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RESEARCH ARTICLE

Acute and Resistant İntraoperative Hypertension during Mitral Valve Replacement: A Case Presentation

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ABSTRACT

Background: Intraoperative hypertension continues to be a significant cause of morbidity and mortality for patients undergoing surgery. In this case presentation, an instance of sudden, unexpected, and resistant hypertension during surgery will be discussed in a patient who underwent mitral valve replacement.

Case report: A 75-year-old patient with known coronary artery disease and advanced mitral stenosis underwent mitral valve replacement surgery. During the intraoperative process, after the completion of cannulation procedures and transitioning to partial bypass, a sudden increase in blood pressure was observed in the patient. Blood pressure was only brought under control through the intravenous administration of a triple antihypertensive agent. Upon postoperative evaluation following a smooth completion of the surgery and discharge, findings suggested that the cause of the hypertension in the patient might be renovascular hypertension.

Conclusions: Intraoperative hypertension, when unexpected and sudden, can lead to an increase in mortality and morbidity. During the intraoperative process, efforts should be made to identify the underlying causes promptly, and appropriate interventions should be carried out. In the postoperative period, patients should be thoroughly evaluated for underlying reasons.

Keywords: Antihypertensive drugs, cardiac surgery, mitral stenosis, renovascular hypertension

Introduction

Hypertension primarily affects vital organs such as the heart, kidneys, and brain, while also posing a risk to all other systems in the body. Globally, an estimated 1.28 billion adults aged 30-79 are hypertensive. Alarmingly, 46% of these individuals are unaware of their condition[1,2]. The underlying cause remains unknown in most hypertensive adults, with only 10% having a specific, modifiable reason for their hypertension[3].

Intraoperative hypertension remains a significant concern for anesthesiologists. Patients with a known history of hypertension undergo necessary precautions during the intraoperative process before the surgery begins. However, managing unexpected increases in blood pressure during surgery poses a significant challenge for anesthesia management. Intraoperative hypertension, which may be unexpected and resistant, can have various underlying causes[4]. Factors related to the patient, as well as issues arising during surgery such as inadequate analgesia, anesthesia-induced increased sympathetic activity, or occurrences like hypoxia or hypercarbia, can contribute to this scenario[4]. Once reversible causes are ruled out, intervention with intravenous antihypertensive drugs is crucial during intraoperative hypertension, followed by necessary investigations into the underlying causes during the postoperative period[5].

This case presentation outlines an instance of sudden, unexpected, and resistant hypertension occurring intraoperatively in a patient with suffering from advanced mitral stenosis, necessitating mitral valve replacement.

Case report

A 75-year-old male with a medical history of coronary artery disease, atrial fibrillation, class I hypertension, and mitral stenosis underwent percutaneous coronary intervention (PCI) following an acute inferior myocardial infarction in 2018. Warfarin therapy was initiated. In March 2022, the patient was admitted to the cardiology department with a suspected warfarin overdose and urinary tract infection. Transthoracic echocardiography(TTE) revealed dilation in the left atrium and right heart chambers, global hypokinesis of the heart, and advanced mitral stenosis. Consultation with the cardiovascular surgery (CVC) unit resulted in the decision to perform mitral valve replacement in December 2022.

The preoperative assessment of the patient revealed the use of verapamil, enoxaparin sodium, furosemide, digoxin, budesonide, ipratropium bromide, and salbutamol. Additionally, it was noted that the patient is an active smoker with no history of allergies. The mildly dyspneic patient had no history of surgeries except for a PCI procedure in 2018. During the physical examination of the patient with normal vital signs, bilateral rhonchi were auscultated in the lungs, there was prolonged expiration, and accentuation of S1 and diastolic murmurs were observed in cardiac auscultation. Abdominal examination revealed normal findings, no pretibial edema was observed, and peripheral pulses were palpable.

The preoperative laboratory values indicated an INR value of 1.81, while other parameters were within normal limits. The electrocardiogram revealed rapid ventricular response in atrial fibrillation, and the chest X-ray was assessed as normal for the patient. (Figure 1)

The patient, who did not fully comply with the respiratory function test, had an FEV1/FVC ratio of 57.5%, and the FEF25-75 value was determined as 45. In the preoperative period, coronary angiography revealed plaque in the left anterior descending artery (LAD) with open stents in diagonal 1-2-3, a proximal open stent and plaque in the mid-region of Cx, and plaque in the proximal and distal parts of right coronary artery (RCA), with an open stent in RCA. Bilateral carotid artery color doppler ultrasonography (USG) indicated increased intima-media thickness in both carotid arteries, a calcified plaque on the right, 50-60% stenosis in the right internal carotid artery, and a calcified plaque on the left. In the preoperative repeat TTE examination, enlargement of the left atrium and left ventricular hypertrophy were noted along with an ejection fraction of 50%. Rheumatic mitral and aortic valves, advanced mitral stenosis, mild mitral regurgitation, mild-moderate aortic regurgitation, pulmonary hypertension, maximum mitral transvalvular gradient of 21 mmHg, mean gradient of 9 mmHg, and a valve area of 1.04 cm2 were reported.

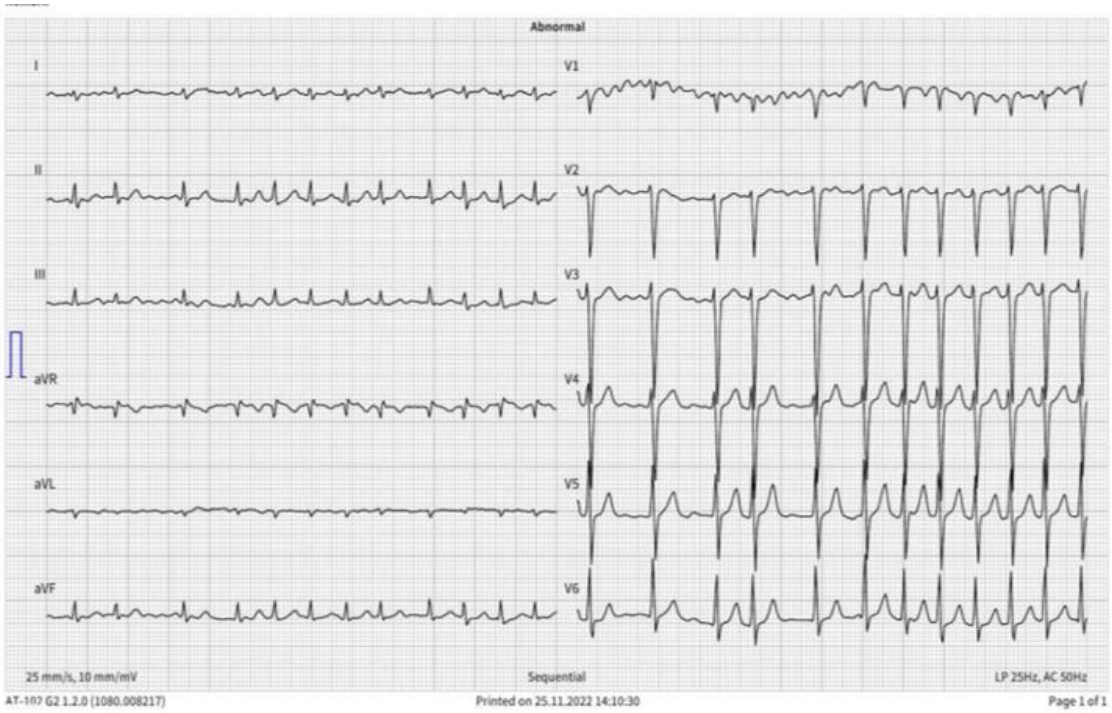

Figure 1. The preoperative electrocardiogram of the patient

The patient underwent necessary preoperative consultations, followed the recommendations, and underwent preoperative anesthetic assessment, leading to planning for surgery with American Society of Anesthesiologists (ASA) III classification. The patient was taken to the operating room, where monitoring was initiated. Prior to induction, the heart rate (HR) was 90/min, blood pressure was 130/85 mmHg, and SpO2 was 94%. Sedoanalgesia was achieved with 2 mg midazolam and 50 µg fentanyl administered as an intravenous bolus. Invasive blood pressure monitoring was established by cannulating the right radial artery. Preoxygenation was performed with 100% oxygen via a mask for 2-3 minutes to ensure deep breathing. Anesthesia induction included controlled intravenous administration of 0.01 mg/kg midazolam, 5 µg/kg fentanyl, and 100 mg propofol. After the loss of spontaneous respiration, the patient, comfortably ventilated with a mask, received 0.6 mg/kg intravenous rocuronium as a neuromuscular blocker. After approximately 3 minutes of mask ventilation, the patient was intubated with an 8.5 mm endotracheal tube. Following tube confirmation, the patient was connected to a mechanical ventilator. Sevoflurane+O2+air mixture and remifentanil infusion were used for anesthesia maintenance. Apart from mild hypotension post-induction, there were no significant changes in the patient's vital signs. After intubation, the HR was 98/min, arterial blood pressure (ABP) was 116/78 mmHg, and SpO2 was 99%. The patient was placed in an appropriate position, and central venous catheterization was performed through the right internal jugular vein. A Foley catheter was inserted to monitor urine output. Before the surgical incision, an additional dose of 10 mg rocuronium, 50 µg fentanyl, and 1 mg midazolam were administered as intravenous bolus. Following the additional dose, the surgery commenced. Mediastinal access was achieved through mid-sternal incision and sternotomy. No significant hemodynamic changes were observed after sternotomy. After pericardial opening and heart suspension following heparinization, arterial and bicaval venous cannulation were performed. After completing the cannulation procedures and initiating partial bypass, a sudden increase in ABP values was noted in the patient. At this point, ABP was measured at 185/105 mmHg, and HR was 100/min. With the continuation of remifentanil infusion at 0.2 mcg/kg/min and ensuring adequate anesthesia depth, intravenous nitroglycerin infusion was initiated at 0.5 µcg/kg/min and increased up to 2 µcg/kg/min as an antihypertensive agent. Upon the blood pressure values dropping to 150/85 mmHg, esmolol infusion was initiated as the second antihypertensive agent with an intravenous bolus of 500 µg/kg and an intravenous infusion at a rate of 50 µg/kg/min. Despite nitroglycerin at 2 µcg/kg/min and esmolol at 300 µcg/kg/min, BP values were 142/79 mmHg, prompting the administration of 25 mg urapidil, a selective alpha-1 adrenergic receptor blocker, as a third antihypertensive agent through slow IV bolus. With BP values at 110/64 mmHg, intravenous urapidil

infusion was initiated at 2 mg/min, and the patient, with BP dropping to 69/45 mmHg, was placed on total bypass to continue the operation. During total bypass, all antihypertensive drugs were gradually reduced and stopped. Subsequently, the patient smoothly came off total bypass without any intraoperative issues. After total bypass, the patient, with low BP values, received infusions of norepinephrine and dopamine. The cross-clamp time during the approximately 4-hour operation was 87 minutes, and the pump time was 124 minutes. The patient was transferred to the ICU with inotropic support (norepinephrine at 0.1 µcg/kg/min and dopamine at 8 µcg/kg/min), showing an HR of 125/min and BP of 105/50 mmHg. The sedated and intubated patient was admitted to the intensive care unit (ICU). The patient, connected to the mechanical ventilator in P-SIMV mode, received dexmedetomidine infusion for postoperative sedoanalgesia. During follow-ups in the ICU, when the first reduced inotropic support was stopped and the patient's blood pressure (BP) began to rise, intravenous nitroglycerin infusion was initiated. With no response, intravenous urapidil infusion was added, effectively controlling the patient's BP values. The patient, extubated at the 5th postoperative hour, received oxygen support via a mask. Within the first 24 hours, the patient experienced 780 cc of bleeding and 2400 cc of urine output. After the 24th hour, intravenous antihypertensive agents were gradually discontinued, and there was no further increase in BP values. With stabilized vital signs, mobilization, and initiation of oral intake, the patient did not develop any additional complications. The patient was transferred to the ward on the 3rd postoperative day, and after uneventful follow-ups, was discharged on the 10th postoperative day.

After stabilizing the patient, investigations were conducted during the postoperative period to identify the causes of secondary hypertension. Examination of blood samples taken from the patient revealed no issues in the complete blood count and biochemical tests. Serum electrolyte values and thyroid function tests (fT4, TSH) were found to be within normal limits.

On abdominal USG, several cortical cysts were identified, with the largest measuring 47 mm in the right kidney and 36 mm in the left kidney. Bilateral renal artery color Doppler USG revealed a 5.5x6 cm calcified cyst at the lower pole of the right kidney, a 2x2 cm septated cyst at the upper pole, and a renal artery resistance index (RI) value of 0.83 in the right kidney. (Renovascular hypertension ?)

Discussion

Hypertension is a significant risk factor for various serious health issues such as coronary artery disease, stroke, heart failure, and kidney failure. The prevalence of hypertension in patients undergoing cardiac surgery can reach up to 80%[6-8]. Additionally, there are less common but challenging forms of resistant and sudden-onset secondary hypertension that can arise[3]. Whether due to primary or secondary causes, the intraoperative anesthesia management of hypertensive patients requires special attention. Failure to manage this condition appropriately can significantly increase the risk of mortality along with myocardial and renal damage during intraoperative hypertension[9,10]. Control of the acute hypertensive episode during cardiac surgery aims to reduce oxygen consumption by minimizing surgical bleeding and myocardial wall tension[8,9,11]. In this patient group, inadequately controlled intraoperative hypertension can lead to a substantial increase in postoperative morbidity and mortality. In our case, we have a patient with a history of rheumatic valve disease causing severe mitral stenosis, along with a history of coronary artery disease, class I hypertension, and atrial fibrillation. The patient experienced an unexpected, sudden, and resistant hypertensive attack during the intraoperative period. This attack could only be brought under control by the combination of three intravenous antihypertensive drugs.

Intraoperative hypertension can stem from various causes. One of the primary considerations and exclusions among these causes is inadequate depth of anesthesia and analgesia. Additionally, factors such as laryngoscopy, endotracheal intubation and extubation procedures, surgical stimuli, hypoxia, hypercarbia, hypervolemia, bladder distension, and increased intracranial pressure can contribute to intraoperative hypertension[12]. Another significant factor is the failure of patients with known hypertension to take their routine antihypertensive medications during the preoperative period. The current guideline recommends that patients continue taking their antihypertensive medications (with a few exceptions, such as ACE inhibitors and ARBs) until the day of the surgery[3,4].

Other rare factors that can lead to intraoperative hypertension are secondary causes of hypertension. These factors can be present in 5-10% of all patients diagnosed with hypertension and may be a cause of intraoperative resistant hypertension[13]. Various reasons, including obstructive sleep apnea syndrome, renovascular diseases, and primary aldosteronism can contribute to secondary

hypertension[3,13]. In our case, the finding of a RI of 0.83 in the bilateral renal artery color Doppler ultrasound performed in the postoperative period raised the possibility that renal artery stenosis could be the cause of intraoperative hypertension in this case. Further investigations for other secondary causes of hypertension in the patient revealed no pathological findings, except for several cortical cysts in the renal parenchyma.

In renovascular hypertension, a common cause of secondary hypertension in adults, the underlying reason is often stenosis in the renal arteries, typically associated with atherosclerosis[13,14]. Stenosis in the renal arteries leads to decreased renal blood flow and activation of the renin-angiotensin-aldosterone system, causing the development of hypertension[13,14]. In our case, considering the timing of the onset of elevated blood pressure (at the start of partial bypass), a sudden decrease in renal blood flow leading to the activation of the renin-angiotensin-aldosterone system due to underlying stenosis may have contributed to the severity of the condition. Additionally, the presence of coronary artery disease in the patient's medical history raised the possibility that the cause of renovascular hypertension could be atherosclerosis. Unfortunately, since the patient was referred to another center for further investigation and treatment, subsequent results and outcomes are not available.

Intraoperative hypertension management begins with identifying the underlying cause and then implementing targeted treatments. Adequate anesthesia depth and analgesia should be ensured with appropriate medications[12]. After reviewing correctable factors contributing to intraoperative hypertension, intravenous treatment is initiated. Short-acting agents are preferred initially. Beta-blockers like esmolol, labetalol, or metoprolol, ultra-short-acting calcium channel blockers like clevidipine, or vasodilators like nicardipine or nitroglycerin can be chosen based on the response to bolus dosing and administered as intravenous infusions. In this case, nitroglycerin was initially used as the antihypertensive agent, followed by esmolol if the response was inadequate, and ultimately, intravenous infusion of urapidil, a selective alpha-1 adrenergic receptor blocker, successfully brought intraoperative hypertension under control.

In conclusion, intraoperative resistant hypertension in a patient undergoing mitral valve replacement, with a medical history of coronary artery disease and atrial fibrillation, was determined to be attributed to renovascular hypertension—a common secondary cause. It is crucial to note that intraoperative hypertension can stem from various factors. Identifying the specific cause and managing blood pressure appropriately are imperative for minimizing morbidity and mortality. Postoperatively, a thorough investigation into the potential causes of secondary hypertension is warranted.

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