Histopathologically, the most prominent feature in 81% of the DLE patients was hyperplasia of the elastic tissue, very much identical to the findings in solar elastosis. Previous workers have not reported such changes in DLE of the scalp. It seems that the DLE lesions on the sun-exposed site like scalp develop basophilic degeneration of collagen, which on elastic tissue staining gives the appearance of elastic material. This further supports the views of many authors[2,3] that sunlight is one of the triggering factors in DLE.

The most striking features in the PP group were the total disappearance of pilosebaceous follicles and the presence of perpendicular columns of elastic fibres. Sub-epidermal loss of elastic fibres was also observed in 80% cases in another series.[4] The term fibrosing alopecia, first described by Pinkus,[4] is applicable to a more diffuse clinical thinning of the hair, and in which elastic fibre formation is not a feature of the cutaneous scar formation. It is distinguished from PP by the minor amount of perifollicular infiltrate and by the development of elastic fibres around the lower portion of the hair follicle.

In FD, 2 biopsies were performed, one from the centre of an atrophic smooth hairless area and a second from an area of the lesion towards the border which contained a pustule. The histopathologic findings in the central portion of the bald area seemed almost identical in all respects to those of PP, but no section showed vertical fibrous remnants of the atrophic hair follicles. In the sections containing the pustules, a marked perifollicular infiltrate was seen with a preponderance of polymorphs along with a considerable number of lymphocytes. Intrafollicular infiltrate was not seen in any section. The perifollicular infiltrate contained a good number of plasma cells, which were absent in other groups of scarring alopecias studied. Blood vessels were dilated in all the cases which was very striking.

In the present series of LPP, none of the sections showed sub-epidermal infiltrate typical of lichen planus, while hydropic degeneration of the basal cells was practically absent. These could be explained by the fact that most of our cases were in the late stages, hence basal cells were probably replaced by squamous cells.

In general, the sebaceous glands were lost, whereas sweat glands and arrectores pilorum were present in almost all the sections of DLE, PP, LPP and FD irrespective of their stage. This suggests that probably sebaceous glands are affected first in cicatricial alopecia, and thus their absence should make the dermatologist and pathologist suspicious of the diagnosis. Morphoea looked the same histopathologically as elsewhere in the body.

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RENAL INVOLVEMENT IN LEPROSY

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Renal involvement in 20 lepromatous leprosy (LL) and 5 non-lepromatous patients was assessed by (a) biochemical analysis of blood and urine, (b) renal functional tests, and (c) histopathological examination of renal biopsies. Ten age-matched healthy normals formed the control group. LL patients had a varying degree of renal involvement as indicated by the presence of pus cells, granular, hyaline and red cell casts, reversal of albumin/globulin ratio and lowered creatinine clearance rates. Renal biopsies showed significant histopathological lesions in 50% of lepromatous as compared to 20% of the non-lepromatous patients. The pathological changes were predominantly of chronic glomerulonephritis followed by chronic pyelonephritis and interstitial nephritis. Surprisingly, none of the patients studied showed granulomas, acid fast bacilli or amyloid in the kidney.

Key words : Leprosy, Renal, Pathology, Kidney.

Lepromatous leprosy (LL) is a systemic, disseminated form characterised by dermal, neural and systemic granulomatous disease, abundant *Mycobacterium leprae*, lowered cell mediated immune responses, increased specific and non-specific humoral response, autoantibodies and circulating and tissue-localised immune complexes.[1,2] Renalin volvement was first reported by Mitsuda and Ogawa[3] in 1937. Non-specific renal lesions, such as glomerulonephritis, pyelonephritis, interstitial nephritis were variously reported by subsequent Indian workers.[4-6] Whereas a high incidence of 45 to 55% of secondary renal amyloidosis was reported by Western authors,[7,8] reports on Indian patients showed only 8-10% involvement.[4,9] With a view to further evaluate the geographic pattern of renal lesions in lepromatous leprosy, patients from the Baroda district of Gujarat were studied, using biochemical, microbiological and histopathological criteria.

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Materials and Methods

Twenty five patients of leprosy consisting of 20 lepromatous leprosy (LL) and 5 non-lepromatous leprosy attending from June to November 1982 were included in the study. They were classified according to the clinico-pathological criteria of Ridley and Jopling.[10] The clinical details are given in table I. Fifteen of the patients received anti-leprosy treatment with dapsone (DDS). Detailed physical and clinical examination was carried out prior to the study to exclude any associated illnesses such as nephritis, diabetes mellitus, hypertension and pulmonary tuberculosis. Ten normal healthy subjects were also investigated and formed the control group.

Both the patients and the controls were subjected to : (a) estimation of hemoglobin, total and differential leucocyte counts, erythrocyte sedimentation rate and blood smear examination; (b) urinalysis for the presence of albumin, sugar, pus cells, red blood cells and casts if any; (c) serum electrolytes and blood urea, serum and urinary creatinine, creatine clearance, 24 hours albumin, serum proteins with albumin/globulin ratio, serum cholesterol, serum calcium, phosphorus and alkaline phos-

Table I. Clinical details and treatment status of leprosy patients.

Type of leprosy	Number of cases			Duration of illness (years)			Duration of treatment (years)			
	Total	Male	Female	<1	1 to 5	5	Nil	<1	1 to 5	>5
Tuberculoid	2	2	—	2	—	—	1	1	—	—
Borderline tuberculoid	2	1	1	1	1	—	2	—	—	—
Borderline	1	—	1	1	—	—	—	1	—	—
Borderline lepromatous	5	4	1	2	3	—	2	1	1	1
Lepromatous	15	10	5	5	4	6	5	3	1	6
Total	25	17	8	11	8	6	10	6	2	7

phatase, (d) urine culture for pyogenic organisms and the presence of acid fast bacilli (AFB), (e) The dermal bacillary index in the patients was evaluated by slit smears of 6 sites in the body.

Percutaneous renal biopsies were done in all patients using Franklin's modification of Vim-Silvermann's needle. The biopsies were fixed in buffered formalin and the paraffin blocks were sectioned and stained for haematoxylin and eosin, PAS, congo red, and methyl violet. Acid fast bacilli were stained by Wade and Fite method.

Results

Of the 25 leprosy patients, 17 were male. They ranged in age from 15 to 70 years. Sixteen of the patients had received less than one year's antileprosy treatment with dapsone. None of the patients were noted to have other systemic diseases such as hypertension, diabetes or tuberculosis. All non-lepromatous patients except one (BB) in reactional phase were bacteriologically negative. All lepromatous patients showed bacilli in the skin with BI ranging from 3+ to 5+.

Routine urinalysis of LL patients showed significant numbers of pus cells in 4 cases, granular, hyaline and red cell casts in 3, and 10-15 red cells per high power field in 5. Three patients showed 1.5 to 2 gm per day of proteinuria. Two patients having erythema nodosum leprosum showed smoky urine. Bacterial culture of urine samples revealed *E. Coli* and *Klebsiella* in 3 and 2 lepromatous patients respectively.

Table II gives the data on serum proteins, serum cholesterol and blood urea of patients and controls.

Table II. Comparison of serum proteins, cholesterol and blood urea of the leprosy patients with normal controls.

Subjects	Serum proteins			Serum cholesterol	Blood urea
	Total gm%	Albumin gm%	Globulin gm%	mg%	mg%
Non-lepromatous	6.8±1.1	3.9±1.0	2.9±0.6	170±35	29±3.0
Lepromatous	7.5±0.5	4.1±0.4	3.1±0.5	160±30	28±5.0
Normal controls	7.1±0.2	4.6±0.2	2.5±0.1	174±20	30±3.0