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Case Report

Blood Pressure Fluctuations Due to Lurasidone Reversal: A Case Report

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Abstract

Background of study: Although catecholamine medications are frequently employed to promote circulation, in certain circumstances they may unexpectedly result in hypotension. Here, we report the first instance of hypotension that was unanticipated in response to catecholamine drugs.

Case presentation: A 36-year-old guy from Addis Ababa,Ethiopia was brought to our emergency room due to schizophrenia. He was unconscious and in shock. We administered catecholamine agents after fluid resuscitation, however the catecholamine infusion caused his blood pressure to drop to 62/45 mmHg. However, his blood pressure significantly improved once we started vasopressin, and he eventually became stable. He acknowledged taking a lot of lurasidone on day two, and we determined that he had overdosed on the medication. We surmise that the lurasidone-induced α-adrenergic blockade was the reason for this unanticipated hypotension in response to catecholamine infusion. Research on animals demonstrated that concurrently administering adrenaline and α-adrenergic inhibition caused a drop in blood pressure, a phenomenon known as "adrenaline reversal." We refer to this occurrence as "catecholamine reversal" since in our situation, catecholamine infusion under the α-adrenergic blocking effect of lurasidone may have induced a drop in blood pressure through the same mechanism. Since vasopressin functions differently in some situations than catecholamines, we advise using it to keep blood pressure stable.

Conclusions: We presented the first documented clinical instance of "catecholamine reversal" and emphasized that we should be suspicious of the use of α-adrenergic antagonists if unexpected hypotension results from a catecholamine infusion. Instead, we ought to think about giving vasopressin in these circumstances.

Keywords: Cardiovascular; Emergency medicines; Cardiac shock; Electrocardiogram; Intensive care units

Introduction

Often used to promote circulation, catecholamines act differently on various receptors depending on the agent [1]. Catecholamine activity improves chronotropic and inotropic effects on β1 adrenergic receptors, increases vasodilation on β2-adrenergic receptors, and promotes peripheral vasoconstriction on α-adrenergic receptors [2]. We often employ catecholamine drugs, such as dopamine and noradrenaline, to enhance vasoconstriction in distributive shocks like septic shock since they primarily impact the α-adrenergic receptor [3]. Nonetheless, in some circumstances, these catecholamine medications may result in unanticipated hypotension [4]. Here, we report a case of unanticipated hypotension brought on by the use of catecholamine medications.

Presentation of Case Study

A 36-year-old guy from Addis Ababa, Ethiopia was brought to our emergency room due to schizophrenia. He arrived in a state of shock and unconsciousness. His body temperature was 36.6°C, his heart rate was 140 beats per minute (bpm), his respiratory rate was 40 breaths per minute, and his blood pressure was 62/45 mmHg. (E1V1M1) was his Glasgow Coma Scale score. Due to his shock and unconsciousness, we conducted intubation right away. His blood pressure rose to 75/30 mmHg after receiving 3000 ml of crystalloid fluid resuscitation, but his shock remained. We tentatively started dopamine infusion at 5μg/kg per minute, then raised it to 10μg/kg per minute be-

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Table 1: Findings on arrival.

Blood CP		Clotting	
WBC	5700/µl	PT-INR	1.06
RBC	450×104/µl	APTT	22.6
Hb	12.1 g/dl	Fib	220 mg/d
Chemical lab values		**ABGs**	
ALT	17 U/L	pH	7.31
AST	20 U/L	pCO2	37.7 mmHg
CPK	134 U/L	pO2	482 mmHg
Cr	1.20 mg/d	HCO-	20.1 mmol/l
Na	145 milli Eq/l	-	-
K	2.5 milli Eq/l	-	-
Cl	106 milli Eq/l	-	-

Table 2: ABGs in ICU.

Agents	Values
Partial pressure of CO_2	35.8 mmHg
Partial pressure of O_2	216 mmHg
Bicarbonate ions	14.7 mmol/l
Base excess	−11.2 mmol/l
Sodium	142 mmol/l
Potassium	2.6 mmol/l
Chloride	106 mmol/l

fore inserting a central venous line; nonetheless, his hypotension deteriorated with time to 50/30 mmHg. After arriving for seventy minutes, we started the infusion of noradrenaline at 0.1 µg/kg per minute and then raised it to 0.3 µg/kg per minute by inserting the central venous line. Furthermore, we started dobutamine at 5 µg/kg every minute 95 minutes after arriving. His blood pressure suddenly dropped to 50/35 mmHg, though. The etiology of the coma and hypotension was not found using head computed tomography, improved chest-abdominal computed tomography, point of care sonography, or laboratory data (Table 1). He had an extremely low Systemic Vascular Resistance Index (SVRI) of 430 dynes/second/cm/m2, which is within the normal range of 1970 to 2400 dynes/second/cm/m2. We thus suspected an unknown distributive shock that was resistant to a significant infusion of catecholamines. Thus, 165 minutes after arrival, we started vasopressin infusion at 3 U/hour in addition to catecholamine infusion. Due to an improvement in his metabolic acidosis: pH is 7.36; partial pressures of carbon dioxide and oxygen in arterial blood are 39.2 and 73.6 mmHg, respectively; bicarbonate (HCO-) is -1.2 mmol/l, lactate (Lac) is 1.6 mmol/l, and the Fraction of Inspired Oxygen (FiO2) is 0.4. We stopped the vasopressin injection on day two. His status was stable upon extubation: BP of 124/78 mmHg and HR of 100 bpm. We moved him to our general ward. He acknowledged taking about 150 mg of lurasidone in an effort to end his life. His blood sample had a very high concentration of lurasidone upon admission; on day two, that value had dropped to 3.58 ng/ml. As a result, we identified a lurasidone overdose. After that, nothing happened to his condition, and on day five, he was sent to a psychiatric facility for mental health treatment.

Discussion

We hypothesize that the catecholamine action under the α-adrenergic blockade effect of lurasidone is the pharmacological phenomena that caused the unanticipated hypotension we observed in response to the catecholamine infusion. When α-adrenergic receptor blockers, such phentolamine, are given alongside adrenaline in animal tests, the actions of the α-adrenergic receptor are obscured and the effects of the β2-adrenergic receptor are primarily amplified [5]. Vasodilation therefore takes place, and blood pressure drops. We refer to this unusual occurrence as "adrenaline reversal."[6]. In clinical settings, adrenaline reversal has also been seen; in a case of significant quetiapine overdose, paradoxical hypotension resulting from adrenaline infusion has been documented due to quetiapine's α-adrenergic blocking action [7]. According to this study, adrenaline reversal happens even when a significant overdose of antipsychotics occurs [8]. We disagree with this report's recommendation to use noradrenaline for hypotension caused by α-adrenergic blockade effects, such as quetiapine overdose. This is due to the fact that additional animal studies demonstrated noradrenaline might also result in the same phenomena known as "nor-adrenaline reversal" [9]. Despite this, noradrenaline has α-adrenergic effects that are greater than β-adrenergic actions. As a result, we recommend that in these circumstances, we stay away from noradrenaline. Additionally, both dopamine and dobutamine have α- and β-adrenergic actions [10]. We speculate that catecholamine reversal may likewise be caused by dopamine and dobutamine. Therefore, in individuals who have taken α-adrenergic antagonists, catecholamine medications other than adrenaline may cause "catecholamine reversal" [11]. Due to the α-adrenergic blockade action of lurasidone, hypotension may have been produced by a substantial quantity of catecholamine injected under the influence of this blockade. Conversely, vasopressin is a kind of vasoactive drug that acts through V1 receptors to enhance peripheral vasoconstriction [12] and is frequently used, especially in distributive shock, to preserve vasoconstriction [13]. Vasopressin treatment for our patient's acute hypotension resulted in an instant improvement. This may be explained by the fact that catecholamines and vasopressin work through distinct mechanisms. Vasopressin, therefore, could be helpful in helping patients who have taken α-adrenergic antagonists maintain circulation [14]. The term "catecholamine reversal" has never been used to characterize sudden hypotension in a clinical setting before. Since most anti-psychotic medications block α-adrenergic receptors, this instructive instance emphasizes the need to choose the appropriate vasoactive agent for the patient taking these drugs [15].

Conclusions

We presented the first documented clinical instance of "catecholamine reversal" and emphasized that we should be suspicious of the patient's usage of α-adrenergic antagonists if unexpected hypotension follows catecholamine infusion. Instead, we ought to think about giving vasopressin in such a circumstance.

Author Statements

Competing Interests

The authors declare that they no known competing financial interests or personal relationship that could have appeared to influence the work reported in this paper.

Author Contributions

All authors contributed to the study conception and design. Material preparation, data collection by [Vemparala Priyatha] and [Sowmya Durga Subhasri Guna] analysis were performed by [Motuma Gonfa Ayana] and [Arwa Taha Fadlalla Elkhalifa] The first draft of the manuscript was written by [Jyothika Venkata swamy Reddy] and [Simon Tsegaye Geleta] literature review by [Ruth Betremariam Abebe] and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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