

RENAL DISEASE PRESENTING AS ASYMPTOMATIC PROTEINURIA

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In 14% of the patients subjected to renal biopsy over a 5-year period, proteinuria was detected accidentally as a result of screening or of occupational fitness tests. Microscopic and immunohistologic studies of the biopsy specimens of the practically asymptomatic patients showed the most different and in more than one case advanced, renal processes in the oligosymptomatic clinical disguise. This justifies a close investigation and follow-up in every case of newly detected persistent proteinuria. The importance of early detection of proteinuria and screening of the population, as the most efficient means to this end, are emphasized.

Introduction

The meaning of the definition “persistent, asymptomatic urinary abnormality” is being increasingly realized. This is not only because the cases of identified origin are steadily increasing in number, but also because it has been confirmed that asymptomatic proteinuria and haematuria are in the majority of cases a disguise of histologically and immunologically active renal processes. “Persistent asymptomatic urinary abnormality” is merely a collective term covering renal conditions of the most diverse aetiological, microscopic and clinical types.

Even the closest clinical study fails in the majority of cases to provide adequate clues to the nature or to the microscopic and immunohistological types of the disease, since the meagre symptomatology may be the expression of anything between an acute nephritis under way of recovery and of an oligosymptomatic chronic nephritis. This is consistent with extensive observations, including those by Muth [17] which were, however, not concerned with immunohistological features, Cameron [6, 7], Szabó [25], Varga [32] and by the present authors [14, 23]. Study of the microscopic and immunohistologic features is thus of prime importance by furnishing diagnostic evidence and by providing therapeutic and prognostic guidelines. In the present study, the cases of asymptomatic proteinuria in a 5-year biopsy material have been analysed, consideration being given to the clinical, microscopic and immunohistologic features.

Patients and methods

The present review covers those practically sign- and symptom-free cases in which renal disease has been detected accidentally as a result of screening or in the course of occupational fitness tests. A total of 63, i.e. 14.3%, of 440 patients subjected to renal biopsy between 1975 and 1980, suited these criteria. The 28 cases in which proteinuria had been detected at pregnancy clinics, have not been included in the study, since it is impossible to ascertain retrospectively whether proteinuria had been in existence earlier or whether it was connected with pregnancy.

The types of medical examination resulting in the detection of proteinuria and the respective number of patients are presented in Table I.

Table I
Types of screening

Type of tests	Number of patients
Test for occupational fitness	24
Blood donor tests	15
Driving license tests	9
Conscription	3
Screening of schoolchildren	2
Other types of screening	10
Total	63

All further data refer to the time of biopsy.

The patients were divided into age- and sex-related groups. In the history, possible aetiologic factors, in the first place bacterial infections, were given particular attention.

The microscopic abnormalities were correlated to the four basic clinical features of nephritis: proteinuria, haematuria, oedema and hypertension.

The incidence, nature and quantity of immune deposits were registered on the basis of immunohistologic studies.

The functional state of the kidneys was assessed by creatinine clearance (C_c) and concentration test.

Results

Of the patients 78% were males and 22% were females (Table II).

The age distribution revealed a prevalence of young patients. The age groups below 30 years accounted for 65% of the cases, a further 14% of the

Table II
Sex distribution

Sex	n	Per cent
Females	14	22
Males	49	78
Total	63	100

Table III
Age distribution

Age (years)	n	Per cent
14-20	8	12.7
21-30	33	52.4
31-40	10	15.9
41-56	12	19.0
Total	63	100

Table IV
Infections in history

Antecedents	n	Per cent
Recurrent tonsillitis and/or tonsillectomy	29	46
Chronic osteomyelitis	1	1.5
Chronic otitis media	1	1.5
Chronic adnexitis	1	1.5
Total	32	50.5

Table V
Microscopic appearance of kidney and clinical features

Microscopic appearance	P	P H	P Hy	P Oe	P _{Oe} Hy	P _H Hy	Total
Mesangioproliferative GN	2	2					4
Mesangioproliferative GN + focal segmental sclerosis	12	7	3		1	5	28
GN with progressive focal sclerosis	2	3	1				6
Membranoproliferative GN	1	1					2
Membranous GN	1			1	1		3
Chronic GN	1	1	2			1	5
Chronic pyelonephritis	1	2					3
Intact renal tissue	4	1	1				6
Unsuited for evaluation	1					1	2
Other types	1	1	2				4
Total	26	18	9	1	2	7	63
	n						
	per cent	41.2	28.5	14.2	1.5	3.1	100

P = proteinuria; Hy = hypertension; H = haematuria; Oe = oedema; GN = glomerulonephritis.

patients were between 31 and 40, and only 19% were beyond 40 years of age (Table III).

The history revealed earlier tonsillitis or other bacterial infections in 50% of the patients (Table IV).

Light microscopic study of the biopsy specimens revealed various types of glomerulonephritis, with a prevalence of proliferative forms. The cases in which the majority of glomeruli had undergone hyaline degeneration were assigned to the group of chronic glomerulonephritis. In two cases the glomeruli were too small in number to admit of any definite opinion. In four cases the microscopic features were inconclusive; these were listed under the heading "other abnormalities". These included moderate thickening of Bowman's capsule without any other sign of an active glomerular process, focal interstitial fibrosis, focal hyaline degeneration, signs of hypoxaemic renal damage. At the time of renal biopsy, proteinuria had been joined by one more sign in

Table VI
Mesangial immune deposits

Amount	IgG	IgM	IgA	C3	Fibr.
	n				
±	8	15	8	18	3
+	8	2	3	8	4
++	4	1	8	5	1
+++	—	—	5	—	—
Total n	20	18	24	31	8
per cent	31.7	28.5	38	49.2	12.7

± traces; + minor; ++ medium; +++ major

Table VII
Immune deposits on the GBM

Amount	IgG	IgM	IgA	C3	Fibr.
	n				
±	17	27	21	28	29
+	6	4	5	10	4
++	1	—	—	1	—
+++	—	—	—	—	—
Total n	24	31	26	39	33
per cent	38	49.2	41.2	61.9	52.4

± traces; + minor; ++ medium; +++ major