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Case Report

Renal Involvement in Leprosy: A Case Report

A K M M Islam[1], E Alam[2], A Rezzak[3], N Huda[3]

Abstract

Leprosy is a chronic granulomatous multisystem disorder. Renal involvement is one of the dangerous complications of leprosy. Kidneys are usually involved during splanchnic localization of leprosy. The histopathological renal lesion spectrum includes spectrum of glomerulonephritis, renal amyloidosis and interstitial nephritis. Here we report a case of leprosy who presented with diffuse membranoproliferative glomerulonephritis with renal failure.

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Introduction

Of the diseases that have affected man kind from its earliest days, including plague, cholera, smallpox and tuberculosis, leprosy also known as Hansen's disease (HAD) – undoubtedly remains an emergency in certain countries.

Leprosy, a chronic, granulomatous, multisystem disease caused by *Mycobacterium leprae*, primarily affects the skin and peripheral nervous system and presents with two types of granulomatous lesions:

Macrophagic (Lepromatous leprosy), which is marked by a strong immunologic response, and epitheloid (Tuberculoid leprosy), which is characterized by a weak or negative immunologic response.[1]

This disease continues to be prevalent in most parts of Asia, Africa and Latin America. The global number of leprosy cases was estimated around 10 to 12 million in the 1970's and 1980's, but according to newer estimates (WHO 1991), the number has decreased to about 5.5 million.

Bangladesh was considered as high endemic country. About 13 million cases were registered in 1993. About 90% cases were tuberculoid. Lepra type II reaction mostly causes the glomerulonephritis.[2]

The kidney is one of the target organs during the splanchnic localization of leprosy. The histopathological renal lesion spectrum includes GN (diffuse endocapillary proliferative, membranoproliferative, focal proliferative, membranous and crescentic), renal amyloidosis (RA) and interstitial nephritis.[3, 4, 5]

Case Summary

A 40-year-old married male, day-labour from Natore was admitted in the Nephrology ward of Rajshahi Medical College Hospital, Rajshahi with

[1] Associate Professor, Department of Nephrology, Rajshahi Medical College, Rajshahi.
[2] Registrar, Department of Department of Nephrology, Rajshahi Medical College, Rajshahi.
[3] Indoor Medical Officer, Department of Medicine, Rajshahi Medical College, Rajshahi.

the complaints of non-healing ulceration over the dorsum of feet and legs. Those ulcers were painless, deep with sloughing of tissue and healed poorly with antibiotics. About 15 days later the patient noticed gradual swelling of the whole body, started initially in the legs and then involved the face and abdomen. At the same time, patient noticed high colored urine with reduction in volume. The patient did not give any history of loin pain or dysuria or frequency of micturition or any other urinary abnormalities.

On past medical history the patient was diagnosed as a case of lepromatous leprosy by Danish-Bangladesh leprosy Mission, Nilfamary (DBLM). The diagnosis was confirmed by slit-skin smear test. He was treated by three drugs (Multi-drug Treatment – MDT, Rifampicin, Dapsone, Clofazimine) for 5 years. But he recovered with deformity. He had amputation of several fingers and toes and developed planter ulcer due to anaesthetic feet. On examination he was ill looking with puffy face, mildly anaemic, grossly oedematous, mildly hypertensive and without any organomegaly.

On neurological examination, loss of pain, touch and temperature sensation over the lower legs, feet and hands were noted. Other systems revealed no abnormality.

On investigations, urine analysis revealed gross proteinuria, haematuria, Pus cells- 15-20/HPF and there were granular casts. Urinary total protein (UTP) was 3.6 gm/24 hours. Blood examinations showed Hb- 6gm/dl, ESR- 32 mm in 1st hour, TLC- 7500/mm^3, DLC- N- 65%, L- 28%, M- 04%, E- 03%, serum total protein was 6.4 gm/dl, and s/albumin was 3.5gm/dl, RBS- 6.0 mmol/L, serum cholesterol - 150mg/dl.

His serum creatinine initially was 2.5 mg/dl (01.07.04) and gradually became 5.0 mg/dl (26.07.04). On ultrasonographic examination, kidney size was normal with increased cortical echogenicity and poor cortico-medullary differentiation.

Renal biopsy report showed diffuse membranoproliferative glomerulonephritis. His clinical diagnosis was leprosy with diffuse membranoproliferative glomerulonephritis with renal failure.

Discussion

Leprosy is a chronic granulomatous disorder caused by acid fast bacillus Mycobacterium leprae. The usual mode of presentation is with anaesthetic skin lesions, peripheral neuropathy and palpable enlargement of peripheral nerves.

Renal involvement was studied in 70 patients with leprosy by urine analysis, detailed bio-chemical investigations and renal histopathology. Creatine clearance was reduced in 20 patients.

Renal biopsies were studied in 50 cases; of which in 13 cases abnormal histopathological lesions were found by light microscopy. Amyloidosis was seen in only one lepromatous patient. No acid fast bacilli and leproma like lesion were demonstrated in any case.[6] Another study revealed that the duration of the disease has a significant relationship with renal involvement, proteinuria, microscopic haematuria, granular and hyaline casts are mainly seen in lepromatous cases and specially with lepra reaction (100%) while few of the non-lepromatous (2%) cases may show these abnormalities. Impaired renal functions are mostly observed in lepromatous cases.[7] Significant histopathological lesions were observed in lepromatous patients (50%) as compared to non-lepromatous patients (20%). The pathological changes were predominantly of chronic glomerulonephritis followed by chronic pyelonephritis and interstitial nephritis.[8]

Renal involvement in 13 non-lepromatous and 17 lepromatous leprosy patients were assessed by routine urinalysis, detailed biochemical analysis of blood and urine and by renal histopathological studies and compared with 10 normal healthy controls. The presences of RBC and pus cells were detected in the urinary deposit of only one lepromatous leprosy patient in reactional phase. 47% of the non-lepromatous and 46% of the lepromatous patients had proteinuria. The creatinine clearance was low in 82.3% of the non-

lepromatous and in all of the lepromatous patients. Twenty one percent renal biopsy specimens showed non-specific pathological changes such as nephritis of various varieties in 71.4% of the specimens.

Among the lepromatous group, renal involvement was observed in 5 out of 9 cases (55.6%) and in the non-lepromatous group 10 out of 12 cases (83.3%). No acid fast bacilli, amyloid and granuloma were seen in any of the renal tissues studied. None of the patient showed any clinical evidence of renal involvement.

Renal involvement was found in our patient. On renal biopsy he was diagnosed as a case of diffuse membranoproliferative glomerulonephritis with moderate renal failure.

It is concluded that leprosy is a chronic granulomatous multisystem disorder. Renal involvement is one of the dangerous complications of leprosy.

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All correspondence to:
A.K.M. Monoarul Islam
Associate Professor & Head,
Department of Nephrology
Rajshahi Medical College, Rajshahi.