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A Histopathologic Study of Renal Biopsies in Fifty Cases of Leprosy[1]

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Although clinical manifestations of renal involvement in leprosy are often observed, the exact nature of pathologic lesions with special reference to specific lesions of leprosy in the kidney which might be responsible for such manifestations is little noted. Most authors in studies of kidney tissue in leprosy have reported amyloidosis in varying incidence, and nephritis of all types such as chronic pyelonephritis, interstitial nephritis, chronic diffuse glomerulonephritis in varying frequencies ([2, 6, 7, 10, 12, 13, 15–17, 19]). Specific lesions, i.e., lepromas, have been reported only by Powell and Swan ([15]) in 2 of their 15 necropsies. Sainani and Rao ([16]) described the presence of leproma-like lesions composed of mononuclear cells with vacuolated cytoplasm without demonstrable acid-fast bacilli in 1 of their 60 cases and suggested greater possibility of development of such lepromas in cases of longer duration. Most of the other workers in this country as well as in other countries have failed to observe any such lesions ([1, 2, 5, 10, 11]). In India, 11.2% of the cases of leprosy have been reported as dying of renal failure ([2, 12]).

In the present study, histopathologic changes in renal biopsies in cases of leprosy, including lepromatous and nonlepromatous types, have been studied with the special interest of discovering if any specific lesions such as lepromas or granulomas are seen in kidneys, as has been reported for other tissues of the body such as liver, striated muscle and lymph nodes.

MATERIALS AND METHODS

Renal biopsy tissues from 50 cases of leprosy, including 45 cases of lepromatous type and 5 cases of tuberculoid type, attending the skin, V.D., and leprosy department of the Medical College Hospital in Jabalpur during the period 1974-1975, have been studied in detail.

Thirty-five of the patients were males and 15 were females. They ranged in age from 7 to 70 years, 41 patients being in the age period of 21 to 50 years. Duration of illness ranged from seven days to four years in the tuberculoid group, and three months to twenty years in the lepromatous group. Diagnosis of leprosy was made in all cases on the basis of clinicobacteriologic criteria and was confirmed by histopathologic examination of skin biopsies. Renal tissue, obtained by biopsy in all cases, was studied for histopathologic changes and for the presence of acid-fast bacilli, employing H & E stain, Congo red stain for amyloid, Masson's trichrome stain and Ziehl-Neelsen stain.

OBSERVATIONS

Histopathologic features observed in the renal biopsies of 50 cases are recorded in Table 1.

Specific lesions of leprosy such as lepromas or granulomas or acid-fast bacilli could not be detected in renal tissue from any of the 50 cases of leprosy, whether of lepromatous or tuberculoid type. Further, none of these cases showed evidence of amyloidosis. The significant pathologic changes noted in a portion of the cases consisted of increased cellularity, hyalinization and congestion of glomeruli, periglomerular fibrosis, albuminoid degeneration, atrophy and hyalinization of the tubules, and a certain amount of fibrosis with chronic inflammatory cell infiltrate in the interstitial tissue and occasional arteriosclerotic changes in the blood vessels. On the basis of these changes, the definite histopathologic diagnoses that could be made are shown in Table 2. Only 18 cases (40%) of lepromatous leprosy and 2 cases of tuberculoid leprosy showed significant pathologic lesions, suggesting chronic pyelonephritis (12%), acute interstitial nephritis (6%), chron-

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TABLE 1. *Histopathologic features of renal biopsies in 50 cases of leprosy.*

Histopathologic changes	No. of cases	Percent
I. Changes in glomeruli		
1. Cellularity of the capillary tuft		
a. mild	15	30
b. moderate	30	60
c. severe	4	8
2. Congestion	6	12
3. Hyalinization of glomerulus	6	12
4. Periglomerular fibrosis	8	16
II. Changes in tubules		
1. Albuminoid degeneration	45	90
2. Atrophy	6	12
3. Tubular hyaline-like cast	6	12
III. Changes in the interstitial tissue		
1. Fibrosis		
a. mild	8	16
b. marked	11	22
2. Cellular infiltration (diffuse and local) of lymphocytes, plasma cells and less frequently neutrophils.		
a. mild	12	24
b. moderate	10	20
c. severe	9	18
IV. Changes in the vessels: arteriosclerosis	7	14
V. Amyloid deposition	Nil	—
VI. Acid-fast bacilli	Nil	—

TABLE 2. *Histopathologic diagnoses in 50 cases of leprosy.*

Histopathologic diagnosis	No. of cases	Percent
Lepromatous leprosy (45 cases):		
1. No significant pathologic lesions detected	27	54
2. Significant pathologic lesions	18	36
a. Chronic pyelonephritis	6	
b. Acute interstitial nephritis	3	
c. Chronic interstitial nephritis	6	
d. Chronic diffuse glomerulonephritis	3	
Nonlepromatous leprosy (5 cases):		
1. No significant pathologic lesions detected	3	6
2. Chronic interstitial nephritis	2	4

ic interstitial nephritis (16%) and chronic diffuse glomerulonephritis (6%). The remainder (60%) did not show any significant pathologic lesions.

DISCUSSION

The present study of renal biopsies from 50 cases of leprosy, including 45 lepromatous and 5 tuberculoid, failed to demonstrate any specific lesion of leprosy such as a granuloma or a leproma or the presence of acid-fast bacilli. These observations are in accordance with those reported by earlier workers such as Kean and Childress [11], Desikan and Job [2], Furata and Ozaki [5], Johny and Karat [10], and Bernard and Vanquez [1], except for the occasional cases of leproma in kidney reported by Mitsuda and Ogawa [14], Powell and Swan [15] and Sainani and Rao [16]. It raises an important question as to why, when

lepromas can be identified in other tissues such as the liver ([20-22]), striated muscles ([9]) and lymph nodes ([3, 18, 19]), similar lepromas do not occur in kidney tissues in cases of leprosy of either type and of any duration when bacteremia in leprosy is held to be responsible for the lepromas and granulomas appearing in various tissues. Why the kidney escapes remains a problem. It has not been determined if this is due to greater immunologic resistance of renal tissue or to the possibility that renal tissue constitutes an unfavorable site for the settling and multiplication of acid-fast bacilli due to physiochemical factors such as the rapid flow of blood through the kidneys. Drutz and Gutman ([4]) also regarded the kidney to be highly resistant to leprous affliction.

No amyloid deposit could be demonstrated in these 50 cases of leprosy, although amyloid deposit in kidneys in leprosy has been reported by some workers ([1, 2, 10, 12, 16, 19]). In this institution, amyloid deposit also could not be demonstrated early in liver ([8]) and skeletal muscle tissue ([9]) from cases of leprosy. It is again rather difficult to explain the absence of amyloid in the tissues of our cases. It has been reported by some authors in this country despite the fact that the incidence of amyloidosis in this country, in general, is considered to be lower than in Western countries. Only nephritis of different types could be recognized in a portion of the cases in this study. The possible role of leprosy itself in the causation of these diseases is debatable. Either leprosy does not influence their occurrence and they may be occurring in those patients as much as in nonleprosy individuals, or cases of leprosy suffer from an altered immunologic state, which renders them more prone to such renal lesions. Although the former postulate cannot be ruled out or established, we feel the latter might be playing some role in producing such nonspecific renal lesions in leprosy patients, with resultant clinical manifestations, despite the absence of any specific lesions of leprosy.[3]

[3]The authors agree that the reaction of renal tissues to *M. leprae* is different from that of other tissues, but have postulated that it could be due to various local immunologic or physiochemical factors. In respect to renal tuberculosis, however, they have observed very extensive inflammatory reaction and tubercle formation in renal tissues in the past. Involvement of liver has been noted more often in leprosy than in tuberculosis in terms of specific granulomatous lesions.

SUMMARY

Renal biopsies from 50 cases of leprosy, including 45 cases of lepromatous and 5 cases of tuberculoid, have been studied in detail histopathologically with special reference to any specific leprosy lesion such as the presence of leproma or granuloma, the presence of acid-fast bacilli and the occurrence of amyloid deposit. Leproma or granuloma, acid-fast bacilli and amyloid deposit could not be detected in any of these cases. Pathologic features of nephritis of various types were seen in only 40% of cases. Similar observations made by previous authors have been reviewed.

The question is raised as to why kidney tissue should escape from developing specific leprosy lesions in either type of leprosy when other tissues such as liver, striated muscles and lymph nodes are known to develop such lesions. A greater immunologic resistance of the renal tissue to lepra bacilli or local physiochemical factors which may render renal tissue an unfavorable site for the settling and multiplication of lepra bacilli are considered as possible related factors.

RESUMEN

Se hizo el estudio histopatológico en biopsias renales de 50 casos de lepra (45 lepromatosos y 5 tuberculoides). Se tuvo especial cuidado en buscar lesiones específicas de la lepra. En ninguno de los casos se pudo demostrar la presencia de lepromas o granulomas, de bacilos resistentes al alcohol ácido o de depósitos de amiloide. Sólo en el 40% de los casos se observaron evidencias patológicas de nefritis de varios tipos. Otros autores han hecho observaciones similares.

Se plantea la pregunta de porqué el tejido renal escapa al desarrollo de lesiones específicas de la lepra (en cualquier tipo de lepra) mientras que tales lesiones se presentan con frecuencia en el hígado, el músculo estriado y los ganglios linfáticos. Posiblemente esto puede explicarse por una mayor resistencia immunológica del tejido renal al bacilo de la lepra, o por factores fisicoquímicos locales que pueden hacer del tejido renal un sitio desfavorable para el establecimiento y multiplicación del bacilo de la lepra.

RÉSUMÉ

On a procédé à une étude histopathologique détaillée de biopsies rénales provenant de 50 cas de lèpre, parmi lesquels on comptait 45 cas de lèpre lépromateuse et 5 cas de lèpre tuberculoïde. Les observations ont été particulièrement mises

en rapport avec toutes lésions lépreuses spécifique telles que l'existence de lépromes ou de granulomes, la présence de bacilles acido-résistants, et l'apparition d'un dépôt amyloïde. Chez aucun de ces cas on n'a pu mettre en évidence de lépromes ou de granulomes, de bacilles acido-résistants, ou de dépots amyloïdes. Ce n'est que chez 40 pour cent des cas seulement que l'on a pu déceler des caractéristiques pathologiques de néphrites de divers types. Les observations analogues faites antérieurement par divers auteurs ont été passées en revue.

On s'est posé la question de savoir pourquoi le tissu rénal échapperait au développement de lésions lépreuses spécifiques, de l'un ou l'autre type de lèpre, alors que d'autres tissus, tels que le foie, les muscles striés et les nodules lymphatiques, sont connus pour développer de telles lésions. On a considéré comme explication possible l'existence d'une plus grande résistance immunologique du tissu rénal à l'égard des bacilles de la lèpre, ou des facteurs physicochimiques locaux, qui pourraient faire du tissu rénal un lieu peu favorable à l'implantation et à la multiplication des bacilles de la lèpre.

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REFERENCES

1. BERNARD, J. C. and VAZQUEZ, C. A. J. Visceral lesions in leprosy. Int. J. Lepr. **41** (1973) 94-101.
2. DESIKAN, K. V. and JOB, C. K. A review of postmortem findings in 37 cases of leprosy. Int. J. Lepr. **36** (1968) 32-44.
3. DESIKAN, K. V. and JOB, C. K. Leprous lymphadenitis. Demonstration of tuberculoid lesions. Int. J. Lepr. **34** (1966) 147-154.
4. DRUTZ, D. J. and GUTMAN, R. A. Renal manifestation of leprosy: glomerulonephritis a complication of *erythema nodosum leprosum*. Am. J. Trop. Med. Hyg. **22** (1973) 496-502.
5. FURATA, M. and OZAKI, M. Calcium oxalate crystals in the kidney and thyroid of leprosy patients. Int. J. Lepr. **38** (1970) 286-293.
6. GLENNER, G. G., TERRY, W., HARADA, M., ISERSKY, C., and PAGE, D. Amyloid fibril proteins: proof of homology with immunoglobulin light chains by sequence analyses. Science **172** (1971) 1150-1151.
7. GREBSTALD, H. and SWEN, L. Genitourinary lesions in leprosy with reference to the problem of atrophy of testis. JAMA **1149** (1952) 1287-1291.
8. GUPTA, J. C., GUPTA, D. K. and GUPTA, A. K. Hepatic lesions in leprosy. Lepr. India **46** (1974) 226-233.
9. GUPTA, J. C., JESUPADAM, T., GUPTA, M. C. and GUPTA, D. K. Changes in striated muscle in leprosy. Int. J. Lepr. **43** (1975) 348-355.
10. JOHNY, K. V. and KARAT, A. B. A. Renal biopsy studies in cases of leprosy. J. Assn. Phys. India **19** (1971) 117-118.
11. KEAN, B. H. and CHILDRESS, M. E. A summary of 103 autopsies on leprosy patients on the Isthmus of Panama. Int. J. Lepr. **10** (1942) 51-59.
12. KRISHNAMURTHI, S. and JOB, C. K. Secondary amyloidosis in leprosy. Int. J. Lepr. **34** (1966) 155-158.
13. LEDINGHAM, J. M. *The Medical Annual*, Bristol: John Wright & Sons, Ltd., 1972, pp 384-388.
14. MITSUDA, K. and OGAWA, M. Study of 150 autopsies on cases of leprosy. Int. J. Lepr. **5** (1937) 53-60.
15. POWELL, C. S. and SWAN, L. L. Leprosy: Pathologic changes observed in 50 consecutive necropsies. Am. J. Pathol. **31** (1955) 1131-1147.
16. SAINANI, G. S. and NARAYAN RAO, K. V. Renal changes in leprosy. J. Assn. Phys. India **22** (1974) 659-664.
17. SATYANARAYANA, B. V., RAJU, P. S., KUMARI, K. R. and REDDY, C. R. R. M. Amyloidosis in leprosy. Int. J. Lepr. **40** (1972) 278-280.
18. SHARMA, K. D. and SHRIVASTAV, J. P. Lymph nodes in leprosy. Int. J. Lepr. **26** (1958) 41-50.
19. SHUTTLEWORTH, J. S. and ROSS, S. H. Secondary amyloidosis in leprosy. Ann. Intern. Med. **45** (1956) 23-38.
20. SOOD, V. K. and GRUEBER, H. L. E. Correlation of histopathologic changes in the liver and bone marrow of leprosy patients. Int. J. Lepr. **37** (1969) 28-39.
21. THOMAS, E., RAMACHANDRAN, P., THANGAVELU, M. and CHARY, A. Liver changes in cases of leprosy. Indian J. Pathol. Bacteriol. **9** (1966) 331-336.
22. VERGHESE, A. and JOB, C. K. Correlation of liver function with the pathology of the liver in leprosy. Int. J. Lepr. **33** (1965) 342-348.