Acute eosinophilic glomerulonephritis with Bancroftian filariasis

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Summary

A case is reported of Bancroftian filariasis and acute immune complex glomerulonephritis in a 44-year-old man. Renal biopsy showed microfilariae and large numbers of eosinophils in the glomeruli (eosinophilic glomerulonephritis). The absence of other aetiological factors suggests that the glomerulonephritis may have been of filarial origin.

Introduction

Large numbers of eosinophilic leucocytes can be seen in the glomeruli of patients with acute nephritis if they have peripheral blood eosinophilia. The glomerulonephritis and the eosinophilic leucocytosis are most often due to unrelated causes, as when post-streptococcal glomerulonephritis occurs in patients who have intestinal helminthiasis (Date, Jacob and Johny, 1977). The unusual association of an eosinophilic glomerulonephritis with Bancroftian filariasis is now reported and the possibility of an aetiological relationship between the 2 conditions is discussed.

Case report

A 44-year-old man presented with pedal oedema and puffiness of the face for 15 days and increasing exertional dyspnoea for 12 days. There was no history of sore throat, skin disease, hypertension, diabetes or urinary abnormality. On examination the patient had moderate congestive cardiac failure with a BP of 190/120 mmHg. No other clinical abnormality was detected. Results of laboratory tests are shown in Table 1.

The patient was admitted to hospital and treated with diuretics, anti-hypertensive agents and diethyl-carbamazine. There was a low grade fever initially but the temperature returned to normal by the end of the second week of treatment at which time there was considerable improvement in renal function with the following laboratory results: urinary output 1290 ml/24 hr; blood urea 6.6 mmol/l; serum creatinine

221 μ mol/l; sodium 146 mmol/l; potassium 4.5 mmol/l; bicarbonate 33 mmol/l. The BP was 140/90 mmHg. The patient was now given a course of steroid therapy and showed continuing improvement in renal function at the time of discharge from hospital 10 days later.

Renal biopsy findings

Approximately 50 glomeruli were present in the tissue processed for light microscopy. There was a diffuse glomerular hypercellularity and the tufts contained variable numbers of eosinophilic and neutrophilic leucocytes (Fig. 1), with an average of 15 eosinophils and 17 neutrophils per glomerulus in a 3- μ m section. Microfilariae were present in 8 of the glomeruli. Transverse and longitudinal sections of the parasites were easily identified in the glomerular capillaries in sections stained by HE and PAS methods (Fig. 2). The eosinophils did not appear to be preferentially located near microfilariae. Mast cells were not present. The extraglomerular tissues were normal except for a solitary aggregate of inflammatory cells, including eosinophils, in the interstitium.

Seven glomeruli were present in the sections stained with fluorescein-labelled antisera. Conspicuous irregular granular discontinuous deposits of IgG and C3 were seen distributed along the glomerular capillary walls. Fibrin, IgA and IgM could not be detected.

Discussion

The diffuse proliferative exudative glomerulonephritis seen on light microscopy, the granular deposits of IgG and C3 detected by immunofluorescence microscopy and the hypocomplementaemia indicate that this patient had an acute immune complex type of glomerulonephritis (Germuth and Rodriguez, 1973). The glomerular morphology was unusual in that large numbers of eosinophils were present in addition to neutrophils. It is known that both eosinophils and neutrophils are attracted by

TABLE 1. Results of laboratory investigations

Investigation	Result
24-hr urine volume	820 ml
24-hr urine protein	6.9 g
12-hr Addis count:	
Erythrocytes	120 million
Leucocytes	9.6 million
Blood urea	33.9 mmol/l
Serum creatinine	322.7 μ mol/l
Serum cholesterol	4.4 mmol/l
Total serum protein	57 g/l
Serum albumin	28 g/l
Serum sodium	137 mmol/l
Serum potassium	5.3 mmol/l
Serum bicarbonate	17 mmol/l
Peripheral blood smear	<i>Microfilaria bancrofti</i> present No malarial parasites
Haemoglobin	11.6 g/dl
Total leucocyte count	$13.6 \times 10^9/l$
Differential count:	
Neutrophils	44%
Eosinophils	34%
Lymphocytes	22%
Absolute eosinophil count	$6.8 \times 10^9/l$
Erythrocyte sedimentation rate	75 mm/1st hour
Platelet count	$150 \times 10^9/l$
Prothrombin time	13.5 sec (control 13 sec)
Antistreptolysin O titre	170 Todd units
Anti-hyaluronidase titre	64 units
Anti-DNase B titre	150 units
Serum total complement	60% (control 92%)
C3	10%
C4	120%
Lupus erythematosus cell test	Negative (on 3 occasions)
Anti-DNA antibody	Negative (on 2 occasions)
VDRL	Non-reactive
Serum HBs Ag	Negative
Urine and stool inoculation into primary monkey kidney tissue culture	No viral isolate
Stool microscopy	Negative

FIG. 1. A typical glomerulus showing a hypercellular tuft containing many neutrophilic and eosinophilic leucocytes (HE, $\times 515$).

FIG. 2. Glomerulus with a capillary loop containing longitudinal sections of a microfilaria (PAS, $\times 560$).

immune complexes (Litt, 1964; Henson, 1971) and are present in the glomerular exudate in acute glomerulonephritis in the same proportion as in the peripheral blood (Date *et al.*, 1977). Eosinophilic glomerulonephritis is therefore a reflection of a high blood eosinophil count which in this patient was due to filariasis.

Acute immune complex glomerulonephritis is most often poststreptococcal in origin, but it is an unlikely aetiological factor in this patient since there was no preceding skin or throat infection, and antibody titres to all 3 streptococcal antigens studied were normal. The investigations (Table 1) would also appear to exclude other well known causes of glomerulonephritis. These negative results and reports of glomerulonephritis occurring in association with many types of human filariasis (Bariéty *et al.*, 1967; Zuidema, 1971; Pillay, Kirch and Kurtzman, 1973; Couzineau, Bourée and Molimard, 1973; Chugh *et al.*, 1978) suggest that the glomerulonephritis in this patient may have been of filarial origin.

The patient was seen last at the nephrology clinic 5.5 months after his discharge from hospital. He was asymptomatic with no oedema or haematuria, and with a BP of 130/80 mmHg. Results of laboratory investigation were as follows: serum creatinine, 106 µmol/l; sodium, 147 mmol/l; potassium, 3.7 mmol/l; bicarbonate, 28.5 mmol/l; blood urea, 5.8 mmol/l; serum total protein, 59 g/l; albumin, 36 g/l; urinary protein excretion, 0.8 g/24 hr; urine volume, 1220 ml/24 hr.

Acknowledgments

We are grateful to Mr M.S. Irudayaraj for typing the manuscript and to Mr Roy Charles Nayagam and Mr K. Bhaskar Babu for technical assistance.

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