Membranoproliferative Glomerulonephritis Presenting as Malignant Hypertension

E.	Ellen	Burgess
M.	Michael	Curtis
H.	Halgrimur	Benediktsson

Departments of Medicine and Pathology, University of Calgary, Alta., Canada

Dr. Ellen Burgess, 1403-29 Street NW, Calgary, Alta. T2N 2T9 (Canada)

Dear Sir,

Malignant hypertension is defined as the presence of a diastolic blood pressure in excess of 140 mm Hg with grade IV retinopathy. It has been reported in association with renal parenchymal disease [1, 2] and in chronic glomerulonephritis, but has not been reported in specific association with membranoproliferative glomerulonephritis (MPGN). Although hypertension may be present in 25-37% of cases with MPGN [3, 4], accelerated or malignant hypertension has not been reported. Herein is reported a case of MPGN presenting as malignant hypertension.

A 40-year-old male Caucasian with chronic headaches presented with progressive increase in severity and frequency of headaches and sudden onset of right central scotoma 4 weeks prior to presentation. Medical history was significant for smoking, nocturia $\times$ 2, and family history of stroke and myocardial infarction at late age. There was no previous documentation of hypertension.

Initial examination revealed a blood pressure of 210/ 140 mm Hg in all limbs and pulse 70/min.

Funduscopy showed grade IV retinopathy, with a right-eye central visual field defect.

Cardiovascular examination revealed a normal S1, increased A2, S4 present, and 2/6 systolic ejection murmur. No bruits were detected, and the remainder of the examination was normal.

Significant laboratory findings included serum creatinine 177 μ mol/l, serum urea 16.4 mmol/l, fasting cholesterol 7.12 mmol/l, total serum protein 44 g/l, and serum albumin 27 g/l. Urine microscopy revealed 5-8 white and

20-25 red blood cells per high-power field and hyalin and granular casts. A 24-hour urine collection revealed a creatinine clearance of 0.88 ml/s (corrected for body surface area and 19.85 g protein. Electrocardiogram met voltage criteria for left ventricular hypertrophy.

The patient underwent closed renal biopsy. The biopsy specimen had 14 glomeruli, 5 globally and 2 segmentally sclerosed. Glomeruli showed marked diffuse increase in cellularity with lobar accentuation and increased mesangial matrix. Tuft adhesions were prominent, and no cellular crescents were seen. The capillary walls were thickened with extensive tramtracking. There was moderate tubular atrophy and focally prominent vacuolization of tubular cell cytoplasm. Larger arteries showed intimal thickening; arterioles showed hyaline deposits and focal mucoid intimal thickening. Electron microscopy demonstrated basement membrane reduplication in glomerular capillary walls with mesangial inter-cation in glomerular capillary walls with mesangial interposition and subendothelial dense deposits, endothelial cell proliferation, and mesangial dense deposits. Immunofluorescence revealed granular deposits of C3 in glomeruli, basement

membranes of some tubules, arterioles, and small arteries. The biopsy was consistent with type I MPGN.

The patient has been maintained on verapamil, 160 mg twice daily, Persantine, and ASA and follows a low-protein diet. Papilledema resolved by 16 weeks of follow-up, and at 1 year creatinine clearance has fallen to 0.51 ml/s.

Malignant hypertension has been reported in 12.9–37.2% of the cases with chronic glomerulonephritis [2], but specific classification of the glomerulonephri-

370
Burgess/Curtis/Benediktsson

tides was not noted. Similarly, hypertension may be present in MPGN, but malignant hypertension has not been reported. This case illustrates a patient with MPGN presenting with malignant hypertension.

Habib R, Kleinknecht C, Gubler MC, et al: Idiopathic membranoproliferative glomerulonephritis in children. Report of 105 cases. *Clin Nephrol* 1973;1:194–214.

Donadio JV, Slack TK, Holley KE, et al: Idiopathic membranoproliferative (mesangiocapillary) glomerulonephritis: A clinicopathologic study. *Mayo Clin Proc* 1979;54:141–150.

References

Kincaid-Smith P, McMichael J, Murphy EA: The clinical course and pathology of hypertension with papilloedema (malignant hypertension). *Q J Med* 1958;105:117–153.

Kincaid-Smith P: Malignant hypertension: Mechanisms and management. *Pharmacol Ther* 1980;9:245–269.

Announcement

Invitation for Submissions for the Nils-Alwall Prize 1990

The Arbeitsgemeinschaft für Klinische Nephrologie e.V. would be pleased to receive submission of papers from candidates for the Nils-Alwall Prize. This prize is awarded to young scientists engaged in clinical research in German-speaking regions in the field of nephrology, with particular emphasis on activities involving dialysis or similar methods of extracorporeal elimination or kidney transplants. The award comprises the Nils-Alwall Medal, a certificate and DM 15,000.- prize money.

The prize may be awarded to up to three nominees. Candidates up to 45 years of age are required to submit either non-published work, work published not more than 1 year before submission or a

summary of original extended research work (but not a habilitation thesis). If this work involved patients, it must explicitly confirm that all relevant ethical principles have been observed (Helsinki Agreement 1964, Tokyo Agreement 1975).

A total of six copies of the paper are required. These are to be in anonymous form and accompanied by a short curriculum vitae and information regarding the candidates present professional activities. These documents are to reach the Chairman of the Prize Committee not later than April 15, 1990, at the following address: