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Case Report

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HYPERTENSIVE URGENCY WITH BEYOND-THE-SPHYGMOMANOMETER SYSTOLIC BLOOD PRESSURE IN GENERAL HOSPITAL OJI RIVER, ENUGU STATE, SOUTHEAST NIGERIA

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ABSTRACT

Hypertensive urgency is defined as severe hypertension (with diastolic pressure > 120 mm Hg) without end-organ damage. The case reported was that of a 64-year old woman, a known hypertensive, who presented to our hospital, General Hospital Oji River, with a systolic blood pressure (BP) that was above 300 mmHg, and therefore could not be measured. Her general condition was stable, hence she was managed as a case of hypertensive urgency with a parenteral antihypertensive drug after a period of rest. After about 4 hours on admission, following the initial treatment, her systolic BP came down to 220/80 mm Hg. She was discharged home the next day to continue with her oral drugs, and to return to the hospital in one week's time for a follow-up.

KEYWORDS: Hypertension, urgency, severe, General Hospital, Oji River.

INTRODUCTION

Hypertensive crisis is characterized by severe and abrupt elevation of the blood pressure (BP), usually defined by diastolic pressure above 120mm Hg.[1] It is classified as a hypertensive urgency when there is no end-organ damage, and as hypertensive emergency when there is a risk of death evidenced by end-organ damage.[2] A third entity known as hypertensive pseudocrisis, is characterized by a transient elevation of the BP during painful or emotional events such as headache, rotational dizziness, anxiety, or panic syndrome.[3]

The major causes of hypertensive crisis are inadequate or poor adherence to current antihypertensive regimens and inaccurate BP measurements that underestimate or do not detect increased BP at all. Although the pathophysiology of hypertensive crisis is complex and still remains incompletely understood, an initial vascular resistance seems to be a necessary first step. This increased vascular reactivity may be precipitated by the release of vasodynamic substances such as norepinephrine and angiotensin II, and can occur as a result of relative hypovolaemia.[4,5] The complementary endothelial vasodilatory responses are overwhelmed, leading to endothelial decompensation, which promotes further rises in BP.[4]

The clinical picture of hypertensive crisis may range from asymptomatic presentation to specific symptoms characterizing acute organ damage (dyspnoea, chest pain, neurological disorders). In the absence of organ manifestations, the patient may complain of non-specific symptoms such as palpitations, headache, and dizziness.

Aggressive treatment of hypertensive urgency is not recommended. The BP reduction should be gradual, using oral medications, as no benefit, but potential harm, may be associated with a rapid BP decrease, due to a rightward shift in the pressure/flow auto-regulatory curve in critical beds (cerebral, coronary and renal).[6,7] The initial treatment involves rest. If after 30 minutes of rest, and re-evaluation of the BP shows that it is still high and no treatable causes found, the doctor should consider altering his chronic antihypertensive regimen to promote long-term BP control. The use of sublingual nifedipine is contra-indicated, as it causes an unpredictable, and often too rapid and large, decrease in BP reduction.[8]

In the epidemiology of hypertensive crisis, it has been found that hypertensive urgencies are more prevalent than hypertensive emergencies. A number of past studies have proved this to be true. For instance, a 1992 Italian study showed that hypertensive crisis represented 3% of all medical emergencies, with a prevalence of 24% and 76% respectively for emergencies and urgencies.[9] Risk factors possibly promoting a hypertensive crisis, as revealed by the study, included female sex, higher degree of obesity, hypertensive or coronary heart disease, higher number of antihypertensive drugs, and most importantly, non-adherence to medications.[10] In a similar study in

Uganda, the prevalence of hypertensive crisis was found to be 5.1%, while hypertensive urgencies were in 32.5% of the cases.[11]

This case was considered worthy of reporting because the patient's systolic BP could not be measured as it was found to be higher than the maximum calibration of the measuring sphygmomanometer (>300 mm Hg).

CASE REPORT

A 64-year old woman presented to our hospital, General Hospital Oji River, Enugu State, Southeast Nigeria, on the 16thof September 2020 with complaints of headache, dizziness and generalized body weakness. Past medical history revealed that she has been hypertensive for over ten years now, but not adherent to her antihypertensive drugs. She was lost to follow-up for about seven months before the present events. One week prior to the present visit, her BP was 210/90 mm Hg.

Family history revealed that HTN runs in her family, as her sister is also hypertensive. Patient is a farmer who resides in a rural area. Her education did not go beyond the primary level. She is obese (obesity class III) with a body mass index (BMI) of 44kg/m^2. Her systolic BP was not measurable as it was over 300 mm Hg, beyond the calibration of the sphygmomanometer. Her diastolic BP was 130mm Hg. Her BP was recorded as >300/130mm Hg. Her general condition was stable, as she did not manifest any signs of end-organ damage. Full blood count, urinalysis and serum urea, electrolytes and creatinine were all normal.

The patient was admitted on the general ward for subsequent management. After about 30 minutes of rest, her systolic BP was checked again and it was still beyond the sphygmomanometer calibrations (>300 mm Hg). She was injected with 20 mg of hydralazine intramuscularly. Three hours after the hydralazine injection, her BP came down to 220/80 mm Hg. She was then switched back to her oral antihypertensive drugs. The next day she was discharged home to continue with her oral drugs. She was booked for a revisit after one week.

DISCUSSION

We consider the case we reported a hypertensive urgency because it did not have the characteristics of an emergency or pseudocrisis. In spite of the extremely high systolic BP, our patient was still apparently stable. This could be considered a case of an exceptional compensatory mechanism of the body, for this level of the BP could easily tip the patient into hypertensive emergency, yet it did not. We suspected that non-adherence to her antihypertensive drugs had played a major role in the development of hypertensive urgency in this patient. This finding affirms what had been found by other researchers in the past.[12,13] Other factors that could have contributed to this include higher degrees of obesity (BMI of 44kg/ m ^{2}), the female sex, and illiteracy. To a large extent, these factors are the same as those that had been earlier reported by.[13]

The line of management we adopted was justified by the fact that our setting (rural setting and illiteracy on the part of the patient, which is largely responsible for her non-adherence to her drugs) would not permit outpatient management with oral drugs. Moreover, we suspected that it was non-adherence to her drugs that precipitated the present crisis and there was no guarantee that she would act differently this time. Furthermore, we did not know what the systolic BP was since it had gone beyond the last calibration of the sphygmomanometer. Why this patient had remained stable despite the extremely high systolic BP was not clear to us. This was one reason we considered this case worthy of being reported.

CONCLUSION

Although hypertensive urgency is a severe type of HTN, there is no urgency in its management as demonstrated by the case reported. We therefore consider hypertensive urgency a misnomer, since even the standard management protocol does not reflect any sense of urgency at all. This is why we strongly align with those who are questioning the usefulness of this term. In the light of this, we recommend a change of nomenclature for this entity, to reflect the true situation. In its place, we are of the opinion that asymptomatic severe HTN, as has been suggested by some researchers, or compensated severe HTN, might be more appropriate. Finally, we suggest an extension/expansion of the upper limit of the sphygmomanometer calibration by instrument manufacturers, to about 350-400mm Hg, in order to take care of systolic BPs that might rise above 300mmHg.

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