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Hypertensive emergency occurred due to forgetting to take antihypertensive medication: A case study

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Abstract

A life-threatening condition known as a "hypertensive emergency" is marked by a severe increase in blood pressure together with acute or significant target-organ damage. On 1 June 2022, a 67-year-old black male farmer was admitted to the emergency department with a major chief complaint of breathing difficulty. The patient was traveling to the village for work and forgetting his medication at home, and he was losing consciousness and motor activity at his workplace. He presented with symptoms of shortness of breath, confusion, dizziness, nausea, vomiting, blurred vision, and faintness. An abnormal cardiac region was visible on chest X-rays, and there were no changes to the pulmonary parenchyma or fluid overload. Upon admission, hydralazine (5 mg) intravenously was administered immediately, and he was reassessed after 20 min and kept at the emergency department. The next day, sustained-release nifedipine (20 mg) was initiated orally twice a day for the patient, and he was transferred to the medical ward. In the medical ward, the patient was assessed for 4 days, and in those 4 days, he showed marked improvement. Hypertensive emergency treatment intends to reverse target-organ damage, readily lowering blood pressure, decreasing adverse clinical complications, and enhancing the quality of life.

Keywords

Antihypertensive medication, case report, hydralazine, hypertension, hypertensive emergency

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Introduction

According to the 2020 International Society of Hypertension Global Hypertension Practice Guidelines, "hypertension emergency" is defined as "substantially elevated blood pressure associated with acute hypertension-mediated organ damage."[1] Typical target-organ damage correlated with hypertensive emergencies comprises cerebral infarction, intracerebral bleed, hypertensive encephalopathy, acute pulmonary edema, acute congestive heart failure, acute myocardial infarction, aortic dissection, and eclampsia.[2,3] The commonly known factors that contribute to the development of hypertensive emergencies include elevated sympathetic nervous activity, elevated renin–angiotensin–aldosterone system activity, lowered nitric oxide due to oxidative stress, fluid retention or edema, inflammation, and endothelial dysfunction.[4,5] The goal of treatment in a hypertensive emergency is to lower mean arterial blood pressure by ⩽25% within minutes to an hour after the presentation, followed by a reduction in blood pressure to 160/110 mm Hg or less within the next 2–6 h, and then gradually tend to normal within 2 days.[6] Hydralazine is a direct vasodilator of smooth muscle in the arteries and is administered as an intravenous or intramuscular bolus.[7] Hydralazine stimulates the sympathetic nervous system resulting in tachycardia and activation of the renin–angiotensin–aldosterone system.[8] Hydralazine is the first-line treatment for a hypertensive emergency due to its ability to lower blood pressure rapidly within minutes; its peak action occurs at 10–80 min.[9] This case report demonstrates a hypertensive emergency that occurred due to forgetting to take antihypertensive medication in a youngest-old male patient.

Case presentation

On 1 June 2022, a 67-year-old black male farmer who had been experiencing significant chief complaints of shortness of breath for the previous 4 h and an elevated basal blood

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Table 1. Vital signs of five consecutive days from admission to discharge of the patient.

Days	Typical vital signs			
	Blood pressure (mm Hg)	Heart rate (bpm)	Respiratory rate (bpm)	Saturated oxygen (%)
Day 1	194/106	93	20	91
Day 2	189/101	84	17	88
Day 3	175/99	92	19	93
Day 4	159/96	75	16	98
Day 5	155/93	73	14	97

pressure of 187/102 mm Hg at a nearby clinic for the previous 3 h was admitted to the emergency room. The patient had a 5-year history of hypertension, type 2 diabetes mellitus, *Helicobacter pylori*-positive peptic ulcer disease, and anemia. The patient had taken medications such as hydrochlorothiazide, metformin, glibenclamide, enalapril, and ferrous sulfate, and triple therapy for peptic ulcer diseases such as omeprazole, amoxicillin, and clarithromycin. The newly hospitalized patient had no prior history of using tobacco, consuming alcohol, or drinking black tea. The patient who was admitted did not know his family's past drug and medical backgrounds. The patient was traveling to the village for a 2-day job but forgot his medication at home, and he lost his consciousness and motor activity at the work site. The patient who was admitted was unconscious and unresponsive for 24 h. A rapid rise in blood pressure brought on by non-compliance with antihypertensive medication is the direct cause of the hypertensive emergency in this patient. The admitted patient presented with symptoms of shortness of breath, disorientation, dizziness, nausea, vomiting, blurred vision, and faintness.

His vital signs upon admission showed a high blood pressure of 194/106 mm Hg, a heart rate of 93 beats per minute, a body temperature of 35.8°C, a respiratory rate of 20 breaths per minute, a weight of 63 kg, a height of 1.76 cm, body mass index of 20 kg/m^2, and an oxygen saturation of 91% on room air (Table 1).

Laboratory investigations of the patient revealed: blood urea nitrogen of 29 mg/dL (normal value = 6–20 mg/dL), potassium of 3.7 mmol/L (normal value = 3.6–5.2 mmol/L), sodium of 168 mEq/L (normal value = 135–145 mEq/L), fasting blood glucose of 134 mg/dL (normal value = 100–126 mg/dL), higher density lipoprotein of 56 mg/dL (normal value = 40–60 mg/dL), lower density lipoprotein of 122 mg/dL (normal value = 100–129 mg/dL), triglycerides of 145 mg/dL (normal value = <150 mg/dL), hemoglobin of 13.9 mg/dL (normal value = 13.8–17.2 mg/dL), hematocrit of 47% (normal value = 41%–50%), and serum creatinine of 1.4 mg/dL (normal value = 0.7–1.3 mg/dL), urinalysis revealed urine crystals, white blood cell of 11,340 cells/mm^3 (normal value = 4500–11,000 cells/mm^3), neutrophils of 67% (normal value = 55%–70%), and lymphocytes of 27% (normal value = 20%–40%). The troponin level in this patient is normal because his hypertensive crises did not lead to a myocardial infarction.

In the course of his physical examination, which included a cardiovascular examination, the heart auscultation of S1 and S2 revealed a heart murmur, an increased jugular venous pressure, crackles, and gallops. An abdominal examination showed abdominal swelling correlated with moderate epigastric tenderness. A head, ear, eye, nose, and throat examination revealed pink conjunctiva. Lung auscultation revealed no pulmonary edema and tiny crackles in the respiratory system. The Glasgow Coma Scale, to determine level of consciousness, gave the right arm a score of 2/5, the right leg a score of 3/5, the left arm a score of 2/5, and the left leg a score of 2/5. All four limbs were feeble. The patient scored a total of 9 on the Glasgow Coma Scale (2 + 2 for the upper extremities and 3 + 2 for the lower extremities). Her lower limbs had no edema and symmetric palpable pulses. No space was found to be occupied by a brain lesion, according to a brain computed tomography scan. The absence of any localizing signs was revealed by magnetic resonance imaging (MRI). An abnormal cardiac region was visible on chest X-rays, and there were no changes to the pulmonary parenchyma or fluid overload. Although there was a rapid heartbeat on the electrocardiogram (ECG), there was no evidence of an arrhythmia or a change in the ventricular repolarization of the anterolateral wall. An echocardiogram revealed healthy cardiac structure and function.

Upon admission, hydralazine (5 mg) intravenously was administered immediately, and he was reassessed after 20 min and kept at the emergency department. After receiving two injections of 5 mg of hydralazine, the patient developed reflex tachycardia. The physician then added 50 mg of atenolol, taken orally once a day, to the treatment plan. On the next day, sustained-release nifedipine (20 mg) was initiated orally twice a day for the patient, and he was transferred to the medical ward. In the medical ward, he was assessed for 4 days, and in those 4 days, he showed marked improvement. The patient received advice as he followed a non-pharmacological approach to managing his hypertension, such as consuming a diet high in whole grains, fruits, and vegetables high in nitrates (leafy vegetables) as part of a dietary strategy to treat his condition. In addition to cutting back on items heavy in sugar, saturated fat, and trans fats, he also avoids adding salt when making food. On 6 June 2022, the physician added nifedipine to his past antihypertensive medication for 1 month and discharged him back to his home. The patient was advised to have a monthly ambulatory care check-up.

Discussion

Hypertensive emergency is an acute and severe elevation in blood pressure correlated with target-organ damage.[10] The common symptoms of a hypertensive crisis include headache, blurred vision, nausea, vomiting, chest discomfort,

decreased urine production, shortness of breath, and changes in mental status.[11] The patient who was admitted for this study displayed shortness of breath, confusion, nausea, vomiting, blurred vision, unresponsiveness, and faintness as symptoms. The frequently occurring target-organ damage comprises neurologic, kidney, ocular, myocardial impairment, which manifested as encephalopathy, seizures, blurred vision, and electrocardiography dysfunction, and damaged kidney function.[12] The patient in this study suffered from damage to several organ targets, including the optical system (blurred vision), the heart (dysfunctional ECG), and the brain (neurological deficits). Target-organ damage and severity of blood pressure elevation at the time of presentation of hypertensive emergency are associated with failure of the normal autoregulatory function and abrupt elevation in systemic vascular resistance.[13] Angiotensin II, noradrenaline, or other endogenous compounds are playing a crucial role in the pathophysiology of hypertensive emergencies and causes elevation of vascular resistance.[5] The development of hypertensive emergency linked with the renin–angiotensin–aldosterone system, and polymorphisms of angiotensinogen encoding genes are correlated with early onset hypertension.[14] About 5%–10% of arterial hypertension cases are caused by secondary hypertension, of which primary hyperaldosteronism has an incidence of 0.05%–2% in hypertensive people and is characterized by hypokalemia, increased aldosterone production, decreased or suppressed renin, an elevated aldosterone/renin ratio, and metabolic alkalosis.[15] In this case report, primary aldosteronism is caused by increased sodium level and low potassium level on laboratory tests. The patient in this study had an excess of aldosterone, which raised his blood pressure by reducing potassium and raising sodium levels.

Troponins (chest pain or anginal equivalent), chest X-rays (congestion or fluid overload), transthoracic echocardiograms (cardiac structure and function), computed tomography or MRI of the brain (cerebral hemorrhage or stroke), and computed tomography-angiography of the thorax and abdomen (acute aortic disease) are specific tests for hypertensive emergencies.[1] The imaging analysis of the brain computed tomography scan in this study showed no evidence of a brain lesion. The absence of any localizing indications was revealed by MRI. An abnormal cardiac region was visible on chest X-rays, and there were no changes to the pulmonary parenchyma or fluid overload. The ECG revealed a fast heartbeat, although the anteromedial wall ventricular repolarization was unaffected. An echocardiography revealed a healthy heart structure and function. The main intention of hypertensive emergency management is to control blood pressure within hours and admitted the case to a critical care setting.[7] The goal of hypertensive emergency treatment is to lower the mean blood pressure by ⩽25% in the first 8 h after presentation, followed by gradual normalization of mean arterial blood pressure over the next 26–48 h.[16] Following an intravenous infusion of hydralazine, a 5 mg bolus of hydralazine was administered intravenously over a period of 5–10 min. This was repeated every 20 min, up to a maximum of 20 mg.[17] Hydralazine is a direct vasodilator of smooth muscle in the arteries and is administered as an intravenous or intramuscular bolus.[18] Hydralazine peak action occurred 10–80 min after intravenous administration, and its shelf-life on mean arterial blood pressure is only about 3 h.[19] As a result of hydralazine's stimulation of the sympathetic nervous system, tachycardia, activation of the renin–angiotensin–aldosterone system, and relaxation of the blood vessels, the body's blood flow is improved.[20] In this study, the patient was given 5 mg of hydralazine intravenously right away. Twenty minutes later, he was re-evaluated and retained at the emergency room. Following two injections of 5 mg of hydralazine, the patient experienced reflex tachycardia. The physician then prescribed 50 mg of atenolol daily for the patient. The following day, the patient began receiving sustained-release nifedipine 20 mg twice daily. Due to concerns that it might trigger an acute ischemic event and also cause hypotension, short-acting nifedipine is not advised for rapid blood pressure reduction during hypertensive emergencies.[21]

Conclusion

Hypertensive emergency is an acute, sudden, and severe elevation in blood pressure and correlated with progressive signs of target-organ damage, initially cardiovascular system, brain, and kidneys. The admitted patient in this study presented with symptoms of shortness of breath, disorientation, dizziness, nausea, vomiting, blurred vision, and faintness. In a hypertensive emergency, urgent reduction in mean arterial blood pressure is frequently required. An initial dose of hydralazine 5–10 mg was initiated repeatedly every 20–30 min; a maximum dose of 20 mg should be given until blood pressure is lowered by 25% within minutes to 2 h, and then the mean arterial blood pressure decreased to 160/100 mm Hg within 2–6 h.

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