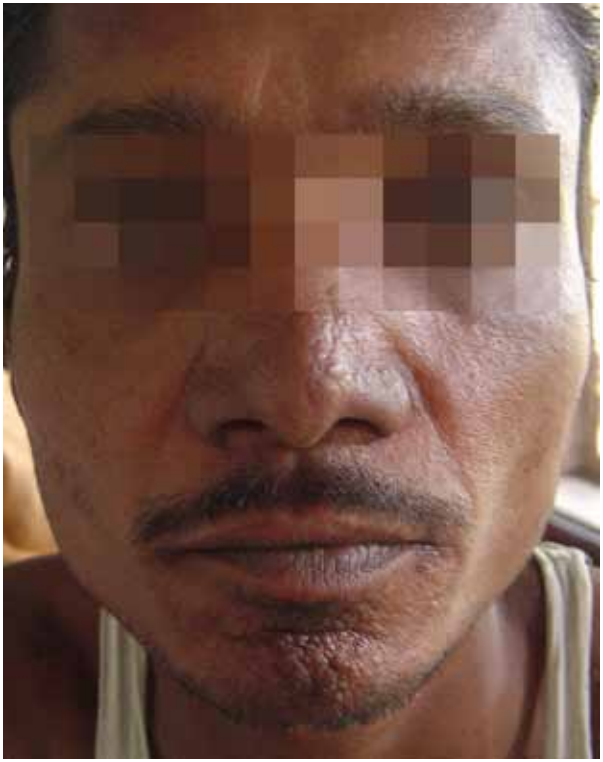


273 PKDL: macular lesions, some are confluent.

274 Large areas of confluent macules leaving some islands of normal skin

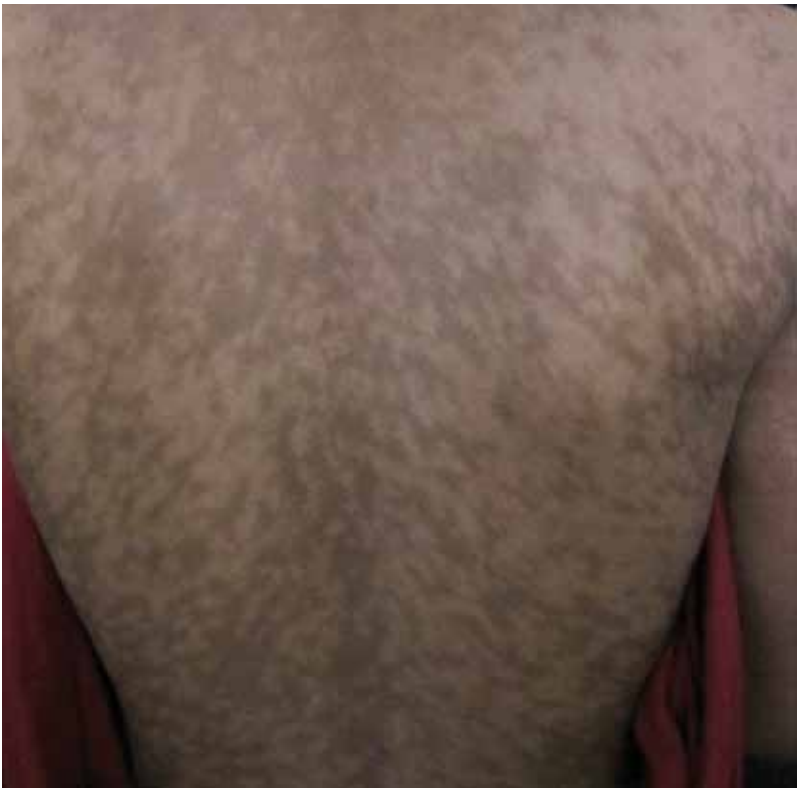


275



276

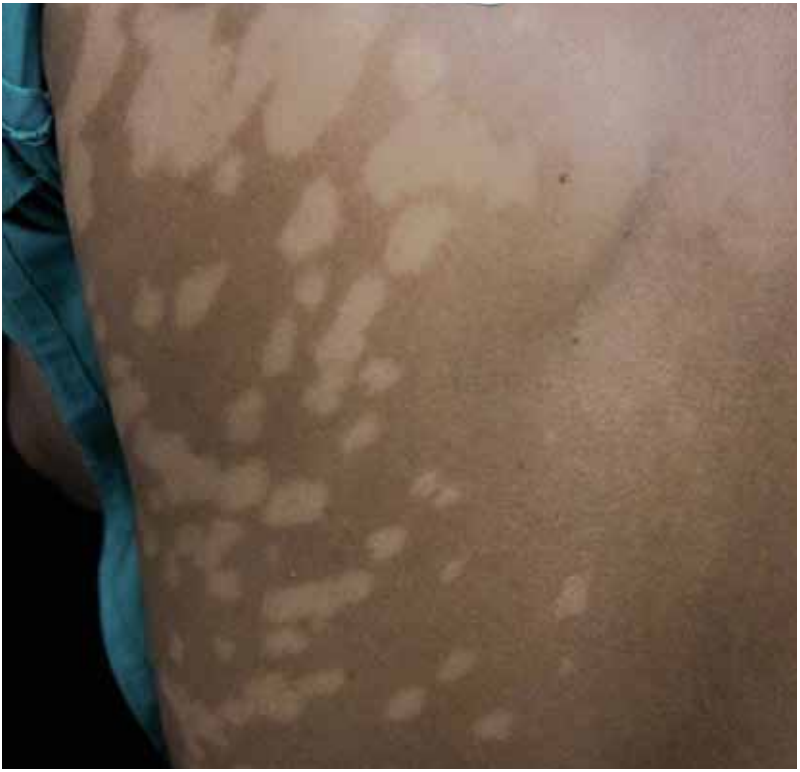
Combined confluent macular lesions on the arms (275) and papulonodular lesions on the face (276).



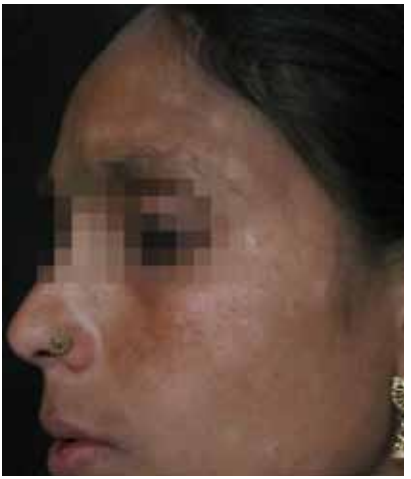
277

Extensive confluent macules on the back and face leaving little normal skin.

278



279 Extensive confluent macules on the back.

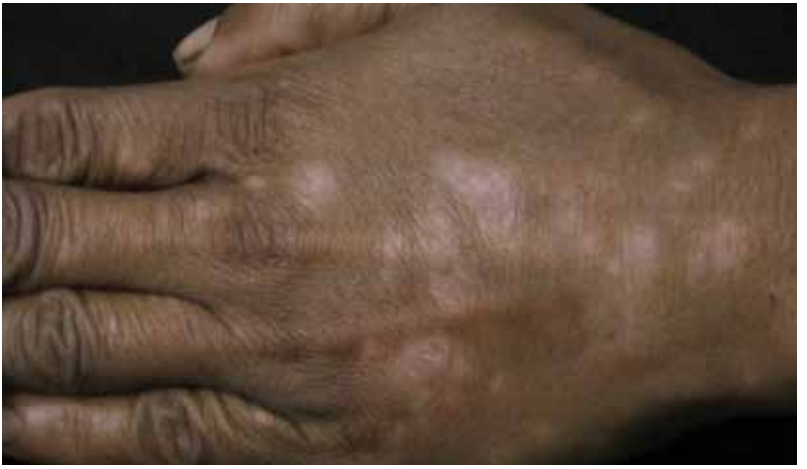


280

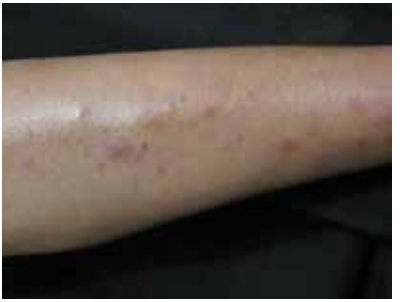
281

Mostly macular rash with papular rash on the shin.

282



283



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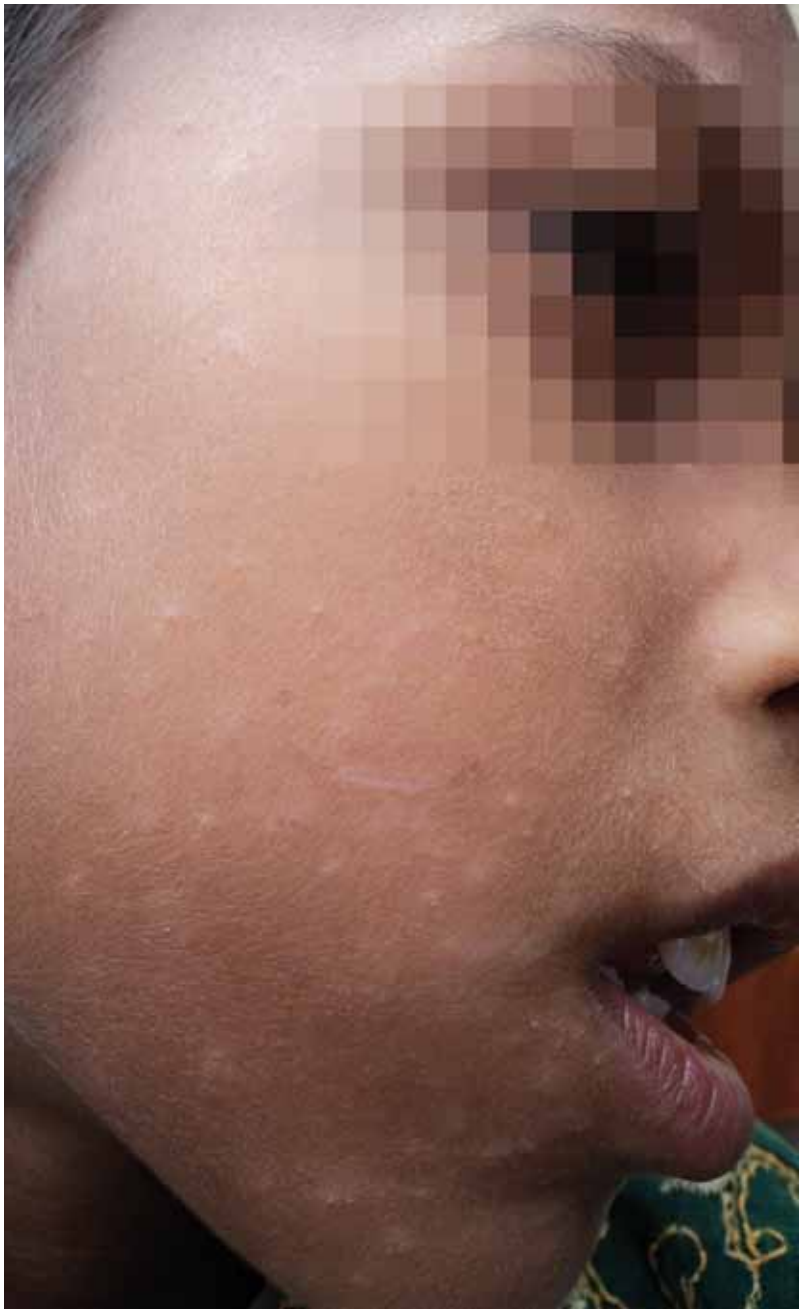
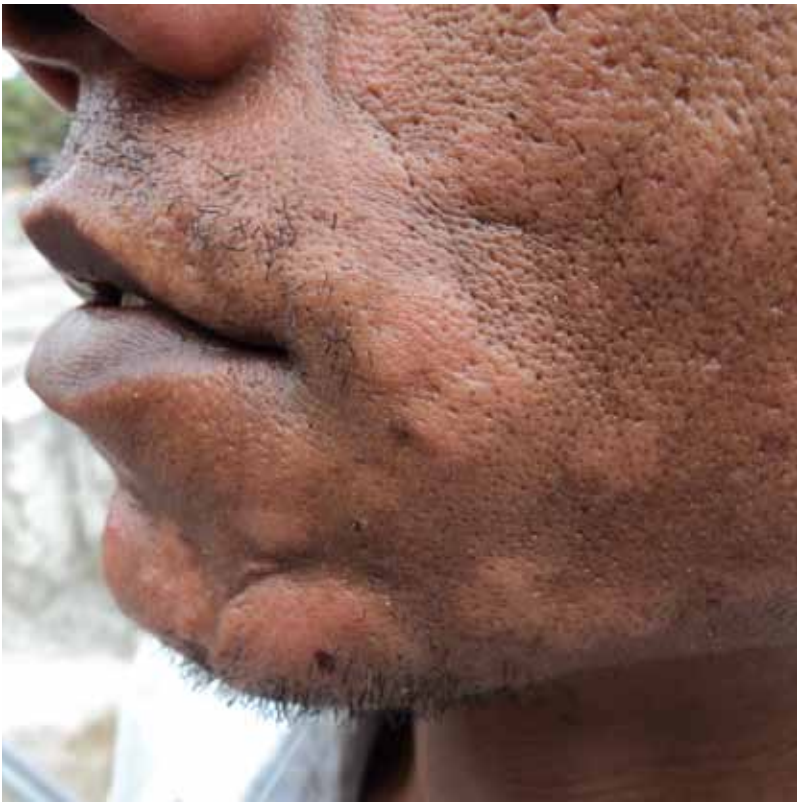


285
Maculopapular rash.



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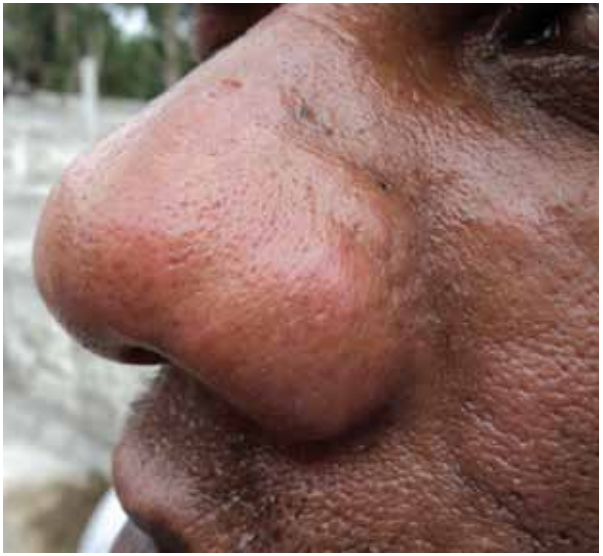


Nodular rash with infiltration of the skin in particular on the nose.

287

288

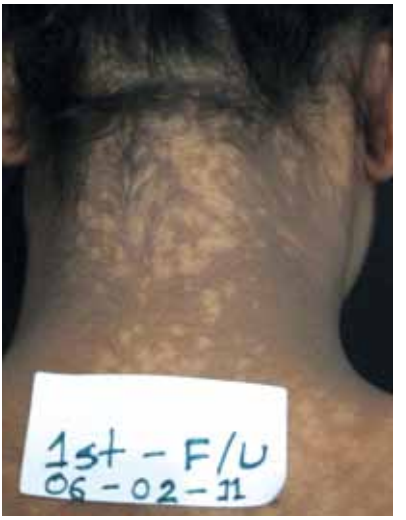
Macular lesions on chest and nodular lesions on arms and fingers.

289

Figures 290-318: In this section, a number of figures are shown of patients who were treated with liposomal amphotericine 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



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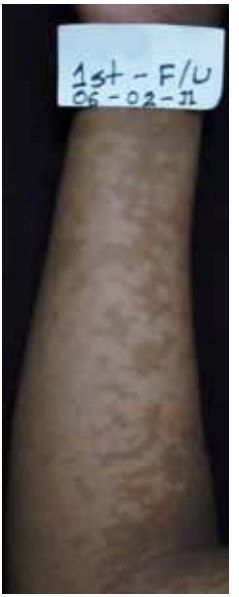
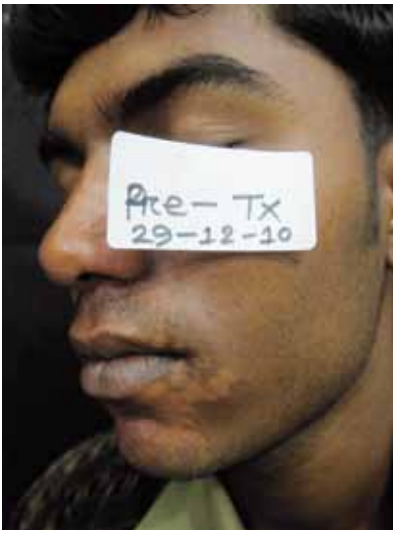


Macular rash before and after 6 weeks; no response can be noted.

293

Response to treatment



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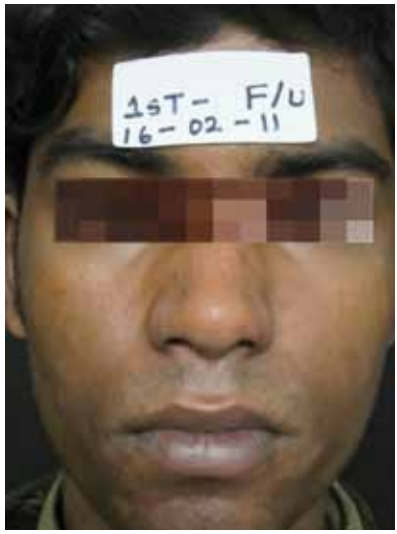
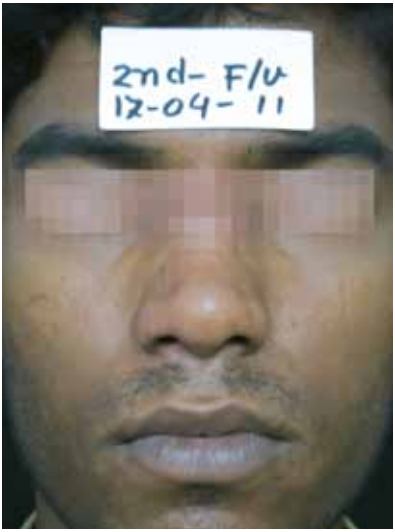


295

Macular rash before and after 6 weeks, 4 months and 7 months; note the slow recovery (e.g. around the mouth).

296



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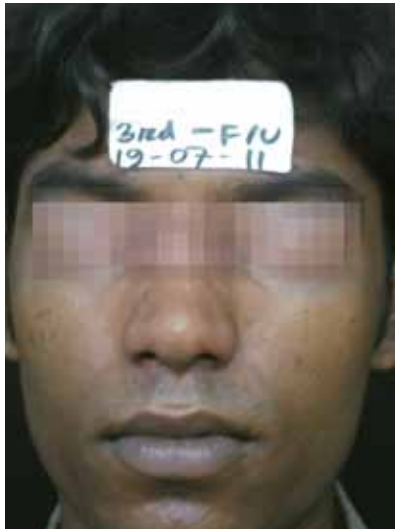

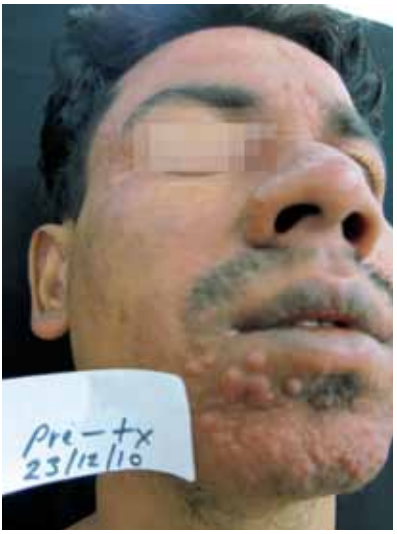

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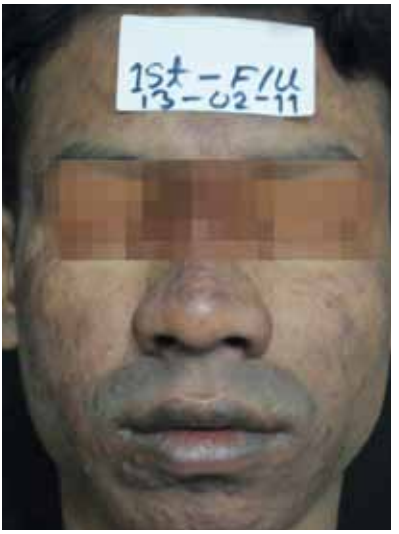
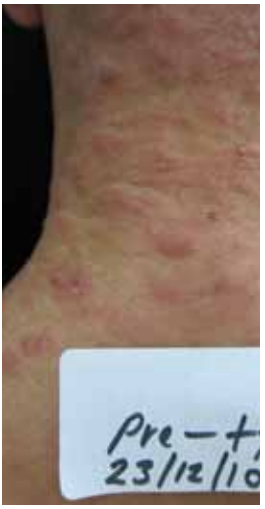


299

Residual lesions can be seen 10-11 months after treatment (301).

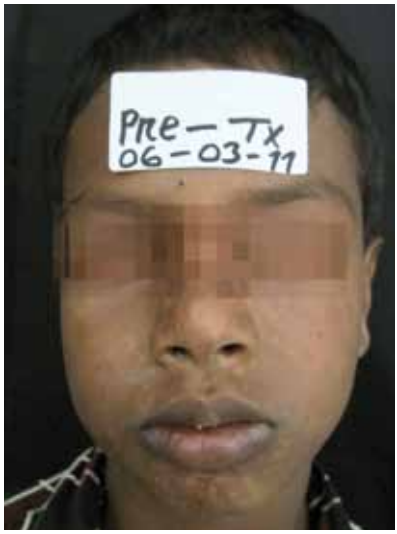


300

301

302

Response to treatment



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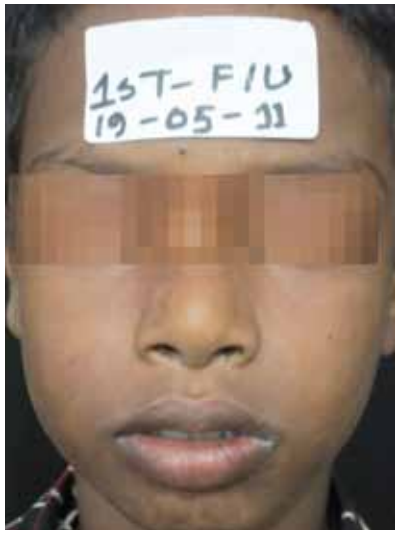

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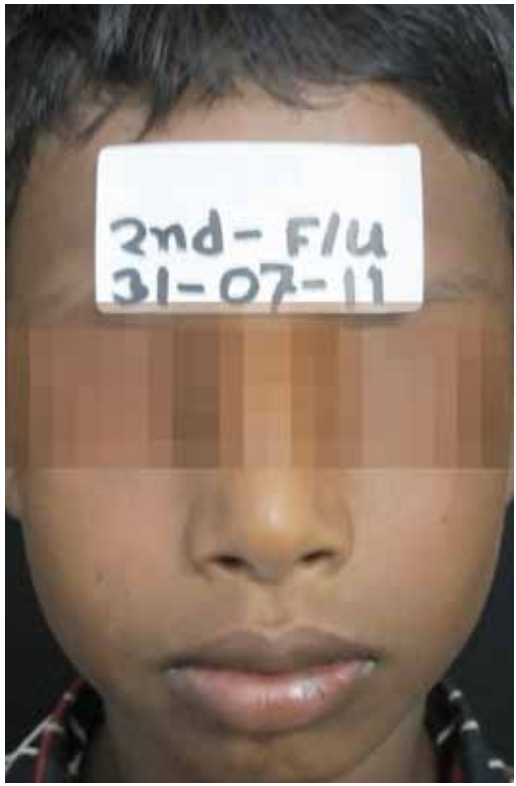
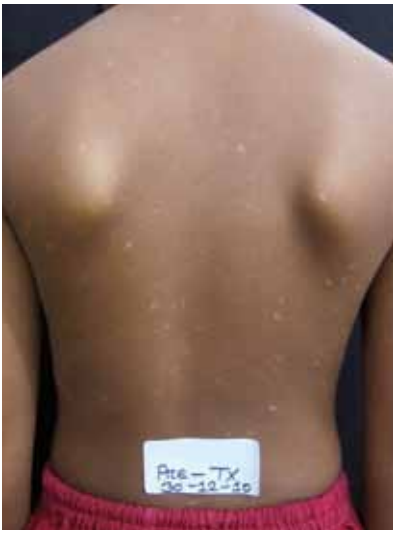


Almost complete disappearance of lesions 4 months after treatment

305



306

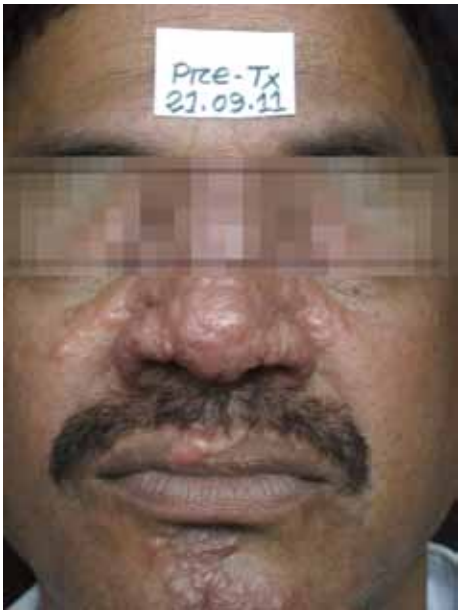
307

Good response 7 months after treatment.

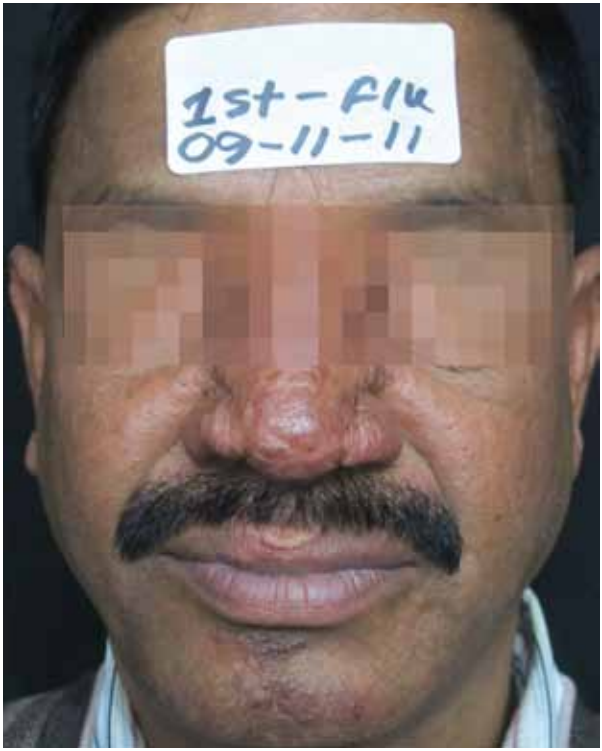
308

Response to treatment

309



Good response of nodular lesions 6 weeks after start of treatment.



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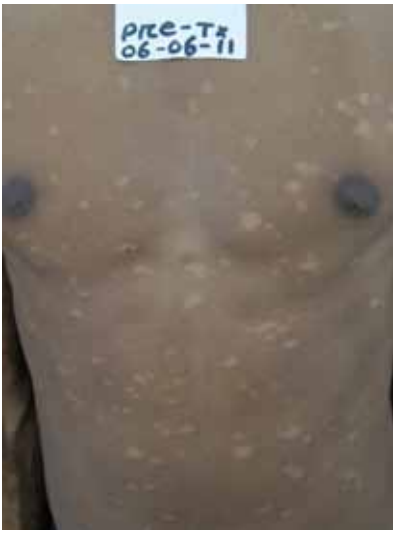

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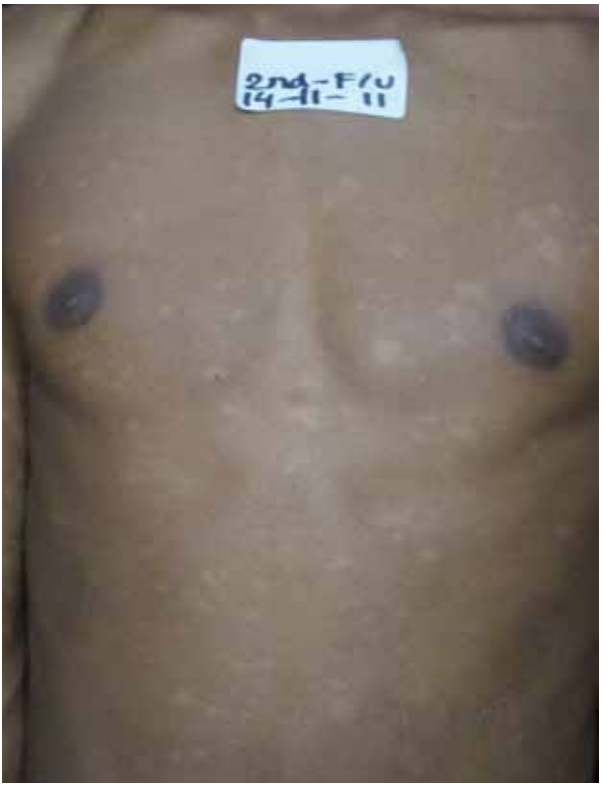
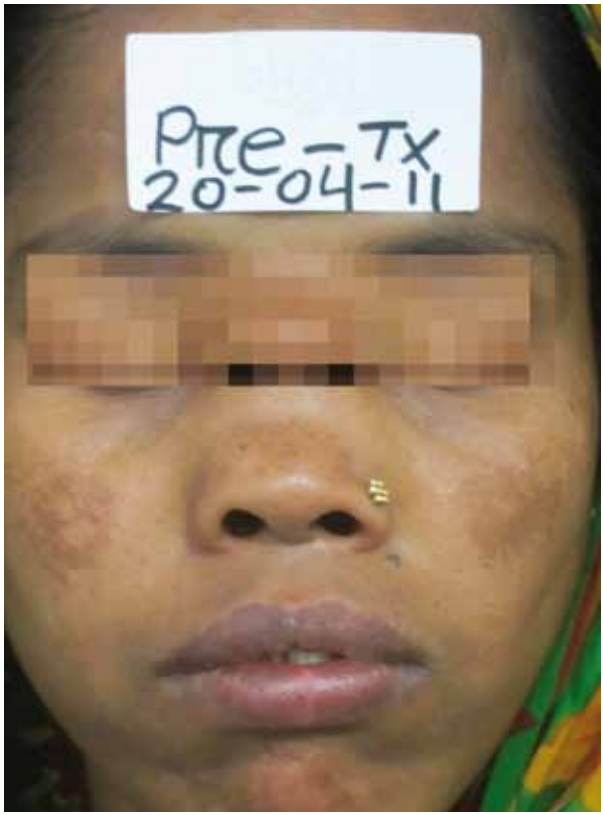
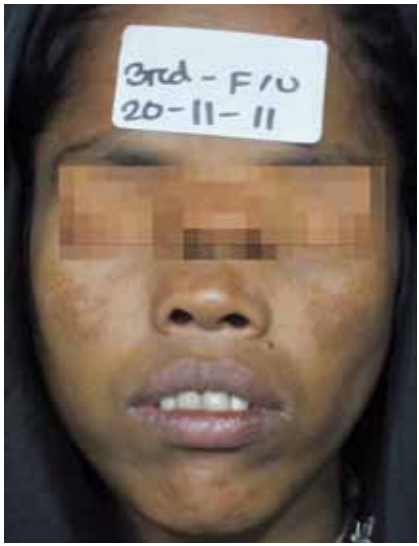


Clear residual lesions 5 months after start of treatment.

313

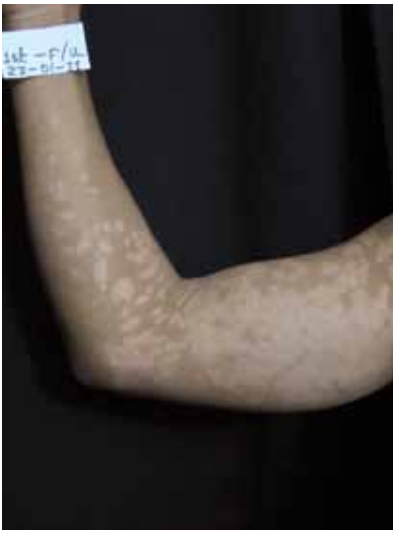
314





Poor response to treatment; lesions are virtually unchanged.

315



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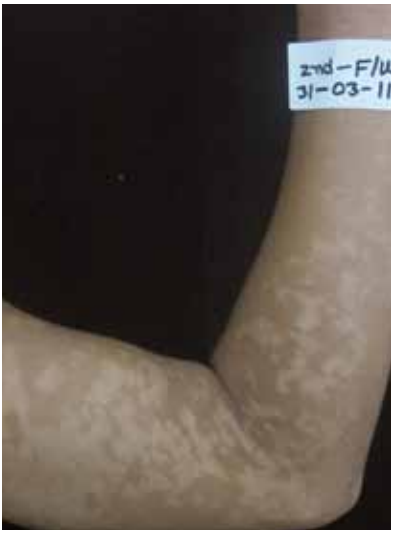


317

318

Gradual disappearance of the macular lesions 9 months after start of treatment

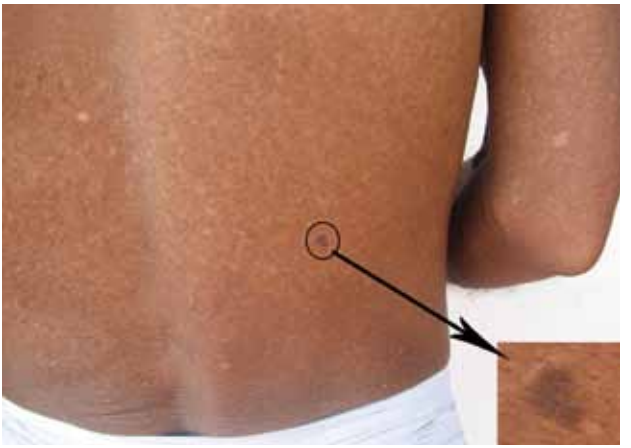
Differential diagnosis

Table 6: Differential diagnosis of PKDL in Asia (most important)

Macular rash	Papulonodular
Leprosy Chronic arsenic poisoning Pityriasis versicolor Vitiligo Pityriasis alba	Leprosy Neurofibromatosis Secondary syphilis

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. **319**

320



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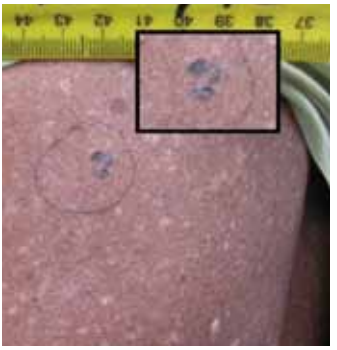

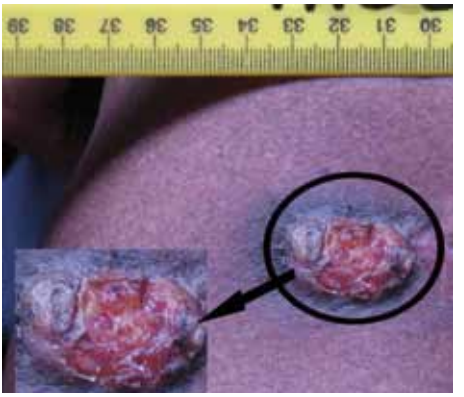

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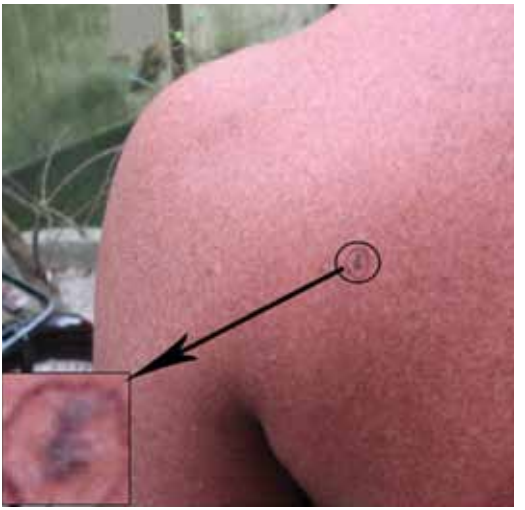
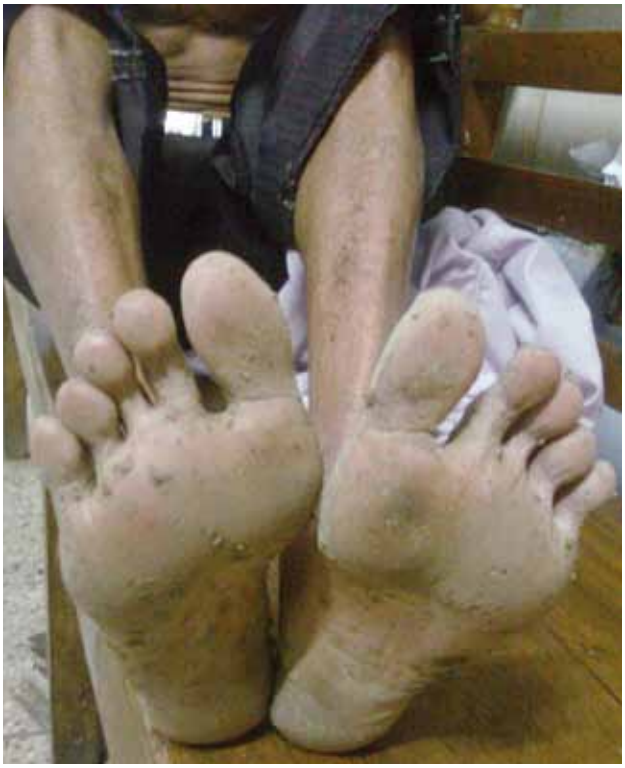


323

Complications of **chronic arsenicosis**: solar elastosis (320), squamous cell carcinoma (321,322) and lentigo simplex (323). These complications are not seen in chronic PKDL.

324



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.

325

4. PKDL in other areas

a. PKDL in China

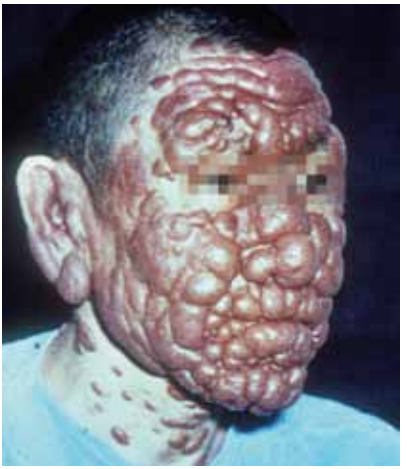
b. PKDL in Brazil

PKDL in China

326

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

327



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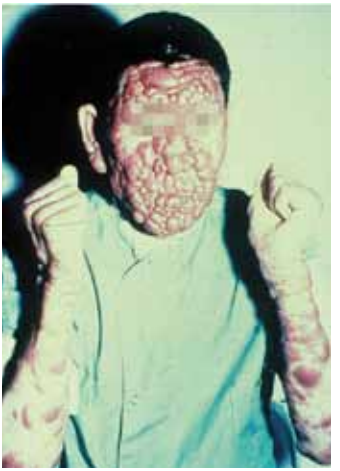
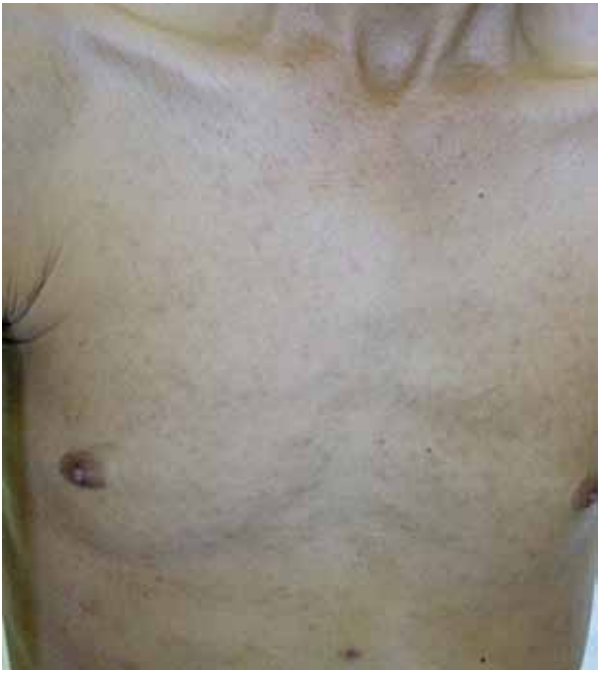


PKDL in Brazil



329

PKDL from 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.

330

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.

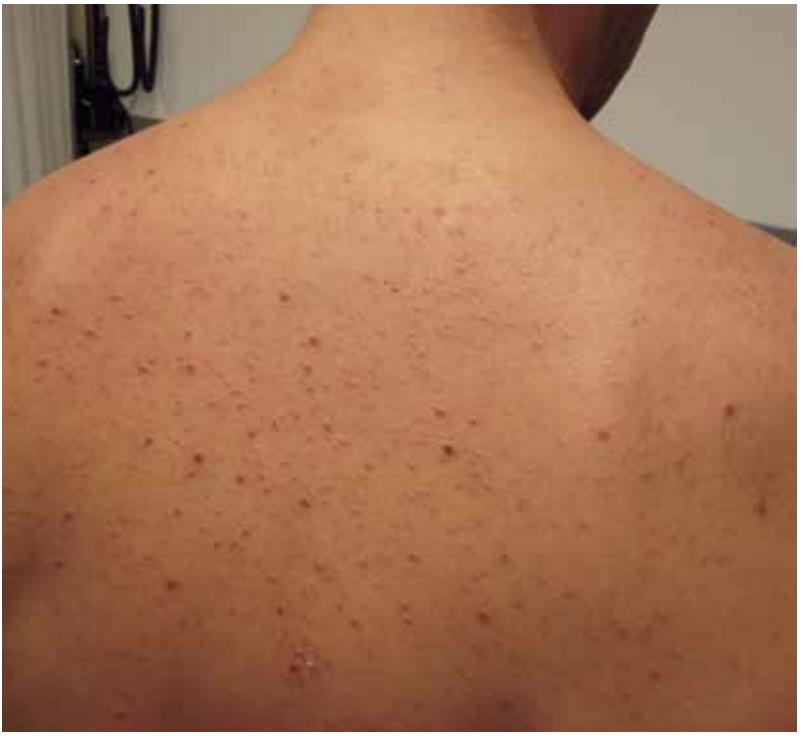
The clinical presentation and characteristics may differ from those found in immunocompetent patients; the most important differences are the atypical distribution and evolution and the abundance of parasites in mainly nodular lesions (Table 7).

Table 7: Differences between PKDL in immunocompetent and immunocompromised patients

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

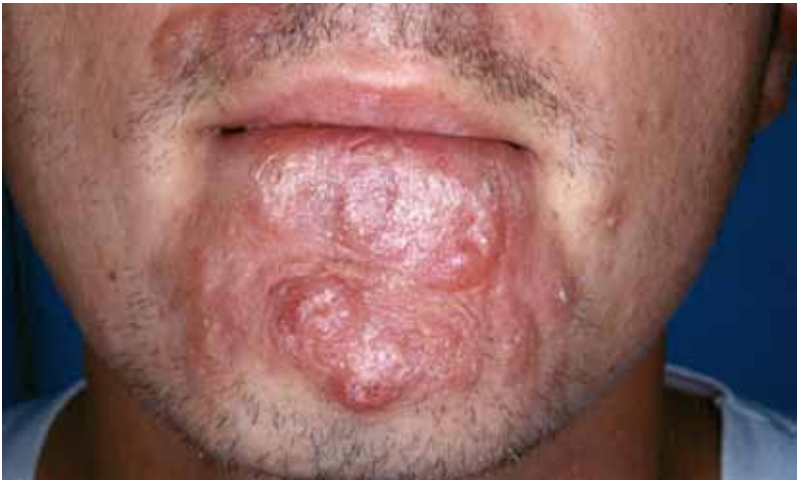
Table 8: Clinical entities in HIV infection with disseminated cutaneous *Leishmania* lesions, parasites isolated and areas from where reported

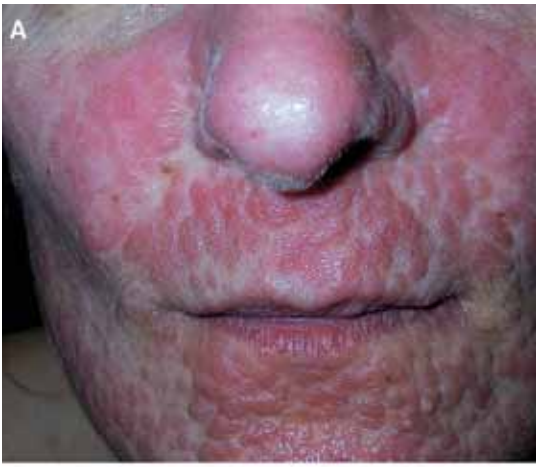
Disseminated cutaneous leishmaniasis without VL (history or present):
L. tropica (India)
L. chagasi / *L. infantum* (Nicaragua)
Disseminated cutaneous lesions preceding VL:
L. infantum (France)
Disseminated cutaneous lesions with concomitant VL
L. donovani (Ethiopia)
L. infantum + *L. donovani* (Brazil)
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)



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.





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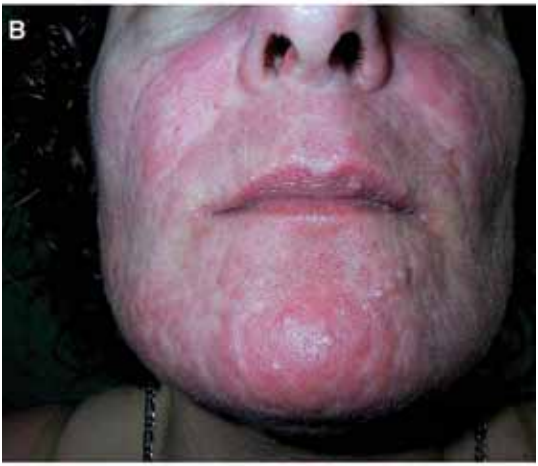

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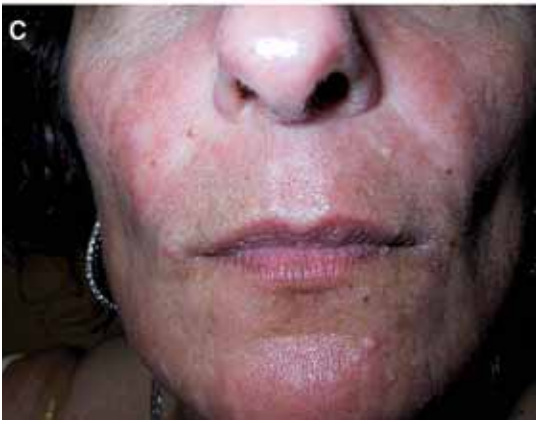
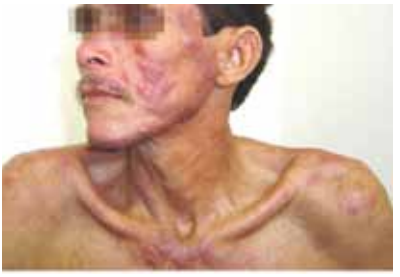
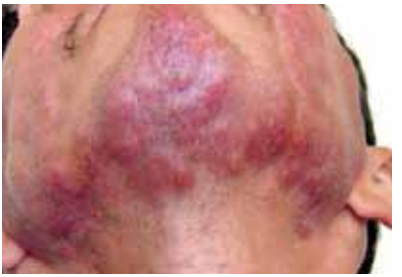


335

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.



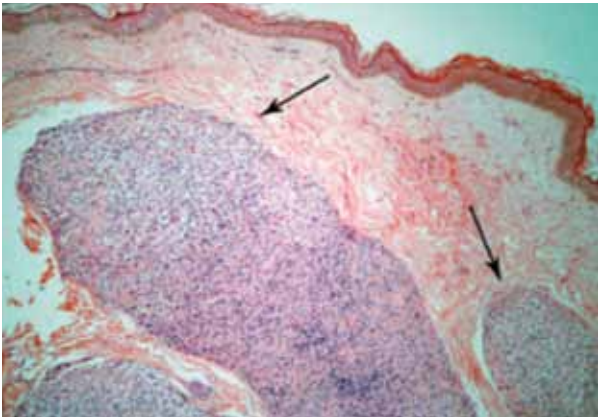
336



337

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).

338



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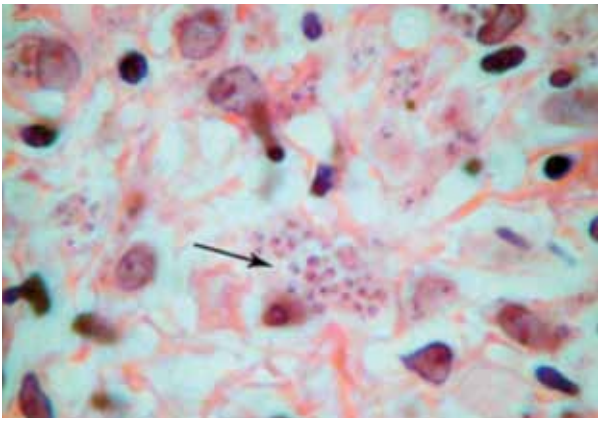
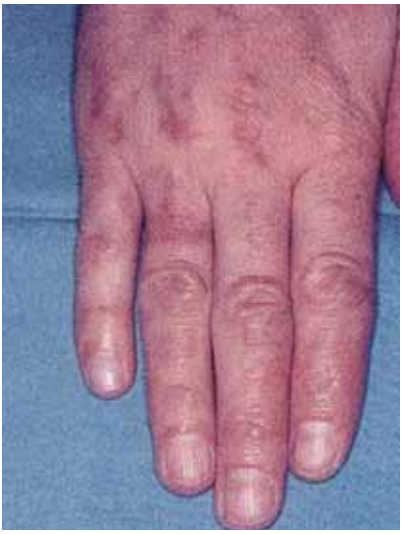
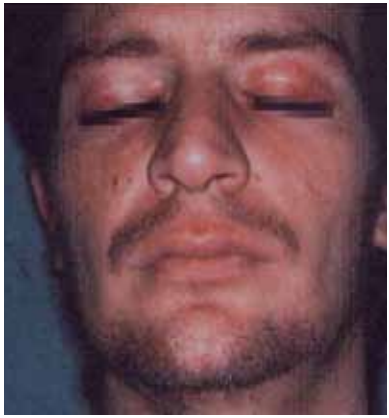




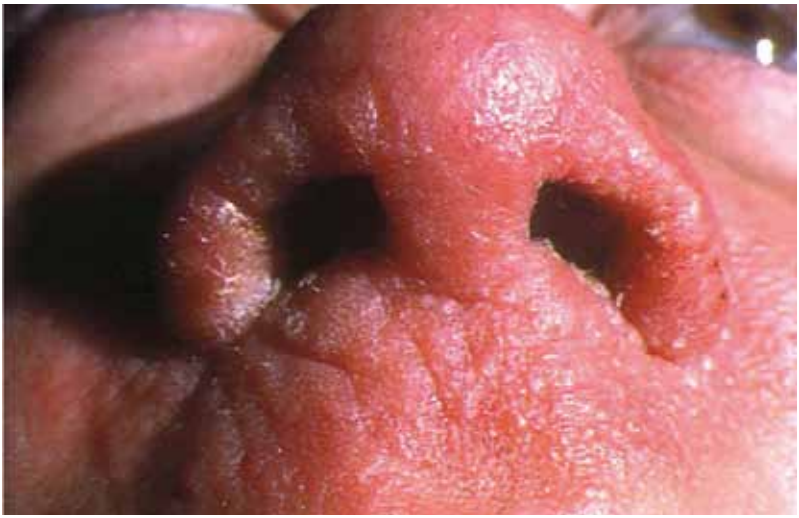
340

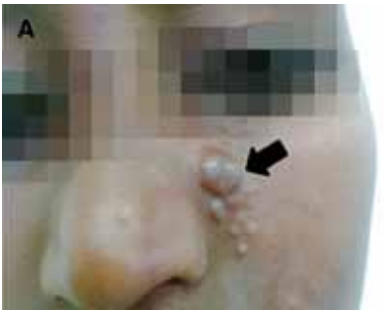
PKDL in 2 HIV positive patients from Spain. Dermatomyositis-like presentation showing erythematous plaques with periungual erythema on dorsum of hands (340) and in the face particularly on the upper eyelids (341).

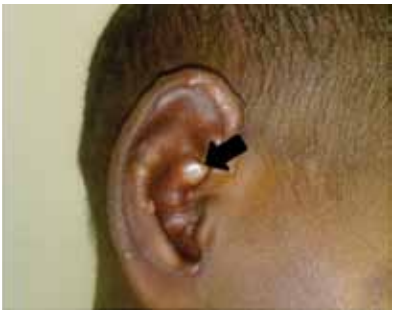
341



342
Diffuse infiltration around the nose.



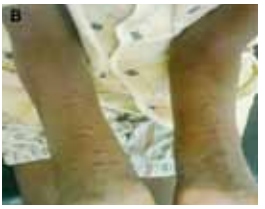




343 **344**

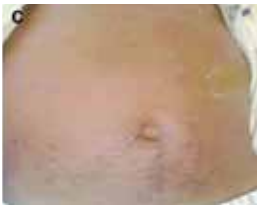
Three patients from Ethiopia.
HIV-positive with papules and nodules in the face, abdomen and extremities. Amastigotes were demonstrated in spleen aspirate and slit skin smear.

345

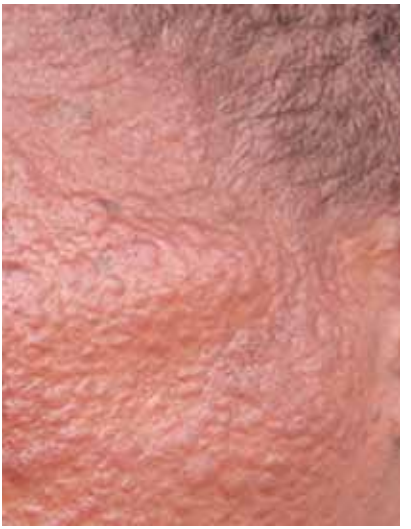


346

347

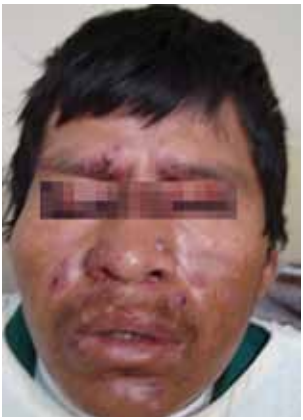


349
HIV-positive with amastigotes demonstrated from bone marrow and skin scrapings.

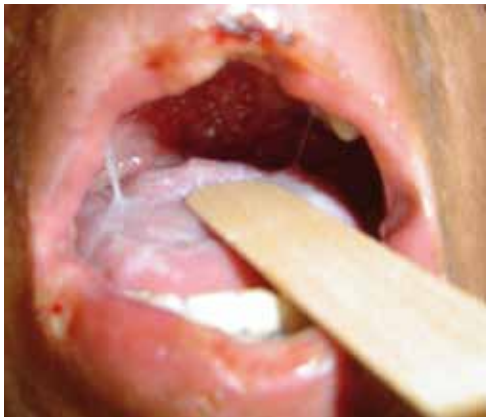


348
HIV-positive and PKDL with Kaposi's sarcoma-like lesions on the lower legs; amastigotes were found in a skin scraping.

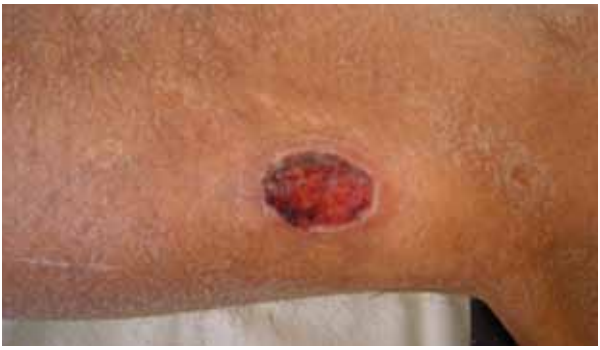
Disseminated cutaneous lesions and mucosal lesions in HIV infection without VL



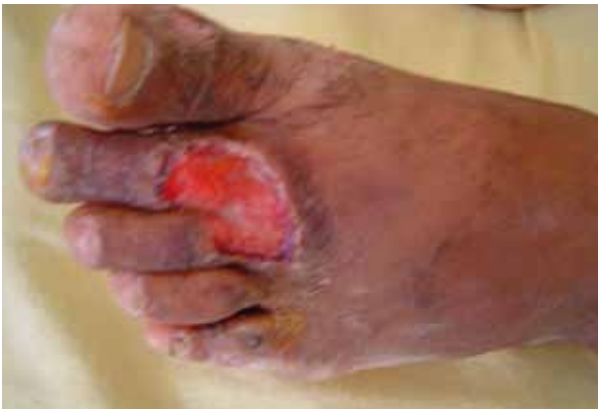
350



351



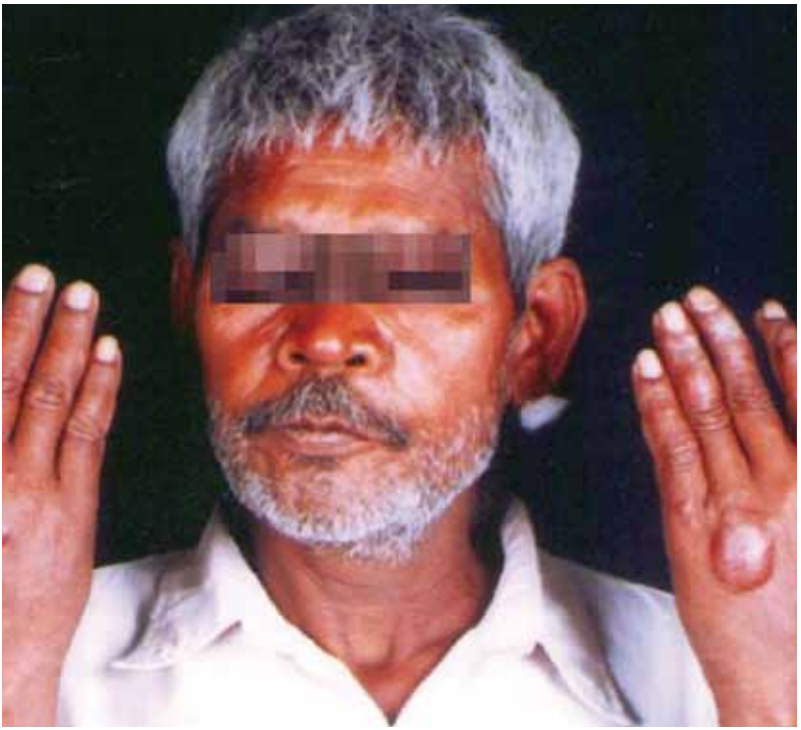
352



353

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.

Disseminated cutaneous lesions and mucosal lesions in HIV infection without VL

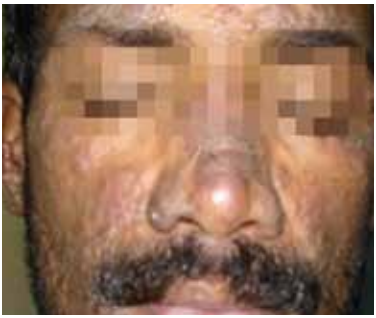


354

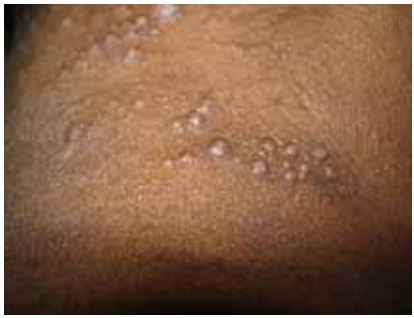
355

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).

Disseminated cutaneous lesions and mucosal lesions in HIV infection without VL

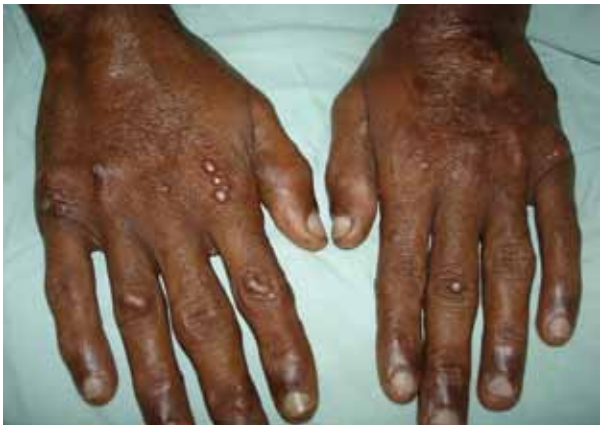


356

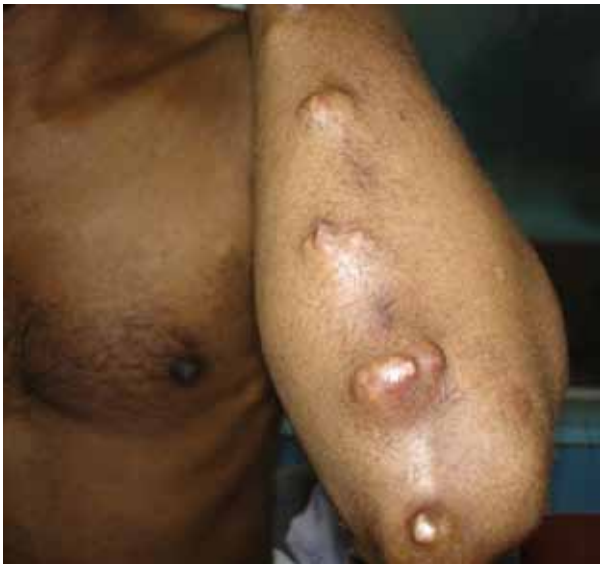


357

358

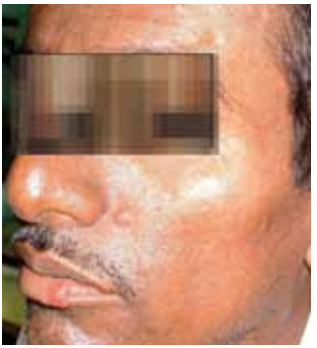


Diffuse cutaneous leishmaniasis in a patient who was HIV-positive patient for 8 years and on antiretroviral therapy. This patient was from Kerala, South India. There was no previous history of VL. *Leishmania* amastigotes were demonstrated in a smear.

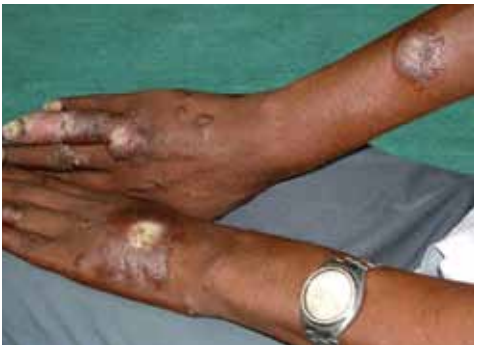


359

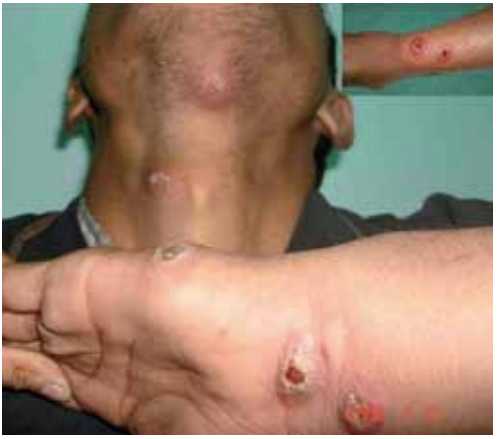
Disseminated cutaneous lesions and mucosal lesions in HIV infection without VL



360

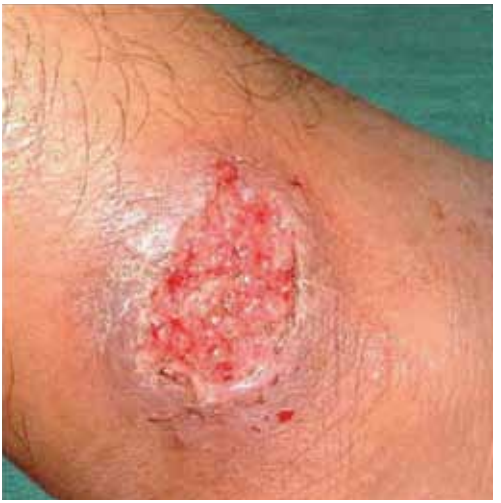


361



362

Three HIV positive patients from India with multiple CL lesions due to *L. tropica*.



363

364

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

Note

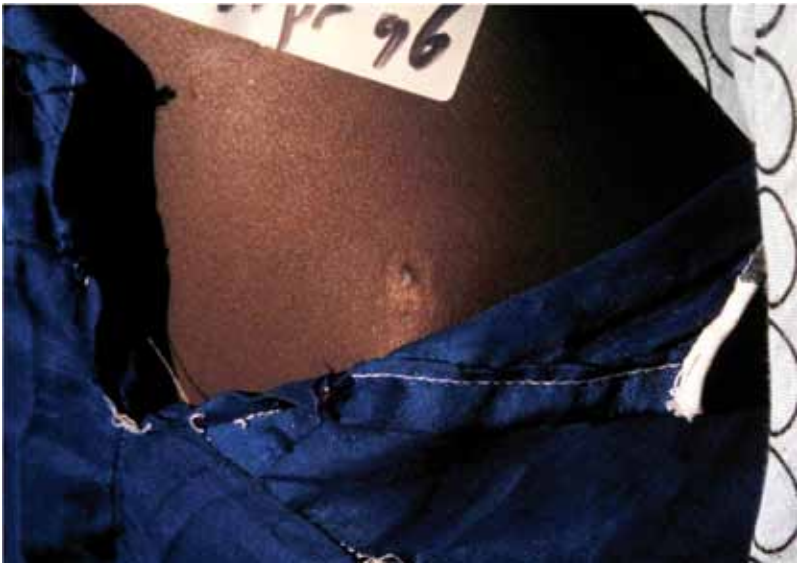
The conditions shown do not necessarily overlap in epidemiology with PKDL.

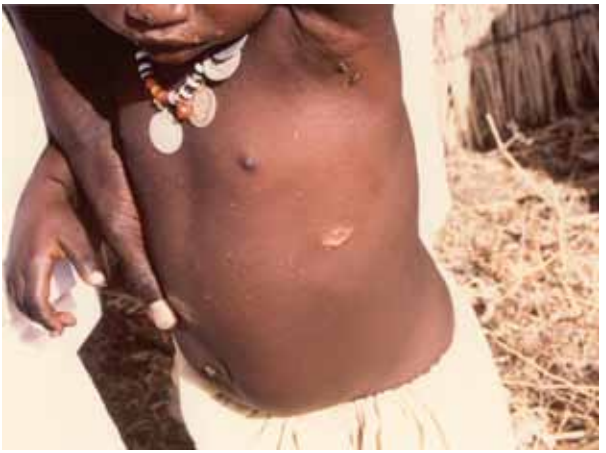
Leishmanioma in Sudanese patients

365

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).

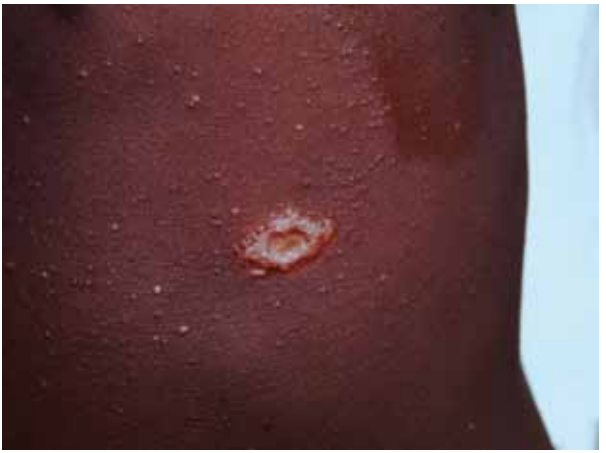
366



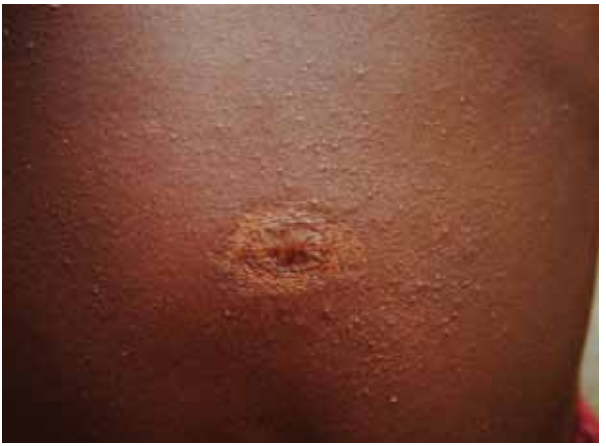


367

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



368

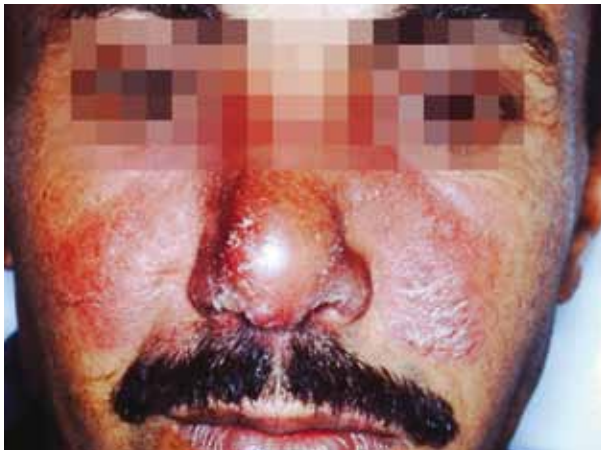


369

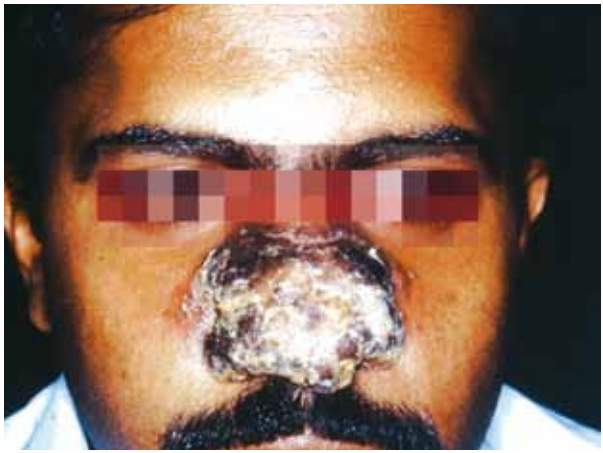
Healed leishmanioma .

Cutaneous leishmaniasis in three Sudanese patients

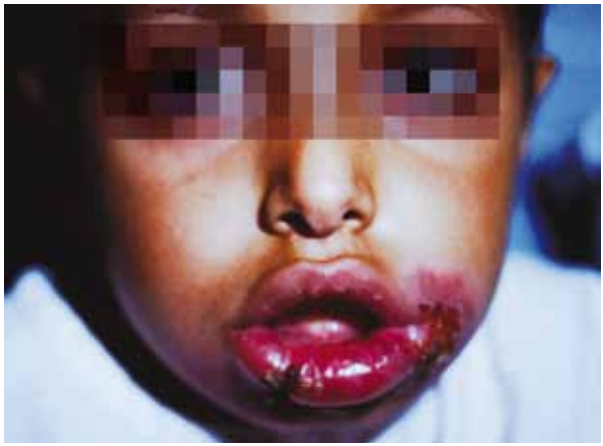
370
Erysipeloid infiltrative lesion of the nose and cheeks.



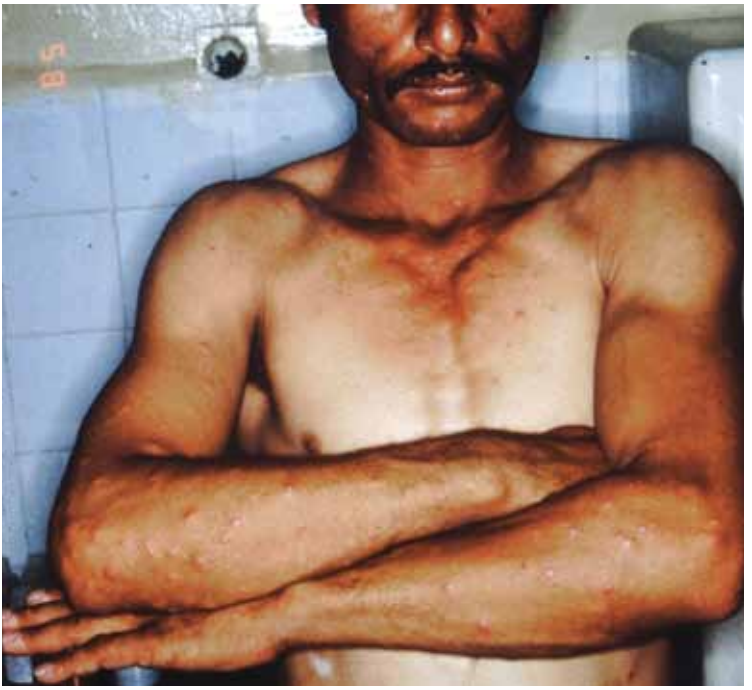
371
Note swelling, crusts and scales.



372
Facial lesion with cheilitis.

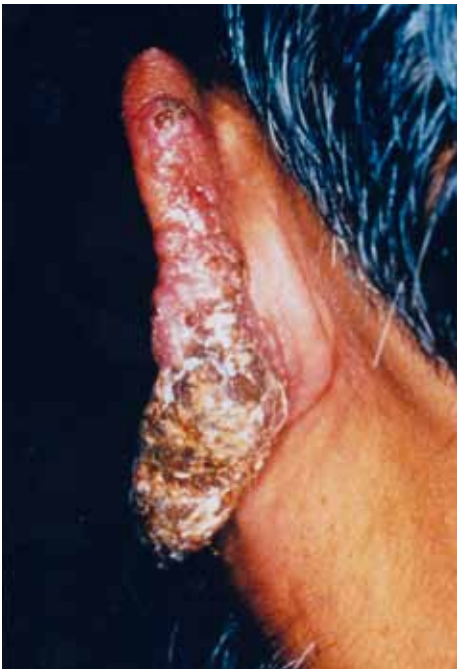


373



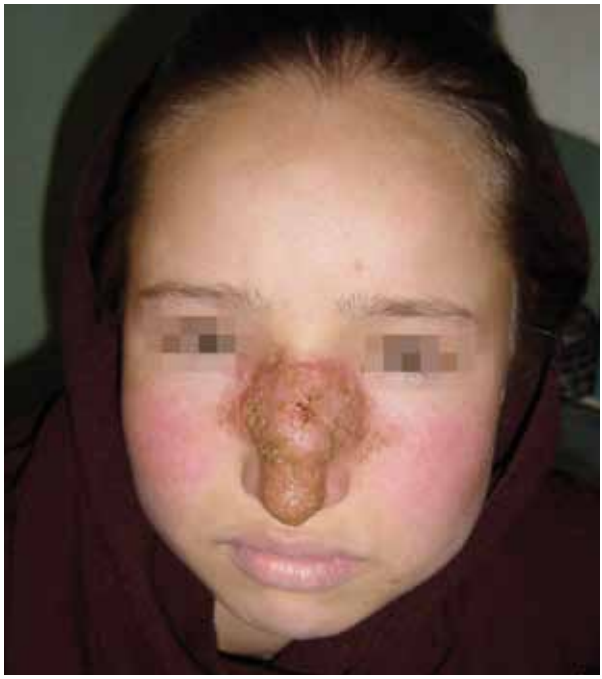
374

Multiple disseminated micro-nodular lesions. Note the face is free of lesions, which is unusual in PKDL.



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

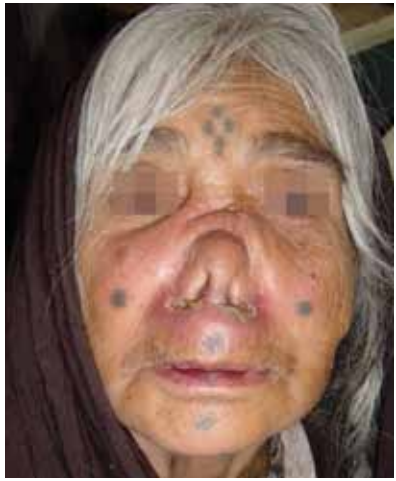
Cutaneous leishmaniasis *(L. tropica)* from Afghanistan



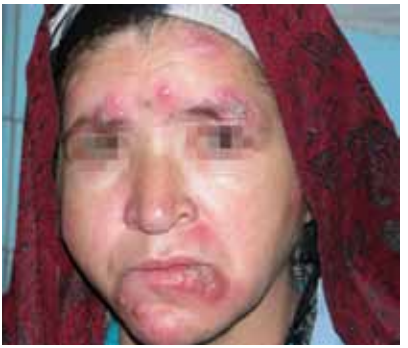
375

Clinical manifestations of CL showing various degrees of ulceration.

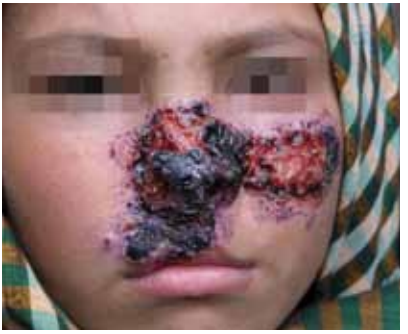
376



377



378

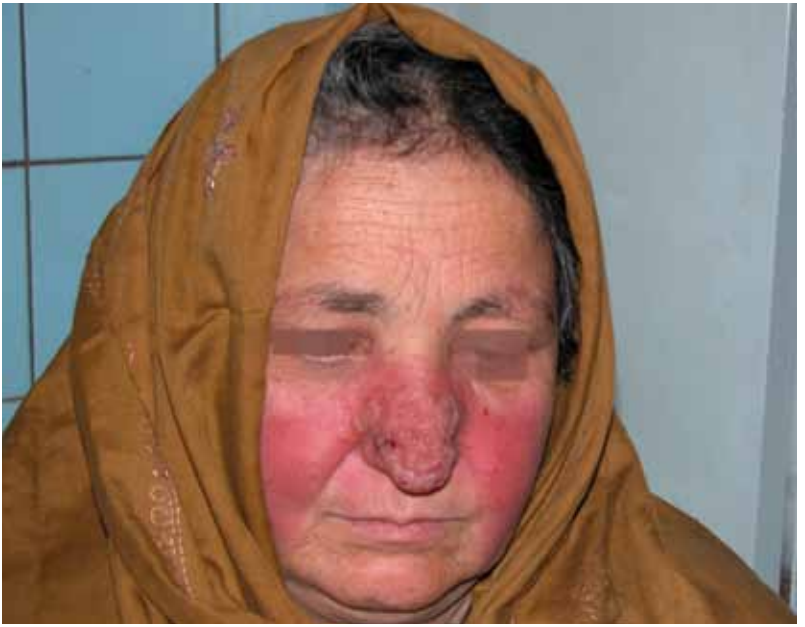


379

Various degrees of ulceration in cutaneous leishmaniasis *(L. tropica)* from Afghanistan.

380

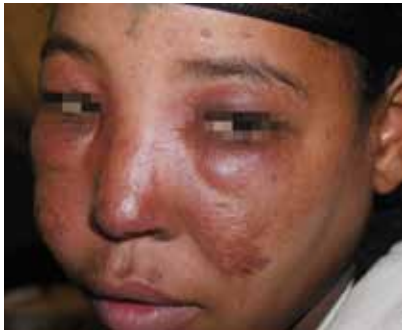
Lupoid form. **381**



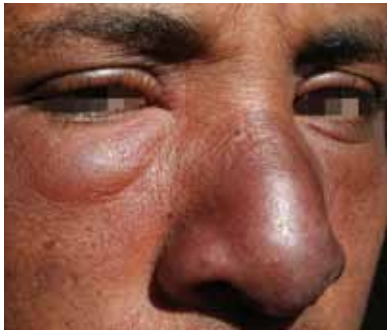
Cutaneous leishmaniasis *(L. aethiopica)* from Ethiopia

382

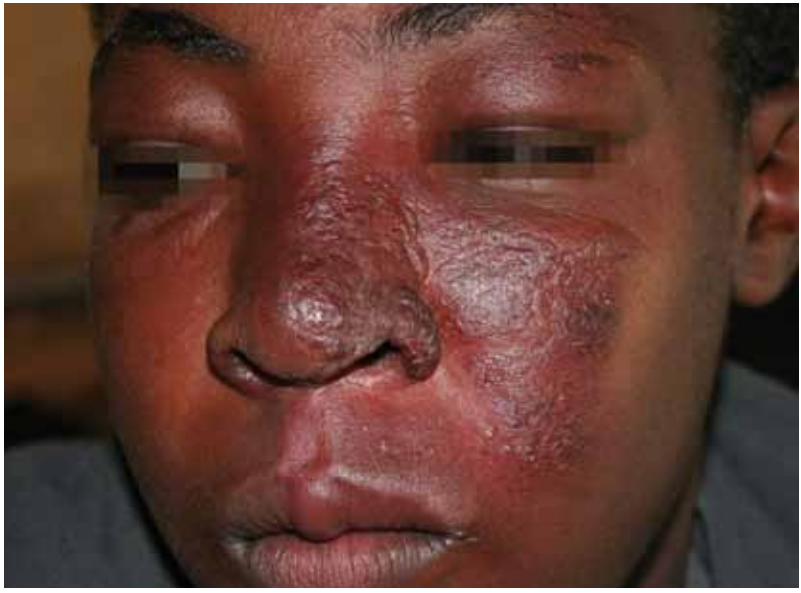
CL due to *L. aethiopica*: diffuse infiltration of the skin



383



384



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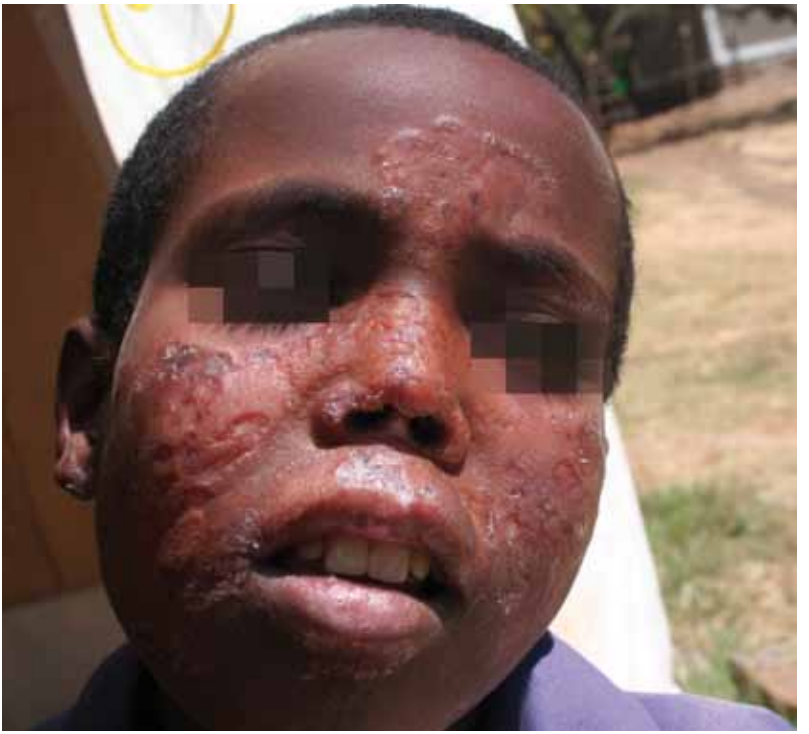
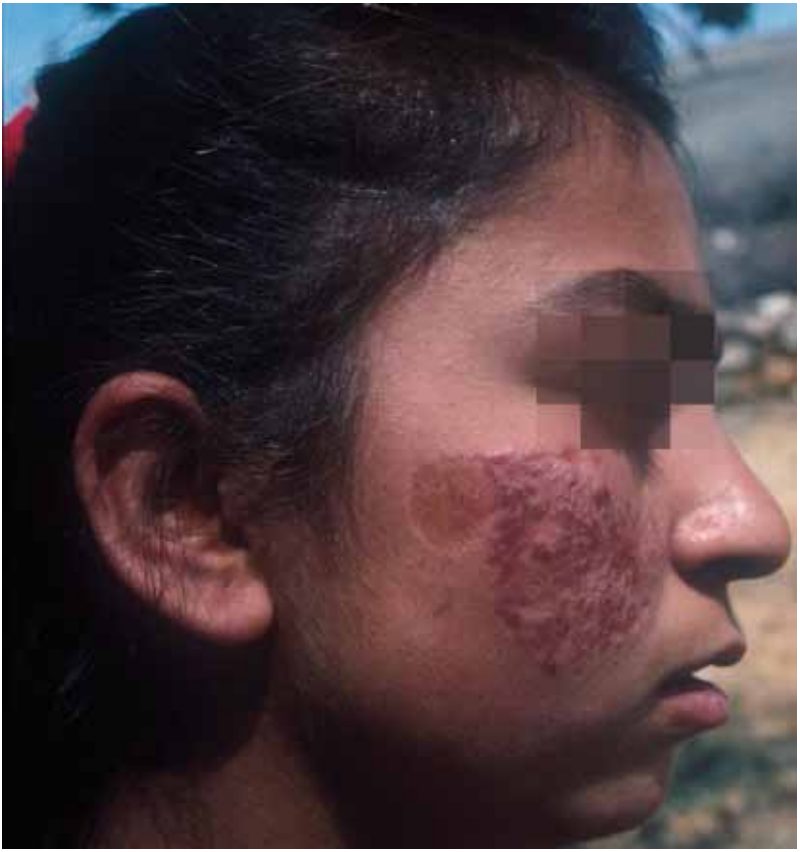
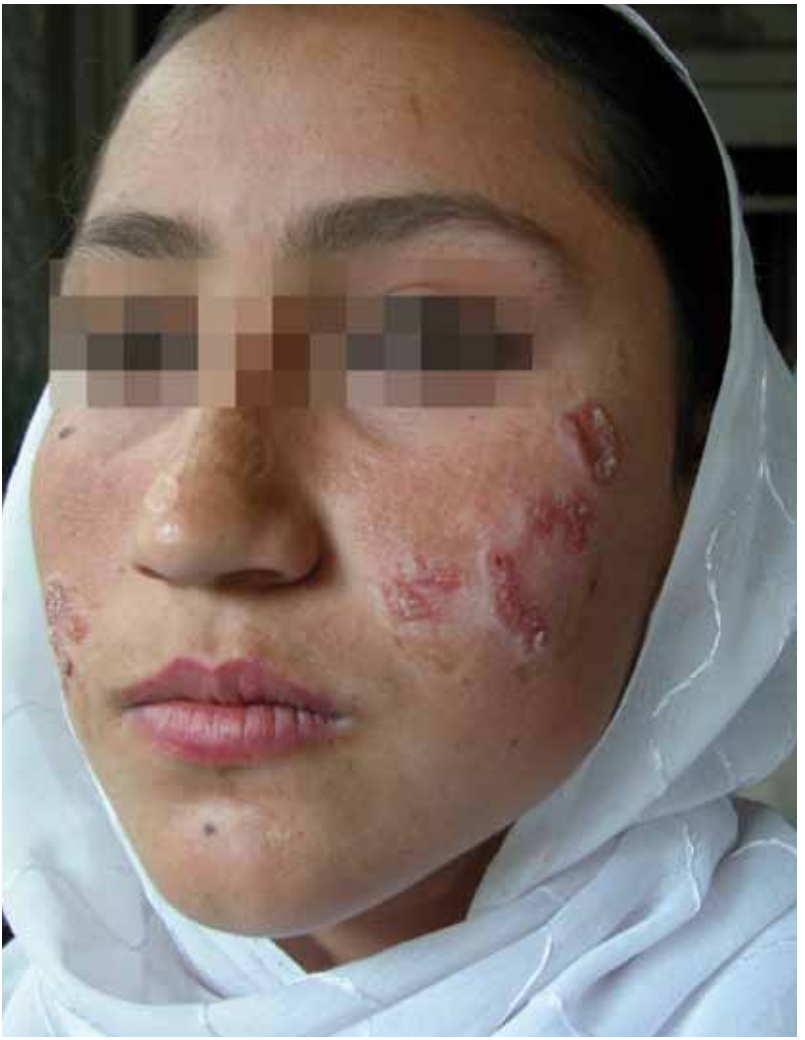
386



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

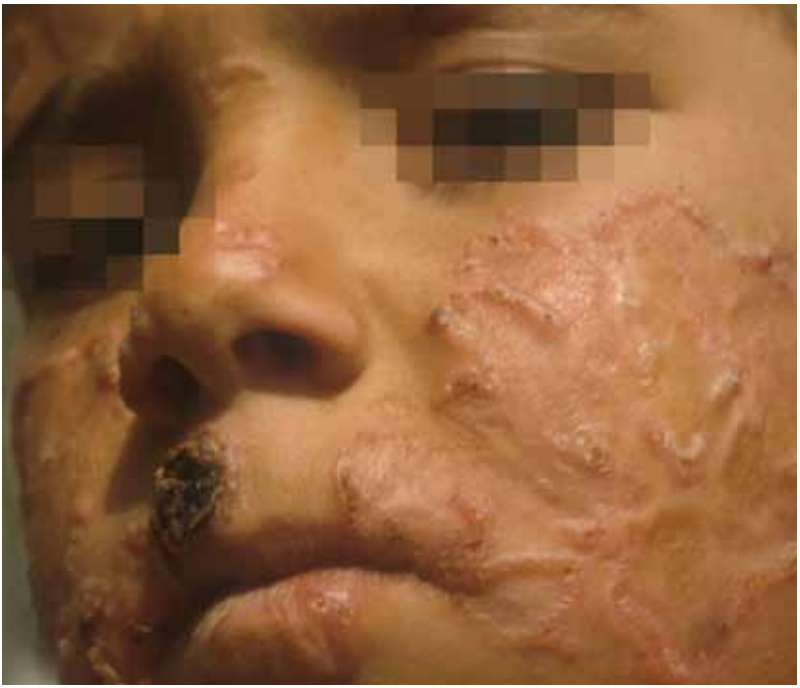


388 Leishmaniasis recidivans due to *(L. tropica)* from Morocco. Note the healed scar from which new lesions develop.



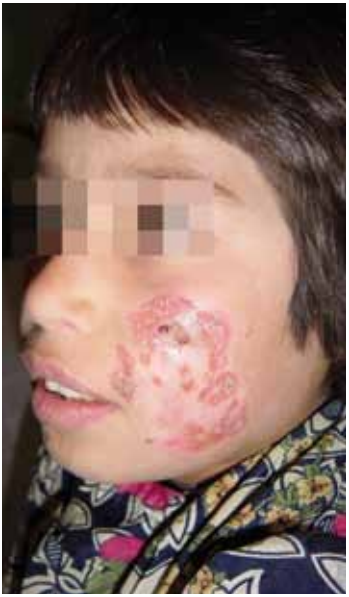
389 Leishmaniasis recidivans due to *(L. tropica)* from Afghanistan. Note the healed scars from which new lesions develop.

Leishmaniasis recidivans



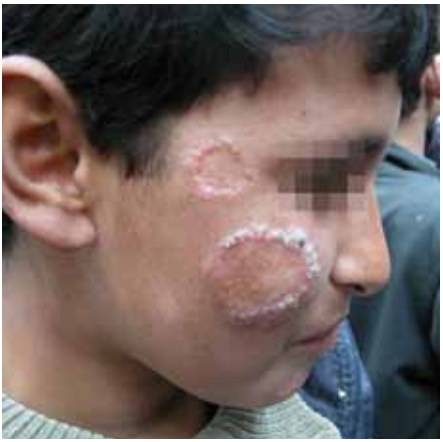
390

391



Leishmaniasis recidivans due to *L. tropica* from Morocco (390,) and Afghanistan (391, 392).

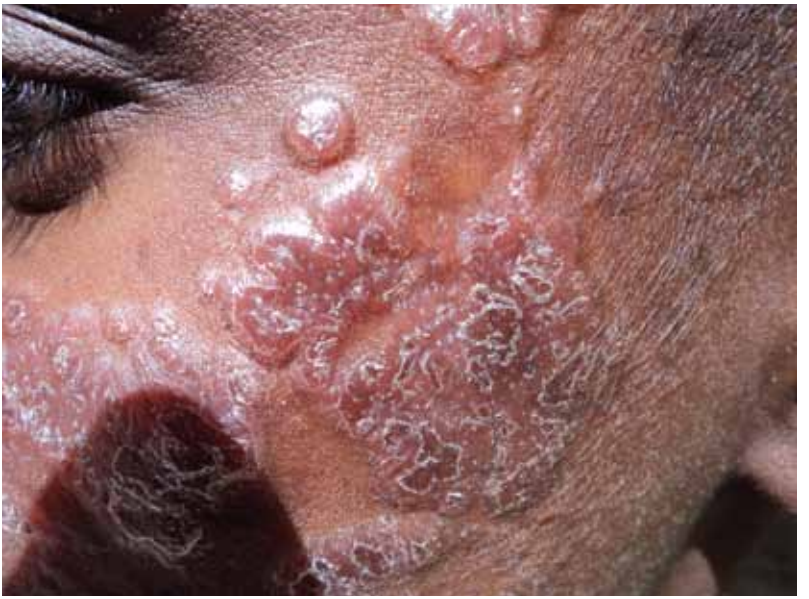
392

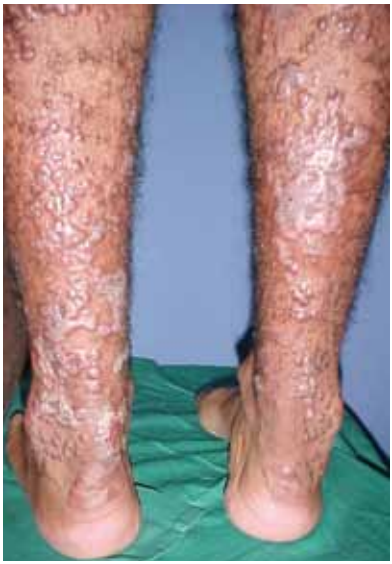


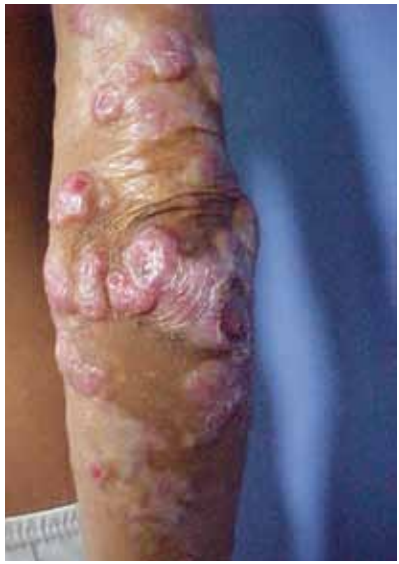
393 **Diffuse Cutaneous Leishmaniasis (DCL)** from Ethiopia. The patient originating from the Highlands where CL and not VL is endemic; there is no previous history of VL. *Leishmania* parasites were found in a skin scraping.

394

395

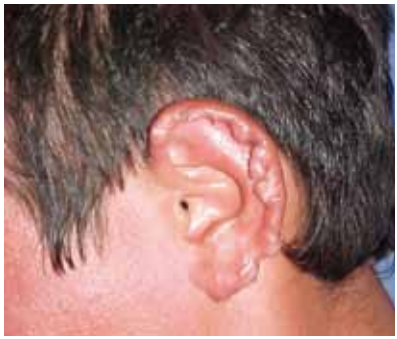


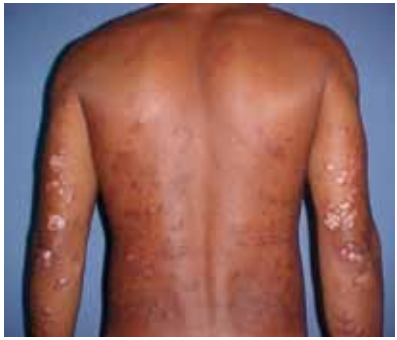




396 **Diffuse Cutaneous Leishmaniasis (DCL)**; Venezuela. **397**

398





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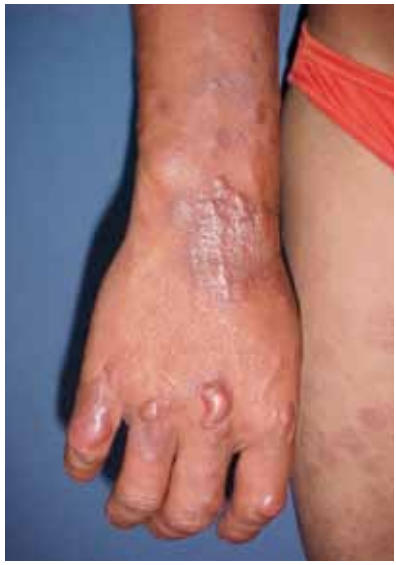
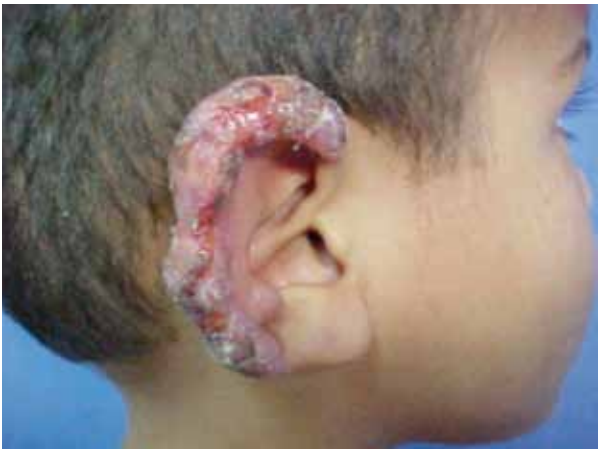
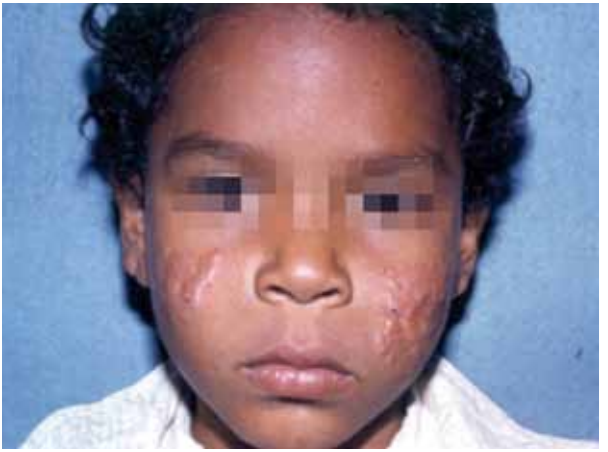


400

Diffuse Cutaneous Leishmaniasis (DCL); Venezuela.

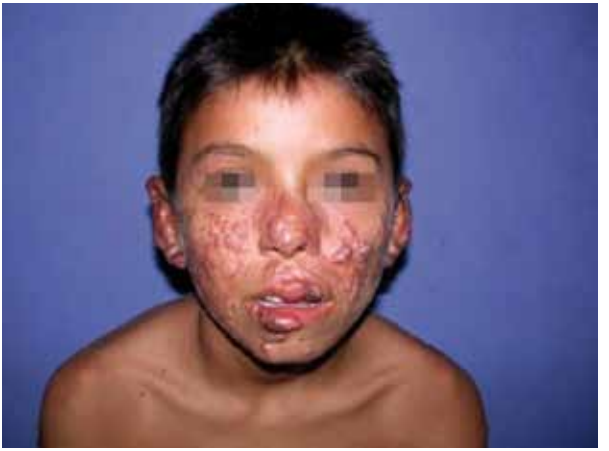
401





402

Diffuse cutaneous leishmaniasis (DCL) in Venezuela is normally by *L. amazonensis.*



403

404

Diffuse cutaneous leishmaniasis; 405
Venezuela (see also Figs 357, 358 an 361 for comparison)

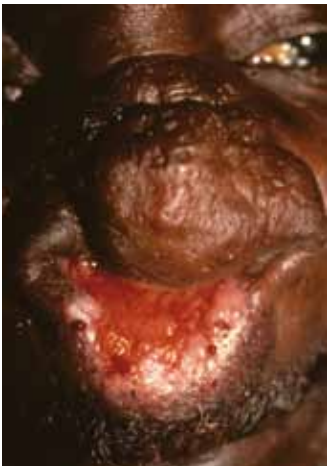
406

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.

408

Mucosal leishmaniasis (Sudan). Before and after treatment with Pentostam®.

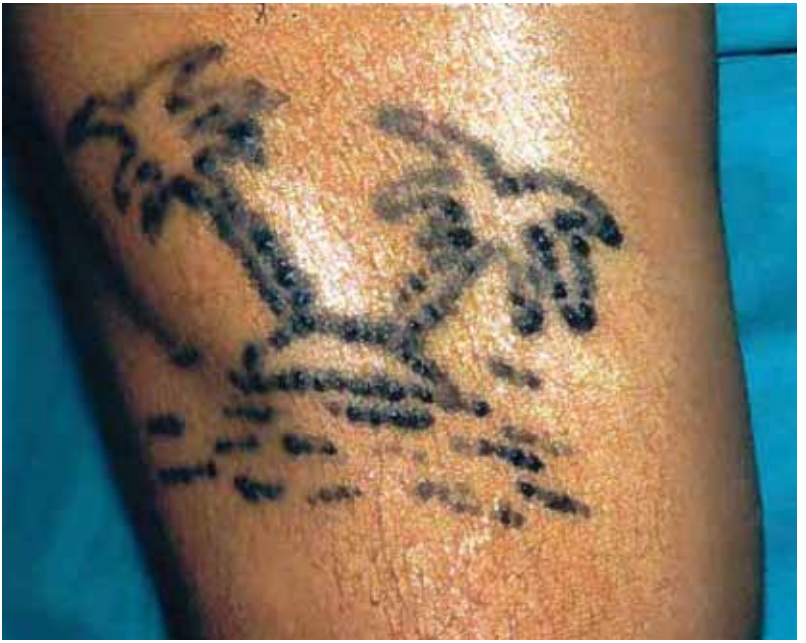
407



409

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 Köbner's phenomenon, Figs 186-189).

410



7. Literature

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8. Acknowledgements/ List of contributors

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