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CASE REPORT

Hypertensive Urgency and Anterior Epistaxis Caused by Antihypertensive Medication Noncompliance: A Case Report

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Background and Aims: A sudden increase in blood pressure without serious, life-threatening symptoms or indications of immediate target organ damage is referred to as "hypertensive urgency." This case study revealed the rare direct cause of epistaxis and the direct cause of hypertensive urgency in an elderly man due to antihypertensive medication noncompliance.

Case Presentation: A black male farmer, age 63, was brought to the emergency room on June 1st, 2022, with chief complaints of breathing difficulties, epistaxis, and disorientation. The patient was hospitalized after exhibiting symptoms of breathlessness, malaise, nausea, and vomiting. Magnetic resonance imaging, an echocardiogram, and a computed tomography scan of the brain are all clear. For the treatment of epistaxis, he received 1g of tranexamic acid intravenously three times a day for two days. He received intravenous labetalol, which was effective in treating his hypertensive urgency and rebound hypertension, utilizing repeated dosages of 5–20 mg. The patient's intravenous labetalol and previous enalapril were switched to captopril 25 mg orally three times a day for one month after starting drugs per os.

Discussion: The patient's hypertensive urgency is directly caused by forgetting to take his blood pressure medication and by not adhering to his previous antihypertensive drugs as prescribed. In this study, the patient's hypertension had been uncontrolled for the previous six months despite his treatment plan. Unaware that he had missed two doses of his antihypertensive medication, he was admitted to the emergency room with progressive anterior nose bleeding that persisted for four hours. The patient's elevated arterial blood pressure is what's causing the patient's nose to bleed.

Keywords: antihypertensive medication, anterior epistaxis, case report, hypertensive urgency, noncompliance

Introduction

Patients frequently visit healthcare institutions for hypertensive crises, which are clinical syndromes that develop as side effects of untreated or improperly managed hypertension.[1] In 2020, the International Society of Hypertension reported According to the Global Hypertension Practice Guidelines, "hypertensive urgency" is "seriously high blood pressure without severe hypertension-mediated organ damage".[2] End-organ damage is not usually seen in hypertensive urgency. Symptoms of hypertensive urgency often include headaches, epistaxis, tiredness, psychomotor agitation, dyspnea, chest discomfort, and vertigo.[3] Antihypertensive drug non-adherence is thought to be a significant factor in the development of hypertensive urgency, which leads to repeated visits to the emergency room and extended hospital stays.[4] Patients with hypertension were 1.47 times more likely to experience epistaxis than those without hypertension.[5] Captopril is known to be both safe and effective in lowering blood pressure in hypertensive urgencies. When taken orally, its hypotensive action becomes apparent within 15 to 30 minutes.[6] The antidote for fibrinolytic drugs is tranexamic acid. Finally, it stops bleeding by preventing both the production of plasmin and its effects.[7] This case study revealed the rare direct causes of epistaxis and the direct cause of hypertensive urgency in an elderly man due to antihypertensive medication noncompliance.

Received: 4 December 2022
Accepted: 27 January 2023
Published: 3 February 2023

Case Presentation

On June 1st, 2022, a 63-year-old black male farmer was brought to the emergency room with symptoms of disorientation and breathing difficulties. The patient was brought into the emergency room four hours earlier with significant anterior nose bleeding. The physician took the patient's blood pressure and discovered that it was 196/113 mmHg based on the patient's historical history after his caregivers informed him of his previous medical and drug histories. The physician recommended tranexamic acid 1 g intravenously three times a day for two days after the blood pressure was checked and determined to be raised without any laboratory investigations, physical examinations, or imaging evaluation. The patient had a sixteen-year history of type 2 diabetes mellitus and a nine-year history of hypertension. The patient was treated for his prior type 2 diabetes mellitus with metformin and glibenclamide. He used enalapril and furosemide for his prior hypertension. He had previously smoked cigarettes and consumed alcohol. Five years prior, he gave up drinking alcohol and smoking cigarettes. Due to the patient's failure to take his antihypertensive medications consistently during the previous six months, atenolol was added to his treatment regimen. The patient who was admitted had no prior medical or drug history in his family. The patient's hypertensive urgency is a direct result of forgetting to take his blood pressure management drugs and of not adhering to his previous antihypertensive drugs as prescribed. The patient was hospitalized after exhibiting symptoms of breathlessness, malaise, nausea, and vomiting. The patient lost consciousness and lost motor function while traveling to the village for a two-day business trip and left his medicine at home.

At the time of admission, his vital signs included a high blood pressure of 196/113 mmHg, a heart rate of 93 beats per minute, a body temperature of 35.8 °C, a respiratory rate of 20 breaths per minute, a height of 1.70 m, a weight of 83 kg, a body mass index of 28.7 kg/m2, and an oxygen saturation of 91% on room air. Up on his admission blood chemistry 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), 2-hour postprandial blood glucose of 203 mg/dL (normal value: 140–180 mg/dL), higher density lipoprotein of 59 mg/dL (normal value: 40–60 mg/dL), lower density lipoprotein of 130 mg/dL (normal value: 100–129 mg/dL), triglycerides of 145 mg/dl, (normal value: < 150 mg/dL), tests for coagulation are normal, hemoglobin of 13.5 mg/dL (normal value: 13.8–17.2 mg/dL), hematocrit of 37% (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%).

His cardiac auscultation revealed that he had not have elevated jugular venous pressure, crackles, and gallops in addition to no heart murmurs in S1 and S2. An abdominal examination revealed no edema and a link to some minor epigastric discomforts. Pink conjunctiva was discovered during a head, ear, eye, nose, and throat examination. During the neurological examination to determine his degree of awareness, it was found that he was unconscious and had eyesight problems (blurred vision rather than optic neuritis). The right arm received a score of 3/5, the right leg received a score of 3/5, the left arm received a score of 2/5, and the left leg received a score of 3/5 on the Glasgow Coma Scale, which was used to measure level of consciousness. On the Glasgow Coma Scale, the patient received an overall score of 11 out of 15. Lung auscultation revealed no pulmonary edema and minute crackles in the respiratory system. Even though the electrocardiogram (ECG) showed a rapid heartbeat, there was no sign of an arrhythmia or a change in the ventricular repolarization of the anterolateral wall. The electrocardiogram's readings were normal, and the adjusted QT interval was 0.78 seconds. Chest X-rays revealed an aberrant cardiac area (which shows the length of the cardiac cycle is changed without a compensatory change in the length of the refractory period), although the pulmonary parenchyma and fluid overload were unaffected. Magnetic resonance imaging, an echocardiogram, and a computed tomography scan of the brain are all clear. There is no sign of any bruising or petechiae that could point to an underlying hematologic disorder.

After taking tranexamic acid for 48 hours, the patient was able to recover from epistaxis. He had received doses of 5–20 mg of intravenous labetalol, which effectively treated his hypertensive urgency and rebound hypertension. The patient's intravenous labetalol and previous enalapril were switched to captopril 25 mg orally three times until hypertensive urgency was managed after he began taking the medication orally. In addition to his existing hypertension medication regimen, which included furosemide, atenolol, and captopril, the physician added amlodipine 5 mg orally,

once daily for 30 days. The patient was instructed to engage in regular aerobic exercise as well as follow a dietary approach to prevent hypertension.

Outcome and Follow-Up

He was observed for seven days in the medical ward, and by day 10, he had significantly improved. His heart rate dropped to 78 beats per minute, and his blood pressure recovered to 149/97 mmHg after 10 days. He returned home after being discharged. He was advised to have a monthly ambulatory care follow-up.

Discussion

Hypertension urgency lacks aortic dissection, abrupt pulmonary edema, intracerebral hemorrhage, unstable angina, eclampsia, or hypertensive encephalopathy, examples of acutely increasing target organ damage that separate from hypertension emergency.[8] In this study, there was no target organ damage. Due to missed antihypertensive medication, prior medical history, and non-adherence to previous antihypertensive drugs, the patient had continuously raised blood pressure and was experiencing hypertensive urgency.

Acute bleeding into the nostril, nasal passage, or nasopharynx is known as epistaxis.[9] Epistaxis is thought to be brought on by a variety of factors, including dry air (changes in humidity), infection, allergies, trauma, alcohol addiction, intranasal tumors, and the use of anticoagulants. One of the few direct risk factors for epistaxis among the various risk variables is hypertension.[10] In this study, the patient's hypertension was uncontrolled for the previous six months despite his treatment plan. Unaware that he had missed two doses of his antihypertensive medication, he was admitted to the emergency room with worsening anterior nose bleeding that persisted for four hours. His arterial blood pressure is elevated, which is what is causing this patient's nose to bleed. A blood vessel that supplies the nasal mucosa can rupture due to high blood pressure. Because high blood pressure affects blood vessels, it's possible that the blood vessels in the nose are more prone to harm and more likely to bleed as a result or

Elevated blood pressures ➔ rupture of blood vessel in nasal mucosa ➔ anterior nose bleeding

➔Extend the duration of the nose bleeding episode (epistaxis).

The patients in this study had class I, or low-risk obesity, which increases fat storage, and a commensurate rise in leptin signals that the body has enough energy reserves. The body mass index for the patient in this study was 28.7 kg/m^2. Hypertension is brought on by obesity, which stimulates the hypothalamus's blood vessel development. Due to his comorbidity, type 2 diabetes mellitus destroys his body's tiny blood vessels, stiffening their walls, which raises pressure and results in high blood pressure. The patients in this study had a history of smoking and drinking, which are risk factors for the hypertensive crisis known as hypertensive urgency.

Due to humoral vasoconstriction, there is a sudden increase in systemic vascular resistance during a hypertensive episode. Elevated blood pressure results in fibrinoid necrosis and intimal proliferation via increased endothelial permeability and local activation of the clotting cascade (platelet and fibrin deposition). As a result, the endothelium is unable to self-regulate or respond to variations in blood pressure.[11] By inhibiting fibrinolysis when first administered, tranexamic acid is used to halt nasal bleeding.[12] By reducing excessive bleeding and preventing rebleeding, tranexamic acid has an antifibrinolytic effect. Usually, bleeding is treated with tranexamic acid for a brief period of time. To treat other bleeding that does not cease on its own, this medication is used for seven days or four days if you have heavy periods.[13] Due to the patient's quick admission to the emergency room shortly after the anterior nose bleeding started, the patient in this study recovered from epistaxis after receiving tranexamic acid for 48 hours.

Hypertensive urgency management's goal is to gradually lower blood pressure to equal or less than 160/100 mmHg over the course of several hours to days, not readily. Contrary to a hypertensive emergency, hypertension urgency can be treated with oral or injectable medicines to resolve spontaneously or within 24 to 48 hours.[14,15] In the first 24 hours, the mean arterial blood pressure should not drop by more than 25%.[16] After receiving intravenous labetalol in this study, the patient's blood pressure fell by less than 160/100 mmHg after eighteen hours. He was administered labetalol 20 mg

intravenously every 20 minutes until his target blood pressure was reached. The patient's intravenous labetalol and previous enalapril were switched to captopril 25 mg orally three times a day for the whole day after he began taking the medication orally. He stayed in the medical ward for seven days, and by day 10, he had partially improved and was sent back to his home. He recommended going on follow-up at the ambulatory care center monthly.

Conclusion

A clinical condition known as hypertensive urgency occurs when the blood pressure is extremely high yet there are little or no symptoms or indicators of recent target-organ damage. Epistaxis affects hypertensive people more frequently, maybe as a result of the nose's vascular fragility brought on by a chronic disease. The patient in this study had class I obesity, advanced age, and other concomitant diseases, including type 2 diabetes mellitus and hypertension, which are risk factors for his hypertensive urgency. His prior medical history, the last six months of non-compliance with his previous antihypertensive medications, and forgetting to take his hypertension prescription are the main causes of his hypertensive urgency. He had received doses of 5–20 mg of intravenous labetalol, which effectively treated his hypertensive urgency and rebound hypertension.

Informed Consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

Acknowledgments

The author is grateful to the study participant for his willingness to respond to all questions.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors'.

Disclosure

The authors declare no conflict of interest in this work.

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