MALIGNANT HYPERTENSION WITH MULTI-ORGAN FAILURE IN A YOUNG NIGERIAN: A CASE REPORT

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Abstract

Malignant hypertension is a known complication of primary hypertension and its multi-organ involvement leads to increased morbidity and mortality. A fact that should prompt its early diagnosis and treatment. We present the case of a 36-year old man who had no prior history of hypertension but presented on account of a persistent headache, amaurosis fugax and blurring of vision which have failed to respond to several self-administered medications. He was found to have a severe systemic hypertension on clinical evaluation and laboratory investigations confirmed the multi-organ involvement of malignant hypertension in him

Keywords: malignant hypertension, amaurosis fugax, retinopathy, multi-organ involvement

Case Report

A 36-year old man who sells computer hardware presented with a 3-month history of persistent frontal headache relieved by analgesics. The headache was accompanied by nausea, though no vomiting or fever. He had also been increasingly fatigued and breathless on moderate exertion, in addition to transient episodes of orthopnea and palpitation. He had no cough, chest pain, paroxysmal nocturnal dyspnea nor ankle swelling. He reported an incidence of transient visual loss lasting a few seconds just two days before he presented in the hospital which was different from the blurred vision he had been experiencing a week earlier. Except for a recent onset of nocturia, he had no other symptoms referable to the kidneys.

He had no prior history of hypertension or diabetes. He had a significant alcohol consumption and cigarette smoking. No history of hypertension or diabetes in his first degree relatives.

His physical examination showed pallor, tachycardia (heart rate of 120beats/min) and a blood pressure of 220/150mmHg.The apex was displaced to left anterior axillary line in the 6^{th} intercostal space. A 4^{th} heart sound and a loud pan systolic apical murmur were heard in addition.

His respiratory rate was 36cycles/min with flaring of his ala nasae and coarse crackles in right lower zone posteriorly.

Fundoscopy revealed features of grade 4 hypertensive retinopathy.

Laboratory investigations showed a PCV of 28%, serum urea of 59mg/dl (normal 15-45mg/dl), serum creatinine of 2.4mg/dl (normal 0.5-1.2mg/dl), and serum potassium of 3.3mmol/l.

Other hematological and biochemical results were within normal limits.

Urinalysis showed traces of protein but no red cells. Chest radiograph showed a hazy right lower lung zone, cardiomegaly (CT ratio 58%) with left ventricular preponderance. and unfolding of the aortic arch. The electrocardiogram revealed evidence of left atrial and ventricular enlargements and anterolateral wall infarct. Echocardiography also revealed Septal Hypertrophy. His abdominal ultrasound scan revealed neither enlarged nor shrunken kidneys but financial constraints precluded an Intra-Venous Urogram (IVU) and other tests (hormonal) to exclude causes of secondary hypertension.

His blood pressure was controlled within 72 hours of admission with parenteral and oral anti-hypertensive agents and he was discharged to the outpatient clinic on oral nifedipine and lisinopril for follow-up. The pansystolic murmur was no longer audible at discharge.

Literature Review

Malignant hypertension is one of the complications of hypertension and is characterized by severe elevation of the systemic blood pressure (diastolic blood pressure >130mmHg[1]) accompanied by damage to end organs (usually the brain, heart and kidneys). The presence of end-organ damage differentiates hypertensive emergencies from urgencies and this differentiation is essential in its proper treatment.[2]

Malignant hypertension can occur in the course of both primary and secondary hypertension[1] and may rarely be the initial presentation of systemic hypertension[3]

Less than 1% of hypertensive patients develop malignant hypertension[1] but there is an increased risk in patients with secondary hypertension.

It is commoner in blacks[4] and in men. The average age at presentation is 40 years[5]

Malignant hypertension can be considered an extreme phenotype of rennin-mediatedhypertension[6]. However, the variations in renin-angiotensin system (RAS) activation in malignant hypertension are not completely understood[7] but two main mechanisms are involved in the clinical manifestations.

The first is the loss of cerebral autoregulation due to sustained, marked blood pressure elevation and this result in cerebral arterial dilation, increased cerebral blood flow and cerebral edema that is responsible for the features of encephalopathy seen in them.

In addition, diffuse fibrinoid necrosis of small arteries and arterioles that are characteristic of malignant hypertension give rise to micro-angiopathic hemolytic anemia as red cells traverse these vessels.

A proposed mechanism for ongoing RAS activation is the presence of microangiopathic hemolysis resulting in renovascular ischemia[7.] This contributes to the renal function impairment seen in them.

Drugs such as monoamine inhibitors, oral contraceptives, cocaine and withdrawal of beta blockers, clonidine, alcohol and steroids have been associated with hypertensive emergencies.[5]

Early occurrences of organ damage, mainly in the cardiovascular system, the central nervous system, the kidney or the retina, are hallmarks of a malignant course in arterial hypertension.[8]

Clinical features therefore include elevated blood pressure which may be accompanied by severe headache, vomiting, visual disturbances, seizures, focal neurological deficits and coma in varied combination. Features of other organs involved such as dyspnea, angina, pulmonary edema, myocardial infarction or oliguria may be present. Patient may have lateralizing signs making it difficult to distinguish from intracerebral hemorrhage. However, involvement of other organs and a grade III-IV retinopathy will leave the diagnosis in no doubt.

Laboratory tests are required to establish the involvement of many organs as well as establish the secondary causes of hypertension such as phaeochromocytoma, thyrotoxicosis and intrinsic renal diseases.

In treating hypertensive emergencies, blood pressure should be reduced by about 10% during the first hour and another 15% gradually over the next 2 to 3 hours to prevent cerebral hypoperfusion[9]. Therapy in malignant hypertension is directed at reducing the diastolic BP by a third but not < 95mmhg within 24hours[1] and correcting the complications resulting from the involvement of the end-organs. Hospital admission is mandatory as intravenous antihypertensives will be needed to quickly reduce the blood pressure and stem the damage to the end-organs. Some of the drugs used include hydralazine, labetalol, enalaprilat, esmolol, metoprolol, nicardipine, and fenoldopam especially in patients with renal insufficiency. Phentolamine is used in phaeochromocytoma.[1]

Prior to the availability of effective therapy life expectancy was less than 2 years, survival rate was less than 25% in 1year and < 1% in 5 years, with most deaths resulting from stroke, renal or cardiac failure. [5]

With current therapy improved survival rate of >90% in 1year and >80% in 5years are now achieved. [5]

Biblography

1. **Harrison's Principles of Internal Medicine** 16TH Edition, part VIII, section 4, chapter 230, page 1480.
2. **Shafi T**. Hypertensive urgencies and emergencies. *Ethn Dis*. 2004; 14(4):S2-32-7.
3. **Muñoz S, Martín N, Arruga J**. Malignant hypertension: ocular manifestations. *ScientificWorldJournal* 2006; 26;6:122-4
4. **van den Born BJ, Koopmans RP, Groeneveld JO, van Montfrans GA**. Ethnic disparities in the incidence, presentation and complications of malignant hypertension. *J Hypertens* 2006; 24(11):2299-304.
5. **van den Born BJ, van Montfrans GA, Uitterlinden AG, Zwinderman AH, Koopmans RP**. The M235T polymorphism in the angiotensinogen gene is associated with the risk of malignant hypertension in white patients. *J Hypertens* 2007;25(11):2227-33.
6. **van den Born BJ, Koopmans RP, van Montfrans GA**. The renin-angiotensin system in malignant hypertension revisited: plasma renin activity, microangiopathic hemolysis, and renal failure in malignant hypertension. *Am J Hypertens* 2007; 20(8):900-6
7. **John D Bisognana**. Hypertension,malignant. 2006: *eMedicine*
8. **Schaller G, Wolzt M**. Malignant and refractory hypertension. *Wien Med Wochenschr*. 2006;156(17-18):488-93
9. **Feldstein C**. Management of hypertensive crises. *Am J Ther* 2007;14(2):135-9.