also reported in their series of 25 HIV-positive patients presenting with proteinuria, 2 patients with cryptococcal renal infiltration. The patient described in the case report had evidence of macroalbuminuria and tubular dysfunction as evidenced by significant urinary sodium loss on the 24-h urine specimen. This may be attributable to both glomerular and tubulo-interstitial cryptococcal invasion. Unlike the index patient who had a minimal inflammatory response, attendant inflammation evoked by the organisms may also contribute to the proteinuria in patients with cryptococcal nephritis.

Conclusion

This case report highlights the varied aetiology of proteinuria in the HIV-infected patient and brings to the fore the fact that not all gross proteinuria in an HIV-infected patient is secondary to HIV-associated nephropathy.

Conflict of interest statement. None declared.

[1]Department of Medicine
[2]Department of Pathology, The Aga Khan University Hospital, Nairobi Kenya
E-mail: jay_shavadia@aku.edu

Jay Shavadia[1]
Shahin Sayed[2]
Gunturu Revathi[2]
Ahmed Sokwala[1]
Mohamed Said Abdullah[1]

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Diabetic glomerular disease: pitfalls in diagnosis

Sir,

Diabetic nephropathy is a common but not an invariable complication of type 1 and 2 diabetes mellitus. However, diabetes mellitus itself is very common, and therefore, other causes of renal impairment co-exist in this population. Even with a renal biopsy, the correct diagnosis may not always be immediately evident as other pathologies can also mimic diabetic nephropathy. We present a case where diabetes mellitus was incorrectly assumed to be the cause of the patient's renal impairment.

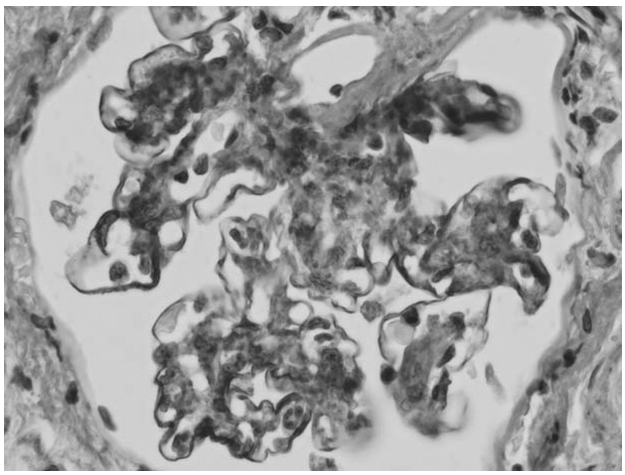

Fig. 1. Mesangial deposits of IgA × 400.

The case is of a 54-year-old man diagnosed with type 1 diabetes mellitus 20 years earlier. He had proliferative retinopathy and autonomic neuropathy but no proteinuria and no hypertension. He developed sudden onset nephrotic syndrome with eGFR >60 ml/min/1.73 m^2. On renal biopsy, the light microscopic appearance was of a diffuse and nodular diabetic glomerulosclerosis with arteriolar hyalinosis. However, the degree of mesangial and endocapillary proliferation was atypical. EM examination disclosed large mesangial, para-mesangial and small extramesangial sub-endothelial electron-dense deposit but no specific features of light chain deposition disease. Over the next 6 months, his eGFR deteriorated to 9 ml/min/1.73 m^2. The repeat biopsy revealed a new focal necrotising and crescentic glomerulonephritis. On immunohistochemistry, there were IgA and C3 deposits in the glomerular mesangium and extra-mesangial capillary walls (Figure 1). The final histological diagnosis was that of diffuse (endocapillary) proliferative glomerulonephritis–IgA nephropathy superimposed on diffuse and nodular diabetic glomerulosclerosis that later evolved into a focal and necrotising crescentic glomerulonephritis. The patient's renal function deteriorated further and he started dialysis.

The sudden onset of nephrotic syndrome in our case should suggest an alternative diagnosis to diabetic nephropathy, as this alone would usually be preceded by lesser degrees of proteinuria.

Despite diabetic nephropathy being the leading cause of end-stage renal failure in the Western world, we must remain suspicious for alternative non-diabetic causes of renal impairment that may co-exist with diabetic nephropathy. Furthermore, other diseases, in particular light chain deposition disease, can mimic the typical nodular lesions of diabetic glomerulosclerosis. Our case emphasizes the importance of immunochemical and ultrastructural evaluation of biopsy tissue submitted from all such patients [1].

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[1]Department of Nephrology and Transplantation, Guy's & St Thomas' NHS Foundation Trust
[2]Department of Renal Medicine King's College Hospital NHS Foundation Trust
[3]Department of Pathology, Guy's & St Thomas' NHS Foundation Trust
[4]School of Clinical Medical Sciences, Newcastle University, UK
E-mail: Neil.Sheerin@newcastle.ac.uk

Victoria Ingham[1]
Satish Jayawardene[2]
Patrick O'Donnell[3]
Neil Sheerin[4]

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How to define a cut-off value of tumour markers in haemodialysis patients?

Sir,

Biological tumour markers in haemodialyzed patients suffer from a high false positive rate, particularly CEA, C19-9 and CA 125. We conducted a study in haemodialysis patients without diagnosed malignancy to evaluate if a threshold value, defined by the 95th percentile of this cohort, could be proposed for these markers in this population.

A total of 105 dosages of each marker were done on 75 patients (immunometric assay, Immulite 2000, DPC). For very high values, markers were monitored at least twice and major causes of elevated level were checked. Twenty patients with normal or high values undertook a second sample to study dosage variability.

In 75 patients, the mean value of CEA, CA 125 and CA 19-9 was equal respectively to 4.8 ± 3.9 ng/mL, 25 ± 51 ng/mL and 47 ± 122 U/mL (Table 1). The false positive rate of each marker was concordant with the literature: CEA 34%, CA 125 33% and CA 19-9 22% (Tables 2–4). The 95th percentile of each marker was equal to CEA 12.7 ng/mL, CA 125 119 ng/mL and CA 19-9 294 U/mL. The very high level of the 95th percentile of CA 125 and CA 19-9 does not permit us to define a threshold value. Some very high levels of CA 125 were associated with fluid overload and lessened with the decrease of the dry weight of the patients. The 95th percentile of CEA stands in common values known to be frequent in patients with non-malignant causes of elevated level of this marker. A CEA cut-off value around 13 ng/mL in haemodialysis patients could be proposed using immunometric assay.

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[1]Nephrology Department
[2]Biology Department, Pitié Salpétrière Hospital, 83 bd de l'hopital, 75013 Paris, France
E-mail: lucile.mercadal@psl.aphp.fr
[3]Department of Biostatistics and Medical Informatics and Pitie-Salpetriere Charles-Foix Clinical Research Unit, Modeling in Clinical Research, EA 3974 University Pierre et Marie Curie Paris, France Assistance Publique-Hopitaux Paris, France AP-HP

Lucile Mercadal[1]
Sylvie Cormont[2]
Sophie Tezenas-du-Montcel[3]
Sabria Hacini[1]
Marcia Venditto[1]
Gilbert Deray[1]

Table 1. Results of the tumour markers in a cohort of 77 haemodialysis patients on 105 dosages

	CEA (ng/mL)	CA125 (ng/mL)	CA199 (U/mL)
Mean	4.7	50.2	27.4
Standard deviation	3.9	120.2	49.9
Range	0.8–21	<1–722	<2.5–389
% False positive rate	34%	33%	22%
Median	3.6	11.3	8.66
95th percentiles	12.7	119	294
Reference values in healthy population	<5	<21	<33

Table 2. CEA in haemodialysis patients in the recent literature

HD	*n*	Dosage	Reference cut-off	CEA mean (ng/mL)	CEA false-positive rate
Filella, *Int J Biol Markers*, 1990 [1]	36	Abbott, IRMA	3.5 ng/mL	5.05 ± 5.02	47%
Eskiocak, *Nephrol Dial Transplant*, 1995 [2]	32	IRMA		6.03 ± 0.45	
Arican, *Transplant Proc*, 1999 [3]	50	Abbott, IRMA		5.87 ± 11.1	
Zeferos, *Nephron* 1991 [4]	23	IRMA		5.45 ± 0.9	
Walz, *Am J Nephrol*, 1988 [5]	93	Abbott, IRMA	5	3.93	
Odagiri, *Am J Nephrol*, 1991 [6]	144	Dinabot, RIA	2.5	2	25.7%
Polenakovic, *Int J Artif Organs*, 1997 [7]	62	Cobas, EIA		4.06	41%
Arik, *Intern Urol Nephrol*, 1996 [8]	35	Abbott, IRMA		2.6 ± 0.3	
Nomura, *Oncol Rep*, 1998 [9]	73	Eiken, IRMA	2.4	3.4 ± 2.4	