Virchows Arch. A Path. Anat. and Histol. 363, 213—224 (1974)

The Morphological and Clinical Features of Membranoproliferative Glomerulonephritis in Adults* **

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Received February 15, 1974

Summary. A study of 110 patients with membranoproliferative glomerulonephritis (MPGN) revealed that this disease appears in a simple form and in a lobular form. Both forms are characterized by a differently severe proliferation of mesangial cells and by a mostly extensiv thickening of the glomerular basement membrane. The proliferation of mesangial cells and thickening of the glomerular basement membrane is more severe in the lobular form of MPGN. Dense deposit lesions were observed in association with both the simple form and the lobular form of MGPN. An analysis of the morphological and clinical features of MPGN in patients with dense deposits in the glomerular capillary walls failed to indicate that dense deposits represent a unique disease entity.

Twelve years ago Royer *et al.* (1962) described in children 3 cases of chronic glomerulonephritis associated with endocapillary cell proliferation and considerable diffuse thickening of the glomerular capillary walls. They called this disease membranoproliferative glomerulonephritis (MPGN).

Since then this disease has been called persistent or chronic hypocomplementemic glomerulonephritis (West *et al.*, 1965; Herdman *et al.*, 1970), membranoproliferative glomerulonephritis with hypocomplementemia (Michael *et al.*, 1969; Cameron *et al.*, 1970; Michael *et al.*, 1971), mesangiocapillary glomerulonephritis (Churg *et al.*, 1970; Cameron *et al.*, 1973), mixed membranous and proliferative glomerulonephritis (Burkholder *et al.*, 1970), glomérulonéphrite pariétoproliferative (Bariéty *et al.*, 1971) and membranousproliferative glomerulonephritis (Zollinger, 1971; Kettler *et al.*, 1973).

Some investigators claim that MPGN can be found in a simple and in a lobular form (Cameron *et al.*, 1970), while others recognize an additional form with dense deposits firstly described by Berger and Galle (1963) which belongs also to MPGN (Bariéty *et al.*, 1971; Habib and Kleinknecht, 1971). Contrary, other authors (Kincaid-Smith and Hobbs, 1972; Antoine and Faye, 1972) believe that dense deposits disease is a special entity. In order to resolve this controversy and to standardize the nomenclature of this disease we examined the morphological and clinical features of MPGN.

Materials and Methods

One-hundred and ten patients with MPGN examined by the authors between 1964 and 1973 were included in the study. Morphological examination of PAS and Goldner

* Supported by the Deutsche Forschungsgemeinschaft.

** Dedicated to Prof. Dr. W. Doerr for his 60th birthday.

Trichrome stained paraffin kidney sections as well as silver impregnated semithin sections embedded in plexiglass (Movat and McGregor, 1959) was carried out in each patient. In addition, immunofluorescence microscopic examination was performed in 14 patients and electronmicroscopic investigation of the kidney was carried out in 6 patients.

The clinical condition of each patient was noted. Nephrotic syndrome was defined by proteinuria greater than 3 g/24 hr associated with proteinemia less than 6 g/100 ml and albuminemia below 3 g/100 ml. The presence of edema and hypercholesterolemia are not considered essential criteria. Hypertension was defined as systolic blood pressure above 140 mmHg and diastolic blood pressure above 90 mmHg. Hypocomplementemia was defined as β-1c levels under 80 mg/100 ml. A normal level of creatinine clearance were values with 80 ml/min/1.73 m^2 and above 80 ml/min/1.73 m^2. Creatinine clearance levels between 40–79 ml/min/1.73 m^2 were indicative of renal insufficiency without decompensation while values below this range indicated renal insufficiency with decompensation.

Results

The simple form of MPGN was observed in 70 patients while the lobular from occurred in 40 patients. The lobular form was distinguished by the greater proliferation of mesangial cells that accentuated the normal lobular structure in the glomeruli. Otherwise, both forms showed similar lesions in the glomerular capillary walls (Fig. 1a, b). The thickening of the capillary walls was due to the extension of mesangial cells interposed between detached endothelial cells and the glomerular basement membrane (Fig. 2a). An interposition of mesangial cells was occasionally seen throughout the whole subendothelial space in the periphery of the capillary loops. In addition, the precipitation of blood protein detached endothelial cells from the glomerular basement membrane and caused thickening of the capillary walls (Fig. 2b). Interposition of mesangial cells and precipitation of blood proteins were occasionally seen to coexist in the simple and lobular forms of MPGN. Both processus were combined with a new formation of basement-membrane material in the subendothelial space giving the appearance of a splitted or double contoured real basement membrane. Generally, these basement membrane changes coexisted in both forms of MPGN, whereby on the one hand the subendothelial blood protein precipitations were dominant, on the other hand the mesangial interposition. In some cases, even in progressed cases of MPGN the walls of some capillary loops were unchanged.

Besides these presented changes dense deposit lesions (Fig. 2c) were observed in eighteen of the cases with the simple form as well as in four cases with the lobular form of MPGN. The dense deposits which mostly were extended but sometimes affected only a part of the glomerular capillary walls, gave in semithin sections a ribbon-like appearance to the basement membrane. There was a deficiency of silver impregnation in the affected areas seen in semithin sections (Fig. 3). In electronmicroscopic studies the dense deposits described by Berger and Galle (1963) were distinguished by their electron density (Fig. 4). Sometimes the dense deposits included the whole breadth of the basement membrane; sometimes a small border of basement membrane was spared at the subendothelial or subepithelial side. The dense deposits could appear in the same glomerulus besides the other basement membrane lesions mentioned above. Finding these dense deposits in the glomerular basement membrane the same changes could appear, even if less intensive in most cases, in the basement membranes of the Bowman's capsule, the tubuli and the intertubular capillaries.

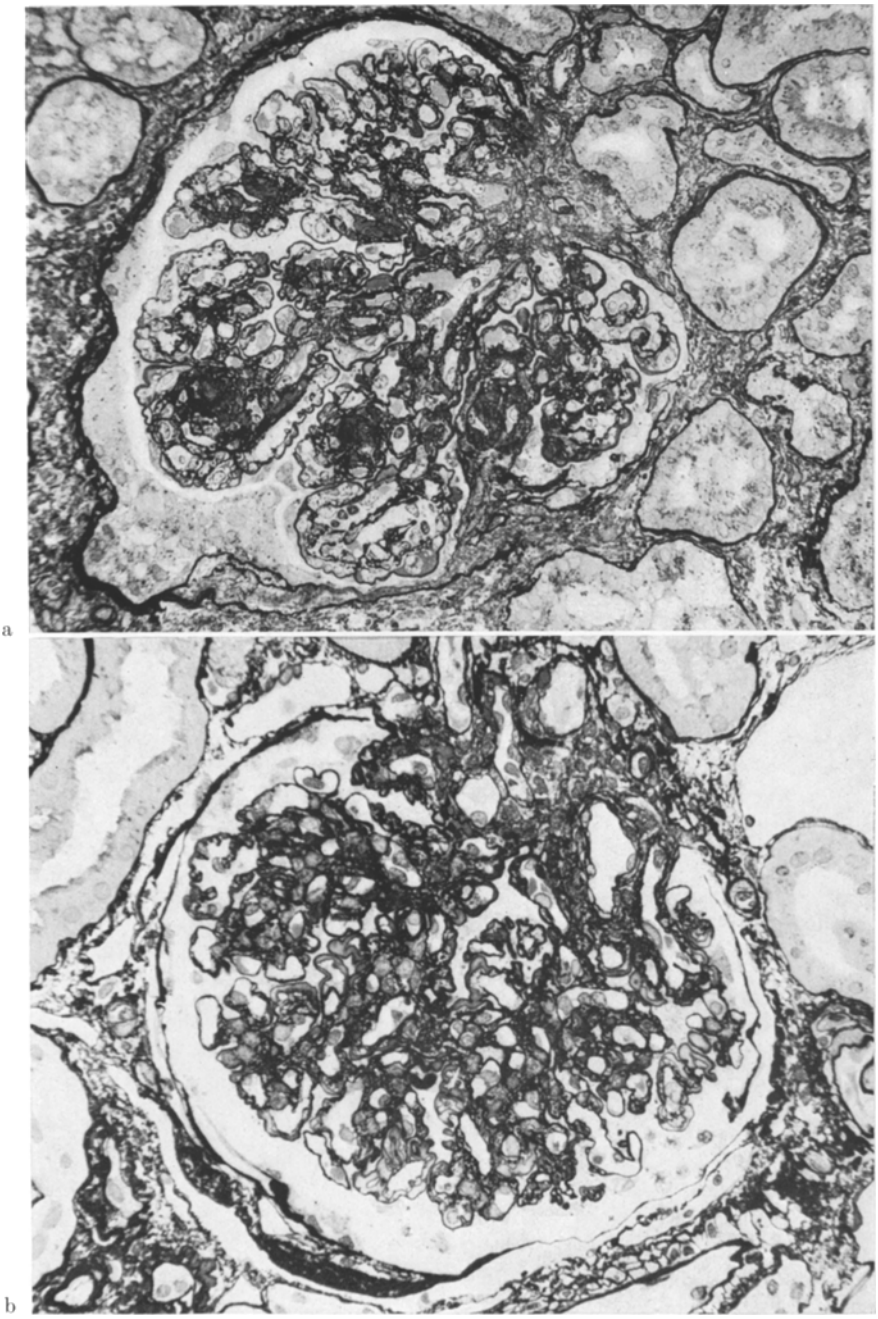

Fig. 1. a The first renal biopsy from a patient with the lobular variant of MPGN. Notice "splitting" of the basement membrane and dense deposits within the basement membrane. Silver impregnation of semithin section 360:1. b The third renal biopsy from the patient shown in Fig. 1a three and one-half years after the first renal biopsy. The simple form of MPGN with "splitting" of the basement membrane and dense deposits within the basement membrane is present. Some areas present a normal thin basement membrane. Silver impregnation of semithin section 360:1

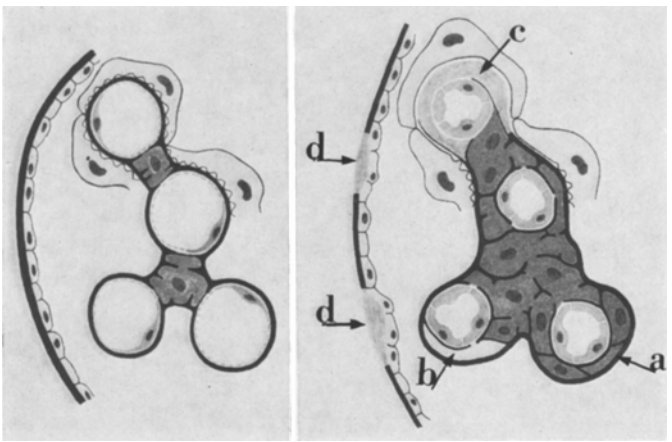

Fig. 2a—d. Cross-section of a normal glomerular lobulus (on the left), and a glomerular lobulus of MPGN (on the right). Notice the thickening of the glomerular capillary wall in the right lobulus, caused a) by the mesangial interposition between the basement membrane and the endothelial cells, b) by deposition of protein precipitates in the subendothelial space and c) by distention of the basement membrane after deposition of dense-deposits within the basement membrane, sometimes sparing a small border of the basement membrane subendothelial and subepithelial. Notice the dense deposits in the basement membrane (d) of the Bowman's capsule

Table 1. A morphometric comparison of normal glomeruli and glomeruli of membranoproliferative GN

The total number of glomerular cells, the total glomerular area, the total glomerular cells/1000 sq. μ of total glomerular area observed in the renal biopsies taken from patients with normal kidneys and with membranoproliferative GN. Furthermore the number of mesangial and endothelial cells and the number of mesangial and endothelial cells/1000 sq. μ of total glomerular area (mesangial and endothelial cells = endocapillary cells)

	Normal n = 10	Membranoproliferative glomerulonephritis $n = 27$
Total number of glomerular cells	46.9 ± 6.3 ± 2.0	102.8 ± 31.7 ± 6.1*
Total glomerular area (sq. μ)	20414 ± 4353 ± 1376	27593 ± 10397 ± 1998*
Total glomerular cells/1000 sq. μ of total glomerular area	2.3 ± 0.5 ± 0.2	3.9 ± 0.84 ± 0.16*
Number of mesangial and endothelial cells (endocapillary cells)	29.4 ± 8.5 ± 2.7	88.2 ± 31.9 ± 6.1*
Number of mesangial and endothelial cells/1000 sq. μ of total glomerular area (endocapillary cells)	1.54 ± 0.3 ± 0.06	3.30 ± 0.71 ± 0.14*

Values are mean ± standard deviation ± standard deviation of the mean. n = number of patients, p = probability, * $p < 0.001$.

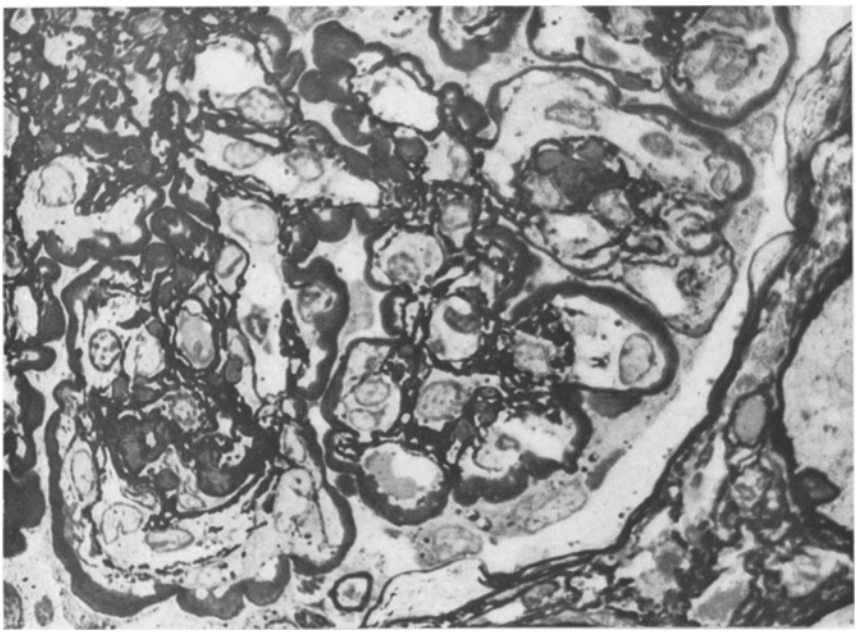

Fig. 3. Part of a glomerulus of MPGN (lobular form, the same case as in Fig. 1a) with dense deposits within the glomerular basement membrane. Notice the deficiency of silver impregnation of the dense deposits. Silver impregnation of semithin section 1000:1

Table 2. Immunofluorescence studies in 14 cases with MPGN: positive immunofluorescence findings in 7 cases of the simple form of MPGN without "dense deposits", in 4 cases of the simple form of MPGN with "dense deposits", and in 4 cases with the lobular form of MPGN without "dense deposits". In all cases deposition of β-1c, in most of the cases additional JgG or/and JgM, in few cases or/and JgA. Nearly all cases with the same granular or coarse pattern along the glomerular basement membrane, sometimes like a garland in the periphery of the glomerular capillary loops. Values in parenthesis are the percentages for the number (n) of cases

Immuno-fluorescence findings with deposition of:	Membranoproliferative glomerulonephritis						
	simple form ($n=11$)				lobular form ($n=3$)		
	without "dense deposits"		with "dense deposits"		without "dense deposits"		with "dense deposits"
	$n=7$	(%)	$n=4$	(%)	$n=3$	(%)	
JgG	7	(100)	2	(50)	2	(67)	—
JgM	5	(71)	2	(50)	3	(100)	—
JgA	3	(43)	1	(25)	1	(33)	—
β_{1c}	7	(100)	4	(100)	3	(100)	—

Table 1 presents the results of a *morphometric investigation* performed in a random sample of the patients with MPGN. The glomerular cell number, glomerular area, glomerular cells per unit area, endocapillary cell number (mesan-

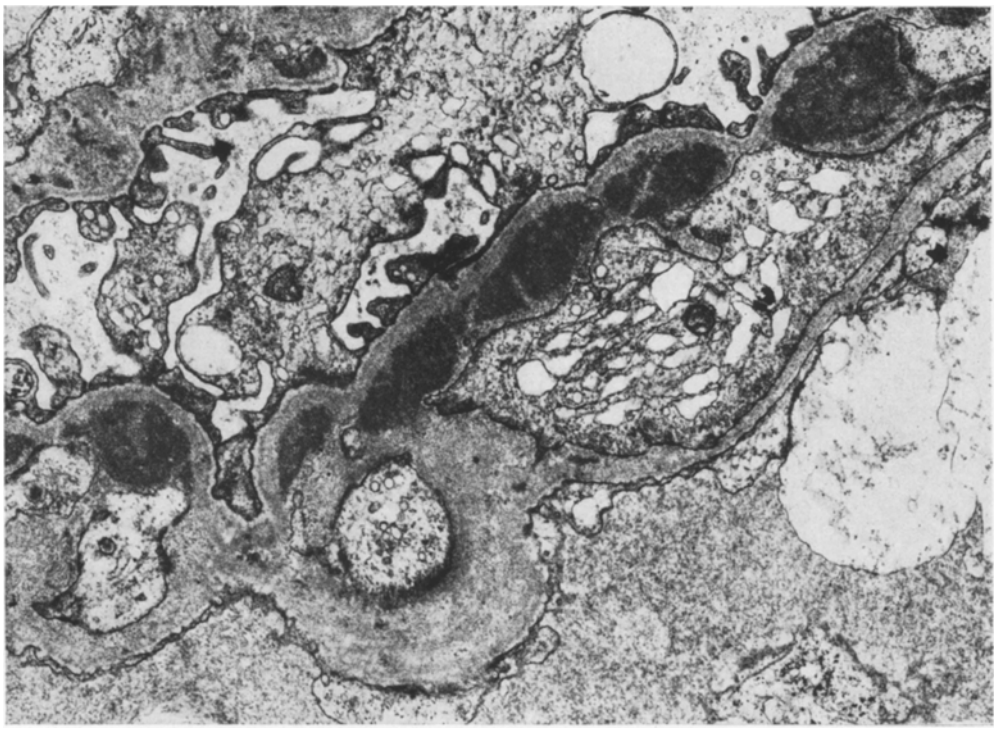

Fig. 4. Electron micrograph of the lobular variant of MPGN with deposition (dark areas) of dense deposits within the thickened basement membrane, sparing a small border of the subendothelial and subepithelial basement membrane. Notice the "splitting" of the basement membrane with the mesangial interposition between the real basement-membrane, containing dense deposits, and the new formed subendothelial basement membrane. 10000:1

gial and endothelial cells) and the number of endocapillary cells (mesangial and endothelial cells) per unit area were significantly elevated in the group of patients with MPGN compared to normal values. In addition, random samples show that glomeruli of the lobular form compared with the simple form have more cells in the mean.

Immunofluorescence studies carried out in a random sample of patients with MPGN failed to reveal significant differences between the frequency of occurrence of JgG, JgM, JgA, and β-1c in the different morphological groups (Table 2).

Examination of the *clinical parameters* failed to reveal significant differences between the patients with the simple, lobular or dense deposit form of MPGN. The mean age of the patients at the time of the first renal biopsy was 35, 32 and 31 years old, respectively. Table 3 presents the clinical findings in the patients with MPGN. Patients with dense deposits in combination with either the simple or lobular form of MPGN were combined as a group in the analysis. The sex-ratio male: female with a moderate predominance of women in two of the three groups is similar. The percentage of patients that had tonsillitis at some time prior to the onset of renal disease was comparable in the three groups. There was a

Table 3. The clinical features of membranoproliferative glomerulonephritis in patients that showed various types of changes in the glomeruli. Values in parenthesis are the percentages for the number (n) of cases

	Simple form $n=52$		Lobular form $n=36$		Dense deposits form $n=22$	
	n	(%)	n	(%)	n	(%)
		Sex-ratio				
Male: Female		44:56		50:50		37:63
		Preceding Diseases				
Tonsillitis	19	(37)	11	(31)	6	(27)
Unspecific bacterial and viral respiratory tract infection	5	(10)	5	(14)	0	
Elevated ASO titer	5	(10)	3	(9)	0	
		Initial symptoms				
Edema or Edema with Proteinuria	22	(46)	12	(35)	10	(45)
Edema with Hypertension	4	(8)	6	(18)	—	
Asymptomatic Proteinuria	6	(12)	6	(18)	3	(14)
Hypertension	—	—	1	(3)	2	(9)
Hypertension with Edema and Proteinuria	2	(4)	1	(3)	3	(14)
Hypertension, Proteinuria and Hematuria	4	(8)	4	(12)	2	(9)
Uncharacteristic Symptoms (Fatigue, Weakness, Headache)	10	(21)	4	(12)	2	(9)
	(4 cases without informations)		(2 cases without informations)			
		Symptoms at the time of renal biopsy				
Systolic blood pressure						
Normal (0–140) mmHg	15	(29)	6	(17)	5	(23)
Elevated (>140) mmHg	37	(71)	30	(83)	17	(77)
Diastolic blood pressure						
Normal (0–90) mmHg	20	(38)	10	(28)	10	(46)
Elevated (>90) mmHg	32	(62)	26	(72)	12	(54)
Nephrotic syndrome	39	(75)	26	(72)	15	(68)
Hematuria mild (+)	16	(31)	14	(39)	6	(27)
moderate (++)	11	(21)	6	(17)	7	(32)
severe (+++)	12	(23)	12	(33)	5	(23)
Creatinine clearance[b]						
Normal	25	(68)	14	(48)	9	(56)
Renal insufficiency						
without decompensation	11	(30)	12	(41)	5	(31)
with decompensation	1	(3)	3	(11)	2	(13)
Decreased β-1c[a]	9	(60)	9	(75)	3	(75)

[a] β-1c complement levels were examined in 15 cases of simple, 12 cases of lobular and 4 cases of dense deposit changes. (n = number of cases.)

[b] Creatinine clearance: (15 cases without informations) 7 cases without informations) 6 cases without informations)

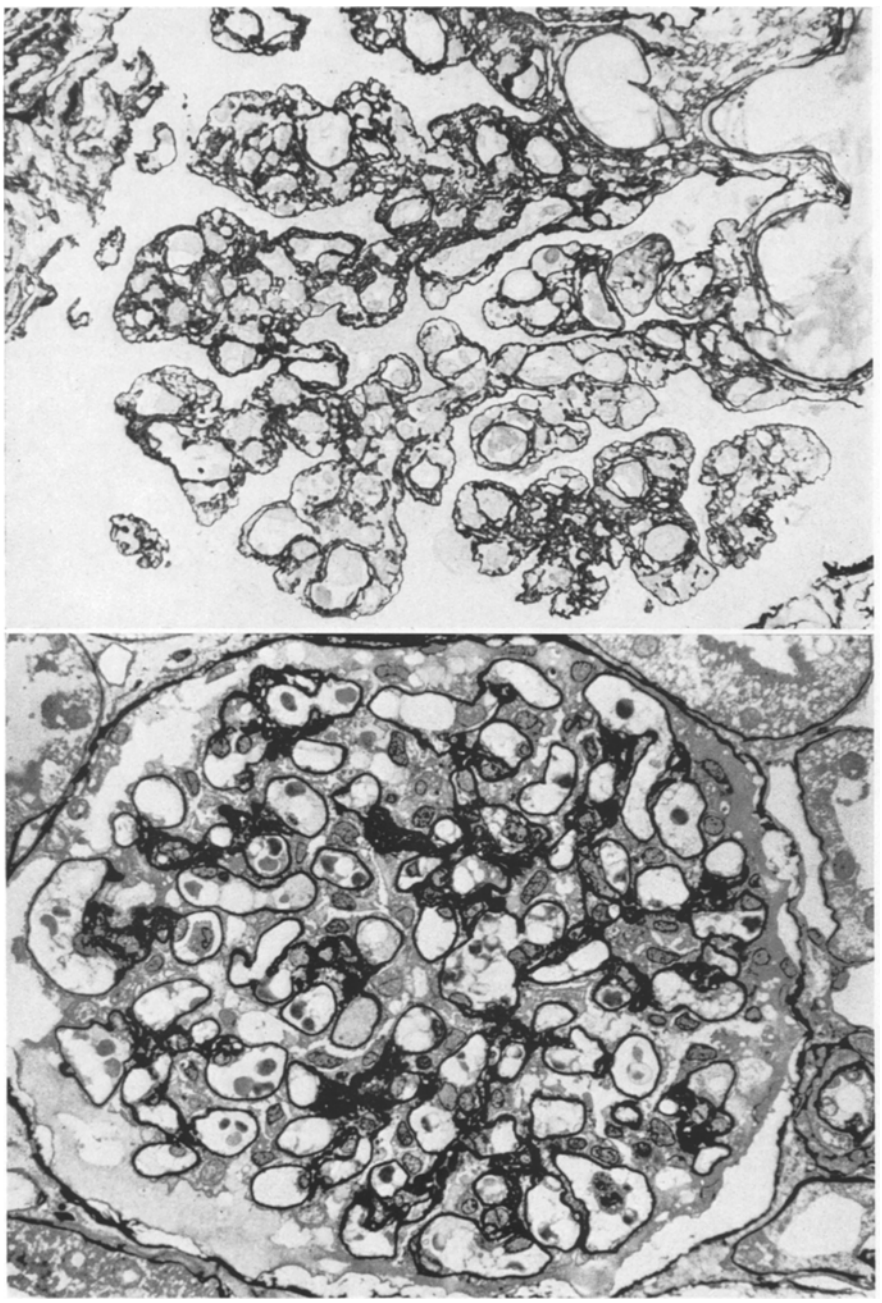

Fig. 5. a The first renal biopsy from a patient with the lobular form of MPGN. Notice the distinct "splitting" of the glomerular basement membrane. Silver impregnation of semithin section, 940:1. b The second renal biopsy from the patient in Fig. 5a three and one-half years after the first renal biopsy. The patient was clinically cured at this time. Notice the complete disappearance of the "splitting" of the basement membrane and the absence of the accentuated lobular structure. Silver impregnation of semithin section 590:1

Table 4. The clinical and morphological course of MPGN in relation to therapy. Details in the text

	n	Clinical			Morphological		
		+	Ø	−	+	Ø	−
Without therapy	3	0	1	2	0	1	2
Steroids	3	1	1	1	0	1	2
Indomethacine	8	2	1	5	0	1	7
Immunsuppr. drugs	6	4	0	2	4	0	2

n = number of cases, + = improved or cured, Ø = unchanged, − = deteriorated.

tendency for the frequency of respiratory tract infections and elevated ASO titers prior to the onset of renal disease to be reduced in the patients with dense deposit lesions. The percentage of patients that showed edema, proteinuria, hypertension or hematuria as initial symptoms was very similar in the three groups. The frequency of uncharacteristic symptoms as fatigue, weakness and headache as initial symptoms appeared to be slightly higher in the patients with the simple form of MPGN.

At the time of the first renal biopsy, a comparable percentage of patients in the three groups showed elevated systolic and diastolic blood pressure, whereby the blood pressure in patients with the lobular form was more often elevated than in the other groups.

The frequency of the nephrotic syndrome was similar in the three groups, the intensity of hematuria tended to be different in the three groups. Normal values of creatinine clearance were more often seen in patients with the simple form and the dense deposits form of MPGN than in the lobular form. Renal insufficiency without decompensation occurred more often in patients with the lobular form, while the occurrence of renal insufficiency with decompensation appeared to be rarer in patients with the simple form compared with the other two forms. There was no marked difference in the presence of a decreased β-1c complement level in the three groups of patients.

The morphological and clinical course of serial renal biopsies, performed in 20 patients with MPGN in a time interval of 6–40 months were summarized in Table 4.

It should be noticed that 2 of the 4 patients, with a morphological and clinical improvement under azathioprine therapy showed in renal biopsy the simple form of MPGN, the other 2 patients the lobular form. It was remarkable that one of the latter received azathioprine only for 8 days during the course of 3.5 years passed from the first to the second renal biopsy. The remaining time the treatment was homoeopathically (Fig. 5a, b).

Discussion

Our findings agree with reports that MPGN can be found in a simple and in a lobular form (Cameron *et al.*, 1970; Kincaid-Smith and Hobbs, 1972; Habib *et al.*, 1973). We view these forms as two variants of one disease. The lesions of

the glomerular capillary walls in the simple and the lobular forms of MPGN are principally identical. They can be caused by interposition of cellular and fibrillar material of the increased mesangial matrix in the subendothelial space, deposition of blood protein precipitates within the subendothelial space, or distension of the basement membrane in the area of the dense deposits. In the lobular form, however, the proliferation of mesangial cells is exceptionally severe and the natural lobular structure of the glomeruli appears to be accentuated. The lesioned appearance of the basement membrane distinguishes the lobular form of MPGN from pure lobular glomerulonephritis, which has none of the demonstrated basement membrane lesions described above. The other differences between the lobular form of MPGN and the pure lobular glomerulonephritis will be discussed at another place.

However our findings disagree with reports that the dense deposit form described by Berger and Galle (1963) is a special entity of disease (Kincaid-Smith and Hobbs, 1972; Antoine and Faye, 1972). This conclusion is based on several observations. First all three characteristical types of lesions of the glomerular capillary walls in MPGN can coexist in one kidney, even in the same glomerulus. Dense deposits lesions were always associated with the simple as well as with the lobular form of MPGN. Second, the clinical features of the patients with dense deposit lesions were essentially the same as in patients with the simple or the lobular form of MPGN without dense deposit lesions. The observation that hypertension and severe hematuria occur more often in the lobular form corresponds to the often more serious course of the disease in these patients. Third, the course of the disease was comparable in the patients with MPGN, irrespective of the presence of dense deposit lesions, which supports the view that the group of MPGN is homogenous. Follow-up examinations in 20 patients revealed that the clinical condition improved in 7 and deteriorated in 10 cases, irrespective of the variant of the disease or whether steroid, indomethacine or immunosuppressant drug therapy was employed. In addition, improvement in the morphological condition of the glomeruli failed to correspond to the variant of the disease or the type of therapy. Our findings agree with those of others that the prognosis of the simple and lobular forms of MPGN is generally unfavorable (Herdman *et al.*, 1970; Mandalenakis *et al.*, 1971; Horvarth *et al.*, 1971; Holland and Bennett, 1972; Kincaid-Smith, 1972; Cameron *et al.*, 1973). We also found that the simple form can proceed to the lobular from (Burkholder *et al.*, 1970; Mandalenakis *et al.*, 1971) while the lobular form can pass into the simple form (Fig. 1a, b) and in seldom cases be cured completely by decrease of the endocapillary cells, and disappearance of the so-called splitting of the basement membrane and the dense deposits (Fig. 5a, b).

The etiology of MPGN remains still unknown. Our findings support the view that MPGN is not a poststreptococcal glomerulonephritis (Mandalenakis *et al.*, 1971; Michael *et al.*, 1971), even though splitting of the basement membrane also occurs in poststreptococcal glomerulonephritis (Bohle and Krecke, 1955; Churg and Grishman, 1957). But it is not even clear, if MPGN, which can reappear in renal transplants (Bohle, 1972) is an immunopathy (Herdman *et al.*, 1970; Holland and Bennett, 1972; Nagi, 1972). Our immunohistological findings in MPGN confirm previous reports (Habib and Kleinknecht, 1971; Bariéty *et al.*, 1971; Mandalenakis

et al., 1971; Kincaid-Smith and Hobbs, 1972; Holland and Bennett, 1972) and additionally show that patterns of immunoglobulins and complement in the different forms of MPGN and in the different basement membrane changes, also in the dense deposits form, are essentially identical. Furthermore it seems proved today, that the β-1c levels fail to correlate with the activity and the course of the disease (West and McAdams, 1970; West, 1971).

We recommend that the term membranoproliferative glomerulonephritis, originally proposed by Royer and coworkers in 1962, is used as the official name for this disease characterized by thickening of the glomerular basement membrane and endocapillary cell proliferation. This name is especially appropriate for the morphological features of the disease. Use of only this name is a step toward standardization of the nomenclature for glomerulonephritic diseases.

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