Tropical Nephropathy

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By
Hani Abd El-Ghani Mohamed
M.B.B.Ch.

Supervised by:
Prof. Dr./ Mohamed Mahmoud Abd El-Ghany
Professor of Nephrology
Faculty of Medicine – Ain Shams University

Prof. Dr / Osama Mahmoud Mohamed
Assistant Professor of Nephrology
Faculty of Medicine – Ain Shams University

Dr / Essam Nour Eldin Afiffy
Lecturer of Nephrology
Faculty of Medicine – Ain Shams University

Faculty of Medicine
Ain Shams University
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Abbrev	
ARF	Acute renal failure
BCG	Bacille calmette-guérin
CAPD	Chronic ambulatory peritoneal dialysis
ESRD	End-stage renal disease
FSGS	Focal and segmental glomerulosclerosis
GN	Glomerulonephritis
HAART	Highly active antiretroviral therapy
HIV	Human immunodeficiency virus
HIVAN	Hiv associated nephropathy
HUS	Haemolytic uraemic syndrome
Ig	Immunoglobulin
MARF	Malarial acute renal failure
MGN	Membranous glomerulonephritis
MPGN	Membranoproliferative glomerulonephritis
NHANES	National health and nutrition examination survey
VL	Visceral leishmaniasis
INF	Interferon

Introduction

The word 'tropics' is more than a geographical expression. The tropics are the home of large numbers of poor disadvantaged and malnourished people, in whom severe bacterial infections are an important cause of death. These unfortunate people have autopsy findings rarely seen in the Western developed world during the last half century. Many cases with severe infections had secondary renal lesions capable of causing acute renal failure (ARF). These included multiple renal abscesses, intravascular coagulation and acute tubular necrosis ***(Date, 1994)***.

Tropical nephropathy covers renal diseases commonly seen in the tropics and specific tropical renal diseases seen mostly or only in the tropical area. Pathologically, all renal structures can be affected. There is, therefore, a broad spectrum of pathologic changes, and clinical renal manifestations vary from mild urinary sediment changes to acute renal failure. Inflammatory processes play an essential role in the pathogenesis of renal involvement in infection and toxin groups ***(Sitprija, 2003).***

Renal diseases unique to the tropics are those that occur in association with infectious diseases including dengue hemorrhagic fever, typhoid fever, shigellosis, leptospirosis, lepromatous leprosy, malaria, and schistosomiasis. These renal complications can be classified on the basis of their clinical and pathologic

characteristics into acute transient reversible glomerulonephritis, chronic progressive irreversible glomerulonephritis, amyloidosis, and acute renal failure (ARF) resulting from acute tubular necrosis, acute tubulointerstitial nephritis, and thrombotic microangiopathy ***(Boonpucknavig and Soontornniyomkij, 2003)***.

In areas where human immunodeficiency virus (HIV) and malaria are common, these may be factors predisposing to renal disease. Primary glomerulonephritis also occurs in these regions, and the nature of the renal lesion can only reliably be determined if renal biopsy and expert histologic analysis are available ***(Mathieson, 2006)***.

Acute renal failure may be encountered in several parasitic infections, including those which lead to profound systemic illness, leading to acute tubular necrosis (e.g. falciparum malariae); those associated with acute interstitial nephritis (e.g. leishmaniasis) and occasionally those associated with the acute nephritic syndrome (e.g. trichinosis) ***(Barsoum, 1999)***. Renal involvement in visceral leishmaniasis (VL) is very frequent but the pathogenesis of this nephropathy is poorly understood ***(Prianti et al., 2007),***

One third of the cases of ARF, in tropics, are due to the haemolytic uraemic syndrome (HUS) complicating bacillary dysentery, and particularly those resulting from

infections caused by Shigella dysenteriae serotype. Ganulomatous interstitial nephritis seen commonly as a reaction to drugs in the Western countries is most often the result of tuberculosis in the tropics ***(Date, 1994).***

Tropical nephropathy is caused by several mechanisms varying according to the cause. For example, with visceral leishmaniasis, most probably the glomerular alterations have been considered to be a consequence of the immune response and immune complex deposition ***(Costa et al., 2000)***, while the direct infection of renal epithelial cells by the HIV virus is linked to the most common cause of chronic renal failure in HIV positive patients so called HIV associated nephropathy ***(Holden and Brook. 2002)***

Aim of the Work

This essay aims to review the tropical diseases which are associated with nephropathy, and to highlight what is common in Egypt with special emphasis on the methods of diagnosis and treatment.

Epidemiology of Tropical Nephropathy

In the mid 1970s a 'tropical nephropathy' was described in Senegalese patients with an NS showing a 'curious progressive and segmental glomerulosclerosis characterized by a flaking or fibrillary splitting of the capillary wall, seen in quartan malarial nephropathy' ***(Morel-Maroger et al., 1975)***.

Barsoum (1999) concluded that glomerular immune complex deposition in a variety of parasitic diseases may lead to 'mild and self limiting glomerulo-nephritis (GN), reflecting the critical balance of concomitant immunity'. This author proposed that some other additional autoimmune pathogenetic mechanisms may be superimposed to the parasitic process inducing progression to an NS.

Thus, the question of a possibly distinct renal pathology diagnostic of a tropical NS may be raised. In fact, the pattern of childhood nephrotic syndrome in Africa varied according to geographical, racial, environmental and even historical and methodological factors ***(Doe1 et al., 2006)***.

In most North African countries the literature on geographical pathology of NS shows a profile which seems to be similar to that in Europe ***(Elzouki et al., 1984)***, with the exception of Egypt where schistosomal–salmonella

associated glomerulopathy was described ***(Bassily et al., 1976)***.

The situation is less clear in West Africa. ***Adu et al. (1981)*** reported in the early 1980s MCNS in 14/25 children and FSGS in 12/61 patients of all age groups. By contrast, Nigerian children had a high proportion of cases with MPGN and quartan malaria nephropathy in the 1980s and MPGN in the 1990s ***(Asinobi et al., 1999)***. In the early 1970s, ***Hendrickse et al. (1972)*** had described a peculiar type of quartan malaria nephropathy with lacunae in the glomerular basement on electron microscopy; however, these latter findings were never confirmed and may have been due to an artefact during fixation.

In Nigeria, the response of the NS to corticosteroids was poor; however, 20 years later 52% of patients responded to PRED ***(Ibadin and Abiodun, 1998)***. There was also relatively high corticosteroid sensitivity in children from Togo ***(Gbadoe et al., 1999)***.

In the late 1960s, 17/25 nephrotic patients from the Ivory Coast showed a peculiar type of glomerulopathy, with focal and segmental lesions due to hyalinosis of some glomerular loops probably affecting some more than others ***(de Paillerets et al., 1972)***.

Today, there seems to be a high proportion of patients in the Ivory Coast suffering from secondary NS due to

systemic lupus and amyloidosis ***(Adonis-Koffy and Timite-Konan, 2003)***.

The HIV glomerulopathy was found in patients from Benin ***(Attolou et al., 1999)***. The analysis of 55 cases with NS in Senegal revealed 27% with MCNS, 14% with FSGS and 10% with MPGN as the most frequent histological types ***(Satgé et al., 1970)***. The literature on NS in the Sudan is scarce ***(Musa et al., 1980)***. ***Doe1 et al. (2006)*** study on renal function in 218 Sudanese school children and 58 adults failed to detect a significant number of patients with schistosomal glomerulopathy.

Concerning East Africa, John Kibukamusoke and Michael Hutt were pioneers in the study of NS in Uganda in the 1960s; however, recent studies in this country are scarce, showing a high rate of nephrotic patients with a low selective protein index in the urine, thus suggesting a low sensitivity to steroids ***(Odiit and Tindyebwa, 1997)***.

Nephrotic children and young adults in Kenya showed a high rate of mesangioproliferative glomerulonephritis (25%), MCNS (18%) and FSGS (15%) ***(McLigeyo, 1994)***.

There are few reports on NS from Central Africa, e.g. in Cameroon, 38% of patients had MCNS, 31% membranous glomerulo-nephritis (GN) and 24% proliferative GN ***(Mbakop et al., 1990)***. In Zaire, 22% of nephrotic children had MCNS, 18% amyloidosis, 16% proliferative GN, 14% MPGN and 10% membranous GN.

The FSGS was found in 6% only ***(Pakasa and Kalengayi, 1984)***.

The literature on NS in the Southern parts of Africa, especially in the South African Republic itself, is rich. The patterns of GN in Zimbabwe showed predominantly proliferative GN in 51%, followed by membranous GN and FSGS ***(Seggie et al., 1984)***. Proliferative GN and FSGS were also dominant in Malawi ***(Brown et al., 1977)***. Namibian children had mainly membranous GN ***(Buuren et al., 1999)***. Glomerulonephritis in South Africa is characterised by a high frequency in Africans, a lesser frequency in Indians and a low frequency in Caucasians ***(Seedat, 1992)***.

Bhimma et al. (1997) reviewed their 20 year experience of 636 children with NS in Durban. Membranous GN was found in 40% of Blacks, mostly associated with hepatitis B. Typical steroid-sensitive NS was uncommon. In Indian children, 47% had MCNS and 21% had FSGS. The FSGS was also frequently found in children from Natal ***(Adhikari et al., 2001)***.

In summary, there seems to be no unique pattern of the underlying histological types of GN in African children with NS and the term ‘tropical nephrotic syndrome’ could be dropped because there is no unique geographical pathology. ***Doel et al. (2006)*** claim that FSGS may be a frequent cause of steroid-resistant NS in West African