

## Refractory Post-Induction Hypotension in Young Hypertension Underwent Humerus Surgery: A Case Report

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Received: 18<sup>th</sup> July 2023

Accepted: 6<sup>th</sup> November 2023

Published: 28<sup>th</sup> February 2024

### Abstract

With changes in lifestyle habits and reduced diagnostic thresholds for hypertension, the prevalence of hypertension among young individuals is expected to rise. We become aware of the adverse effects of pharmacological interventions related to anaesthesia. We present a case in which refractory hypotension emerged after the induction of anaesthesia for orthopaedic humerus surgery. The patient required significantly more pharmacological support over an extended period of the procedure after developing refractory hypotension following an induction phase of anaesthesia that was resistant to fluid resuscitation. We present the case while addressing the current management and potential future management for a foreseeable future strategy.

### Keywords:

Post-Induction Hypotension, General Anaesthesia, Hypertension, Surgery, Case Report

### Introduction

Perioperative management of hypertensive patients undergoing surgical procedures is a well-established practice in modern medicine. However, the occurrence of refractory post-induction hypotension in young individuals with a history of hypertension represents a challenging clinical scenario that warrants closer examination. In anesthesiology practice, hypotension is frequent during induction of general anaesthesia and can affect outcomes.<sup>1</sup> According to a 2019 review, male sex, older age, lower pre-induction systolic arterial pressure, general anaesthesia with propofol, combined general and regional anaesthesia use, emergency surgery, and duration of anaesthesia were predictors of intraoperative hypotension.<sup>2</sup> Additionally, the use of anti-hypertension drugs such as ACE inhibitors, angiotensin II receptor antagonists, alpha-2 agonists, and beta-blockers was linked to hypotension events and cardiovascular morbidity.<sup>2</sup> We present a case of young hypertension that requires extra pharmacological support during operation, since

the hemodynamics did not improve despite fluid resuscitation and boluses of vasopressors. To avoid major complications from intraoperative hypotension, we emphasize early identification and prompt treatment. We discuss about the clinical peculiarities of our patient and compare them with previous reports.

### Case Presentation

We report the case of a 34-year-old Malay gentleman who was 98 kg, had a height of 169 cm, and had a body mass index (BMI) of 34 kg/m<sup>2</sup>. He has been taking tablets of Verapamil 40 mg BD and Prazosin 6 mg TDS for his underlying essential hypertension since 2016. There is no proper follow-up for his antihypertensive with poor compliance. His antihypertensive medications were self-prescribed, as he is working as a doctor. He has also had bronchial asthma since childhood, with the medication of metered-dose inhalers (MDI) Symbicort and Salbutamol. The most recent exacerbation event occurred in August 2022 and was treated with MDI salbutamol. On the other hand, an ultrasound of his hepatobiliary system in 2018 revealed that he had a fatty liver. His prior allergy history was unremarkable, and one month earlier, following a car accident, he underwent open reduction-internal fixation (ORIF) with a Philos Plate of right humerus surgery. At that time, his first operation was uneventful, and there were no reported anaesthesia incidents or any complications that occurred perioperatively.

He was electively admitted for screw fixation with the diagnosis of a closed fracture of the right greater tuberosity of the proximal humerus. Upon arrival in the operating theatre (OT), Glasgow's coma scale was full, pink, and had a good pulse volume. Examination of skin and mucous membranes was good, with capillary refill time (CRT) less than two seconds. No jugular venous distention or oedema in extremities, heart and lung examinations were unremarkable. Vital signs included a blood pressure (BP) of 156/90 mmHg, a pulse rate (PR) of 88, and oxygen saturation (SpO<sub>2</sub>) of 98% under room air. The induction of general anaesthesia was uneventful. He received intravenous (IV) doses of 100 mcg of Fentanyl, 200 mg of Propofol, 80 mg of Rocuronium, and 8 mg of Dexamethasone. We used IV induction agents during induction, and sevoflurane as a volatile agent was used during maintenance. He was intubated using a PVC endotracheal tube, size 8, anchored at 21 cm, and cricoid pressure in one attempt using direct laryngoscopy. Following induction, he had a right interscalene and superior trunk block for the control of intraoperative and postoperative pain. Using a 21G stimuplex block needle of 100mm and a total of 20 ml of chirocaine 0.5%, the block was performed under ultrasound guidance. With good local anaesthetic spread and clear plane visibility, ultrasonography confirmed the block.

However, it was noticed around 15 minutes following induction and already completed interscalene block that the blood pressure was consistently low, between 60 and 90 mmHg systolic and between 30 and 40 mmHg diastolic (Figure 1). His PR was between 60 and 70, and his SpO<sub>2</sub> was between 94 and 96%. His ventilation settings were FiO<sub>2</sub> 0.5, PEEP 8, and RR 14. We excluded several differential diagnoses at that time. No obvious maculopapular rash seen to exclude diagnosis of anaphylaxis. Lungs was clear with good tidal volume and peak pressure ranging from 21 to 23 cmH<sub>2</sub>O to exclude possibility of pneumothorax. It is unlikely to have a fat embolism syndrome or bleeding, as it is not in the acute phase and the operation has not started yet. There is also an unlikely attenuation of systemic stimulus as a result of regional anaesthesia, as the dose is calculated under toxic dose and the landmark was under ultrasound guidance. Vasopressor boluses of various strengths were administered: five times of IV phenylephrine 100 mcg, five times of IV ephedrine 6 mg, and three times of IV adrenaline (1:10000) 1 ml. Inotropes were started after a central venous lumen was placed over the right femoral vein. He began treatment with IVI Noradrenaline at a maximum demand of 0.5 mcg/kg/min and IVI Adrenaline at a maximum requirement of 0.5 mcg/kg/min. An ECG with 12 leads showed sinus rhythm, prolonged QT and no signs of acute ischemia alterations.

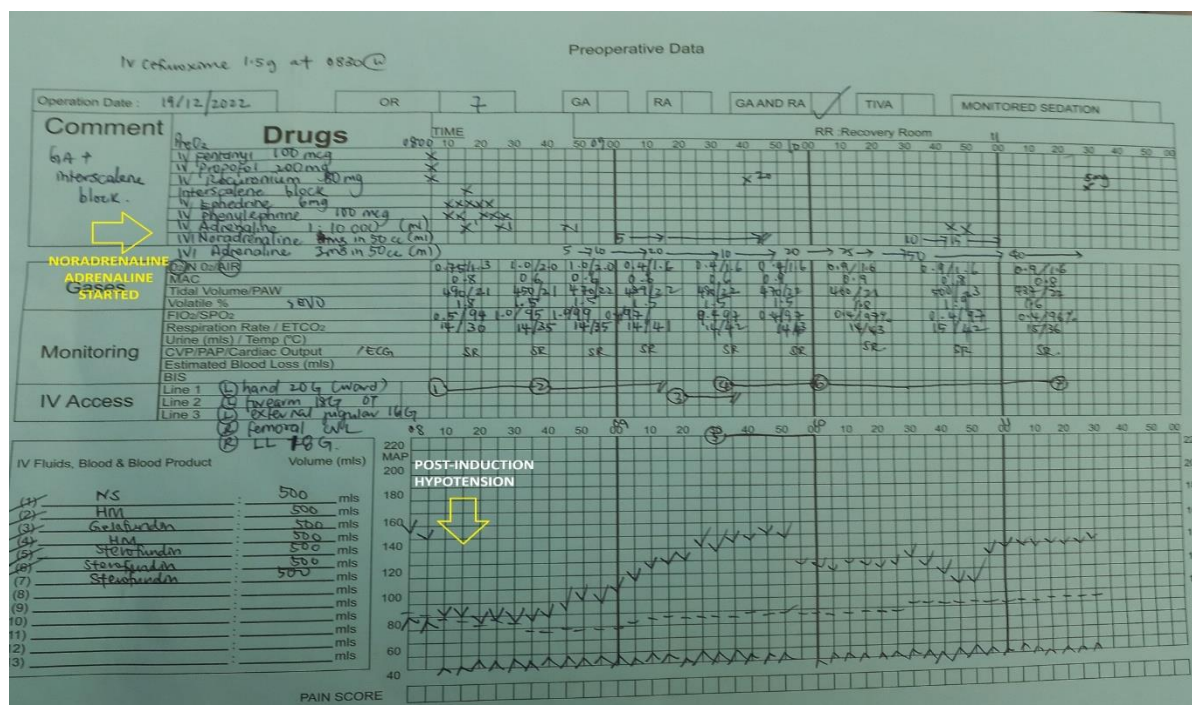


Figure 1: The anaesthetic chart showed a drop in blood pressure following induction of anaesthesia (post-induction hypotension). Blood pressure was maintained with the infusion of noradrenaline and adrenaline with a maximum rate of 0.5 mcg/kg/min intraoperatively

The arterial blood gas showed acceptable oxygenation, but the lactate acidosis was getting worse, with a pH of 7.32 to 7.18 and a lactate range of 1.3 to 7.2. In addition, IV potassium chloride (2 grammes), IV calcium gluconate (10 mmol), IV thiamine (200 mg), and IV NaHCO<sub>3</sub> (8.4%) (20 ml) were administered. Upon further questioning in the ward, the patient was given both tablets of Verapamil and Prazosin before being sent to the operating room. Six pints of crystalloids and one pint of colloids in total were administered, with an estimated 300 ml of blood loss. Resuscitation was done accordingly and was stable enough to proceed with the consultants present in the operation theatre. The ICU team has also been alerted, as operations will proceed with an ICU bed backup postoperatively. The procedure went off uneventfully for three hours. The pharmacological support (IVI noradrenaline and adrenaline) was able to be weaned off subsequently when the operation was done. We postulated that the antihypertension medication effect was wear-off, which explains why the blood pressure normalized at the end of surgery. He was then admitted to the intensive care unit (ICU) for weaning and was successfully extubated two hours after being admitted to the ICU. Our provisional diagnosis was post-induction hypotension secondary to antihypertensive medications. A bedside echo by an ICU consultant revealed good left ventricular (LV) function, no pericardial effusion, good volume and contractility. Bedside lung and abdomen scans showed normal findings with adequate volume status. On the following day, he was transferred to the general ward and subsequently discharged home after four days postoperatively.

## Discussion

The definition of post-induction hypotension (PIH) is the period of time from anaesthesia induction until 20 minutes after induction to ensure detection of arterial hypotension associated with anaesthesia induction without concurrent surgical stimulus, including the early post-induction phase, during which hypotension is most common.<sup>1</sup> An increased risk of postoperative mortality, myocardial injury following non-cardiac surgery (MINS), myocardial infarction, cardiogenic shock, acute renal failure, delirium, and

stroke has been associated with intraoperative hypotension in patients undergoing non-cardiac surgery under general anaesthesia.<sup>2</sup>

On the other hand, in adults between the ages of 20 and 40, hypertension affects 1 in 8 of them, and it is particularly prevalent in young people. To some disagreement, the American College of Cardiology/American Heart Association (ACC/AHA) lowered the diagnostic cut-off for stage 1 hypertension in the 2017 Guidelines from 140/90 to 130/80 mmHg for all age groups.<sup>3</sup> In our case, it should be emphasised that the patient's blood pressure was consistently higher than 130/80 mmHg with medications. He had been diagnosed with hypertension when he was 28 years old. He has been taking verapamil and prazosin for six years. This case highlights an undue sensitivity to the severe cardiac depression caused by a therapeutic oral dose of verapamil and prazosin, as suggested by earlier case reports.<sup>4, 5</sup> The most recent study also found that the independent risk variables for post-induction hypotension were orthopaedic surgery, age greater than 30, American Society of Anesthesiologists (ASA) class II and above, and the induction agent propofol.<sup>6</sup> Therefore, these risks are closely related to our case.

Verapamil is a non-dihydropyridine calcium channel blocker that shares chemical similarities with papaverine, an opium alkaloid.<sup>4</sup> It is used to treat supraventricular dysrhythmias, angina, and hypertension. It blocks voltage-dependent L-gated calcium channels, causing the smooth muscles of the blood vessels to relax and having detrimental inotropic and chronotropic effects on the heart. Additionally, it indirectly reduces the need for oxygen in the myocardium and widens the coronary arteries.<sup>4</sup> In contrast to the prior case report, ours occurred intraoperatively. Regardless of dosage or treatment duration, patients receiving non-dihydropyridine calcium channel blockers (verapamil and diltiazem) who exhibit significant left ventricular failure, cardiogenic shock, or high-grade atrioventricular block should always be taken into consideration. The key to managing such instances and potentially saving lives is early therapy with 10 ml of 10% calcium chloride administered intravenously.<sup>4</sup>

Prazosin is an alpha-1 adrenergic receptor antagonist. Alpha-1 receptors are found on smooth muscle, including the walls of blood vessels, the prostate, urethra, iris dilator muscle, and the brain. By reducing systemic vascular resistance, alpha-1 antagonists can lower blood pressure by relaxing smooth muscle.<sup>7</sup> The blood-brain barrier can be crossed by this medicine. This mechanism permits cognitive impacts, which accounts for its application in post-traumatic stress disorder (PTSD) and nightmares related to PTSD.<sup>7</sup> Previous case reports were inconsistent because of the concurrent prazosin overdose along with acetaminophen and naproxen toxicity. However, there is a scenario where an alpha-1 antagonist needs pharmacological support, which is the similarity.<sup>5</sup> Profound hypotension was seen in our patient, and it was resistant to fluid resuscitation and high-dose epinephrine and norepinephrine infusions. The next step in resuscitation is to begin a vasopressin infusion, as indicated in the prior case report.<sup>5</sup> For patients who have an alpha-1 antagonist, vasopressin is a significant potential therapeutic alternative because it can avoid direct alpha blockage.<sup>5</sup>

For the management of intraoperative and postoperative pain, this patient underwent a right interscalene and superior trunk block. Despite the fact that there is a case report of profound prolonged bradycardia and hypotension following interscalene brachial plexus block, the diagnosis was made by exclusion.<sup>8</sup> This is because the amount administered was below the toxic level and the location was verified using an ultrasound-guided nerve block. Additionally, the period of hypotension began during the induction of general anaesthesia prior to the start of the block procedure. Regarding the preoperative management of antihypertensive drugs, certain drugs were mentioned in the guidelines to be stopped.<sup>9</sup> However, the guideline did not mention stopping alpha blockers like prazosin. On the other hand, the guideline

recommends that when stopping calcium channel blockers, decisions are made on an individual patient basis.

### Conclusion

We recommend the anaesthetist take the patient's age and ASA class into consideration when choosing an induction medication type, as well as whether to optimise orthopaedic patients before surgery. The pharmacology of calcium channel blockers and alpha-1 antagonists should be understood by clinicians because verapamil and prazosin are becoming more widely used. When hypotension is resistant to conventional fluid and catecholamine therapy, clinicians should consider alternative treatments like calcium as well as vasopressin.

The challenges faced in managing refractory PIH in this context necessitate further research into the underlying mechanisms and optimal management strategies. Additionally, preoperative assessment and risk stratification in young hypertensive patients should be more comprehensive, taking into account not only blood pressure control but also potential autonomic dysregulation and individualized response to anaesthesia.

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