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CASE REPORT

Vasogenic Shock Secondary to an Intentional Overdose of Multiple Antihypertensives in a patient with Hypertrophic Cardiomyopathy

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ABSTRACT

Hypotension in patients with Hypertrophic Cardiomyopathy (HCM) can be difficult to manage as increasing blood pressure through inotropes can worsen the left ventricular outflow tract obstruction. An overdose of multiple antihypertensives can further complicate matters regarding the treatment options. We report a case of a young male with a history of hypertrophic cardiomyopathy who presented to the emergency department with an intentional overdose of multiple antihypertensives. He had persistent hypotension irrespective of fluid resuscitation and inotropic support. High dose insulin euglycemic therapy (HIET) was commenced followed by Intralipid infusion but had no improvement. Withdrawal of adrenaline infusion and switching to vasopressin improved the hemodynamics and patient was discharged eventually with full recovery.

KEYWORDS

Vasogenic shock, Hypertrophic cardiomyopathy, HIET, Antihypertensive overdose.

INTRODUCTION

HCM is one of the most common cause of sudden cardiac death in young individuals. [1] It is a disease characterized by a genetic mutation which results in overexpression of cardiac myocytes, especially in the intraventricular septum. [2] This results in left ventricular outflow obstruction especially during periods of stress such as exercise Around 1 in 500 people have this genetic mutation in the American population however its expression, clinical presentation and outcomes are variables. [3] An annual mortality rate of 6% is reported in the adult population, now due to contemporary management techniques such as medical management of dyspneic episodes, surgical options such as septal myomectomy/myotomy or arrhythmia control by implantable cardioverter defibrillators (ICD) has reduced the mortality up to 0.5% per year. [4] Our patient had a life-threatening overdose of multiple antihypertensive agents with vasogenic shock. This posed a challenge in management, as usual management therapies for such an overdose tend to increase inotropy, and increased inotropy can worsen cardiac output in HCM by causing left ventricular outflow tract obstruction.

CASE SUMMARY

A 36-year-old male with a background of HCM, atrial fibrillation (AF), hypertension and hypertensive nephrosclerosis was brought to the emergency department (ED) with a combined overdose of metoprolol, lercanidipine, prazosin, and hydralazine. He took 60 tablets in total, with suicidal intent, the amount of each tablet taken is unknown. Paramedics noted a heart rate of 80 bpm with a blood pressure of 67/46 mm Hg which responded to 500ml crystalloid bolus (94/75). In ED his venous blood gas was normal, and ECG showed broad complex AF which was like previous ECGs.

On toxicology advice, initial resuscitation included boluses of fluid, sodium bicarbonate 8.4% at a dose of 1ml/kg and 40mls of 10% calcium gluconate followed by an infusion at 20ml/hour, aiming for ionized calcium around 2. High dose insulin euglycemic therapy (HIET) was commenced with insulin rate at 1u/kg/hr.

Blood pressure remained in the low systolic range of the 80's and he gradually became more confused, eventually being intubated for airway protection, receiving ketamine 1mg/kg and suxamethonium 1.5mg/kg. Post-intubation sedation was continued

with midazolam and fentanyl infusions. Noradrenaline infusion was commenced prior to intubation aiming to improve hemodynamics. Despite this and quadrupling the dose of HIET, hemodynamics continued to worsen. Adrenaline was added but it only worsened the cardiac output even further. A relationship between heart rate and blood pressure is demonstrated in Figure 1 in our patient. With the commencement of inotropes, particularly adrenaline, the blood pressure plummeted.

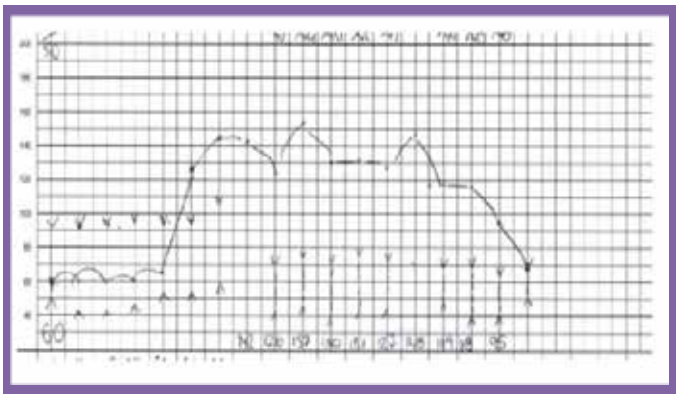

Figure 1: A Patient chart showing the inverse relationship between heart rate and blood pressure

A bedside echo at this stage showed thickened hyperdynamic myocardium and left ventricular outflow tract obstruction. After toxicology consult, an Intralipid bolus of 1ml/kg (10%) and infusion were administered along with methylene blue, all of which failed to improve hemodynamics of the patient.

At this stage, vasopressin infusion was started with adrenaline and noradrenaline gradually tapered. Once the adrenaline was completely stopped, heart rate dropped to around 80 bpm and gradually cardiac output improved, producing a MAP of >65 with good urine output.

An ICU admission followed, with HIET continued for 24 hours and noradrenaline infusion discontinued on day 3. The patient was extubated on day 4 with full recovery. A formal Transesophageal Echocardiogram in ICU showed severe global hypertrophy, preserved function, with features consistent with Fabry's disease.

DISCUSSION

In our literature search, we came across a case report of combined B blocker/calcium channel blocker and amiodarone toxicity in a patient with HCM which responded well to Intralipid. [5] However, to our knowledge, this is the first case report of a life-threatening overdose of multiple antihypertensives including beta blocker, calcium channel blocker, alpha-blocker and centrally acting antihypertensive in a patient with HCM.

In another case report, a patient with HCM had a life-threatening anaphylactic reaction to chlorhexidine during cardiac surgery. [6] The authors had similar difficulty in managing anaphylaxis as epinephrine worsened the hemodynamics of the patient (heart rate went up to 110-120 bpm whereas MAP dropped to 30) confirmed by urgent transesophageal echo (deterioration in LVOT gradient and obstruction) and they had to resort to cardiopulmonary bypass as a last-ditch effort. The report concluded that had the patient not been in a cardiac center, her survival would not have been possible and cautioned that these patients should undergo surgeries at tertiary care as in case of deterioration, management is complicated.

A recent systematic review on the effect of epinephrine in cardiac arrest in patients with HCM revealed that epinephrine could have a different than expected effect on this population as some of the patients in the reported literature had paradoxical or no effect. [7]

In our case, novel therapies such as intralipid and methylene blue did not make any difference. We think earlier commencement of pure a-agonists such as vasopressin or phenylephrine would have been beneficial to increase systemic vascular resistance. Other options could have been extracorporeal membrane oxygenation (ECMO) but our center did not have such a facility. Upon reflection, perhaps a lower blood pressure and MAP would have been acceptable to avoid inotropic agents which were likely counterproductive.

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