CASE REPORTS

Malignant Hypertension With Intestinal Ischemia Secondary to Juxtaglomerular Cell Tumor

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• **Malignant hypertension is a well-defined condition associated with high blood pressure and acute target-organ damage. Although 95% of cases are secondary to essential hypertension, its etiological profile is broad. Juxtaglomerular cell tumor is a rare condition, with only approximately 65 cases reported to date. We describe a patient with malignant hypertension with acute renal failure and intestinal ischemia secondary to a juxtaglomerular cell tumor. We believe this is the first case of juxtaglomerular cell tumor causing malignant hypertension. The diagnostic approach and treatment are discussed.** ***Am J Kidney Dis*** **46:957-961.**
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INDEX WORDS: Juxtaglomerular cell tumor; malignant hypertension; renal failure.

MALIGNANT HYPERTENSION is a well-known condition associated with high blood pressure, acute target-organ damage, and papilledema with hemorrhages and/or exudates. It occurs in up to 1% of hypertensive patients.[1] Presentation generally occurs with encephalopathy, acute renal failure, microangiopathic hemolytic anemia, and other severe organ damage. Intestinal ischemia caused by obliterant endarteritis is an unusual feature, described in isolated reports.[2] The cause of malignant hypertension is broad, with essential hypertension responsible for approximately 95% of cases. Secondary causes include renovascular hypertension, primary hyperaldosteronism, pheochromocytoma, polyarteritis nodosa, and renal parenchymal disorders.

Juxtaglomerular cell tumor is a rare condition, with only approximately 65 cases reported in the literature to date. The tumor originates from modified smooth muscle cells of the juxtaglomerular apparatus. It contains renin-secreting cells, causing secondary hyperaldosteronism and hypertension. We describe a case of malignant hypertension with renal failure and intestinal necrosis secondary to a juxtaglomerular cell tumor. Our MEDLINE search spanning 1966 to September 2004 using the terms "juxtaglomerular cell tumor" or "renin-secreting tumor" showed no reports on such an association. We found only 1 citation correlating juxtaglomerular cell tumor to malignant hypertension; however, no case description was provided.[3]

CASE REPORT

A diagnosis of arterial hypertension was made for a 32-year-old woman 4 years before, during her third pregnancy, which was uneventful with no other complications, similar to what had occurred with the previous 2 pregnancies. One year after the diagnosis of hypertension, she was referred to our center for treatment. At the first evaluation, blood pressure was high (180/110 mm Hg), and her only symptom was daily occipital headache. Family history showed only arterial hypertension in her mother (who had died 6 years before of a stroke). The patient was an irregular user of methyldopa and hydrochlorothiazide. Renal function was normal (creatinine level, 0.7 mg/dL [62 μmol/L]), and mild hypokalemia (potassium, 3.3 mEq/L [mmol/L]) was present. Blood pressure control was achieved with amlodipine, 10 mg/d; hydrochlorothiazide, 25 mg/d; and captopril, 75 mg/d. Before an investigation for secondary hypertension could be carried out, the patient quit medical care.

One year later, the patient was admitted to a medical emergency service with renal failure, severe hypertension (blood pressure, 210/120 mm Hg), blurred vision, and abdominal pain and tenderness, characterizing an acute abdomen. Funduscopy showed retinal hemorrhage, exudates, and papilledema. Initial laboratory test results showed the following values: hemoglobin, 6.4 g/dL (64 g/L); hematocrit, 18.9%; creatinine, 3.4 mg/dL (301 μmol/L); lactate dehydrogenase, 918 U/L; and reticulocytes, 3.9%. A laparotomy was performed, and multiple small ulcerations associated with

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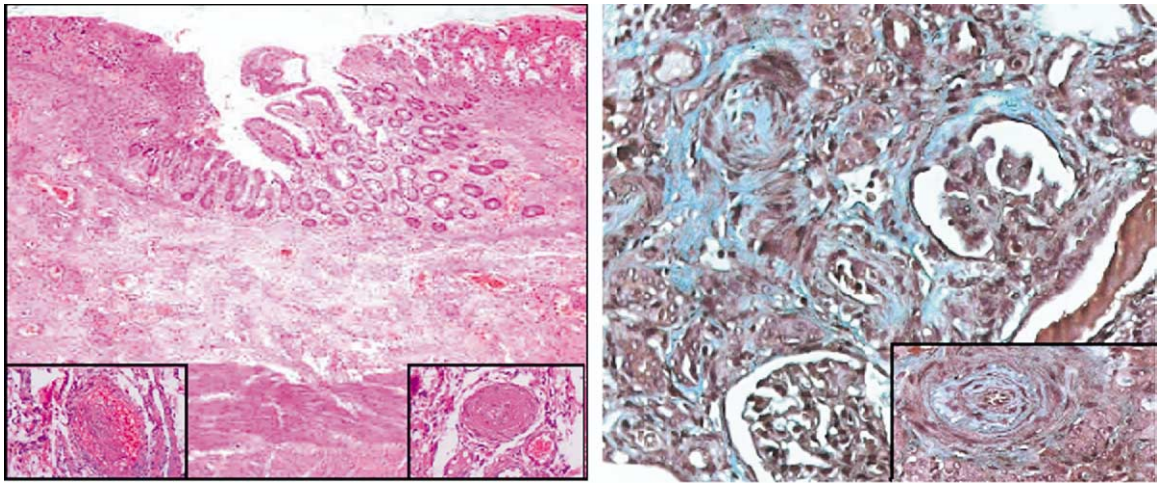

Fig 1. Histological features of malignant hypertension: (A) ischemic enteritis with arteriolar changes of malignant hypertension in the submucosa and (B) malignant nephrosclerosis in renal tissue adjacent to the tumor, showing obliterant endarteritis and glomerular ischemia. (Original magnification: [A] ×250, [B] ×400.)

extensive intestinal necrosis were found. A partial bowel resection was performed, and blood pressure control was achieved through oral medication. Histological analysis of an intestinal segment showed obliterant endarteritis in multiple organizational stages with occlusive thrombus, compatible with malignant hypertension (Fig 1). After recovery, she was referred to our hospital for further investigation.

At admission, her blood pressure was 150/90 mm Hg on treatment with amlodipine, 10 mg/d; furosemide, 80 mg/d; and clonidine, 0.4 mg/d. Laboratory test results are listed in Table 1. An echocardiogram showed concentric ventricular hypertrophy with a septum thickness of 13 mm (9 to 11 mm) and preserved contractile function. A renal Doppler ultrasound showed bilateral increased renal echogenicity with normal kidney size and a 3.5-cm diameter cortical nodular lesion in the right kidney. No evidence of renal artery stenosis was found. Computed tomography (CT), using no contrast because of renal impairment, and magnetic resonance imagining confirmed the finding of an expansive and solid nodular lesion in the right kidney.

Table 1. Laboratory Findings at Admission and 6 Months After Surgery

	Admission	After Surgery
Hemoglobin (g/dL)	7.2	10.7
Hematocrit (%)	19.7	33
White blood cells ($\times 10^3/\mu$L)	8.14	7.91
Platelets ($\times 10^3/\mu$L)	65	196
Sodium (mEq/L)	140	139
Potassium (mEq/L)	4.6	5.2
Creatinine (mg/dL)	3.1	2.3
Lactate dehydrogenase (U/L)	721	256
Reticulocytes (%)	2.8	1.1
Albumin (g/dL)	3.3	4.3
Urinalysis		
pH	5.0	6.0
Gravity	1,010	1,010
Protein (g/L)	0.25	0.5
White blood cells (/high-power field)	5	2
Red blood cells (/high-power field)	80	Absence
Plasma aldosterone (ng/dL)	46 (9-18)	32
PRA (ng/mL/h)	22 (0.2-2.8)	0.6

NOTE. To convert hemoglobin and albumin in g/dL to g/L, multiply by 10; white blood cells and platelets in $10^3/\mu$L to 10^9/L, multiply by 1; creatinine in mg/dL to μmol/L, multiply by 88.4; aldosterone in ng/dL to nmol/L, multiply by 0.02774; PRA in ng/mL/h to ng/(Lxs), multiply by 0.2778.

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