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CLEVIDIPINE INDUCED INTRAPULMONARY SHUNTING IN A PATIENT WITH ACUTE TYPE B AORTIC DISSECTION

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ABSTRACT

Clevidipine is a short acting calcium channel blocker commonly used to treat uncontrolled hypertension. The common side-effects include depression, dizziness, and headache. Hypoxia from intrapulmonary shunting after clevidipine use has not been widely reported in the literature. We present a case of acute type B aortic dissection treated with esmolol and clevidipine complicated by hypoxia which resolved post discontinuation of clevidipine.

KEYWORDS

Clevidipine, intrapulmonary shunting, aortic dissection

LEARNING POINTS

- Clevidipine may be used as an adjunct to beta blockers if the patient is still hypertensive or beta blockers are contraindicated.
- Intrapulmonary shunting leading to hypoxia is a rare complication of clevidipine therapy and should be suspected in an otherwise hypoxic patient in whom all other diagnoses have been ruled out.

INTRODUCTION

Clevidipine is a short-acting calcium channel blocker that is often used to manage hypertensive emergencies due to its selective arterial vasodilatory effects and rapid titration capability[1]. Clevidipine's potent vasodilatory properties can cause redistribution of pulmonary blood flow leading to ventilation-perfusion mismatch resulting in hypoxemia. This case highlights the aforementioned rare adverse effect of clevidipine in a patient with type B aortic dissection who required rapid blood pressure control for severe hypertension. It emphasizes the need for vigilance when managing critically ill patients without pre-existing respiratory compromise.

CASE PRESENTATION

A 56-year-old male construction worker with a documented history of hypertension (non-compliant with prescribed medications) presented to the emergency department with new-onset sharp, mid-sternal, pleuritic chest pain. This pain began approximately 2 hours prior to arrival and was rated

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as 10 on a scale of 10. The discomfort radiated to the back and was accompanied by several episodes of nausea and vomiting. No exacerbating or alleviating factors. The patient has a three-pack year smoking history and denies the use of illicit drugs or alcohol.

On physical examination, vital signs revealed severe hypertension, with a blood pressure reading of 217/95 mmHg, respiratory rate 16 breaths per minute, heart rate 56 beats per minute and saturating 98% on room air, obtained through symmetrical measurements without significant discrepancy. He appeared diaphoretic and pale. S1 and S2 were audible without murmurs, rubs or gallops, no rales, or rhonchi appreciable on auscultation. The electrocardiogram demonstrated sinus bradycardia at 48 beats per minute, with slight ST elevation in the precordial leads, but no other notable ST segment changes. Troponin levels were negative. Additional laboratory tests are summarized in *Table 1*. Chest X-ray showed a widened mediastinum without any consolidations or effusions. Computed tomography (CT) angiogram of the chest revealed a type III/B aortic dissection characterized by an intimal flap arising just distal to the left subclavian artery, extending in a distal direction. The celiac axis, superior mesenteric artery, and renal arteries were opacified via the designated true lumen. Additionally, pulmonary embolism was ruled out. Vascular surgery opted for medical management with the aim of maintaining a systolic blood pressure of less than 120 mmHg and a heart rate of less than 80 beats per minute.

The patient's pain was effectively managed with morphine. To address his elevated blood pressure, a bolus dose of labetalol was administered, followed by an intravenous infusion. Given the patient's bradycardia, however, the patient was transitioned to clevidipine drip and esmolol was carefully implemented. Concurrently, oral antihypertensive medications were initiated.

During the night, the patient exhibited tachypnea and hypoxia with oxygen saturation of 80% on 4 l/min oxygen via nasal cannula. Blood gas analysis showed pH 7.36, pCO_2 35.9, pO_2 55.7 and oxygen saturation 89.7% on a fraction of inspired oxygen $(FiO)_2$ of 40%. The patient was initially treated with 5 l/min oxygen via nasal cannula, but due to continued desaturation and dyspnea, he was later transitioned to bilevel positive airway pressure (BiPAP) ventilation. Furthermore, inhaler therapy was initiated due to suspected chronic obstructive pulmonary disease exacerbation. Repeat troponin was negative, and chest X-ray was unchanged.

Transthoracic echocardiogram revealed a left ventricular ejection fraction of 60 to 65%, along with mild left ventricular hypertrophy and no evidence of valvular disease. A bubble study was performed and was normal, indicating no intracardiac shunts.

Two days later, a follow-up CT angiogram of the chest, abdomen, and pelvis demonstrated no significant changes from the prior imaging study – no pulmonary embolism or other pulmonary pathology to explain the hypoxia. It was noted that the right renal artery received blood supply from the false lumen, with the intimal flap extending toward the origin of the inferior mesenteric artery, which remained patent. The flap extended slightly into the proximal right common iliac artery yet did not exceed this point. The patient

Laboratory parameter	Patient's laboratory values	Normal range
Sodium (mmol/l)	142	136-145
Potassium (mmol/l)	3.6	3.5-5.3
Chloride (mmol/l)	109	98-110
CO2 (mmol/l)	25.4	20-31
BUN (mg/dl)	13	6-24
Creatinine (mg/dl)	0.88	0.7-1.3
Glucose (mg/dl)	123	70-140
AST (U/l)	26	10-36
ALT (U/l)	40	10-49
ALP (U/l)	96	46-116
Total protein (g/dl)	7.1	6.4-8.4
Albumin (g/dl)	4.5	3.2-4.8
Total bilirubin (mg/dl)	0.6	0.2-1.2
Calcium (mg/dl)	9.4	8.6-10.4
Magnesium (mg/dl)	2	1.5-2.5
LDL (mg/dl)	149	0-100
Triglycerides (mg/dl)	205	0-150
Lactic acid (mmol/l)	1.91	0-2
GFR (ml/min/1.73 m^2)	>90	>90
WBC (×10^3/µl)	8.1	4.4-11
RBC (×10^3/µl)	4.79	4.32-5.72
Hemoglobin (g/dl)	13.9	12-17.5
Hematocrit (%)	41.9	41-50
MCV (fl)	91.9	81.2-95.
PT (seconds)	11.2	10.6-12.9
INR	0.97	0.91-1.1
PTT (seconds)	30.1	26.9-36.3

Abbreviations: CO_2, carbon dioxide; BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; LDL, low-density lipoprotein; GFR, glomerular filtration rate; WBC, white blood cells; RBC, red blood cells; MCV, mean corpuscular volume; PT, prothrombin time; INR, international normalized ratio; PTT, partial thromboplastin time.

Table 1. Pertinent laboratory values.

Are there previously conclusive reports on this reaction?	Yes (+1)
Did adverse event appear after the suspected drug was given?	Yes (+2)
Did the adverse reaction improve when the drug was discontinued or a specific antagonist was given?	Yes (+1)
Did the adverse reaction appear when the drug was readministered?	Not Known or done
Are there alternative causes that could have caused the reaction?	No (+2)
Did the reaction reappear when a placebo was given?	No (+1)
Was the drug detected in any body fluid in toxic concentrations?	Not known or done
Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	No
Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	No
Was the adverse event confirmed by any objective evidence?	Yes (+1)
TOTAL SCORE	8

Table 2. Naranjo scale[4].

was maintained on the clevidipine and exhibited oxygen saturation levels between 90 and 92% while on BiPAP at night and alternating with high-flow nasal cannula therapy during the day at 60% FiO_2.

Eventually, the patient's blood pressure was effectively controlled with oral antihypertensive medications, including nifedipine, hydralazine, labetalol, hydrochlorothiazide, and lisinopril; subsequently, the clevidipine drip was discontinued. Upon discontinuation, the patient's oxygen saturation improved to 94% on 3 liters nasal cannula within a few hours afterwards and slowly weaned from oxygen support by discharge.

DISCUSSION

In aortic dissection, short acting beta blockers, such as esmolol or labetalol, are the treatment of choice. However, in the event of resistant hypertension or if beta blockers are contraindicated, calcium channel blockers are recommended as an adjunct. Our patient developed bradycardia, and as a result, clevidipine was initiated given that it is a short-acting calcium channel blocker that inhibits L-type calcium channels with high clearance and low distribution. Clevidipine also can be rapidly metabolized into the corresponding inactive acid and is rapidly hydrolyzed into inactive metabolites in arterial blood.

Clevidipine is unique since it causes significant arterial dilation with minimal venodilation which is optimal for our patient. Furthermore, its ability to modify afterload prevents it from affecting other hemodynamic parameters, including left ventricular filling pressure and pulmonary capillary wedge pressure. Thus, clevidipine has been used in cases of pheochromocytoma, being treated with surgical adrenalectomy which developed intra-operative hypertension[2]. Clevidipine has also been used to prevent vasospasms after radial and mammary artery grafts during coronary artery bypass grafting.

With regard to adverse events, In the VELOCITY trial, among patients who were treated with clevidipine, 6.3% developed headache, 4.8% experienced nausea, and 3.2% developed vomiting. A rare adverse effect that has been reported when clevidipine was used with propofol in a 27-year-old male, was hypertriglyceridemia causing pancreatitis[3]. Clevidipine should be used with caution since it may also interact with aripiprazole and methylphenidate.

Lastly, pulmonary shunting has also been associated with clevidipine. With its infusion, clevidipine inhibits the physiologic pulmonary hypoxic vasoconstriction resulting in diversion of blood flow away from well-ventilated lung areas, increasing ventilation-perfusion mismatch exaggerating the hypoxia. Typically, pulmonary shunting is associated with conditions such as pneumonia, atelectasis, pulmonary edema, pulmonary embolism, vascular tumors, emphysema, or acute respiratory distress syndrome.

In our patient, pulmonary and cardiac pathologies were ruled out through extensive imaging and physiologic work up. As oral beta blocker therapy along with esmolol proved sufficient for blood pressure control, clevidipine was discontinued, which resulted in improvement of patient's oxygen requirement. The Naranjo scale score *(Table 2)*, used to determine likelihood of a drug causing an adverse reaction is eight, deeming hypoxia a probable adverse reaction of clevidipine[4].

Another case of clevidipine-induced pulmonary shunting has also been reported when clevidipine was utilized for postoperative management of blood pressure, in a neurosurgical patient developing refractory hypoxemia[5]. The patient required prolonged ventilatory support. After all other work-up was negative, clevidipine was discontinued, after which hypoxemia resolved within 1 hour. In our case, hypoxia resolved 5 hours after stopping clevidipine.

Awareness of this potential complication is essential for optimizing outcomes in high-risk settings, particularly since the literature has not clearly defined the incidence of clevidipine-induced pulmonary shunting.

CONCLUSION

Clevidipine, while a very useful drug for various pathologies in the intensive care and step-down units, is usually benign. However, it may cause intrapulmonary shunting leading to hypoxia. Physician awareness about this potential side effect of clevidipine is required for prompt diagnosis, management and prevention of potentially life-threatening complications. Further studies are needed to properly understand pulmonary shunting resulting from clevidipine.

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