A Review of Postmortem Findings in 37 Cases of Leprosy [1]

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There are very few publications in English literature on autopsy findings in leprosy. A detailed description of the pathology of leprosy, based on autopsy findings in 125 cases, was given for the first time by Hansen and Looft ([11]) in 1895. Mitsuda and Ogawa ([16]) published a report in 1937 on gross findings in 150 autopsies. By that time Mitsuda alone had performed autopsies on more than 1,000 cases of leprosy in a period of 40 years. His observations were published in a number of papers in the Japanese language. Illustrations from all his earlier works were compiled as an atlas in English and published in 1952 ([15]). Kean and Childress ([13]), in 1942, published their findings in 103 necropsies in Panama. From the National Leprosarium, Carville, Louisiana, there are two publications, one by Black ([2]) in 1938 on 75 necropsies and the other by Powell and Swan ([17]) in 1955 on 50 necropsies.

There are no autopsy reports from India. A study of the disease in different parts of the world would be very interesting, as it is well known that racial factors influence susceptibility to leprosy as well as manifestations of the disease ([1, 8]).

MATERIALS AND METHODS

All the cases studied on which this report is based, were autopsied in the Christian Medical College Hospital, Vellore, which is a general hospital. From 1941 to the end of 1964, 4,211 autopsies were performed. Among these were 41 leprosy cases. Among these, the tissues in four cases were badly autolyzed and these cases have been deleted from the study. The findings in the remaining 37 cases are presented. Nine of the patients died while being treated in the Christian Medical College Hospital, of complaints other than leprosy. Nineteen were inpatients of the Schieffelin Leprosy Research Sanatorium, Karigiri. The remaining nine died in other institutions and were brought to the Christian Medical College Hospital for autopsy. A full autopsy was performed in 21 cases by the routine technic of the hospital. In the remaining 16 cases, permission was not available for a full postmortem examination. An evisceration technic was therefore adopted, in which the abdominal and thoracic viscera and the testes were removed through an abdominal incision. After gross examination of all the viscera, specimens were taken from representative areas and fixed in 10 per cent neutral formalin. Tissues were processed in the usual way. Paraffin sections 7 microns thick were stained by the hematoxylin and eosin stain. Most of the sections, and in particular those from the liver, spleen, lymph nodes, bone marrow, adrenal, kidney, testes, and skin, were stained by Fite's modification of the Ziehl-Neelsen stain. Congo red and methyl violet stains for amyloid were made on sections from liver, spleen, adrenal and kidney.

OBSERVATIONS

Age, sex, nutritional status and the type of leprosy. The necropsies performed were on leprosy patients in advanced stages of the disease, who ranged in age at the time of death from 20 to 60 years, with an average of 35 years. They were poorly built and undernourished. The average weight of the male patients was 39 kgm., and their average height was 162 cm.

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The disease was of very long duration in most of the cases and all cases showed deformities of limbs of various degrees. The duration of the disease is shown in Table 1. A majority of the cases were of the lepromatous type. The sex and type distributions are shown in Table 2.

The nonlepromatous cases included resolved tuberculoid and polyneuritic types of leprosy. All of them were cases of very long standing, and skin lesions had cleared up in all of them.

TABLE 1. *Duration of leprosy.*

Duration (years)	Number of cases
Less than 5	2
5–10	15
More than 10	20
Total	37

TABLE 2. *Sex and type distribution.*

Type	Male	Female	Total
Lepromatous	29	1	30
Nonlepromatous	5	2	7
Total	34	3	37

Visceral leprous lesions. The nonlepromatous cases did not show leprous lesions in any of the viscera. Of the lepromatous cases 21 were clinically active and their skin was bacteriologically positive. All these cases showed leprous lesions in the internal organs, as described under various systems. The remaining nine cases were bacteriologically negative and clinically quiescent. Of these, six cases showed lesions in internal organs and three did not show any lesions.

Grossly detectable changes were not seen in any of the viscera, except the testes, which were small and firm. Microscopically leprous lesions were seen as collections of foamy macrophages with a few lymphocytes and plasma cells. Acid-fast bacilli were seen in varying numbers, sometimes in clumps and sometimes singly within the cytoplasm of the macrophages. Some of the macrophages were large, ballooned out, and filled with large vacuoles, resembling fat cells. These cells contained very few or no bacilli. It was generally observed that the maximum numbers of acid-fast bacilli were found in those cells that did not contain such large vacuoles, but had granular cytoplasm.

Respiratory system. The incidence of pulmonary tuberculosis was found to be very high in the cases examined. Evidence of active disease with fibrocaseous lesions was seen in nine cases. Fifteen cases showed evidence of healed lesions, such as pleural adhesions, fibrosis or calcified areas. Lepromatous granulomata were not seen in the lungs in any of the cases.

The larynx was examined in nine lepromatous cases. No gross lesion was seen in any one of them. Microscopically eight of the cases showed lepromatous granulomata involving the mucous membrane and submucous tissue. Denudation of the epithelium was seen in all cases, but only microscopic ulcers were found. Dense collections of foamy macrophages were seen around mucous glands and around small nerve trunks. Lepromatous granulomata were seen on the epiglottis, the aryepiglottic folds, in the vestibule, and over the vocal cords. The portion of the larynx below the vocal cords showed no granulomata. The trachea and bronchi were examined in all cases, but did not show any lesion. Leprous lesions were thus seen to stop short at the larynx, not extending into the trachea.

Cardiovascular system. In one case there was tuberculous pericarditis. In all the rest the pericardium was smooth and the sac contained a normal amount of fluid. The coronary arteries were patent and normal in all cases except one. In this case there was narrowing of the lumen by atheromatous plaques. This case also showed fibrosis of the myocardium and evidence of old infarction. One case showed left ventricular hypertrophy, associated with a markedly contracted kidney. Two cases showed mitral stenosis and one had rheumatic vegetations on the mitral valve.

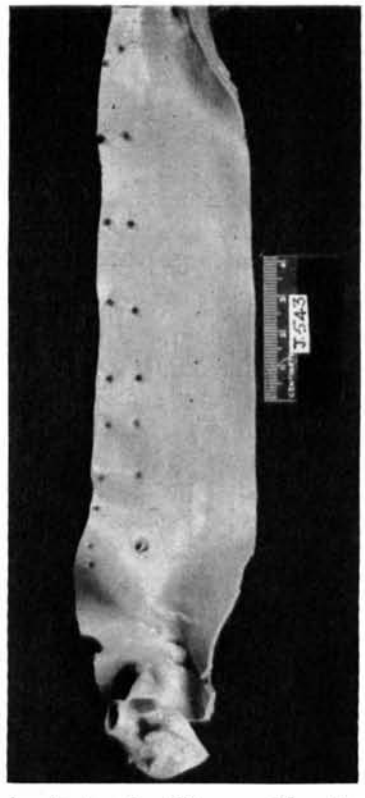

FIG. 1. Aorta of a 53 year-old patient with lepromatous leprosy. The intima is smooth except for a few fatty streaks.

The aorta was remarkably smooth and free from atheromata in 25 cases. In 10 cases, the aorta showed a few fatty streaks or minimal atherosclerosis (Fig. 1). In two only of the 37 cases did the atheromatous plaques reach a moderate degree. Both patients were lepromatous; one was a woman aged 60 years and the other a man aged 45 years. Table 3 shows the incidence of atherosclerotic lesions of the aorta, distributed in two broad age groups.

Alimentary system. The esophagus, stomach, and duodenum did not show any lesions. Tuberculous ulcers in the ileocecal region was seen in three cases. Other cases showed no significant lesions in the bowel. The pancreas showed no lesions.

Hepatic system. The average weight of the liver was 1,163 gm. with extremes ranging from 650 to 1,525 gm. In all cases the outer surface was smooth and reddish-brown in color. The edges were sharp. Grossly no lesions were detected. Microscopically none of the nonlepromatous cases showed leprous lesions. In one there were miliary tuberculous lesions. Two cases showed moderate fatty change. Of the lepromatous cases, 19 showed moderate fatty change. Of the lepromatous cases, 19 showed lepromatous granulomata containing many acid-fast bacilli in the macrophages. These were seen around the portal tracts and also as small focal lesions in the lobules (Fig. 2). Bacilli were seen also in Kupffer cells. In three cases there were extensive tuberculous lesions and it was difficult to exclude coexistent leprous lesions in them. Thus only eight cases were free from granulomata. Three cases showed a moderate degree of fatty change. Cavernous hemangiomata were seen in three cases. Amyloid deposit was detected in two cases. The gallbladder and bile ducts showed no abnormality in any of the cases, and no gallstones were found.

Spleen. The weight of the spleen ranged from 77 to 374 gm. with an average of 201 gm. The outer surface was smooth. The

TABLE 3. *Incidence of atherosclerotic lesions of aorta.*

Age group (yrs.)	Type of leprosy	Total no. cases	Without atherosel.	Fatty streaks	Fibrous plaques	Complicating lesions
20–40	L	21	14	7	—	—
	N	7	6	1	—	—
40–60	L	9	4	3	2	—
	N	—	—	—	—	—
Total		37	24	11	2	—

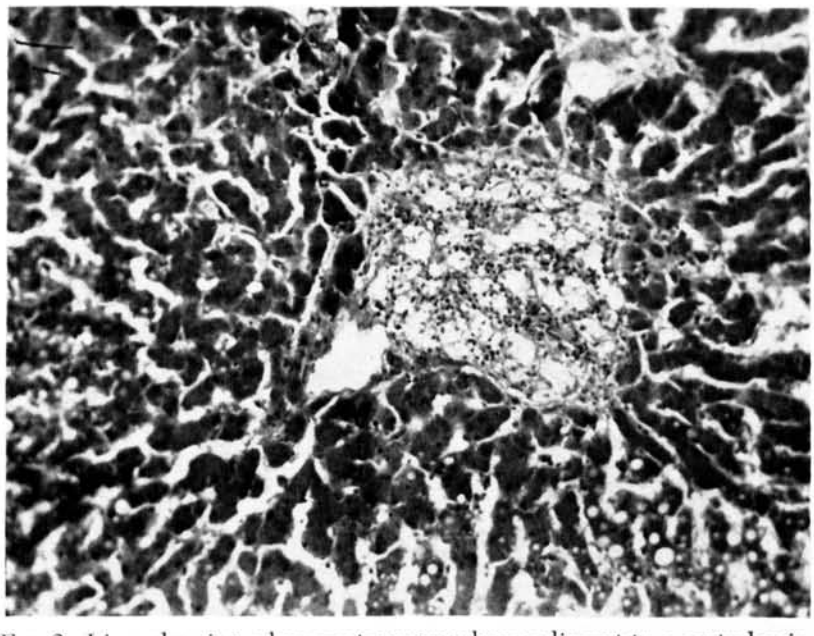

FIG. 2. Liver showing a lepromatous granuloma adjacent to a central vein.

capsule showed no thickening. The cut surface showed a soft, friable pulp. There was infarction in three cases. Microscopically, the nonlepromatous cases showed no lesions. Acid-fast bacilli were seen in 14 of the lepromatous cases. In nine of these, there were discrete lepromatous granulomata, scattered in both the red and white pulp (Figs. 3, 4). The other five cases showed diffuse infiltration of the sinusoids with foamy macrophages, which were filled with acid-fast bacilli. Tuberculous lesions were seen in six cases. Four cases showed amyloid deposit, which was extensive in three of them.

Lymph nodes. Among the nonlepromatous cases, two showed tuberculoid lymphadenitis. The rest showed no significant lesion. Among the lepromatous cases, the lymph nodes were examined in 22 cases, and leprous granulomata were found in 16 of them (Fig. 5). The remaining six cases showed tuberculous lesions. An extensive survey of all the groups of lymph nodes in the body was made in 10 lepromatous cases by sampling lymph nodes from each group. Lepromatous granulomata were seen in all the external groups of lymph nodes draining the skin of the limbs, head and neck. Of the internal groups, the internal iliac, external iliac and paraaortic groups showed extensive granulomata. It was of interest to note that the glands along the porta hepatis showed lepromatous granulomata in all cases except one. The mesenteric lymph nodes did not show leprous lesions. Similarly, nodes in the thoracic cavity were not affected.

Bone marrow. A search for acid-fast bacilli in the bone marrow was made in 15 lepromatous cases. Bacilli were found in 10 cases, but in two of these the bacilli found were probably *M. tuberculosis*, because these two had extensive tuberculous lesions in many organs. A significant finding in the bone marrow was the presence of a large number of plasma cells and a few scattered macrophages, without lepromatous granulomata.

Urinary system. The average weights of the right and left kidney were 117 and 126 gm., respectively; the weights ranged from 50 to 175 gm. None of the lepromatous or the nonlepromatous cases showed specific lesions of leprosy. An outstanding observation was the frequent occurrence of inflammatory lesions of the kidney in lepromatous leprosy. Table 4 shows the incidence of various inflammatory lesions found in the kidneys. No inflammatory lesions were found in six lepromatous cases and five nonlepromatous cases.

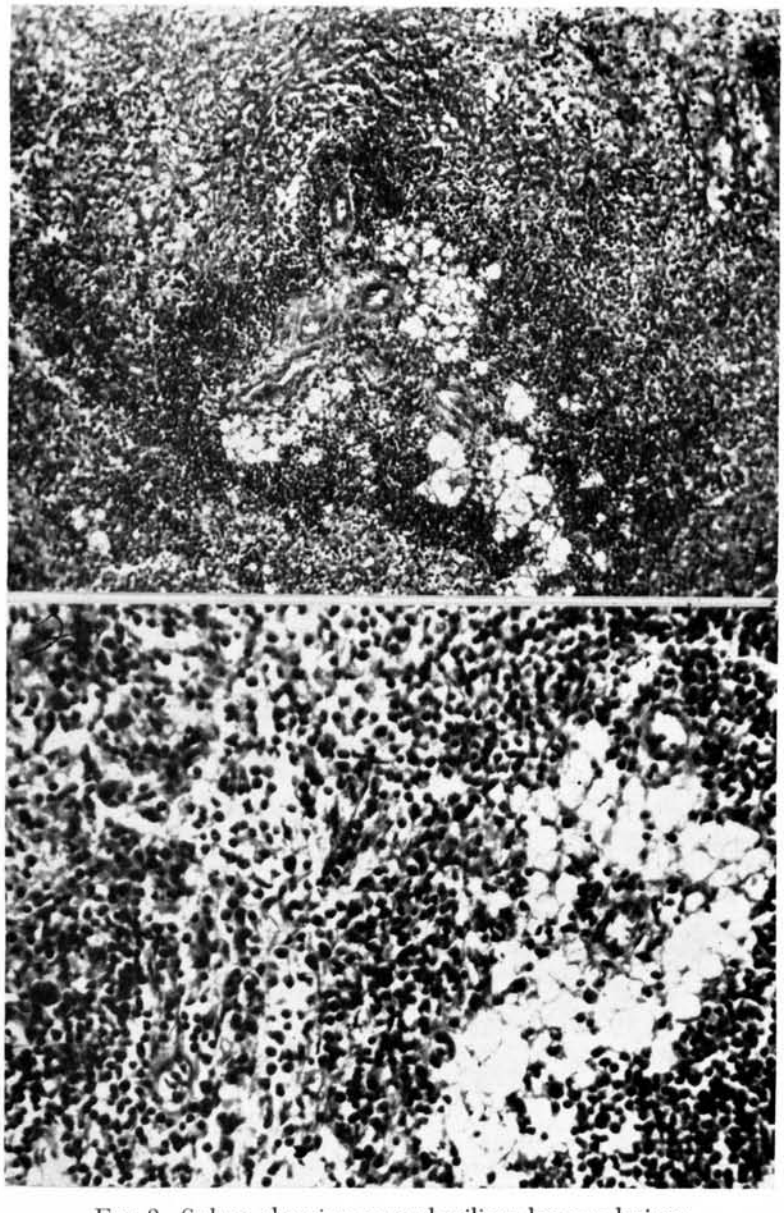

FIG. 3. Spleen showing several miliary leprous lesions.

FIG. 4. A higher power view of the lesion in the spleen (see Fig. 3) showing a granuloma in the white pulp. Acid-fast stains showed numerous bacilli inside the macrophages.

The most frequent inflammatory condition was pyelonephritis, with hyalinized glomeruli, dense infiltration of the stroma with chronic inflammatory cells, and some fibrosis and scarring (Figs. 6, 7). The cases with interstitial nephritis showed some congestion of glomerular tufts, and mild infiltration of the stroma diffusely or in foci with lymphocytes, plasma cells, and, less frequently, neutrophils. Two cases showed a histologic picture consistent with acute glomerulonephritis. The two patients were 20 and 27 years old. Amyloid deposit was seen in three cases (Fig. 8).

Genital system. The right and the left testis had an average weight of 16 and 17

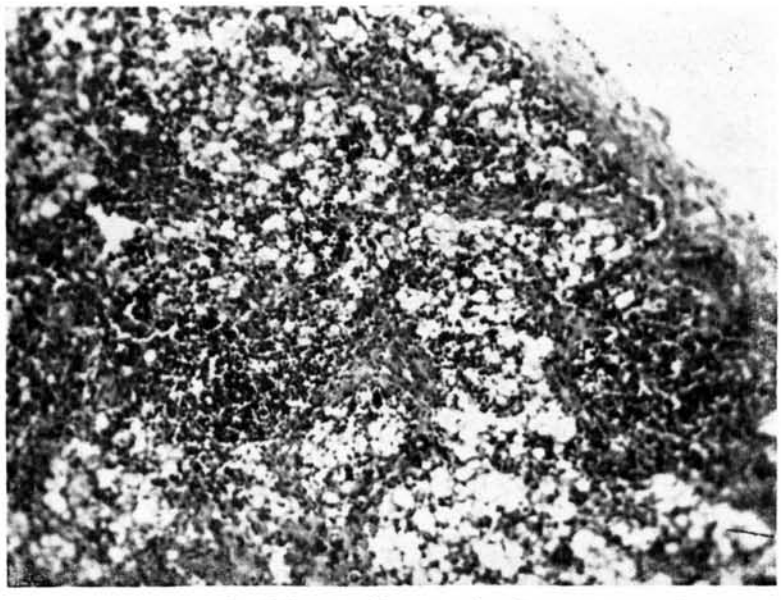

FIG. 5. Lymph node with diffuse infiltration by foamy macrophages. Numerous acid-fast bacilli were seen inside these cells.

TABLE 4. *Inflammatory lesions of kidney.*

	Non-lepromatous	Lepromatous
Acute glomerulonephritis	—	2
Interstitial nephritis	1	9
Pyelonephritis	—	10
Tuberculosis	1	3
No lesion	5	6
Total	7	30

gm., respectively, ranging from 8 to 28 gm. The testes were examined in 25 lepromatous cases. They were normal in two cases. In the remaining cases the testes were small, firm and atrophic. Cut surfaces were of a uniform white appearance, showing dense fibrous strands. The tubules could not be picked up by forceps. Microscopically the seminiferous tubules were markedly atrophic and hyalinized. Sheets of foamy macrophages loaded with acid-fast bacilli were seen replacing the parenchyma. Diffuse fibrosis with areas of hyalinization was seen. The interstitial cells were replaced by lepromatous granulomata or fibrous tissue (Fig. 9). Lepromatous granulomata were seen also in the epididymis. The prostate and seminal vesicles showed no significant lesions. In all the nonlepromatous cases the testes were found to be normal grossly and microscopically.

Endocrine system. The weights of the right and the left adrenal averaged 5.7 and 5.8 gm., respectively. Among the lepromatous cases, granulomata were seen in nine cases, located mostly in the cortex near the junction between the cortex and the medulla (Figs. 10, 11). Tuberculous lesions were seen in two cases. None of the nonlepromatous cases showed any lesions.

The thyroid was examined in 13 cases. The weight averaged 17 gm., ranging from 12 to 25 gm. The gland showed no lesion. In the pancreas no histologically detectable lesions were seen in the islets of Langerhans in any of the cases. The pituitary was examined in 13 cases and showed no significant lesions.

Central nervous system. The brain was examined in 13 cases. The leptomeninges were smooth and normal in all cases. Multiple coronal sections through the cerebrum and brain stem and horizontal sections through the cerebellum showed no gross lesions. Sections of specimens taken from several representative areas of the brain

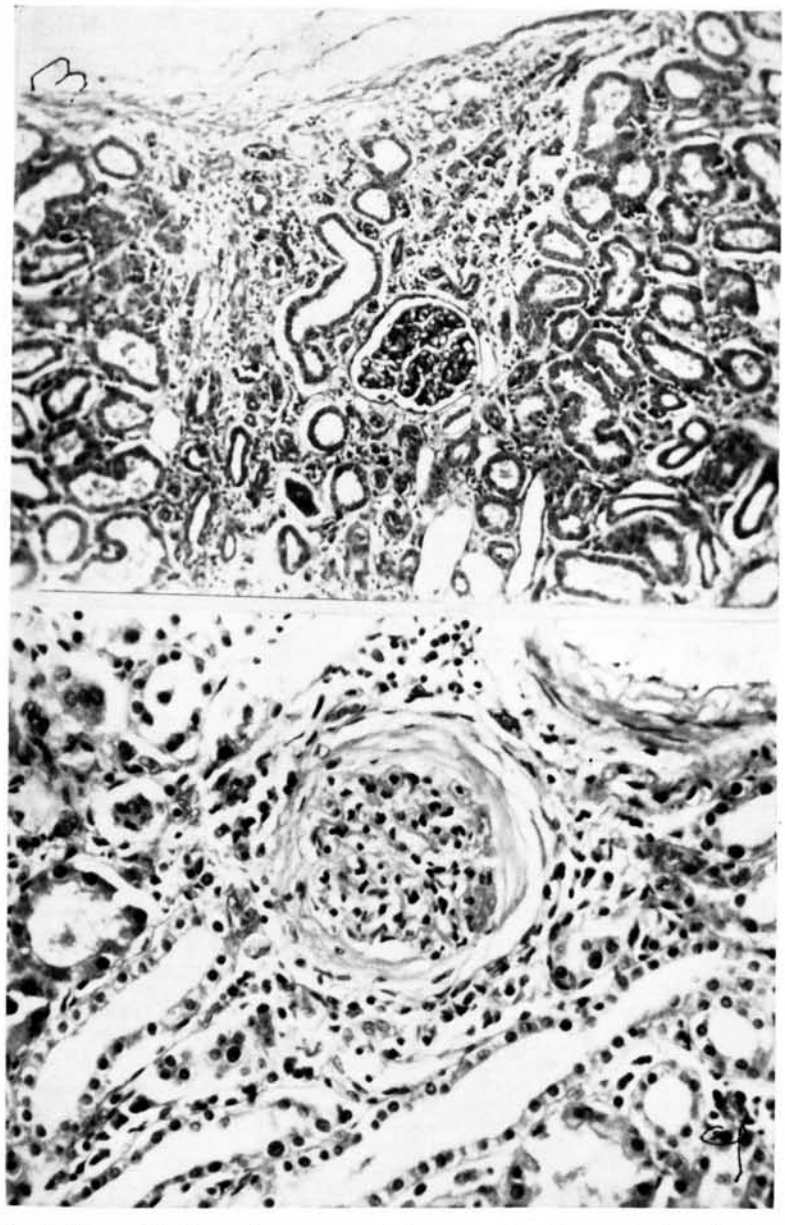

FIG. 6. Section of kidney in a case of lepromatous leprosy, showing an area of scarring, with atrophy of tubules and infiltration by inflammatory cells, consistent with chronic pyelonephritis.

FIG. 7. Kidney in lepromatous leprosy showing pyelonephritis. Note the pronounced periglomerular fibrosis.

showed no significant lesions. The spinal cord was examined in five cases. Sections at various levels showed no lesion.

Associated diseases. Chronic inflammatory lesions of the kidney formed the commonest group of conditions associated with lepromatous leprosy. The condition was chiefly an incidental postmortem finding not clinically recognizable except on careful investigation. Pulmonary tuberculosis was the next most common condition. It was clinically detectable and was the cause of death in many of the cases. Secondary amyloidosis was less common than in reports from other countries. Table 5 presents a list of the diseases associated with leprosy in the cases studied.

TABLE 5. *Diseases associated with leprosy.*

Diseases	No. cases
Chronic inflammatory lesions of the kidney	24
Acute glomerulonephritis	2
Tuberculosis (pulmonary and disseminated)	10
Chronic abscess of the lung	1
Tetanus	5
Secondary amyloidosis	4
Valvular diseases of the heart	3
Hyperpyrexia of unknown cause	2
Syphilitic aortitis	1
Brain tumor (cholesteatoma)	1

TABLE 6. *Cause of death.*

	No. cases
Tuberculosis	10
Tetanus	5
Acute pulmonary infections	3
Nephrosis and renal failure	4
Septicemia	3
Hyperpyrexia of unknown cause	2
Unnatural causes (accidental and suicidal)	2
Severe exacerbations of leprosy	8
Total	37

Cause of death. The causes of death are listed in Table 6. Tuberculosis was the most common. Five patients died of tetanus. In eight cases the death could not be explained by any cause other than leprosy itself. These cases had had repeated severe exacerbations. As a terminal event, five patients had pulmonary edema, one had bronchopneumonia, and two had severe diarrhea.

DISCUSSION

The incidence of visceral lesions in leprosy is a long recognized fact. Our findings in general are consistent with those of Powell and Swan ([17]), Fite ([9]), Mitsuda ([16]), and other earlier workers. Some of the unusual findings and salient features are mentioned below. It is of interest to note that in the present series visceral lesions were seen not only in active cases of lepromatous leprosy but also in the bacteriologically negative and clinically quiescent cases. Lesions in these quiescent cases were seen in the testes in four cases, in the liver in two cases, and in one case each in lymph nodes, adrenal and spleen. The earlier autopsy reports do not mention findings in clinically quiescent or arrested cases. Our findings support the expectation of Cochrane ([5]) that leprosy bacilli might lurk in the internal organs for a long time after the skin had become bacteriologically negative. They also emphasize the danger of relapse in treated cases and the need to continue treatment for a long time after skin lesions have cleared.

The liver and lymph nodes were found to be the internal organs most commonly affected. Lesions were seen also in the spleen. The lesions seen in the spleen and lymph nodes were discrete and well formed lepromatous granulomata and not just infiltration of the sinusoids by bacillus-laden "lepra cells," although such infiltration was also seen as an additional feature in many cases. Similarly the liver also showed discrete miliary lepromata as well as many Kupffer cells filled with acid-fast bacilli. Extensive involvement of the lymph nodes in the abdomen and pelvis was also a salient feature of the present study, as was the fact that the mesenteric nodes and

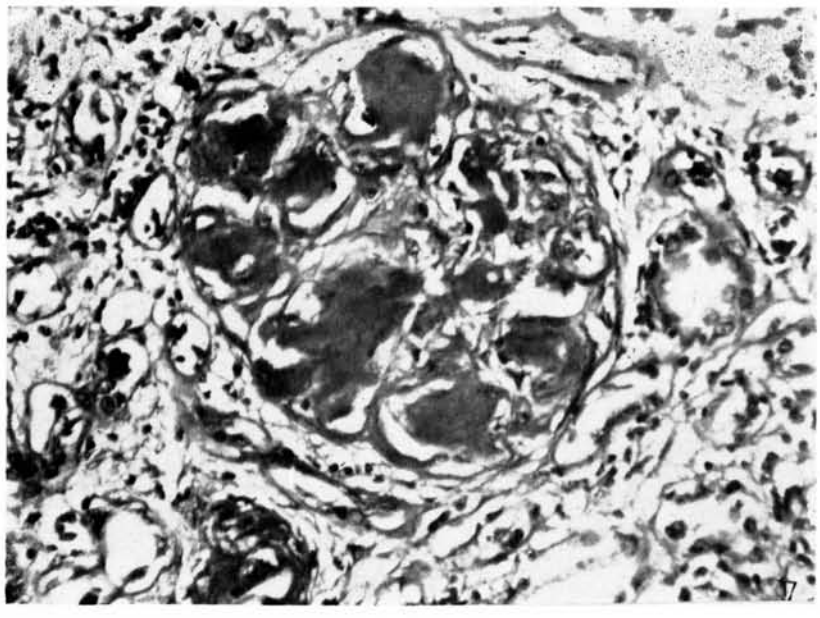

Fig. 8. Kidney showing amyloid deposit in a glomerulus.

FIG. 9. Atrophic testis showing diffuse infiltration by lepromatous granulation tissue and hyalinization of seminiferous tubules.

nodes in the thorax were not involved. A possible explanation of freedom from involvement in the latter two groups was the fact that the organs which they drained, viz., the intestines and thoracic viscera, did not show lepromatous lesions. A striking feature, in contrast, was constant involvement of the lymph nodes along the porta hepatis. This is consistent with the observation that the liver is a frequently affected organ; 19 of the 30 lepromatous cases in the present study showed lepromatous granulomata.

Tuberculosis was the commonest associated disease and the obvious cause of death in most of the cases. Mitsuda and Ogawa ([16]) have reported tuberculosis as the cause of death in 54.7 per cent of cases. They have also quoted figures from the Culion Leper Colony, where 48.6 per cent of 3,155 deaths were due to tuberculosis. Other reports also give a high incidence of tuberculosis in leprosy patients. In the present study, in addition to the 10 active cases, evidence of previous infection by tuberculosis was seen in 15 cases, so that 70 per cent showed active or healed tuberculosis.

There has been much discussion in recent years of the subject of cross immunity between tuberculosis and leprosy. Chaussinand ([3]) was among the first to put forward the theory of cross immunity; it was based on his observation that as tuberculosis increased in Western countries leprosy decreased. Convit and Rassi ([7]) observed that 98 per cent of lepromin-negative reactors could be converted to lepromin-positive reactors by BCG vaccination, and concluded therefore that BCG vaccination gives immunity in 98 per cent of cases. While it is generally observed that BCG induces lepromin positivity, many workers are cautious in accepting the equivalence of hypersensitivity and immunity. In the present study it was not known definitely whether the patients had tuberculosis first and subsequently developed leprosy or vice versa. However, the occurrence of tuberculosis in several lepromatous and nonlepromatous cases should invalidate any categoric statement that tuberculosis always excludes or provides ample immunity against leprosy.

The absence of atheromatous lesions was a significant feature in the present study, and difficult to explain exactly. The role of any geographic factors could be ruled out by comparison with the figures of Samuel *et al.* ([18]), who have reported an overall autopsy study on the incidence of atherosclerosis in patients dying of various other diseases in the same hospital (Table 7). The nutritional status of the leprosy patients may be a possible factor. These patients were very much undernourished, as

TABLE 7. *Comparative study of atherosclerotic lesions of aorta (adapted from Samuel et al. ([18]))*.

Authors	Age group (yrs.)	No. cases	Percentage of total cases			
			Without Atherosel.	Fatty streaks	Fibrous plaques	Complicating lesions
Samuel *et al.*	20–40	98	13.0	67.0	17.0	3.0
	41–60	73	7.0	27.0	48.0	18.0
Total		171				
Present study	20–40	28	71.0	29.0	—	—
	41–60	9	45.0	33.0	22.0	—
Total		37				

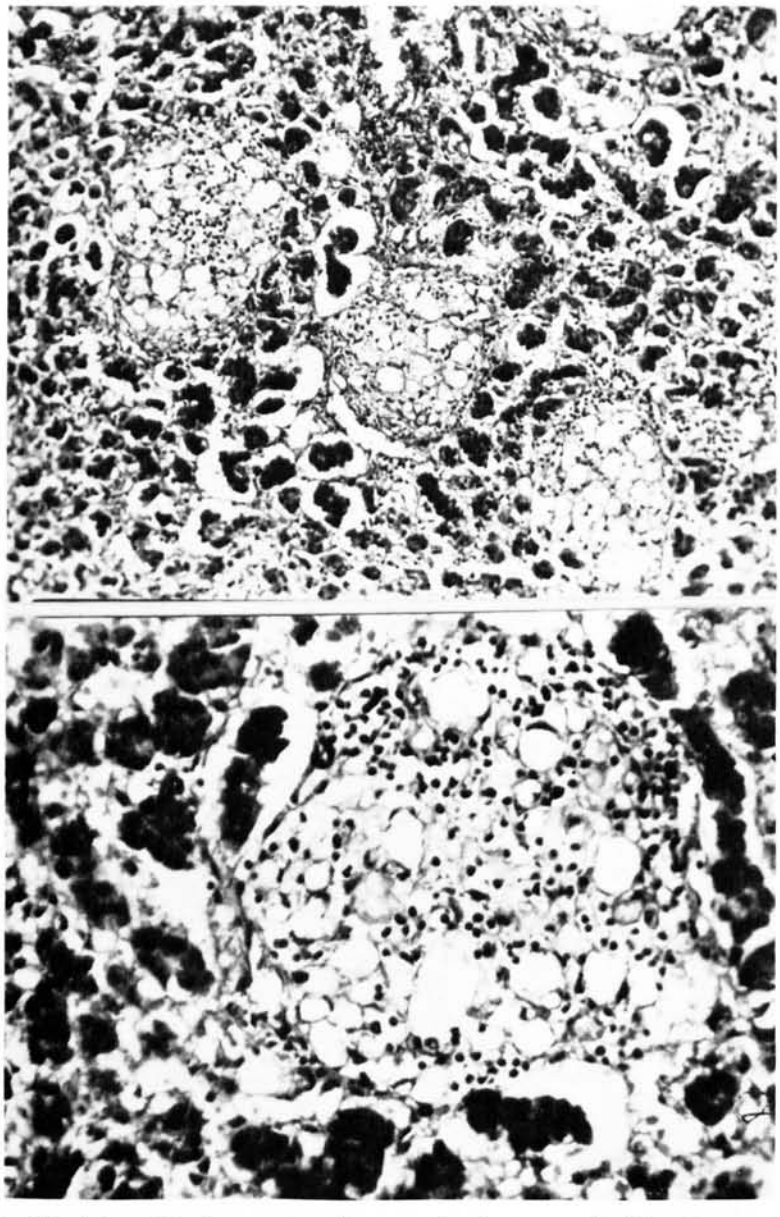

FIG. 10. Adrenal in lepromatous leprosy showing several miliary lepromata.

FIG. 11. A higher power view of adrenal (see Fig. 10) showing a lepromatous granuloma. Acid-fast stains showed many bacilli inside the macrophages.

compared with the other patients in a general hospital.

Another explanation that may be suggested is that these patients had gross testicular atrophy with fibrosis; it is possible that a hormonal imbalance due to diminished androgenic activity might have produced in these patients a female type of atherogenesis. Serum cholesterol estimations were not made in the cases reported. Job ([12]) has observed that the urinary ketosteroids are very low in cases of testicular atrophy, indicating impairment of androgenic activity.

The kidney seems to be the target organ of infection in lepromatous leprosy. The

high incidence of pyelonephritis indicates that the organ is rendered more susceptible to systemic infection. Kean and Childress ([13]) observed all types of nephritis and nephrosclerosis among 51 per cent of their cases, and Guasp ([10]) ascribed 63 per cent of deaths in his series to renal disease. Interstitial nephritis is often associated with some acute infection and toxic conditions, such as diphtheria and scarlet fever. Acute exacerbation of the disease, with erythema nodosum leprosum, represents a toxic state, and this might well cause interstitial nephritis. Two cases were found to show acute glomerulonephritis. Erythema nodosum leprosum is thought to be an allergic manifestation, and the occurrence of acute glomerulonephritis could well be a part of the generalized allergic inflammatory reaction. It was observed by Brusco and Masanti ([4]) that 59 per cent of lepromatous cases in reaction had developed kidney disease by the time of death; this may be compared with 26 per cent among patients without reaction.

The incidence of secondary amyloidosis appears very low in India when compared with figures in Western counties ([14]). Only four of the cases reported here had secondary amyloidosis, and in three of them the amyloid was from minimal to moderate in amount. In contrast, the figures of Powell and Swan ([14]) indicate 38 per cent of their cases as showing severe renal amyloidosis resulting in death with uremia.

While it is generally believed that death in leprosy is due to some other associated disease, it has been observed, as in the present study, that in a small percentage of cases death is due to leprosy *per se*, as a result of severe repeated "lepra reactions." This fact was brought out by Mitsuda and Ogawa ([16]), who found that in four per cent of their cases death was due to a virulent form of leprosy. Cochrane ([6]) also supports the view that prognosis is very grave where the advanced case passes from one "reaction" to another, death resulting from cachexia due to prolonged fever.

SUMMARY

The autopsy findings in 37 cases of leprosy are presented. This is the first report from India on autopsy study of leprosy cases. The findings are in general agreement with the earlier reports from other countries. A few interesting observations were brought out. Lepromatous granulomata were demonstrated in the larynx, liver, spleen, lymph nodes, bone marrow, testes and adrenal, in addition to lesions in the skin and peripheral nerves.

A low incidence of atheromatous lesions in the aorta of leprosy patients was a significant observation. The possible cause of this is discussed. Another interesting finding was the frequent occurrence of interstitial nephritis and pyelonephritis in lepromatous leprosy. Amyloidosis was a rare occurrence as compared with the figures from other countries. Tuberculosis was the commonest disease associated with leprosy in the cases studied and the most frequent cause of death. The possibility of leprosy itself as a cause of death is suggested from observations in some of the cases.

RESUMEN

Se presentan los hallazgos de autopsias en 37 casos de lepra. Esta es la primera comunicación de India sobre estudios de autopsias de enfermos de lepra. Lo encontrado está en general acuerdo con comunicaciones anteriores de otros países. Algunas observaciones interesantes fueron sacadas. Granulomas lepromatosos fueron demostrados en la laringe, hígado, bazo, nódulos linfáticos, médula ósea, testículos y glándulas suprarrenales, además de las lesiones en la piel y en los nervios periféricos.

Una observación significativa fué la baja incidencia de lesiones atheromatosas en la aorta de los enfermos de lepra. Se discute la causa posible de este hallazgo. Otro hecho de interés fué la frecuencia con que se observó la nephritis intersticial y la pyelonephritis en lepra lepromatosa. Amyoidosis fué una observación poco frecuente en comparación con las cifras de otros países. Tuberculosis fué la enfermedad mas comunmente asociada con lepra en los casos estudiados y también la causa mas frecuente de muerte. Se sugiere la posibilidad, con motivo de observaciones hechas en algunos casos, que la lepra en si misma sea una causa de muerte.

RÉSUMÉ

On présente ici les résultats d'autopsie observés dans 37 cas de lèpre. Cette étude constitue le premier rapport en provenance de l'Inde et concernant une étude nécropsique de cas de lèpre. Les observations effectuées sont généralement en accord avec les rapports antérieurs provenant d'autres pays. Quelques observations intéressantes ont été mises en lumière. Des granulomes lépromateux ont été démontrés dans le larynx, le foie, la rate, les ganglions lymphatiques, la moëlle osseuse, les testicules et les glandes surrénales, sans mentionner les lésions de la peau et des nerfs périphériques.

Une incidence peu élevée de lésions athéromateuses dans l'aorte des malades atteints de lèpre constitue une observation significative. La cause possible en est discutée. Une autre observation intéressante fut l'existence fréquente de néphrite interstitielle et de pyélonéphrite dans la lèpre lépromateuse. L'amyloïdose ne survenait que rarement, si l'on se réfère aux chiffres fournis dans d'autres pays. La tuberculose constituait la maladie la plus courante associée à la lèpre dans les cas étudiés, et la cause de mort la plus fréquente. D'après les observations recueillies dans quelques uns de ces cas, il semble que la lèpre elle-même puisse être une cause de decès.

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REFERENCES

1. Austin, C. J. Regional and racial differences in leprosy. A report made from statistics from Dr. C. J. Austin. Leprosy Rev. **19** (1948) 20-22.
2. Black, S. H. The pathology of leprosy. *In* Tuberculosis and Leprosy, the Mycobacterial Diseases. Symposium Series, Vol. 1, American Association for the Advancement of Science. Lancaster, Pa., The Science Press Printing Co., 1938, pp. 97-105.
3. Chaussinand, R. Tuberculose et lèpre, maladies antagoniques. Éviction de la lèpre par la tuberculose. Internat. J. Leprosy **16** (1948) 431-438.
4. Brusco, C. M. and Masanti, J. G. Causes of death of leprosy patients. Influence of lepra reactions and renal disease. Internat. J. Leprosy **31** (1963) 14-25.
5. Cochrane, R. G. Pathology. *In* Practical Text Book of Leprosy. London, Oxford University Press, 1947, p. 33.
6. Cochrane, R. G. Reaction in leprosy. Ibid, p. 98.
7. Convit, J. and Rassi, E. Lepromin and tuberculin tests in Venezuelan leprosy foci. Induction of lepromin reactivity by BCG vaccination. Internat. J. Leprosy **22** (1954) 303-310.
8. Dharmendra. Regional variations in leprosy. Leprosy in India **35** (1963) 7-10.
9. Fite, G. L. Leprosy from the histologic point of view. Arch. Path. **35** (1943) 611-644.
10. Guasp, T. E. Lepra visceral. Rev. Leprol. (Fontilles) **5** (1963) 811-822. (*Abstract in* Trop. Dis. Bull. **61** (1964) 795-796)
11. Hansen, G. A. and Looft, C. Die Lepra vom klinischen und pathologisch-anatomischen Standpunkte. Cassel, T. G. Fisher & Co., 1894, 45 pp. (*Translated by* Norman Walker, Bristol, John Wright & Co., 1895)
12. Job, C. K. Gynecomastia and leprous orchitis. A preliminary study. Internat. J. Leprosy **29** (1961) 423-441.
13. Kean, B. H. and Childress, M. E. A summary of 103 autopsies on leprosy patients on the Isthmus of Panama. Internat. J. Leprosy **10** (1942) 51-59.
14. Krishnamurthy, S. and Job, C. K. Secondary amyloidosis in leprosy. Internat. J. Leprosy **34** (1966) 155-158.
15. Mitsuda, K. Atlas of Leprosy. Okayama, Japan, Chotokai Foundation, 1952.
16. Mitsuda, K. and Ogawa, M. A study of one hundred and fifty autopsies on cases of leprosy. Internat. J. Leprosy **5** (1937) 53-60.
17. Powell, C. S. and Swan, L. L. Leprosy: Pathological changes observed in fifty consecutive necropsies. American J. Path. **31** (1955) 1131-1147.
18. Samuel, I., Gault, E. W. and Sudersanam, D. A postmortem study of the incidence of atherosclerosis in the aorta and the coronary vessels. Seminar on Atherosclerosis and Ischaemic Heart Disease, International Center Medical Research Golden Jubilee, 1961.