

## RAPIDLY PROGRESSIVE (CRESCENTRIC) GLOMERULONEPHRITIS IN *ERYTHEMA NODOSUM LEPROSUM*: CASE REPORT.

Pranesh NIGAM<sup>1</sup>  
K.C. PANT<sup>2</sup>  
R.D.MUKHIJA<sup>3</sup>  
S.P.SHARMA<sup>4</sup>  
S.P.SAXENAS<sup>5</sup>  
Ajax KUMAR<sup>6</sup>  
K.K. KAPOOR<sup>7</sup>  
A.K. GUPTA<sup>8</sup>

**ABSTRACT** — A middle aged man (48 years) with short duration of illness (7 days) was admitted in the state of acute renal failure with erythema nodosum leprosum. He had repeated episodes of *erythema nodosum leprosum* in the past. His blood pressure was normal (150/80 mm Hg). During his hospital stay he was in the state of progressive anaemia (Hb = 8.8 g/dl to 7.2 g/dl), oliguria (urine out-put = 250-350 ml/day), azotaemia (blood urea = 198 mg/dl to 218 mg/dl) and impaired renal function tests with fatal outcome. Kidneys were smooth, congested and weighing 150 g each with histological features of rapidly progressive (crescentic) glomerulonephritis, a result of immune complex deposition from recurrent *erythema nodosum leprosum* episodes.

**Key words:** Leprosy. *Erythema nodosum leprosum*. Glomerulonephritis.

### 1. INTRODUCTION

Lepromatous leprosy is well known for its multivisceral involvement. Several acute clinical manifestations may occur at any stage of the disease. Specific lepromas in kidney are not frequent and a variety of non-specific lesions viz., secondary amyloidosis, acute or chronic glomerulonephritis and pyelonephritis etc., are described<sup>1-4</sup>. Kidney lesions may occur in lepro-

matous leprosy with normal renal functions<sup>5</sup>. However, nephritis complicating lepromatous leprosy may attain alarming significance because of its fatal outcome<sup>6</sup> which may be related to repeated attacks of *erythema nodosum leprosum* (ENL) in the course of the disease.

We report here a case of lepromatous leprosy with recurrent ENL who developed acute renal failure with fatal outcome.

- 
- (1) Reader in Medicine
  - (2) Registrar in Medicine
  - (3) Professor of Skin, V.D. & Leprosy
  - (4) Reader in Pathology
  - (5) Lecturer in Biochemistry
  - (6) Lecturer in Cardiology
  - (7) Lecturer in Medicine
  - (8) Professor of Medicine Head, Principal & Dean

Address: B.R.D. Medical College and Nehru Chikitsalaya - GOBAKHPUR - 273013, INDIA

## 2 CASE REPORT

A middle aged man (48 years) was admitted with fever, vomiting, swelling over the face and decreased urine output for the last seven days. He was taking treatment from Skin, V.D. and Leprosy Out Patient Department of this hospital for the last seven years for diffuse skin infiltration, numbness and epistaxis. During his irregular treatment for leprosy he had repeated episodes of ENL which were relieved with Salicylates, Clofazimine (Hansepran) and Corticosteroids and on admission too he had ENL.

Examination revealed a middle aged man of average built with puffy leonine face, pitting oedema over feet, erythematous tender nodular lesions over the extremities, ear lobules, forehead and plaques of diffuse infiltration and atrophic areas over the chest, abdomen, face and limbs. Distal portion of right great toe, 2nd and 3rd toe showed resorption. Ulnar and lateral popliteal nerves on both sides were cord like and tender with stocking type of hypoaesthesia. His blood pressure was 150/80mmHg. Systemic examination revealed non-tender hepatomegaly of 5 cm and soft systolic murmur (haemic) over the apex of the heart.

Routine investigations revealed mild anaemia (Hb = 8.8 g/dl), TLC 10800/mm<sup>3</sup>, P-46, L-48, E-6 and raised ESR (35mm). The daily urine out-put was 250-350 ml, having urinary proteins 1.5 g per day, 5-8 RBC, 10-15 pus cells, 1-4 granular cast per HPF. Urine culture did not reveal any pathogenic micro-organism. Electrocardiogram was within normal limits while skiagram chest showed changes of chronic bronchitis.

Blood biochemistry revealed azotaemia (blood urea = 198 mg/dl) serum creatinine

5.2 mg/dl, calcium 9.5 mg/dl, phosphate 4.2 mg/dl, protein 7.2 g/dl, albumin 3.5 g/dl, globulin 4.2g/dl, bilirubin 10µmol/l and alkaline phosphatase 32 U<sup>3</sup>. Renal function tests were impaired as evidenced by raised ratio of blood urea (BUN) with creatinine (25:1) and urine to plasma ratio of creatinine (82:1) and urea (16:1). Urea and creatinine clearance were 42 ml/min and 72 ml/min respectively. On 12 hours restriction of fluid the specific gravity of urine was 1.018 and after 12 hours of deliberate fluid intake 1.006

Patient remained oliguric and azotaemic for 3-4 days. The daily urine out-put improved to 500-800 ml. Just before death his haemoglobin came down to 7.2 g/dl, blood urea raised to 218 mg/dl, creatinine to 7.2 mg/dl and alkaline phosphatase to 42 U<sup>3</sup>. Throat swab, blood and urine cultures did not reveal any pathogenic micro-organisms. His condition deteriorated to fatal out-come on 9th day of admission to hospital.

Kidneys (Figure 1) were smooth with congested surface and weighing 150 g each. Histology revealed (Figure 2) diffuse involvement of glomeruli and variable changes of proliferation and sclerosis, some with segmental extracapillary proliferation (crescent — Figure 3). At places an intense inflammatory reaction invaded the glomerular tufts. There was mononuclear cell infiltration in the interstitium with alternating tubular dilatation and atrophy. Multiple lepromatous granulomas were seen in skin, nasal mucosa and liver (Figure 4). Spleen and lymph nodes showed moderate replacement of thymus dependent lymphocytes by foamy cells. *Mycobacterium leprae* could be seen in sections from skin, nasal mucosa and liver.

NIGAM, P. et al. Rapidly progressive (crescentic) glomerulonephritis in erythema nodosum leprosum: case report.

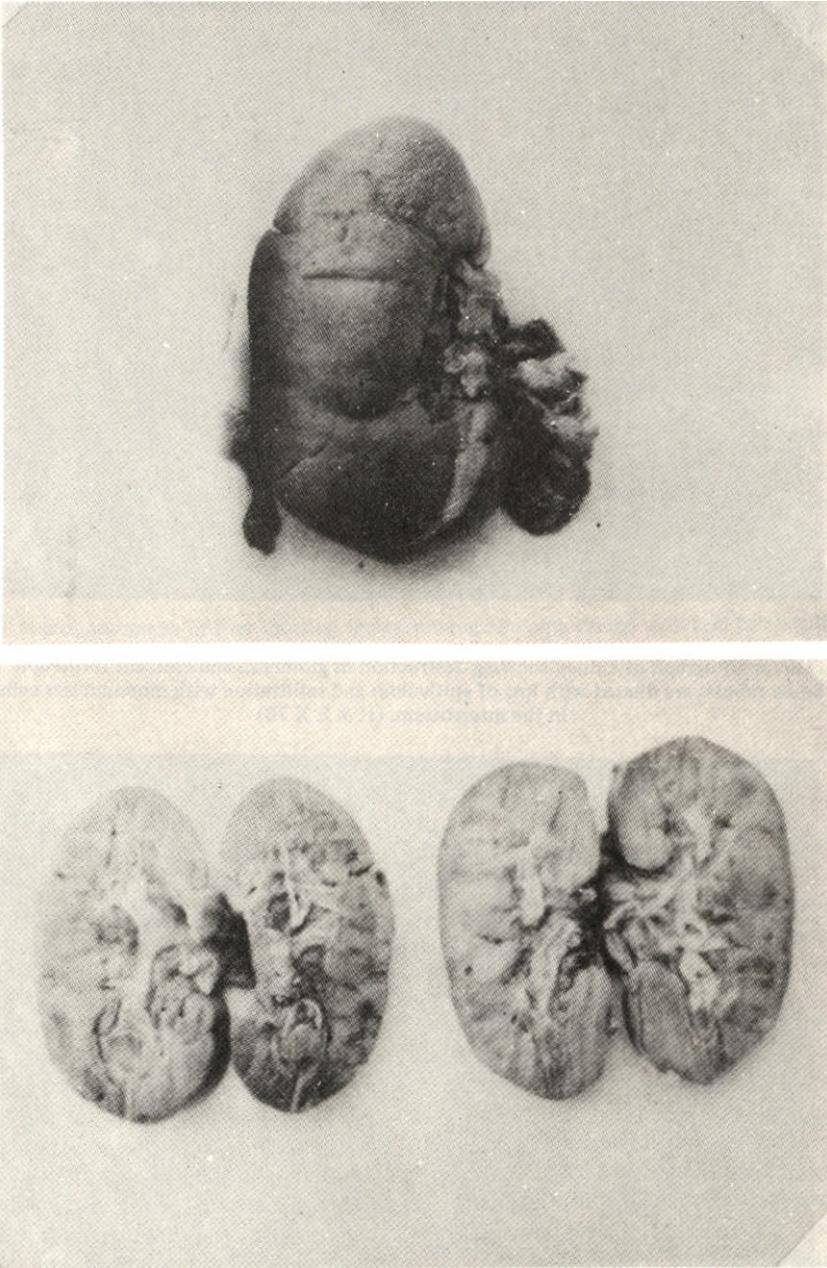


FIGURE 1— Kidneys showing slight increase in size (weight = 150 g) with smooth congested surface.

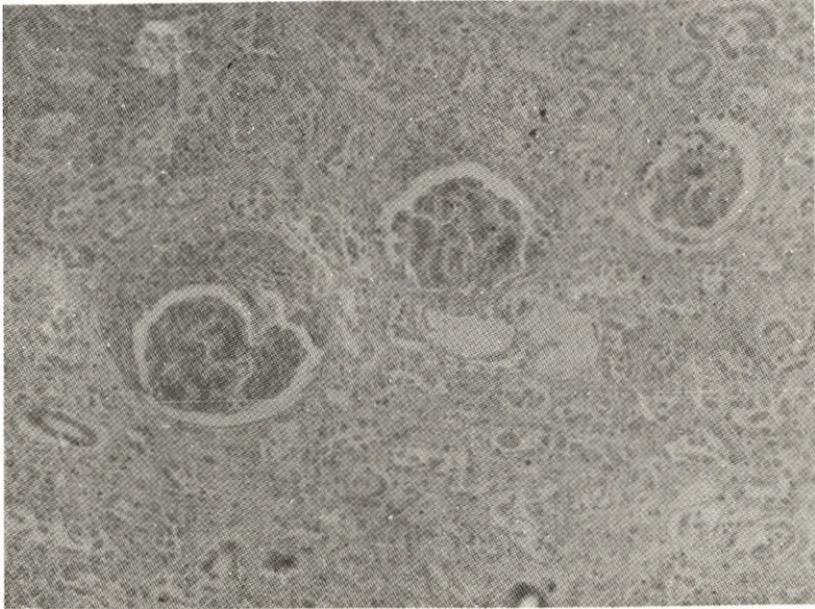


FIGURE 2— Microphotograph of kidney showing destruction of glomeruli and crescent formation. Some tubules are dilated with loss of epithelium and infiltration with mononuclear cells in the interstitium. (H & E X 70)

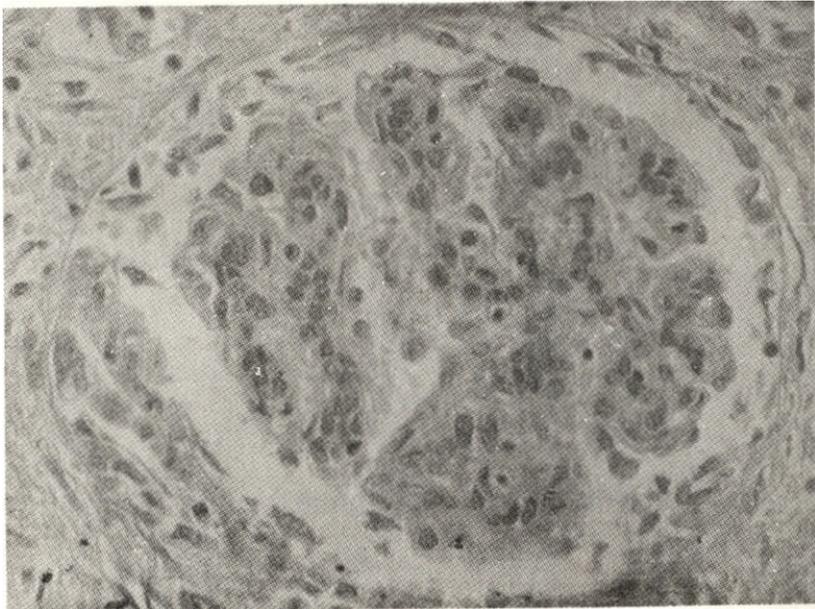


FIGURE 3 — Kidney microphotograph showing hypercellularity of tuft and formation of crescent and suggestive of rapidly progressive glomerulonephritis. (H & E X 280)

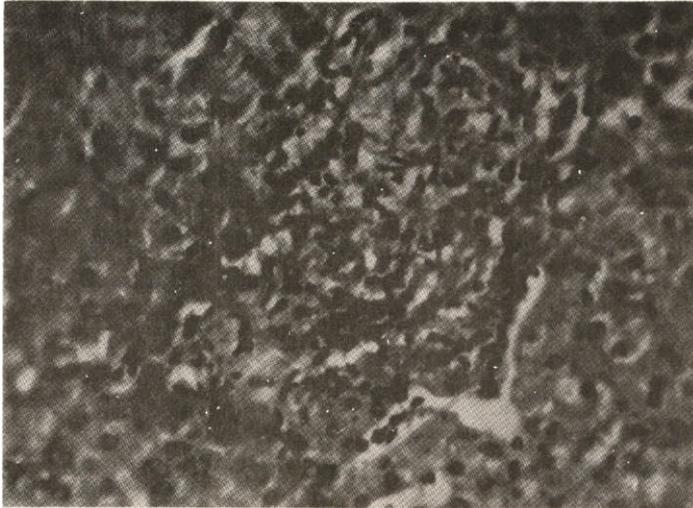


FIGURE 4

Microphotograph of liver showing typical microgranuloma studded with acid fast bacilli. (H & E X 280).

### 3 DISCUSSION

Visceral involvement in leprosy was reported as early as in 1936 by Arning and later on many workers supported his statement.<sup>1-5</sup> Bernard and Vazquez<sup>7</sup> reported in autopsy study that 31.2% of deaths in leprosy were due to renal insufficiency and following that various studies revealed that renal involvement is common in leprosy<sup>4,8,9</sup>, which may be attributed to direct invasion, pathogenic hypersensitivity or degenerative phenomenon. Accordingly, the clinical manifestations are variable in the forms of nephritis and amyloid degeneration. The present case of ENL developed progressive oliguria, anaemia and azotaemia with fatal outcome as a result of acute renal failure and renal histology revealed changes of rapidly progressive crescentic glomerulonephritis which is said to be

a rare event<sup>10</sup>. This entity develops abruptly and displays little tendency for spontaneous or complete recovery resulting to renal failure within weeks<sup>11,12</sup>.

Evidences of renal involvement in cases of lepromatous leprosy especially those who are subjected to ENL is being increasingly recognised<sup>2,13</sup>. Presence of oedema, proteinuria and other biochemical abnormalities in the reactional phase of leprosy are related to repeated episodes of ENL and immune complex deposition<sup>6,10</sup>. The incidence of nephritis as well as amyloid degeneration is allegedly greater in patients having repeated episodes of reactions and being an Indian patient the chances of amyloid change are less<sup>2,4,5</sup>. So immune complex deposition from recurrent ENL might have induced renal damage of the nature of acute proliferative (crescentic) glomerulonephritis in this case.

RESUMO — Um homem de meia idade (48 anos) com doença de curta duração (7 dias) foi admitido com quadro de insuficiência renal aguda e *erythema nodosum hansenicurn*. Tinha apresentado episódios repetidos de *erithemanodosumhansenicurn* no passado. Sua pressão arterial era normal (150/80 thm Hg). Durante nova permanência no hospital apresentou piora progressiva dos quadros de anemia (1-1: 8,8 g/dl até 7,2 g/dl), oligúria (eliminação urinária: 2050-350 ml/dia), uremia (ureia sangínea: 198 mg/dl até 218 mg/dl) bem como dos testes de função renal, vindo a falecer. A necrópsia, os rins apresentaram-se congestionados e pesando 150 g cada, com alterações histopatológicas correspondentes a uma glomerulonefrite de evolução rápida e progressiva (crescátrica), como resultado da deposição de complexos imunes a partir de episódios recorrentes de *erythema nodosum hansenicurn*

**Palavras chave:** Hanseníase. *Erythema nodosum hansenicurn*. Glomerulonefrite.

#### REFERENCES

- 1 BEDI, T.R.; KAUR, S.; SINGHAL, P.C.; KUMAR, B.; BANERJEE, C.K. Fatal proliferative glomerulonephritis in lepromatous leprosy. *Lepr. India*, 49(4): 500-503, 1977.
- 2 BERNARD, J.C. & VAZQUEZ, C.A.J. Visceral lesions in lepromatous leprosy: study of sixty necropsies. *Int. J. Lepr.*, 41(1): 94-101, 1973.
- 3 BRENNER, B.M. & STEIN, J.H., eds. *Acute renal failure*. New York, Churchill Livingstone, 1980. 296p. (Contemporary Issues in Nephrology, v.6)
- 4 DESIKAN, K.V. & JOB, C.K. A review of post-mortem findings in 37 cases of leprosy. *Int. J. Lepr.*, 36(1): 32-44, 1968.
- 5 DRUTZ, D. & GUTMAN, R.A. Renal manifestation of leprosy: glomerulo-nephritis, a complication of erythema nodosum leprosum. *Amer. J. Trop. Hyg.*, 22(4):496-502, 1973.
- 6 GLASSOCK, R.J. & BRENNER, S.M. The major glomerulopathies. In: HARRISON'S principles of internal medicine. 10.ed. Auckland, McGraw-Hill, c1983. p.1632-1642.
- 7 GUPTA, S.C.; BAJAJ, A.K.; GOVIL, D.C.; SINHA, S.N.; KUMAR, R. A study of percutaneous renal biopsy in lepromatous leprosy. *Lepr. India*, 53(2): 179-184, 1981.
- 8 JOHNY, K.V.; KARAT, A.B.A.; RAO, P.S.S.; DATE, A. Glomerulonephritis in leprosy: a percutaneous renal biopsy study. *Lepr. Rev.*, 46(1): 29-37, 1975.
- 9 MITSUDA, K. & OGAWA, M. A study of one hundred and fifty autopsies on cases of leprosy. *Int. J. Lepr.*, 5(1): 53-60, 1937.
- 10 MITTAL, M.M.; AGARWAL, S.C.; MAHESHWARI, H.B.; KUMAR, S. Renal lesion in leprosy. *Arch. Path.*, 93: 8-12, 1972.
- 11 NIGAM, P.; GOYAL, B.M.; SAMUEL, K.C. Study of kidney changes in leprosy. In: REGIONAL CONGRESS ON DERMATOLOGY, 2., Bangkok, 1977. p.17-22.
- 12 RAMANUJAM, K.; RAMU, G.; BALAKRISHNAN, S.; DESIKAN, K.V. Nephrotic syndrome complicating lepromatous leprosy. *Indian J. Med. Res.*, 61(4): 548-556, 1973.
- 13 SAINANI, G.S. & RAO, K.V.N. Renal changes in leprosy. *J. Ass. Phycns. India*, 22(9): 659-664, 1974.