RAPIDLY PROGRESSIVE (CRESCENTRIC) GLOMERULONEPHRITIS IN ERYTHEMA NODOSUM LEPROSUM

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A middle aged man was hospitalised in the state of acute renal failure with erythema nodosum leprosum. He was having progressive anemia, oliguria, azotemia and impaired renal function. The outcome was fatal. Autopsy revealed smooth congested kidney with histopathological features of rapidly progressive (crescentric) glomerulo-nephritis, presumably a result of immune complex deposition from recurrent erythema nodosum leprosum episodes.

Key words : Erythema nodosum leprosum, acute renal failure, rapidly progressive (crescentric) glomerulo-nephritis.

Lepromatous leprosy is well known for its multivisceral involvement. Specific leproma in kidney is infrequent, but a variety of non-specific lesions such as secondary amyloidosis, acute or chronic glomerulo-nephritis and pyelonephritis etc have been described.[1,2] Kidney lesions may occur in lepromatous leprosy with normal renal functions.[3] However, nephritis complicating lepromatous leprosy may attain an alarming significance because of its fatal outcome[4] which may be related to repeated attacks of erythema nodosum leprosum in the course of the disease. We report a case of lepromatous leprosy with recurrent erythema nodosum leprosum who developed acute renal failure with a fatal outcome.

Case Report

A 48 years aged man was admitted with a history of fever, vomiting, swelling over the face and decreased urine output for 7 days. He was taking treatment for the past 7 years for diffuse skin infiltration, numbness and epistaxis. During irregular treatment for leprosy he had had repeated episodes of erythema nodosum leprosum which were relieved with salicylates, clofazimine and corticosteroids. On admission to our hospital too he had erythema nodosum leprosum.

Clinical examination revealed puffy leonine face, pitting oedema over the feet, erythematous and tender nodular lesions over the extremities, ear lobules and forehead, and plaques of diffuse infiltration and atrophic areas over the chest, abdomen, face and limbs. Distal portions of the right great, 2nd and 3rd toes showed resorption. Ulnar and lateral popliteal nerves on both sides were cord-like and tender with stocking type of hypoanaesthesia. Blood pressure was 150/80 mm. Liver was 5 cm enlarged and non-tender. A systolic murmur was audible over the apex of the heart.

Routine investigations revealed hemoglobin 8.8 gm%, total leucocyte count 10800/mm^3, polymorphs 46%, lymphocytes 48%, eosinophils 6% and erythrocyte sedimentation rate 35 mm. The daily urine output was 250-350 ml, having urinary protiens 1.5 gm/day, 5-8 red blood cells, 10-15 pus cells and 1-4 granular casts per high power field. Urine culture did not reveal any pathogenic micro-organisms. Xray chest showed chronic bronchitis and electrocardiogram was normal.

Blood chemistry revealed azotemia (blood urea 198 mg/dl), serum creatinine 5.2 mg/dl,

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calcium 9.5 mg/dl, phosphate 4.2 mg/dl, protein 7.2 gm/dl, albumin 3.5 gm/dl, globulin 4.2 gm/dl, bilirubin 10 μmol/l and alkaline phosphatase 32 U[3]. Renal function tests were impaired, raised blood urea (BUN) and creatinine ratio was 25 : 1, urine to plasma ratio of creatinine was 82:1 and urea 16:1. Urea and creatinine clearances were 42 ml/min and 72 ml/min respectively. On 12 hours restriction of fluid the specific gravity of urine was 1018, and after 12 hours of deliberate fluid intake it was 1008.

The patient remained oliguric and azotemic for 3-4 days. The daily urine output improved to 500-800 ml/day. Before death, his hemoglobin came down to 7.2 gm%, blood urea rose to 218 mg/dl, creatinine to 7.2 mg/dl and alkaline phosphatase to 42 U[3]. Throat swab, blood and urine cultures were sterile. Patient's condition deteriorated and he expired on the 9th day of admission to the hospital. At autopsy, the kidneys were noticed to be smooth with congested surface and weighed 150 gm each. Histopathology (Fig. 1) revealed diffuse involvement of the glomeruli with variable changes indicating proliferation and sclerosis, some with segmental extracapillary proliferation (crescent). At places, an intense inflammatory reaction invaded the glomerular tufts. There was a mononuclear cell infiltration in the interstitium with alternating tubular dilatation and atrophy. Multiple lepromatous granulomas were seen in the skin, nasal mucosa and liver. Spleen and lymph nodes showed moderate replacement of the thymus-dependant lymphocytes by foamy cells. *Mycobacterium leprae* was not seen in the sections from skin, nasal mucosa and liver.

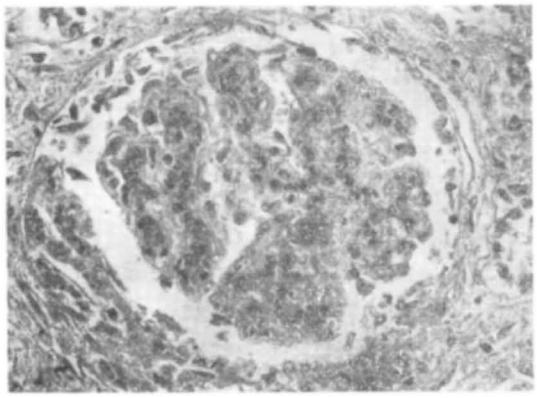

Fig. 1. Microphotograph of kidney showing hypercellularity of tuft and formation of crescent suggestive of rapidly progressive (crescentric) glomerulo-nephritis.

Comments

Bernard and Vazquez[5] reported that 31.2% deaths in leprosy were due to renal insufficiency. Renal involvement in leprosy may be attributable to direct invasion, pathogenic hypersensitivity or degenerative phenomenon.[3-5] In the case of reactions, especially with repeated bouts, it is likely to lead to renal involvement, and sometimes it may be very severe as in the present case. Renal involvement in reactions is caused by the deposition of the circulating immune complexes on the glomeruli which are especially susceptible to damage as the reactants have to pass through them and may get precipitated in them.[6]

Tin Shwe[7] studied the renal biopsy material of 7 patients and concluded that renal impairment is due to deposition of antigen-antibody complexes in the glomeruli. Drutz and Gutman[4] correlated the abnormal urinary findings suggestive of acute glomerulo-nephritis with history of erythema nodosum leprosum, with hematuria, proteinuria and red blood cells or hemoglobin casts and concluded that both glomerular and distal renal function may be impaired, presumably on an immunological basis. Dharmendra[8] and Bullock et al,[9] considered renal changes in leprosy to be toxic in nature caused by the large number of disintegrating leprosy bacilli or their products.

Erythema nodosum leprosum with progressive oliguria, anemia and azotemia and a fatal outcome as a result of acute renal failure and progressive crescentric glomerulo-nephritis is

a rare event.[10] This develops abruptly and displays little tendency for recovery resulting in renal failure within weeks.

Renal involvement in cases of lepromatous leprosy especially those who are subjected to erythema nodosum leprosum is being increasingly recognised.[11] Presence of oedema, proteinuria and other biochemical abnormalities associated with repeated episodes of erythema nodosum leprosum and immune complex deposition,[4,8,10] should alert the physician. The incidence of nephritis as well as amyloid degeneration is allegedly greater in patients having repeated episodes of reactions, although in Indian patients amyloid changes are less frequent.[2,3]

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