Histopathology and Clinico-histopathological correlation in Hansen’s disease

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DOI : 10.5455/jrmds.2014217

ABSTRACT

Background and Objectives: Leprosy (Hansen’s disease) still continues to be an important public health problem. The present study was undertaken to study the histopathological features of leprosy in skin biopsies and to categorize them into various types based on microscopy, bacterial index to correlate with clinical presentations whenever possible.

Methods: Skin biopsies after adequate fixation in 10% formalin, were routinely processed and paraffin embedded sections of 5 micron thickness were stained with Haematoxylin and Eosin stain and were studied microscopically.

Results: 45 skin biopsies were obtained from patients with age range of 7-78 years, majority were in 3rd decade, with male to female ratio of 1:1.1. Borderline Tuberculoid (BT) was the commonest type of leprosy (42%). Most common clinical feature was loss of sensation. Atrophic epidermis and grenz zone were more common in Lepromatous Leprosy (LL), HL (Histoid Leprosy) & Borderline Lepromatous (BL). Majority (55.6%) of biopsies were of paucibacillary type and rest (44.4%) were of multibacillary type. Clinico-histopathological correlation was observed in 34 biopsies (75.5%).

Interpretation and conclusion: For accurate diagnosis, correlation of clinical and histopathological features along with bacterial index appears to be more useful than considering any of the single parameters alone.

Keywords: Leprosy, histopathology

INTRODUCTION

Leprosy is one of the leading causes of physical disabilities contributing to intense social stigma resulting in human discrimination. This chronic infectious disease caused by Mycobacterium Leprae principally affects skin and peripheral nerves; it also involves muscles, eyes, bones, testis and internal organs [1]. The clinical manifestations are varied ranging from an insignificant skin lesion to extensive disease causing profound disability/deformities [2].

Histopathological study of leprosy is very important in understanding the disease, its varied manifestations and complications. Early accurate diagnosis is required for the correct and adequate treatment. So, clinico-histopathological correlation is extremely important in management.

Exact typing of leprosy is sometimes clinically not possible. Also, the poor results obtained by slit skin smear lead to false negative diagnosis. To prevent this, histopathological examination should be done in all suspected cases.

This study was undertaken to know the histopathological features of leprosy in skin biopsies, to categorize these into various types based on microscopy, bacillary index and to correlate with clinical presentations whenever possible.

MATERIAL AND METHODS

The present study was undertaken in Department of Pathology, Government Medical College, Bhavnagar, from October 2012 to August 2013.
Source of Data:

Materials for the study consisted of skin biopsies obtained from patients clinically diagnosed as new cases of leprosy or old cases not responding to therapy, who attended either Dermatology OPD or leprosy clinics of Sir T. Hospital, Bhavnagar. Those not clinically diagnosed as Leprosy were excluded. The sample size was thus limited.

Method of Collection of Data:

Skin biopsies for the study were obtained by punch biopsy by the Dermatologist after taking written consent from the patient (by Dermatologist) and sent to the Department of Pathology in 10% formalin. After adequate fixation for about 8-12 hours, the biopsies were submitted in toto for routine processing, following which the paraffin embedded sections of 5μ thickness were stained with Haematoxylin and Eosin for morphological analysis and studied to observe the various changes that occurred in the epidermis, dermis, neurovascular bundles and adnexae. Then Bacillary index (BI) was calculated using an oil immersion objective (x100) according to the following scale [3]:

1+ 1 to 10 bacilli per 100 OIF – Examine 100 OIF
2+ 1 to 10 bacilli per 10 OIF – Examine 100 OIF
3+ 1 to 10 bacilli per OIF – Examine 25 OIF
4+ 10 to 100 bacilli per OIF – Examine 25 OIF
5+ 100 to 1000 bacilli Per OIF – Examine 25 OIF
6+ more than 1000 per bacilli per OIF – Examine 25 OIF

(Note: OIF=Oil Immersion Field)

Cases with BI 0 were termed paucibacillary and those with BI 1+ or above were termed multibacillary.

The diagnosis of leprosy was confirmed and classified according to Ridley and Jopling classification. Clinico-histopathological correlation was done.

RESULTS

Patients’ age ranged from 7 to 78 years. 28 patients (62%) were in 3rd - 5th decade, 1 (2%) in 1st decade, 5(11%) in 2nd decade, 4 (8.9%) in 6th decade and 7 (15.6%) were above 60 years of age.

There were 21 (46.7%) male and 24 (53.3%) female patients, with male to female ratio of 1:1.1.

Clinically, 41 showed anaesthesia (loss of sensation), 39 hypopigmented skin lesions, 27 nerve thickening, 11 erythematous skin lesions, 4 combinations of lesions, 2 nodules & 2 trophic ulcers.

Table 2 shows that 25 cases (55.6%) were paucibacillary and 20(44.4%) multibacillary.

Table 3 shows that BB, IL and BT showed maximum clinico-histopathological correlation followed by BL. 50% cases of HL & LL showed correlation while TT showed minimum correlation.
Table 1: Histopathological Changes observed in Epidermis & Dermis in Leprosy

<table>
<thead>
<tr>
<th>Epidermis</th>
<th>Cases No.</th>
<th>TT(%)</th>
<th>BT(%)</th>
<th>BB(%)</th>
<th>BL(%)</th>
<th>LL(%)</th>
<th>IL(%)</th>
<th>HL(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrophy</td>
<td>1(100)</td>
<td>10(52.6)</td>
<td>1(100)</td>
<td>5(100)</td>
<td>8(100)</td>
<td>2(25)</td>
<td>3(100)</td>
<td>30(66.7)</td>
<td></td>
</tr>
<tr>
<td>Ulceration</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td></td>
</tr>
<tr>
<td>Unremarkable</td>
<td>0(0)</td>
<td>8(42.1)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>6(75)</td>
<td>0(0)</td>
<td>14(31.1)</td>
<td></td>
</tr>
<tr>
<td>Basement membrane erosion</td>
<td>0(0)</td>
<td>1(5.3)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>1(2.2)</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Dermal changes Type</th>
<th>Cases No.</th>
<th>TT(%)</th>
<th>BT(%)</th>
<th>BB(%)</th>
<th>BL(%)</th>
<th>LL(%)</th>
<th>IL(%)</th>
<th>HL(%)</th>
<th>Total(%)</th>
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<tbody>
<tr>
<td>Epithelioid Granuloma</td>
<td>1(100)</td>
<td>9(47.4)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>10(22.2)</td>
<td></td>
</tr>
<tr>
<td>Giant cells</td>
<td>1(100)</td>
<td>1(5.3)</td>
<td>1(100)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>3(6.7)</td>
<td></td>
</tr>
<tr>
<td>Periappendageal Lymphocytes</td>
<td>0(0)</td>
<td>8(42.1)</td>
<td>0(0)</td>
<td>1(20)</td>
<td>1(12.5)</td>
<td>5(62.5)</td>
<td>0(0)</td>
<td>15(33.3)</td>
<td></td>
</tr>
<tr>
<td>Perivascular Lymphocytes</td>
<td>0(0)</td>
<td>7(36.8)</td>
<td>0(0)</td>
<td>1(20)</td>
<td>1(12.5)</td>
<td>8(100)</td>
<td>0(0)</td>
<td>17(37.8)</td>
<td></td>
</tr>
<tr>
<td>Perineural Lymphocytes</td>
<td>1(100)</td>
<td>7(36.8)</td>
<td>0(0)</td>
<td>1(20)</td>
<td>1(12.5)</td>
<td>1(12.5)</td>
<td>0(0)</td>
<td>11(24.4)</td>
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</tr>
<tr>
<td>Periappendageal Lymphohistiocytes</td>
<td>1(100)</td>
<td>16(84.2)</td>
<td>1(100)</td>
<td>2(40)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>20(44.4)</td>
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<tr>
<td>Perivascular Lymphohistiocytes</td>
<td>0(0)</td>
<td>14(73.7)</td>
<td>0(0)</td>
<td>2(40)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>16(35.5)</td>
<td></td>
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<tr>
<td>Perineural Lymphohistiocytes</td>
<td>1(100)</td>
<td>16(84.2)</td>
<td>1(100)</td>
<td>2(40)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
<td>21(46.7)</td>
<td></td>
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<tr>
<td>Macrophages (periappendageal, perivascular, perineural) foamy &amp; non foamy</td>
<td>0(0)</td>
<td>3(15.8)</td>
<td>1(100)</td>
<td>5(100)</td>
<td>8(100)</td>
<td>0(0)</td>
<td>3(100)</td>
<td>20(44.4)</td>
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<tr>
<td>Grenz Zone</td>
<td>0(0)</td>
<td>7(36.8)</td>
<td>1(100)</td>
<td>4(80)</td>
<td>8(100)</td>
<td>0(0)</td>
<td>3(100)</td>
<td>23(51.1)</td>
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Table 2: Bacillary Index in skin slit smear examination

<table>
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<tr>
<th>Type</th>
<th>No.</th>
<th>Paucibacillary(PB)</th>
<th>Multibacillary (MB)</th>
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<tr>
<td></td>
<td></td>
<td>1+</td>
<td>2+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3+</td>
<td>4+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5+</td>
<td>6+</td>
</tr>
<tr>
<td>TT</td>
<td>1</td>
<td>1 (100%)</td>
<td></td>
</tr>
<tr>
<td>BT</td>
<td>19</td>
<td>1 (5.3%)</td>
<td></td>
</tr>
<tr>
<td>BB</td>
<td>1</td>
<td>1 (100%)</td>
<td></td>
</tr>
<tr>
<td>BL</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LL</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IL</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HL</td>
<td>3</td>
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</table>

Table 3: Clinico-histopathological Correlation

<table>
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<tr>
<th>Clinical diagnosis</th>
<th>TT</th>
<th>BT</th>
<th>BB</th>
<th>BL</th>
<th>LL</th>
<th>IL</th>
<th>HL</th>
<th>aggregate</th>
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<tbody>
<tr>
<td>TT</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>5</td>
<td>8</td>
<td>8</td>
<td>3</td>
<td>33.3%</td>
</tr>
<tr>
<td>BB</td>
<td>17</td>
<td>1</td>
<td>16</td>
<td>5</td>
<td>8</td>
<td>8</td>
<td>3</td>
<td>94.1%</td>
</tr>
<tr>
<td>BL</td>
<td>8</td>
<td>1</td>
<td>5</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>6</td>
<td>62.5%</td>
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<tr>
<td>LL</td>
<td>8</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>6</td>
<td>50%</td>
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<tr>
<td>IL</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100%</td>
</tr>
<tr>
<td>HL</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>50%</td>
</tr>
</tbody>
</table>
DISCUSSION

Leprosy is a slowly progressive infection caused by Mycobacterium leprae affecting the skin and peripheral nerves. Histopathological examination of skin lesion is the gold standard for accurate diagnosis [4].

During the study period of 11 months in the present study, total 45 skin biopsies were of leprosy.

AGE AND SEX DISTRIBUTION

Leprosy can occur at all ages [5]. In the present study, patients of 20-29 years (3rd decade) were affected most and patients below 9 years were affected least. Similar observations were made by Guha et al [6], Kaur S et al [7], Sehgal et al [8], Murthy et al [9] and Kaur I et al [10]. Variable and long incubation period may be responsible for this age distribution [8].

Generally, leprosy is believed to be commoner in males [11]. This is observed in studies by Sehgal et al [8], Nadkarni et al [12], Murthy et al [9] etc. Several factors influence the sex predominance in endemic areas, mainly the opportunity for contact. Practically no difference is noted when the opportunity for contact remains the same[13]. Male predominance may be because of many factors like industrialization, urbanization and more opportunities for contact in males. Social customs and taboos may account for the smaller number of females reporting for treatment to the hospital[8]. Present study shows almost equal incidence in both sexes. Chaturvedi et al showed similar results[13].

COMPARISON OF CLINICAL FEATURES

Present study showed that Anaesthesia was the commonest clinical feature followed by Hypopigmented skin lesions and nerve thickening. Trophic ulcer was rare. Similar observations were made by Verma et al [14]. Since skin and nerves are the commonest sites of M. leprae infection, signs and symptoms related to skin and nerves were common. In contrast, hypopigmented skin lesions were the most common clinical feature in the study by Murthy NB et al [9].

HISTOPATHOLOGICAL FEATURES OF EPIDERMIS (Table 1)

In the present study, in most of the biopsies (66.7%), epidermis was atrophic; in rest 31.1%, the epidermis was unremarkable. Ulceration & basement membrane erosion were rare.

The location of the granuloma mostly in relation to the deep and mid dermal nerves and neurovascular complexes would account for the somatosensory and autonomic neuropathic manifestations of the disease [15].

Also the granuloma situated in mid dermis or in deep dermis and/or small granuloma, is unlikely to cause epidermal changes and hypopigmented skin lesions. When it reaches the superficial dermis and extends to the epidermis, atrophy of epidermis and development of hypopigmented lesions occur [15].

Figures 2 & 3: Histopathological features of Leprosy

HISTOPATHOLOGICAL FEATURES OF DERMIS (Table 1)

Grenz zone was the commonest feature observed in all the biopsies of LL (100%). It was rare in BT (36.8%) and absent in TT. It is widely recognized as a characteristic of non tuberculoid leprosy[5]. It is not diagnostic of leprosy, but helps in considering the diagnosis of leprosy and its types. The following
criteria were used for diagnosis of various types of Leprosy:

1) TT: Collections of epithelioid cells, many lymphocytes peripheral to the granuloma and/or several large Langhan’s giant cells[5,16], or a very large granulomatous nerve with intact perineurium or caseation in a nerve centre or erosion of epidermis by epithelioid cells. Presence of one or few hypopigmented or erythematous, anaesthetic lesions asymmetrical in distribution, with or without nerve thickening was correlated for accurate diagnosis.

2) BT : Presence of epithelioid cell granuloma which was more diffuse than in TT [3], with few small giant cells and moderate number of lymphocytes often within the granuloma [5]. Presence of many asymmetrically arranged hypopigmented/erythematous/anaesthetic lesions with or without enlargement of nerves was correlated for accurate diagnosis.

3) BB: Features of both TT and LL present [17].

4) IL: Mild non-specific perivasular and periadnexal lymphocytic and histiocytic infiltrate in dermis or thickened deep dermal nerve showing intraneural lymphocytic infiltration [16]. Presence of ill defined, hypopigmented skin lesions were correlated for the proper diagnosis.

In IL the histopathological changes are minimal and may be missed unless the biopsy is adequate, including the entire dermis and part of subcutis[16].

5) BL: Diffuse infiltrates of macrophages, foamy macrophages and few lymphocytes seen involving nerves and appendages [17]. Presence of multiple, ill defined, shiny, asymmetrically arranged skin lesions with or without anaesthesia and nerve thickening were correlated for accurate diagnosis.

6) LL: Diffuse infiltrate of macrophages and foamy cells, with few or no lymphocytes [16,17]. Multiple ill defined shiny, symmetrical skin lesions with anaesthesia and with or without nerve thickening were correlated for accurate diagnosis.

COMPARISON OF HISTOPATHOLOGICAL TYPES

The most commonly encountered type of leprosy was BT (42%), followed by LL, IL, BL, and HL. BB and TT (2% each) were the least encountered types. Borderline group constituted the major spectrum (73%), similar to findings of other authors like Murthy NB et al[9], Verma et al[14], Shenoi et al[18], Ashok Kumar et al[19]. A sizable portion of leprosy patients will be in a continuously changing immunological spectrum, i.e., BT, BB and BL[20]. Also, features of both tuberculoid and lepromatous leprosy can occur in a same section or in serial sections or in different lesions of the same borderline cases. Immunological instability in these borderline cases makes them move in either direction along the borderline spectrum. With treatment, they move towards tuberculoid pole while without treatment they tend to move towards lepromatous pole. Biopsy from a case recognized at an earlier stage may show BT while that from a case recognized at a later stage may show BL[3]. Increased awareness of the people to leprosy owing to national programmes makes them present at an earlier stage to leprosy clinics, which may contribute to increased number of borderline group of leprosy.

BACILLARY INDEX

It was highest in LL types and low in BT types. Jopling also observed that the bacilli are scanty or absent in BT, always present in BB and numerous in BL and LL[3]. It also shows the variation of cell mediated immunity and bacillary load as the spectrum of leprosy moves from tuberculoid pole to lepromatous pole. The present study confirms the same.

In few LL and BL biopsies, bacilli were present in sub-epidermal areas, around hair follicles and appendages and also in the vascular endothelial cells. Morphological index is better appreciated in slit skin smear [21]. In present study, result of slit skin smear is used for bacillary index.

<table>
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</thead>
<tbody>
<tr>
<td>TT</td>
<td>100</td>
<td>77.8</td>
<td>50</td>
<td>30.76</td>
<td>33.3</td>
</tr>
<tr>
<td>BT</td>
<td>40</td>
<td>62.2</td>
<td>77</td>
<td>88.18</td>
<td>94.1</td>
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<tr>
<td>BB</td>
<td>100</td>
<td>20.0</td>
<td>25</td>
<td>12.50</td>
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<tr>
<td>BL</td>
<td>40</td>
<td>14.4</td>
<td>43</td>
<td>32.56</td>
<td>62.5</td>
</tr>
<tr>
<td>LL</td>
<td>83.3</td>
<td>100</td>
<td>91</td>
<td>34.78</td>
<td>50</td>
</tr>
<tr>
<td>IL</td>
<td>-</td>
<td>85.7</td>
<td>35</td>
<td>38.46</td>
<td>100</td>
</tr>
<tr>
<td>HL</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>50</td>
</tr>
</tbody>
</table>

In the present study, it was observed that out of 3 clinically diagnosed TT cases, 1 correlated
histopathologically, the remaining 2 were diagnosed as BT. Similar results were seen by few authors[9], while others showed higher percentage of agreement[14,18,20].

The cellular characteristics in leprosy lesions are related to the immunological status of the patient. The widely accepted Ridley and Jopling classification is based on clinical, histopathological and immunological features. Simultaneously clinicians also have adopted Ridley-Jopling nomenclature and at present, the clinical diagnosis is made along the lines of this classification, even when a histopathological examination has not been done; this may lead to discordance between clinical and histopathological diagnosis [20].

TT is slightly different from BT leprosy, both clinically and histopathologically. The line of demarcation often overlaps. Many cases diagnosed clinically as TT have histological evidence of BT [20]. TT is the infection on a knife edge, poised between progression and spontaneous healing. Though the borderline leprosy group is common and BT in particular is the commonest type of leprosy encountered in the world, many disagree with it. Failure to appreciate this fact is due to failure to recognize the exact clinical and histological features and the tendency to classify BT as TT [3].

A sizable portion of leprosy cases are in a continuously changing immunological spectrum. In some early cases, clinical signs and symptoms may precede the presently known characteristic tissue changes and vice versa. If biopsy was taken at an early stage, there are chances of discordance between the clinical and histopathological observation [20].

Some of the lepromatous patients may also show a few lesions of higher spectrum like asymmetrical neuritis, overlapping in histology and variation in smear as well as tissue BI from site to site. These cases can be subclassified as subpolar lepromatous leprosy (LLs). Separation of BL from LLs is very difficult [22], while diagnosing LL, clinical features were also correlated along with strict criteria of paucity in lymphocytes.

Indeterminate leprosy cases appear to be problematic due to the non specific histology of their lesions, variable factors such as nature and depth of biopsy, the quality of section and number of acid fast stained sections examined etc. and inter-observer variations, both clinically and histopathologically [20].

The definitive diagnosis of IL depends on demonstration of nerve lesions and acid fast bacilli, but can be diagnosed even without finding a single bacillus, if clinical and histopathological features are suggestive, especially in endemic areas[20]. IL is an early and transitory stage of leprosy found in persons, whose immunological status is yet to be determined.

Nervous system plays an important role in modulation of the inflammatory response. In areas where modulation has favourably affected the host defense and repair mechanisms, no evidence of disease results. In other areas with different grades of modulation affecting the host defensive response unfavourably, different types of clinicopathological pictures are seen. This concept explains the disagreement in clinical and histopathological classification observed in some cases of leprosy [23].

Clinical diagnosis of early leprosy lesions offer difficulties even to experienced dermatologists. A definitive diagnosis may be possible by histopathological examination [24]. Histopathological features indicate the accurate response of the tissue, while the clinical features indicate only the gross morphology of the lesions which is due to underlying pathological changes.

**IMPLICATIONS OF HISTOPATHOLOGY IN RESOLVING LEPROSY:**

**In Paucibacillary Leprosy:**

The administration of antileprosy drugs kills the organisms thus stopping the multiplication of M. leprae and the continuation of the release of their antigens into the tissues [25].

The granulomatous inflammation degrades the antigens and helps to absorb them resulting in resolution and disappearance of the granuloma. There is no fibrous replacement of the skin adnexa and the dermal collagen tissue has been destroyed during the inflammatory process. Therefore, the healed skin lesions look atrophied and wrinkled. Nerves undergo perineural and intraneural fibrosis. It is at these sites M. leprae get buried and may serve as a focus for relapse [25].
In multibacillary leprosy:

During anti leprosy therapy, as the LL lesions resolve, increasing number of macrophages with pink granular cytoplasm develops intracellular vacuoles and undergoes foamy change until finally all the macrophages in the granuloma are foamy (vacuolated). In some cases lymphocytes and plasma cells infiltrate the granuloma. Invariably granuloma fraction gradually decreases. LL granulomas ordinarily resolve with no residual fibrosis and scar [25].

The Schwann cells in dermal nerves may show foamy change. There is reactive proliferation of the perineurium. As the disease resolves there is fibrous replacement of perineurium and the nerve parenchyma, which finally gets hyalinized.

AFB (Acid fast bacilli) in the granuloma develop granularity and within one to three months of effective therapy all bacilli become granulated. AFB in nerves and wall of blood vessels may persist longer than those inside macrophage. Small collections of foam cells are seen in skin biopsies of inactive patients even years later. Mild non specific chronic inflammation can persist around adnexa in resolved LL lesions [25].

To conclude, the present study emphasizes the importance of histopathological examination and bacillary index in the management of Leprosy. Increased number of borderline cases detected point towards the effectiveness of the National Programmes. However, the sample size was small as the study was conducted in a short duration and in selected cases. Therefore, the results cannot be extrapolated.

CONCLUSION

As there can be some degree of overlapping among different types of leprosy both clinically and histopathologically, clinico-histopathological correlation along with bacteriological index appears more useful for accurate typing of leprosy than considering any of the single parameters alone.

REFERENCES


**How to cite this article:** Suri SK, Iyer RR, Patel DU, Bandil S, Baxi S. Histopathology and Clinico-histopathological correlation in Hansen's disease. J Res Med Den Sci 2014;2(1):37-44

**Source of Support:** None

**Conflict of Interest:** None declared