

The Diagnosis of Leprosy

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Preface

The objective of this LML Distance Learning Guide was to make available a concise and practical guide on how to diagnose leprosy. Since its first publication in 2010 this guide has been enthusiastically received and translated into other languages. This new version is an update of the 2010 guide.

The intention of this learning guide was not to be a substitute for WHO technical guides, ILEP teaching guides, national manuals and guidelines, textbooks or other important learning tools and materials. Nor can any guide or manual replace practical teaching, on-the-job training, learning through collaboration with patients, follow-up of patients on treatment and of patients at risk and/or with complications. The reader is referred to text books and other publications to complete the study of this subject. Many of the concepts reported in the written text are from authors listed in the section Sources and References.

This brief course “The Diagnosis of Leprosy” is made up of a written text and a set of slides. The slides are zipped in order to facilitate transmission via e-mail. Additionally, clinical images can be requested from S. Noto when needed for training purposes and Word/PowerPoint files will be available for downloading from the LML Archives webpage.

The text and related slides have been divided into three parts (“Introduction”, “Cardinal signs” and “Diagnosis and the clinical spectrum of leprosy”). All comments and contributions will be accepted and hopefully included in further editions of the course.

We are very grateful to Enrico Nunzi for his revision of the course and to Augusto da Costa Nery, Antonio Salafia and Grace Warren, for making their slides available, Andrea Clapasson for revising the laboratory section and Anne Chadwick for revising the English.

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Part I. Introduction

1. Definition of leprosy

Leprosy is a chronic, infectious disease caused by *M. leprae*. It affects mainly the skin and the peripheral nerves. (Slide 1)

2. Leprosy a protean disease

Leprosy has been called the “great imitator” and shares this “reputation” with other conditions like syphilis, systemic lupus erythematosus and sarcoidosis (Slide 3). It is indeed a protean disease with innumerable possible clinical frames. The clinical presentations are so many because of the combinations of many different primary skin lesions with or without signs of inflammation, sequelae of these lesions and signs and sequelae of peripheral nerve damage.

The skin may show macules, plaques, papules, nodules, diffuse infiltration, secondary lesions like burns, blisters, fissures, ulcers and scars. The flat lesions, macules and plaques, may be present with extreme variability in colour (also depending on the basic skin colour), size, edge, texture of the surface, location, distribution and number. Papules, nodules and the secondary lesions may also appear in a wide range of modalities.

Moreover the patient may show damage to the eyes, face, nose, hands, feet, testes and other organs and tissues. The damage, again, may vary a lot. It may be relatively minimal and, passing through all degrees, it may become very serious.

At one end of the clinical spectrum a leprosy patient may present only one or a few hypopigmented patches on the skin and nothing else. On the other hand, in the worst scenario, a patient may be blind, with gross destruction of both hands and feet and loss of sensation over most of his skin. The patient is present, “within his anaesthetic skin” but, almost isolated from the rest of the world. Between the two extremes innumerable combinations are possible. Slides 4 - 19 show examples of the same disease, leprosy, but in diverse clinical frames.

3. How to put order in clinical leprosy

The “cardinal signs of leprosy”, the “Ridley-Jopling classification” and, “reactions and nerve damage” are the key concepts that allow order in the extremely various clinical scenario of leprosy (Slide 20).

The cardinal signs are invaluable in guiding the clinician in the careful and systematic assessment of the patient and, in doing so, in reaching the diagnosis (Slide 21). They will be dealt with in this course.

The Ridley-Jopling classification represents a milestone in the field of leprosy. It clearly correlates the immune response of the host to the bacterial load, the histopathological abnormalities and the clinical pictures (Slide 22).

Reactions and nerve damage are critical in order to understand the etiopathogenetic process that links most of the clinical pictures of leprosy. In leprosy the peripheral nervous system can be affected at 3 levels (Slide 23): dermal nerves these are the very fine nerve endings in the skin; cutaneous nerves, these are thicker nerves that run just under the skin and, the major nerve trunks (Naafs B. 1990). Damage to nerve trunks is far more important than damage to dermal or cutaneous nerves. Most of the nerve damage in leprosy takes place during acute exacerbations of the disease called “reactions” (Slide 24).

Classification and reactions and nerve damage will be the topics of forthcoming courses together with the histopathology of leprosy.

4. Tools available for the diagnosis of leprosy

The tools available for the diagnosis of leprosy are very limited and the diagnosis is based mainly on clinical grounds and bacteriological examination.

The careful research of the symptoms and signs of the disease in the skin and peripheral nerves of the patient will allow diagnosis. History taking, physical examination and laboratory investigations are the tools to be used (Slide 25). Physical examination will allow diagnosis in most cases. Physical examination plus slit-skin smear examination will allow the diagnosis in the vast majority of cases.

Histopathology of a skin biopsy and, exceptionally of a nerve biopsy, may be useful. Note however: histopathology can be supportive of the clinical picture, but without any of the cardinal signs present the diagnosis of leprosy cannot be made by histopathology, except when *M. leprae* are found in the biopsy.

There are no laboratory tests which diagnose leprosy, neither serological tests using Phenolic glycolipid 1 (PGL-1), (ELISA, Dipstick, lateral flow) nor the testing of the cell mediated immunity (lymphocyte transformation test [LTT] whole-blood culture), nor the lepromine test (Mitsuda). Even polymerase chain reaction (PCR) and Nucleic Acid Sequence-Based Amplification (NASBA) are of no real help. These tests can be positive in contacts and negative in obvious patients.

5. Clinical examination

This should be possibly performed in the correct environment. The quietness in the examination room, the source of light and the respect of the patient's privacy are important points. In the case of female patients, the presence of a female assistant may be necessary. Examination is better performed in daylight (indirect sunlight), or in a well-lit room (Slides 27-28). The source of light should come from behind the examiner. Look at the skin from a distance and then close up. Examine the whole body from the head to the feet, including the soles. Skin and nerves should be assessed in a systematic way.

6. When to suspect leprosy

Leprosy should be suspected in people:

1. with any of the following symptoms or signs:

- pale or reddish patches on the skin (the most common sign of leprosy);
- loss, or decrease, of sensitivity in the skin patch;
- numbness or tingling of the hands or feet;
- weakness of the hands, feet or eyelids;

- painful or tender nerves;
 - swellings or lumps in the skin and particularly on the face or earlobes;
 - painless wounds or burns on the hands or feet.
2. living or coming from a leprosy endemic area and having a skin disease which does not improve with routine treatment and, especially when there are signs of peripheral nerve involvement.

7. The diagnosis of leprosy

The diagnosis of leprosy is based on the 3 cardinal signs of the disease (Slide 21). Which are:-

1. skin patch with loss of sensation;
2. enlarged peripheral nerve;
3. positive slit-skin smear.

Part II. Cardinal signs

The 1st cardinal sign: skin patch with loss of sensation

Sensory loss in macules or plaques is diagnostic of leprosy (Slide 2). There are very few, if any, skin diseases that present anaesthetic lesions; only when there are very thick squamulae there may be a “pseudo loss” to a very fine touch; indeed never anaesthesia.

Macules and plaques in leprosy may show several other typical abnormalities. The colour can be hypopigmented, hyperpigmented, erythematous or copper-coloured (as mentioned before, also depending on the basic skin colour). The texture of the surface may be dry and rough for loss of sweat in some forms of the disease, or shiny and smooth in others. There may be loss of hair growth. Some macules may show typical streaming on one side of their margins and satellite lesions. The lesions may become acutely infiltrated, swollen and erythematous.

Some leprologists consider “characteristic” skin lesions an additional cardinal sign. “Characteristic” has been explained as: - hypopigmentation in dark skin in tuberculoid and indeterminate leprosy or diffuse infiltration, macules, papules and nodules in lepromatous leprosy. However, **in our point of view**, none of these abnormalities confirms the diagnosis of leprosy unless, there is either a loss of sensitivity, an enlarged nerve or a positive slit-skin smear.

For all purposes in leprosy loss of sensation in a skin lesion is diagnostic of the disease (Slides 5, 6). The loss of cutaneous sensation is often partial; it may be to light touch (anaesthesia), to pain (analgesia) or to temperature discrimination (hot and cold).

Testing for loss of sensation

This is a relatively simple test that confirms diagnosis of leprosy in many cases. Quietness in the environment or in the room where it is performed is important. Both the patient and the examiner must be positioned comfortably while examining.

The simplest and quickest way to test for anaesthesia is to use the tip of your finger to touch the patient. Using the pulp of your little or ring finger, touch the patient very gently. If you can feel it, he should too (Hastings 1985).

More commonly a fine, pointed wisp of cotton wool (Slide 3) is used to touch the part to be tested. First explain to the patient what you will be doing. Then demonstrate while he watches and points carefully to the exact spot touched. When he comprehends fully, then continue testing various sites **in and outside** the lesions but, with the patient's eyes covered (Slide 4). Touch only, do not brush across the skin. Inability to identify the point stimulated at all, denotes loss of sensation to the stimulus used. If he feels it but he cannot point to the exact spot, it is called misreference, and it is the earliest sign of hypoesthesia (Hastings 1985). The patient with closed eyes can either point with one finger to the exact spot where the cotton wool touched the skin or the patient can confirm the exact place verbally when he feels the touch. Test the reliability of the patient by asking where he feels when not touching the skin at all.

Alternatively heat sensation is tested with two test tubes, one containing hot water and the other cold water (Yawalkar S J 2002).

Cotton wool may be too delicate for the thickened skin of palms and soles. Monofilaments or nylon bristles could be used to test for sensory loss in lesions on palms and soles. The Semmes Weinstein monofilament test is nowadays recommended for assessing peripheral nerve impairment. Sensory testing (ST) will be discussed in more detail in the part about "Reactions and nerve damage".

Warnings:

1.

Loss of cutaneous sensation means that the sensation, in particular the touch, in the lesion is diminished in comparison with the surrounding skin. Loss of cutaneous sensation may also be to pain and to temperature.

2.

Sensory changes on face may be less evident than in other areas of the body because of the rich nerve supply of the face.

3.

Towards the lepromatous side of the spectrum, borderline lepromatous and lepromatous leprosy, in early cases often no loss of sensation is found. In advanced lepromatous cases there may be extensive loss of sensation and, bilateral anaesthesia of the glove-and-stocking type.

4.

In the "indeterminate" form of leprosy, loss of sensation cannot be detected; but sometimes loss of autonomic nerve function can be found (e.g. loss of sweating).

5.

Pain sensation is tested by pin-prick (be careful not to damage the skin) and temperature by touching the skin with test tubes containing hot and cold water.

The sweat and histamine tests

Two other tests may be useful in diagnosing leprosy and are used by some leprologists: -

1.

Sweat test: sweating is dependent upon the integrity of parasympathetic nerve fibres. If a hypopigmented patch is due to leprosy the response of the sweat glands to exercise or to a cholinergic drug will be diminished (Slide 8) [Bryceson A, Pfaltzgraff Roy E (1990)];.

2.

Histamine test: the wheal and flare response to histamine is the end product of a local reflex which depends upon the integrity of sympathetic nerve fibres. If a hypopigmented patch is due to leprosy the response of the skin to histamine will be diminished (Slide 10) [Bryceson A, Pfaltzgraff Roy E (1990); Menicucci L. et al.; Rodriguez J. et al (1931)].

The 2nd cardinal sign of leprosy: enlarged peripheral nerve

An enlarged peripheral nerve represents the 2nd cardinal sign of leprosy (Slide 2). Enlarged peripheral nerves are very rarely found except in leprosy. Other conditions which could present enlarged peripheral nerves are: primary amyloidosis and some hereditary peripheral neuropathies (like the neuropathy of Charcot-Marie-Tooth). These are all very uncommon. In a leprosy endemic area, the finding of enlarged peripheral nerves is an important element to establish the diagnosis.

The palpation of the nerves at the “sites of predilection” is performed during the physical examination of the patient. Palpation is performed gently using the pulp of the fingers, not the finger tip or finger nail. Watch the person’s face to make sure you do not cause him unnecessary pain when you touch the nerve. Evaluate the tenderness (spontaneous or when palpating), consistency (soft, hard, irregular) and size (enlarged, normal, small) of the nerve; however, only the size is important for the diagnosis of leprosy (Slide 3). Tenderness when palpating the nerve or spontaneous nerve pain are signs of reaction. Additionally, signs and symptoms of peripheral nerve sensory, motor and autonomic involvement may be present.

It is essential to know the normal limits by constant practice in palpating nerves. During an examination one should always compare nerves on the opposite site of the body.

All peripheral nerves may be enlarged in leprosy. Cutaneous branches associated with a skin lesion may be enlarged as well (Slides 34-35). The two most commonly affected are the ulnar nerve and, in the second place, the lateral popliteal (also called common peroneal) nerve. In the following paragraphs, how to locate and palpate the peripheral

nerves of predilection in leprosy will be illustrated. They will be described systematically starting from the head, then those of the upper limbs and finally those of the lower limbs.

Supraorbital nerve

An enlarged supraorbital nerve is palpable as it passes upwards out of the orbit (Slide 8). To palpate it run your index finger across the forehead from the midline laterally. A branch of this nerve can be seen in Slide 9.

Great auricular nerve

The great auricular nerve can be seen in the neck emerging from the posterior border of the sternocleidomastoid muscle. The patient turns his/her head to one side, thus this muscle is stretched (and the external jugular vein flattened, which otherwise could be mistaken for an enlarged nerve). The great auricular nerve courses anteriorly and superiorly across the muscle towards the earlobe (Slides 10-13).

Ulnar nerve

The forearm of the patient is bent at 90°-110° over the arm. The examiner uses his left hand to palpate the right ulnar nerve and his right hand to palpate the left ulnar nerve. The nerve can be palpated first at the elbow in the olecranon groove, between the olecranon of the ulna and the medial epicondyle of the humerus. Then it can be felt and evaluated immediately above the groove (Slides 15 - 16) and further upwards the arm. In some patients even up till the axilla. In comparing left and right ulnar nerves it is useful to ask the patient to put his hands on the examiner's shoulders; in this case the bending is about 135° (B Naafs, personal communication). Alternatively the patient may hold his own hands in front of him. Branch of the ulnar nerve can be palpated on the dorsum of the hand as it curls round the 5th metacarpal bone. This is a useful confirmatory sign in someone with vague neuritis symptoms in the fingers and no other signs of leprosy (G Warren personal communication).

Radial cutaneous nerve

The radial cutaneous nerve is palpated at the wrist. It can be rolled under the tips of the examiner's fingers as it crosses the lateral border of the radius just proximal to the wrist and courses onto the dorsum of the hand (Slides 17 - 21). The radial cutaneous nerve can

also be palpated as it rolls round the 2nd metacarpal bone. No other clinical or laboratory test has the same high sensitivity and specificity (van Hees C., Naafs B., 2009).

Median nerve

The median nerve is felt in front of the wrist when the wrist joint is semi-flexed, proximal to the flexor retinaculum. It is often easier to see than to palpate due to the presence (if present) of the tendon of the palmaris longus muscle. (Slides 22 - 24).

Lateral popliteal nerve (Common peroneal nerve)

The lateral popliteal nerve can be palpated, with the knee joint semi-flexed, in the popliteal fossa, just medial to the biceps femoralis tendon (Slides 25 - 27) and, as it passes round the neck of the fibula. Alternatively it can be felt with the patient and the examiner, one seated in front of the other. One first feels for the head of the fibula, then moving the fingers backwards and downwards. By moving the fingers up- and forwards one feels the nerve rolling under one's fingers.

Superficial peroneal nerve

The superficial peroneal nerve (also called dorsalis pedis) can be easily palpated on the dorsum of the foot (Slides 28-30).

Posterior tibial nerve

The posterior tibial nerve is palpable as it passes posteriorly and inferiorly to the medial malleolus and supplies the sole of the foot (Slide 32). It is difficult to palpate due to tendons and blood vessels which also pass at the spot.

Sural nerve

The sural nerve can be palpated along the midline of the back of the lower leg. The mid to lower part of the leg, where calf muscles join to the Achilles' tendon. The sural nerve can also be palpated as it runs down behind and under the lateral malleolus and along the lateral side of the foot.

Warnings

It is not uncommon in a leprosy endemic area to find people with an enlarged great auricular nerve or radial cutaneous nerve without any other clinical sign of leprosy (including nerve function impairment) or positive bacteriology. Such patients are not put

on treatment but observed and told to come back if anything changes or if the patients develop skin lesions. The enlargement of these two nerves has no direct clinical relevance.

In early cases of leprosy nerve enlargement may not be very great, the nerve may not be tender and hard palpation may not even cause discomfort. The hardness is the clue in this cases! Thin hard nerve may still be palpable years later and confirm a self healed case years after the active disease (Grace Warren, personal communication).

The 3rd cardinal sign of leprosy: positive slit-skin smear

Leprosy is the only disease in which there can be a massive invasion of the dermis or nasal mucosa with acid-fast bacilli (AFB). In some forms of the disease bacilli are demonstrated in slit-skin smears or in nasal mucus or scrapings.

Leprosy bacilli are extremely scanty in lesions of some forms of leprosy, but are present in enormous numbers in lesions of other forms of the same disease. One gram of skin tissue in lepromatous leprosy may contain as many as 7000 million leprosy bacilli (Yawalkar S J 2002).

In the context of leprosy control activities and programmes it is important to organize services for the collection of slit-skin smear (or skin smear) and their processing. Quality control and continuous supervision and monitoring of this activity are necessary in order to ensure uniformity, reliability and a high level of performance standards.

Bacteriological examination is an essential screening procedure for all patients in whom the diagnosis of leprosy is suggestive after a detailed clinical examination. It assists in: 1.

The diagnosis of leprosy; 2. The classification of leprosy; 3. Monitoring of the response to treatment in skin smear positive patients; and 4. Excluding the diagnosis of leprosy.

1.

The presence of AFB bacilli confirms the diagnosis of leprosy. A positive slit-skin smear examination is the 3rd cardinal sign of leprosy (Slide 2).

2.

Bacteriological examination is useful in classifying leprosy within the Ridley and Jopling spectrum and between the two treatment groups, namely paucibacillary (PB) or skin smear negative leprosy and, multibacillary (MB) or skin smear positive leprosy.

3.

The monitoring of the response to treatment in MB patients is assisted by periodical, normally annual, skin smear examination. Viable bacilli will disappear within months from the beginning of treatment. The total number of bacilli will progressively decrease and disappear within years.

4.

In endemic areas skin smears should not only be used to prove that the patient is suffering from leprosy, but also to exclude leprosy in patients with multiple skin lesions.

Skin smears should be taken from all patients suspected of suffering from leprosy. Smears are taken from suspected skin lesions and particularly from the most active-looking edge of the lesion and especially in lepromatous leprosy from sites with a high probability of demonstrating AFB. Such sites with the highest probability of demonstrating AFB are the earlobes, forehead, chin, extensor surface of the forearms, dorsal surface of the fingers, buttocks and extensor surface of knees.

The slit and scrape method

A fold of skin is picked up between finger and thumb and is squeezed to prevent blood flow (Slide 3). A small incision, 7-8 mm length and 1-2 mm deep, is made into the dermis with a scalpel blade (Slide 4). The blade is then turned through 90 degrees and used to scrape the cut surface of the tissue (Slide 5). Care has to be taken to avoid blood mixing

with the smear. The juice obtained is smeared onto a slide (Slide 6) with standard thickness and diameter and, allowed to dry. The slide is then “gently” flamed to fix the smear.

Staining and reading the smears

Smears are stained by Ziehl-Neelsen’s method. After staining, slides are examined using a 100 x oil immersion lens. Bacilli are seen as red rods against a blue background. Living (viable) leprosy bacilli appear uniformly stained; they are described as solid-staining or “solids” (S) bacilli. Dead leprosy bacilli, that stain irregularly, are described as fragmented (F) and granular (G). (Slides 8, 9)

The total number of the bacilli is recorded as the bacterial index (BI). The percentage of solid-staining bacilli is the morphological index (MI) (Slides 10 and 11). Variations of the BI along the spectrum are reported in Part I., Slide 22.

Part III. Diagnosis and the clinical spectrum of leprosy

1. Diagnosis and the clinical spectrum of leprosy

Systematically searching for the 3 cardinal signs will allow the diagnosis of leprosy in most of the cases but, 3 concepts need to be explained:- firstly the cardinal signs are just part of the vast clinical picture of leprosy; secondly their relevance is in relation to the clinical spectrum of the disease and, therefore with its classification; thirdly there are a few, mostly early, leprosy cases where none of the cardinal signs is present.

The cardinal signs are just part of the vast clinical picture of leprosy.

The reader will be able to conveniently cover the vast clinical picture of leprosy after studying the following subjects: - history taking, physical examination, classification, reactions and nerve damage and, disabilities. Herewith we deal with the signs and symptoms of the disease that are relevant to the 3 cardinal signs. Other important signs and symptoms are mentioned only.

The relevance of the cardinal signs is related to the clinical spectrum of the disease and, therefore to its classification.

Knowledge of the classification of the disease has to guide the physician or the paramedical worker during the assessment of the patient. In tuberculoid (TT) leprosy the slit-skin smear examination is negative. In borderline tuberculoid (BT) leprosy slit-skin smear examination is normally negative but, sometimes it may be positive (Part I. Introduction Slide 22.). In these (TT and BT) forms of leprosy the diagnosis should be based on the presence of loss of sensation in skin lesions and/or enlarged peripheral nerves. In mid borderline (BB) leprosy the slit-skin smear examination is normally positive, sensation on skin lesions may be preserved or lost. Here the diagnosis can be based upon all of the 3 cardinal signs. In borderline lepromatous (BL) leprosy slit-skin smear examination is positive, sensation on skin lesions may be preserved or lost. Here the diagnosis can be based upon all of the 3 cardinal signs. In lepromatous (LL) leprosy slit-

skin smear examination is positive but sensation on skin lesions is often preserved. Here the diagnosis is based on positive slit-skin smear examination and or enlarged peripheral nerves.

There are leprosy cases where none of the cardinal signs is present.

In the indeterminate (I) form of leprosy the 3 cardinal signs are negative. In this form of the disease diagnosis is based on the development of the lesions over time and on histopathology. In pure neural leprosy all the 3 signs may be absent.

2. When diagnosis cannot be made during the first examination

If a diagnosis cannot be made during the first examination, the patient may be asked to come back in 3 months time or earlier, in case the skin lesion increases in size or, if more lesions appear. On first examination, place, size and appearance of the lesions should be noted down carefully. With no signs of a reaction or peripheral nerve damage, the diagnosis and the beginning of treatment can wait. However signs of a reaction and peripheral nerve damage always have to be treated as an emergency.

Although the majority of leprosy patients have straightforward skin lesions which are easily visible, experienced workers know that there is a great variety in the skin lesions of leprosy. Some skin lesions are not well defined and are difficult to distinguish from normal skin: in these cases the other symptoms and signs become important. The most difficult cases to diagnose are those of patients who present one or two pale patches, or with very vague skin patches or diffuse infiltration (Slide 11.) without loss of sensation or other signs of leprosy (and no skin smear services available). In these cases, there are three options:

1. refer: know where to refer cases that are difficult to diagnose; discuss cases with colleagues who have experience in managing leprosy;
2. consider the possibility of another skin disease and treat appropriately;
3. wait 3 – 6 months and review the skin lesions again; if it really is leprosy, loss of sensation may now be found and a diagnosis can be made. Always advice the patient to come back earlier if there are significant changes!

Some forms of leprosy like pure neural leprosy and indeterminate leprosy may require biopsy and histopathology to make the diagnosis. In indeterminate leprosy carefully describe and record the lesion and see the patient back in 3 months. If it is gone or changed place it is not leprosy. If it enlarged it could be leprosy and the cardinal signs should be looked for again. It can be done in this way because an indeterminate leprosy patient is most likely not infectious and, leprosy develops slowly.

Many children from leprosy families have suspect dyschromic patches with no sensory change and no signs of large nerves. Encourage the parents to bring them back for review in about 3 months; it is better reviewing them in person, not just told to return if worse.

3. Clinical cases (Grace Warren)

Case 1. Not leprosy

A small boy brought in with a hypopigmented patch on the face. He was malnourished, was anaemic and had worms and other infections. He had had contact with active leprosy but it was decided that the face lesions were due to malnutrition. He was given intensive treatment with Multivitamins and iron and seen again in 3 months time with a complete change in his condition; no suggestion of leprosy.

Case 2. Lepromatous (LL) leprosy

A girl of about 6 years old arrived with what the mother said were many insect bites on her abdomen, that did not go away as expected. She was light skinned and the abdomen showed many slightly erythematous spots about 1 cm across. They were not itchy, no loss of feeling or altered sensory perception, certainly no large nerves but when one looked carefully one realised that some "SPOTS" were already infiltrated. Slit-skin smear examination was positive and allowed diagnosis. Biopsy confirmed they were indeed LL leprosy lesions.

Case 3. Early lepromatous (LL) leprosy

A boy of about 10 with an olive coloured skin, was brought in with what was said to be tinea versicolor of neck and chest but, on examination of the rest of the body other lesions were found with mild infiltration (Slide 8.). Lesions caused no discomfort and, the patient did not feel sick; they were “just there”. There were no signs of nerve damage or large nerves but there were many bacilli in some lesions, this proved it was leprosy not tinea.

Case 4. Leprosy (BB-BL)

A teenaged girl was brought in because her arms presented many ulcers and post infection scars but not really definite lesions with edges (See slide 13.). On testing it was obvious that ulcers were the result of burns as she could not feel heat and cold. Then, we discovered her grandmother had LL leprosy and the child was highly positive for AFB. There were no definite skin lesions (some hypopigmentation was present on the forearm), no really palpable large or hard nerves but, there were important sensory changes. In this case diagnosis was based on the presence of bacilli and loss of heat and cold sensation.

Case 5. Pure neural leprosy

An Indian male, in his mid twenties, seen in Australia about 9 months after leaving India. He presented at a hand clinic, with a left ulnar clawed hand, of 12 months duration. The surgeon did a nerve graft of the arm interosseous nerve to the ulnar at wrist and was surprised when the piece of removed nerve came back as leprosy. Later, I could detect a definitely firm, slightly large nerve crossing the back of the 5TH metacarpal. Other nerves:- well, some were suspiciously firm and/or large but, none grossly enlarged, I assume they were not examined. He had no skin lesions and there were no bacilli in the skin. In this case diagnosis could have been based on peripheral nerve enlargement and damage (and on biopsy).

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