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A TALE OF TWO SYSTEMS: MALIGNANT HYPERTENSION PRESENTING WITH PAINLESS HEMATURIA AND ACUTE PULMONARY EDEMA — A DIAGNOSTIC ODYSSEY

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ABSTRACT

Malignant hypertension is an acute, life-threatening form of severe hypertension characterized by rapidly progressive target-organ damage involving the kidneys, retina, central nervous system, and cardiovascular system. Early recognition is critical to prevent irreversible injury. We report the case of a previously healthy 35-year-old man presenting with one week of painless macroscopic hematuria followed by sudden-onset dyspnea with pink frothy sputum. Evaluation revealed severely elevated blood pressure, grade 4 hypertensive retinopathy, acute pulmonary edema, proteinuric hematuria, hypokalemia, and acute kidney injury. The patient required non-invasive ventilation, intravenous antihypertensive therapy, and multiple sessions of hemodialysis. This case highlights the importance of recognizing hematuria as an early manifestation of hypertensive renal microangiopathy and emphasizes the multi-system involvement that characterizes malignant hypertension.

KEYWORDS: Malignant hypertension • Hematuria • Pulmonary edema • Hypertensive retinopathy • Acute kidney injury • Renal microangiopathy.

INTRODUCTION

Malignant hypertension is defined by a sudden and severe elevation of blood pressure associated with acute target-organ damage, particularly to the kidneys, retina, heart, and central nervous system. The characteristic hallmark is grade 3–4 hypertensive retinopathy, including flame hemorrhages, cotton-wool spots, and papilledema. Renal involvement occurs due to fibrinoid necrosis of afferent arterioles and hyperplastic arteriolosclerosis, leading to hematuria, proteinuria, and rapidly progressive renal failure. Hematuria in malignant hypertension is often glomerular and reflects underlying vascular injury to the renal microcirculation. Although pulmonary edema is a recognized complication due to acute left ventricular afterload mismatch and diastolic dysfunction, its simultaneous occurrence with hematuria in a young individual presents a diagnostic challenge.

Case Presentation

A 35-year-old male presented with bright red, painless gross hematuria for one week. The hematuria was continuous throughout the urinary stream. On the day of admission, he developed sudden severe breathlessness at rest, accompanied by cough producing pink frothy sputum.

On arrival, he was tachypneic with oxygen saturation of 81% on room air, improving to 97% with CPAP support. Blood pressure ranged from 260/130 to 270/140 mmHg in all four limbs. The pulse rate was 116/min and regular.

Cardiovascular examination revealed a loud A2 component. Chest auscultation revealed bilateral coarse crackles consistent with acute pulmonary edema. Fundus examination demonstrated grade 4 hypertensive retinopathy with papilledema and flame hemorrhages.

Neurological and abdominal examinations were unremarkable.

He was stabilized with non-invasive ventilation, intravenous diuretics, and controlled parenteral antihypertensive therapy. Over 48 hours, urine output declined and he developed progressive azotemia requiring hemodialysis.The case was proceeded with renal biopsy which showed **hypertensive arterionephrosclerosis.**

Investigations

	17/2/25	20/2/25	24/2/25
RBS	73	103	101
UREA	109	208	190
CREATININE	4.96	9.9	10.1
SODIUM	136	139	141
POTTASIUM	3.3	4.3	4.7
CALCIUM	8.8mg/dl		

URINE ROUTINE

- Colour : Reddish
- Protein : 3+
- Glucose : Negative
- RBC : 3+
- Numerous RBCs / hpf
- Abnormal RBCs - Nil
- No dysmorphic RBCs
- WBC : 2-3 / hpf
- pH : 7.0

USG ABDOMEN & PELVIS

- Liver – Normal (12.3 cm)
- Spleen, Pancreas – Normal
- Right Kidney – 9.4 * 4.8 cm ; Normal echoes, CMD maintained
- Left Kidney – 10.1 * 4.2 cm ; Normal echoes, CMD maintained
- No free fluid in abdomen / pelvis

ARTERIAL BLOOD GAS ANALYSIS

- pH : 7.49
- pCO2 : 26
- pO2 : 51
- HCO3 : 19.8
- Sodium : 136
- Potassium : 2.6
- Lactate : 0.9

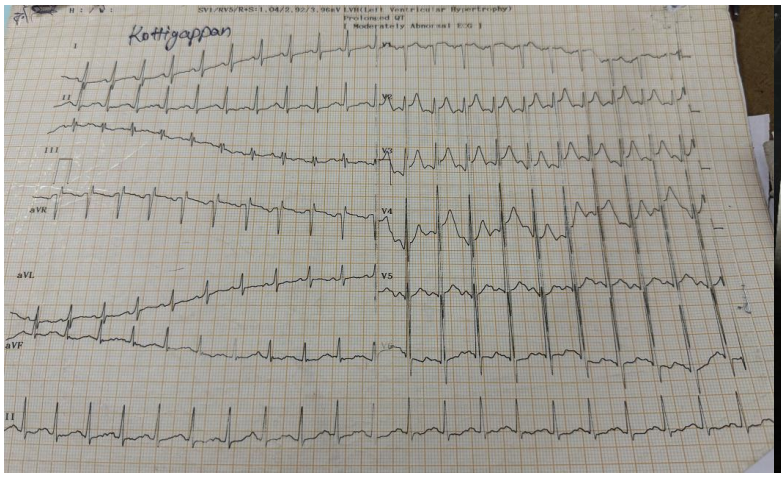
PERIPHERAL SMEAR

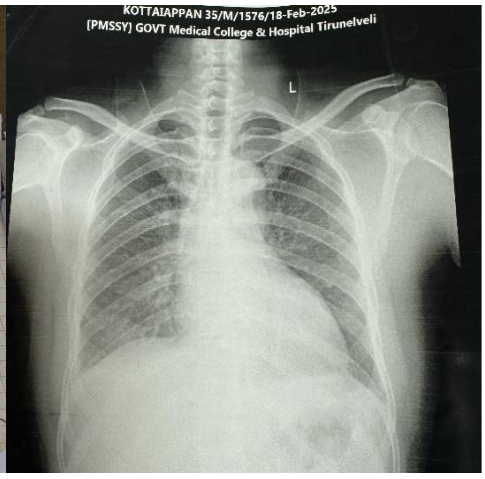
-normocytic normochromic RBC's
-WBC adequate no abnormal cells
-platelet adequate
*Total creatinine kinase-56
*ESR-100mm/hr
*CRP-2.5

ECHOCARDIOGRAPHY

- Normal LVSF
- Concentric LVH +
- No Regional wall motion abnormalities

Coagulation profile with in normal limits





DISCUSSION

Malignant hypertension results from abrupt failure of vascular autoregulation leading to endothelial injury, fibrinoid necrosis, platelet activation, and microangiopathic changes (Harrison's 22e). In the kidneys, hyperplastic arteriolosclerosis and fibrinoid necrosis lead to ischemic injury, glomerular RBC leakage, and proteinuria. The painless hematuria in this patient reflects renal microvascular injury rather than inflammatory glomerulonephritis.

Acute pulmonary edema develops from increased afterload, impaired LV compliance due to concentric LVH, and elevated pulmonary capillary wedge pressure. This mechanism of 'flash pulmonary edema' is characteristic of hypertensive emergencies.

Grade 4 hypertensive retinopathy correlates with severe systemic microangiopathy and confirms the malignant phase. The markedly elevated LDH suggests microvascular hemolysis, though early-stage changes may precede schistocyte formation.

Management involves controlled blood pressure reduction, diuretics, ventilatory support, and renal replacement therapy when indicated. Early recognition and targeted treatment can halt progression of vascular injury and improve outcomes.

CONCLUSION

This case highlights malignant hypertension as a multi-system vascular emergency. Painless hematuria in a young adult may represent renal microangiopathy rather than primary urological disease, especially when accompanied by severe hypertension. The coexistence of pulmonary edema, retinal involvement, and acute kidney injury should prompt immediate evaluation for malignant hypertension. Early diagnosis and carefully controlled BP reduction are essential to prevent irreversible organ damage.

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