Severe hypotension during general anaesthesia in a patient on Tamsulosin: a case report
Saxena A, Mittal A, Trilokchand, Mittal A

ABSTRACT
Tamsulosin is an α1 selective -adrenergic receptor (α1-AR) antagonist commonly used for benign prostatic hypertrophy (BPH). Its interaction with anaesthetic agents has still not been described. We report the case of 64-year-old man undergoing elective right humerus interlocking. The patient was taking tamsulosin 0.4 mg since two years for BPH. The patient developed persistent hypotension during maintenance phase of anaesthesia (nitrous oxide and 1% halothane). The possible reason for this hypotension could be attributed to interaction between inhalational anaesthetic and tamsulosin.

Keywords: tamsulosin, hypotension, hypertension, humerus interlocking

INTRODUCTION
Tamsulosin is a selective alpha1A- and alpha1D-adrenoceptor antagonist. These alpha1-receptors are predominant in prostate, prostatic capsule, prostatic urethra and bladder. The relaxation of prostate and bladder smooth muscles may result in improvement in maximum urine flow (Qmax) and reduction of lower urinary tract symptoms.1 Currently, first-line agents to treat BPH as prescribed by physicians are α1-adrenergic receptor (α1-AR) antagonists. There are three α1-AR subtypes: α1a, α1b and α1d. Terazosin, doxazosin and alfuzosin are α1-AR antagonists that show equal affinity for all α1-AR subtypes.2 Tamsulosin does not interfere with blood pressure control and has a low potential to cause vasodilation.3 Its recommended dose is 0.4 mg or 0.8 mg with a half-life of 9 to 15 hours and is extensively metabolized by the liver. However its effect in lowering blood pressure on interaction with anesthetic agents has not been established.

CASE REPORT
A 64-year-old, 60-kg patient was planned for elective right humerus interlocking. He was being treated with tamsulosin 0.4 mg once daily at night since last two year for BPH. He had no known history of allergy to any drug. Preoperative pulse rate was 86/min, blood pressure was 130/80 mmHg, respiratory rate was 12/min and temperature was 37°C. Laboratory investigations including serum electrolytes, ECG and X-ray of chest were within normal limits.

Ceftriazone 1 g was administered inside the operation theatre. The patient was premedicated with glycopyrrolate 0.2 mg, midazolam 1 mg and fentanyl 100 μg. Three minutes after preoxygenation, rapid sequence induction was done using thiopental 300 mg and succinylcholine 100 mg intravenously. Endotracheal tube (PVC) of 8.5 mm diameter was inserted. Muscle relaxation was achieved by vecuronium 5 mg. Anesthesia was maintained with N2O, O2 and 1% halothane. Within 15 minutes of induction, the patient's BP dropped down to 80/48 mm Hg. His heart rate increased to 94 bpm; oxygen saturation and end tidal carbon dioxide levels were within normal limits. One liter of Ringer lactate was rapidly infused in spite of which the BP remained at 88/50 mm Hg. Ephedrine 20 mg in divided IV doses only transiently improved the hypotension. He was given intermittent phenylephrine bolus (100 μg) to maintain systolic blood pressure above 100 mm Hg.

Skin erythema, urticaria, bronchospasm, facial edema and other features of potential anaphylaxis or anaphylactoid reaction were absent. No ischemic changes were noted on a three-lead electrocardiogram. General anaesthesia was maintained, with strict monitoring of blood pressure; the operation lasted for one hour with an
estimated blood loss of 100 ml. A total of 600 ìg of phenylephrine was required to maintain the blood pressure during surgery.

After surgery, Halothane was turned off, and within 10 minutes the BP improved to 110/76 mm Hg with a heart rate of 85 per minute. Postoperatively, he remained hemodynamically stable with BP in the range of 110/74 to 126/86 mm Hg and heart rate between 70 and 80 per minute.

**DISCUSSION**

BPH becomes clinically significant in older age group. Alpha blockers (technically α1-adrenergic receptor antagonists) are the most common choice for initial therapy. Commonly prescribed alpha blockers include doxazosin, terazosin, alfuzosin, tamsulosin, and silodosin. All are equally effective but have slightly different side effect profiles. A meta-analysis carried out by Nickel et al., concluded that patients receiving alfuzosin, terazosin and doxazosin showed a statistically significant increased risk of developing vascular-related events compared with placebo. Tamsulosin is highly selective for prostatic receptors with minimal affinity for vascular receptors. Therefore, it should have little effect on blood pressure and should not potentiate other agents that have antihypertensive activity. Tamsulosin showed a numerical increase that was not statistically significant. Tamsulosin drug interactions have been studied in humans. In logistic regression analysis, β-adrenoceptor blockers, converting enzyme inhibitors, antidiabetics and diuretics did not significantly affect the odds ratio for having adverse effects. However, concomitant α-adrenoceptor antagonists and treatment with verapamil (which also has α-adrenoceptor antagonist activity) significantly enhanced the odds ratio for having adverse effects. It is contraindicated in patients with orthostatic hypotension.

This case may illustrate a drug interaction between tamsulosin and inhalational anaesthetic agents. Halothane is the most likely anaesthetic drug to have interacted because the phenylephrine requirement lasted until the end of the case, and the hypotension resolved when halothane was stopped. The hypotension in this patient may have been caused by chronic use of tamsulosin. Halothane is a known vasodilator and may have synergetic effect with tamsulosin. This may also explain why ephedrine was less effective than phenylephrine in controlling hypotension. Since phenylephrine is a pure α1-AR agonist and ephedrine has both central and peripheral effects, in clinical doses, its peripheral effects may be less than those of phenylephrine. Ephedrine was administered initially because the differential diagnosis considered at that time was either excessive depth of anesthesia or hypovolemia. When a synergetic effect was suspected, phenylephrine was used instead and patient responded well. Hypovolemia was ruled out when the BP did not rise after administration of 1 L of crystalloid. The degree of hypotension was exceptional compared with the concentration of anesthetic agent used. The other differential diagnosis for this vasodilated hypotensive state could be anaphylaxis or myocardial ischemia. But since the patient did not have any other features of anaphylaxis and no ischemic changes were found in ECG, these diagnoses were ruled out. Other anesthetic drugs used in this patient were thiopentone, fentanyl and vecuronium. Thiopentone and fentanyl can cause vasodilation but can be controlled, if given gradually it and crystalloid can reverse it easily. The increase in BP when halothane was turned off goes in favour of some kind of synergistic interaction between halothane and tamsulosin.

**CONCLUSION**

This case reports hypotension in a patient on tamsulosin under general anesthesia with N₂O/O₂, vecuronium and halothane anesthesia. Other factors could also have contributed to decreased BP, and tamsulosin cannot be implicated definitively. The purpose of this report is to demonstrate the importance of role of tamsulosin in causing unexpected hypotension. If
hypotension does develop in these patients, we should always use a direct-acting vasopressor such as phenylephrine.

**AUTHOR NOTE**

Arpita Saxena, Contact-09927939991,
E-mail-apoorvsn@yahoo.com
(Corresponding Author)
Department of anesthesia and critical care, B.R.D. Medical College, Gorakhpur.

**REFERENCES**


4. Rubenstein, Jonathan; McVary, Kevin T. (February 6, 2008). "Transurethral Microwave Thermotherapy of the Prostate (TUMT)". eMedicine.


