



PERIOPERATIVE BETA BLOCKADE-A CASE REPORT

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About the Author: Dr. Sunitha K. Zachariah is working as Assistant Professor in the Department of Anaesthesia at Christian Medical College, Vellore. Her interests are Paediatric and Cardio-thoracic Anaesthesia.



Abstract: Continuation of antihypertensives preoperatively and their influence on intraoperative haemodynamics is a big concern among anaesthesiologists. A young man with a non-functioning left kidney due to a large renal calculus was posted for laparoscopic nephrectomy. He was a recently diagnosed hypertensive on medication. He was administered general anaesthesia for the procedure, but since he developed severe hypotension on induction which required intensive monitoring and vasopressor infusion, the surgery was deferred. He was monitored in the intensive care unit, investigated and brought for surgery later. On altering the antihypertensive medication the second attempt at anaesthesia was successful and uneventful. Here we are reporting the effect beta blockers on anaesthetic drugs.

Key words: Intraoperative, hypotension, anaesthesia, antihypertensives.

The causes for intraoperative hypotension are multifactorial, including preoperative antihypertensive use, hypovolemia, anaesthetic overdose, sepsis, anaphylaxis and myocardial depression. We report a case in which a patient presented with severe hypotension which persisted for ten minutes on induction of general anaesthesia and did not respond to vasopressors.

Case report: A 38 year old gentleman presented with a history of left flank pain and occasional dysuria of four years duration. Intravenous urogram revealed a large calculus in the left kidney which was in a non functioning status. Incidentally he was detected to have high blood pressure in the outpatient clinic. He had no previous history of palpitations, episodic sweating or chest pain. He had no family history of hypertension or diabetes mellitus. His effort tolerance was normal for his age and sex. Electrocardiogram(ECG)



showed left ventricular hypertrophy and renal function tests were normal. Investigation for causes of secondary hypertension revealed a high urinary metanephrine level but subsequent CT imaging of abdomen ruled out an adrenal mass. He was started on Tab. Atenolol 25 mg once daily and Tab. Prazosin 5mg twice daily.

Two weeks later, the patient was posted for laparoscopic nephrectomy. His blood pressure on the eve of surgery was 130/90mmHg. Prazosin was omitted on the day of surgery but atenolol was continued. Preinduction blood pressure was 130/80mmHg and heart rate was 70/min. Patient was loaded with 400ml of normal saline and induced with 5 mg/kg of thiopentone, 2 mcg/kg of fentanyl, vecuronium 0.1 mg/kg, isoflurane 2% and oxygen. Soon after induction, his blood pressure dropped to a lowest of 44/14mmHg with a heart rate of 90 /min. This was treated with colloids, phenylephrine and ephedrine with which the blood pressure improved marginally. Meanwhile his trachea was intubated and an arterial line was established. After several bolus doses of phenylephrine and ephedrine, inj. Noradrenaline (4 mg in 50 ml of NS) was started as an infusion at a rate of 2-4 ml/hour. The blood pressure improved with a combination of fluids, ephedrine, phenylephrine and noradrenaline to 100/65mmHg after around ten minutes. There were no ECG changes during this time. The surgery was postponed and the patient was shifted to ICU. The infusion of noradrenaline was stopped after a 4-5 hours and the patient was extubated after 12 hours with no post anaesthetic neurological sequelae.

Subsequently, the patient was investigated for other causes of this profound hypotension. Cardiac enzymes were normal which ruled out a perioperative ischaemic event. Possibility of anaphylaxis was considered but due to the probable presence of type 1 hypersensitivity in this patient, allergen testing was not done. Nuclear imaging with Meta Iodo Benzyl Guanidine (MIBG) was done which ruled out phaeochromocytoma and echocardiography was done which ruled out a valvular lesion and obstructive cardiomyopathy.

After discharge from the ICU, antihypertensives were discontinued for a week but were restarted with prazosin 5 mg and metoprolol 12.5 mg twice daily. The patient was posted again for open nephroureterectomy. The night before surgery the patient was given diazepam 5 mg. Prazosin was continued but metoprolol was discontinued the night before and the morning of surgery. He was given a premedication of diazepam 7.5mg and metoclopramide 10mg. The patient came with a heart rate of 130 /min and a blood pressure of 128/84mmHg. General anaesthesia was planned. After placing ECG, pulse oximeter, non invasive blood pressure cuff and end tidal carbon dioxide monitoring, he was preoxygenated, induced with midazolam 2mg, propofol 60mg and 4-6% sevoflurane in 60% oxygen. He was paralysed with 30mg of atracurium. The drugs which were used for the previous anaesthetic were deliberately avoided. Blood pressure was monitored every minute during induction which dropped to 85/42 mm of Hg but came upto 124/86 mm of



Hg with intubation. He was placed in the left lateral deflexed position for the surgery and anaesthesia was maintained with O₂, air, 1.5-2.5% of Isoflurane, boluses of atracurium and 0.15 mg/kg of morphine. The blood pressure remained stable at around 90-110 mm of Hg systolic and 50-70 mm of Hg diastolic. Heart rate was stable at 76-86 /min in the beginning, but as it was going upto 110 /min, i.v metoprolol was given in incremental doses upto 5 mg. The surgery lasted for 3 hours with a blood loss of about 300 ml. The wound was infiltrated with 0.5 % bupivacaine and the patient was reversed with neostigmine and glycopyrrolate and was extubated uneventfully.

Discussion: Intraoperative hypotension is defined as a fall in BP of > 20% of the baseline. The reasons for severe intraoperative hypotension considered in this patient were myocardial infarction, hypertrophic cardiomyopathy, valvular heart disease, phaeochromocytoma, hyper-responsiveness to the preoperative antihypertensives and anaphylactic or anaphylactoid reactions to the drugs used.

The cardiac enzymes and echocardiography were normal in the post operative period which ruled out a cardiac cause for the hypotension. Post operative MIBG scan done did not reveal a phaeochromocytoma. The next probable cause was the exaggerated effect of preoperative antihypertensives and the anaesthetic drugs.

Since the patient had severe hypotension which was not responding to vasopressors during the first anaesthetic, it was decided to stop the morning dose of metoprolol when he came the second time even though the patients on beta-blockers are asked to continue since acute withdrawal can produce rebound hypertension and coronary ischaemia¹. To rule out an allergic cause, midazolam and propofol were used instead of thiopentone and atracurium was used instead of vecuronium. Since the patient did not have an acute drop in BP the second time we came to the conclusion that it could have been due to the effect of antihypertensives.

The antihypertensive action of beta-receptor blockers is due to the reduction of cardiac output. Earlier it was a practice to stop beta blockers two weeks prior to surgery², the view which was supported mostly by anecdotal evidence, but the first detailed study of beta blockade in surgical patients proved that it provided hemodynamic stability on laryngoscopy and intubation and a reduction in the incidence of arrhythmia and myocardial ischaemia^{3,4,5}. Studies on bisoprolol started preoperatively showed 100% reduction in the incidence of myocardial infarction and 80% reduction in cardiac deaths⁶.

Based on large trials and meta analysis, beta-blockers were removed as first line drug in the management of hypertension demonstrating that calcium channel blockers, ACE inhibitors and angiotensin receptor antagonists were superior in preventing the risk of stroke^{7,8}.



American College of Cardiology and American Heart Association guidelines 2002 categorized the use of beta blockers in patients coming for vascular surgery with ischaemia detected during preoperative assessment as class I recommendation whereas for the treatment of preoperatively untreated hypertension in patients with known coronary disease as class IIa recommendation⁹.

The Peri-Operative Ischaemia Study Evaluation (POISE) trial showed a significant reduction of myocardial infarction, need for coronary revascularization and the incidence of atrial fibrillation with metoprolol started 2-4 hours prior to surgery but a significant increase in total mortality and clinically significant hypotension and bradycardia were seen¹⁰. POISE trial concludes that it is inappropriate to start beta-blockers immediately before any surgery. In patients where coronary artery disease is identified at the time of admission for an elective surgery, beta-blockers are found to reduce the incidence of myocardial infarction, but increased the total mortality⁹. If beta-blockers are indicated for intercurrent medical conditions, then it should be started well before, rather than at the time of surgery. Acute withdrawal of beta-blockers is associated with a significant increase in the risk of adverse cardiac outcomes¹¹. From all these evidence we can infer that beta-blockers started just before surgery may increase perioperative morbidity and mortality.

References:

1. Peter K. Lindenauer, M.D., Penelope Pekow, Ph.D., Perioperative Beta-Blocker Therapy and Mortality after Major Noncardiac Surgery, *NEJM* (2005)353:349-361.
2. Viljoen JF, Estafanous FG, Kellner GA. Propranolol and cardiac surgery. *J Thorac Cardiovasc Surg* (1972) 64:826–30.
3. Prys-Roberts C, Foëx P, Biro GP, Roberts JG. Studies of anaesthesia in relation to hypertension. V. Adrenergic beta-receptor blockade. *Br J Anaesth* (1973) 45:671–81.
4. Stone JG, Foëx P, Sear JW, et al. Risk of myocardial ischaemia during anaesthesia in treated and untreated hypertensive patients. *Br J Anaesth* (1988) 61:675–9.
5. Wallace A, Layug B, Tateo I, et al. Prophylactic atenolol reduces postoperative myocardial ischemia. McSPI Research Group. *Anesthesiology* (1998) 88:7–17.
6. Prys-Roberts C, Foëx P, Biro GP, Roberts JG. Studies of anaesthesia in relation to hypertension. V. Adrenergic beta-receptor blockade. *Br J Anaesth* (1973) 45:671–81.
7. Opie LH. Beta-blockade should not be among several choices for initial therapy of hypertension. *J Hypertens* (2008) 26:161–3
8. Wiysonge CS, Bradley H, Mayosi BM, et al. Beta-blockers for hypertension. *Cochrane Database Syst Rev* (2007) CD002003
9. Eagle K-A, Berger P-B, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary. *J Am Coll Cardiol* (2002) 39:542–53.



10. POISE Study Group. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. *Lancet* (2008) 371:1839–47.
11. Shammash JB, Trost JC, Gold JM, Berlin JA, Golden MA, Kimmel SE. Perioperative beta-blocker withdrawal and mortality in vascular surgical patients. *Am Heart J* (2001) 141:148–53.