



## Peri-Operative Steroid Induced Hypertension- A Case Report

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### ABSTRACT

*Hypertension is a common perioperative problem. There are many causes for perioperative hypertension, amongst all, the drug induced hypertension is a relatively rare cause. Here we report a case of hydrocortisone induced hypertension in the postoperative period and its management.*

**Keywords:** Corticosteroids, Perioperative Hypertension, Postoperative Hypertension.

### INTRODUCTION

Hypertension is a common perioperative complication. It can occur during pre-operative period, intra operatively and in the post-operative period. Post-operative hypertension has a variable incidence ranging from 4% to 30%<sup>1</sup>. We report a case of corticosteroid induced hypertension in the immediate postoperative period in a patient, who underwent open reduction and internal fixation (ORIF) for fracture clavicle. All corticosteroids including prednisone can cause sodium retention, resulting in dose-related fluid retention and hypertension. Corticosteroids with strong mineral ocorticoid effects, such as fludrocortisone and hydrocortisone produce the greatest amount of fluid retention<sup>1</sup>.

### CASE REPORT

A 45-year-old male patient was posted for ORIF for fracture medial one third of left clavicle. On pre anaesthetic evaluation, he was found to have

no co-morbid illnesses or drug allergy. On examination, there was no pallor or icterus, BP- 110/70 mmHg, PR- 70/min, RR-14/min. Systemic examination revealed multiple rib fractures on the left side but on auscultation air entry was equal bilaterally with no added sounds. Other systemic examination revealed no abnormality. Airway examination was normal. He was accepted for anaesthesia under American Society for Anaesthesiologists (ASA) grading I. Blood Investigations were within normal limits. Chest x-ray revealed rib fractures involving the 3, 4, 5<sup>th</sup> ribs on the left side with no evidence of underlying lung injury. ECG was normal.

A combined regional (left interscalene block with superficial cervical plexus block) and general anaesthesia was planned. Patient was kept nil by mouth for 8 hours prior to surgery. Informed written consent was obtained after explaining the procedure. Pre medications including tab. Ranitidine 150mg and tab. Alprazolam 0.5 mg PO

were administered before night and 6 am on the day of surgery. Tab Metoclopramide 10 mg PO was administered 6 AM on the day of surgery. In the operating room, baseline monitors were connected (ECG, SPO<sub>2</sub>, Non-invasive blood pressure). An18G IV cannula was inserted on right hand and Ringer Lactate was started. Then he was pre medicated with inj. Midazolam 2mg IV, inj. Fentanyl 50µ IV and oxygen through Hudson mask @ 6L/min. Ultrasound guided left interscalene block was performed with 25 ml of 0.25 % bupivacaine and superficial cervical plexus block was performed with 10 ml of 0.25% bupivacaine. After confirming the effectiveness of blockade at the operating site, Patient was given 50µ IV fentanyl and induced with inj. Propofol 100mg IV and vecuronium 7mg IV. Patient was intubated with 8mm endotracheal tube and tube was secured at 20 cm teeth level after confirming bilateral air entry. He was ventilated with tidal volume of around 500 ml and respiratory rate of 12/min to maintain an ETCO<sub>2</sub> of around 30 mmHg. Anaesthesia was maintained with 0.5 L of O<sub>2</sub> + N<sub>2</sub>O each with isoflurane 1% with intermittent dose of vecuronium 1mg IV. Patient was put in a beach chair position. Duration of surgery was 2 hours. During intra-operative period, BP was within 110 -130 mmHg SBP and 60-80 mm Hg DBP, HR was between 60-80/min. Blood loss was around 150 ml. Intra-operative period was uneventful. Before extubation, inj. Ondansetron was administered for PONV (post-operative nausea and vomiting) prophylaxis. Patient was reversed with myopyrrolate 5ml and extubated in an awake state after thorough oropharyngeal suctioning. Post extubation, on auscultation, there was expiratory wheeze more on the left side, however there was no desaturation. So, patient was positioned propped up, oxygen was supplemented by Hudson mask @ 6L/min and inj. Hydrocortisone 100 mg was administered IV. The patient was observed in OT for 10 mins. The wheeze was found to have reduced and SPO<sub>2</sub> maintained 98% in room air and respiratory rate

around 12 – 14, hence he was shifted to post anaesthesia care unit (PACU).

After 20 minutes of shifting to PACU, patient developed acute hypertension with a BP of 190/120 mmHg which persisted for 20 minutes but heart rate was maintained within 70-80/min, SPO<sub>2</sub> was 98 -100%, RR was 12-14/min at room air, ECG was normal with sinus rhythm and there was no pain at the surgical site. Hypertension was treated with inj. Furosemide 20 mg IV. In the following half an hour, blood pressure came down slowly to SBP 130 mmHg and DBP 90 mmHg. Patient was monitored in PACU for next 24 hours which was uneventful. Post-operative analgesia was maintained by inj. Tramadol 100 mg IV 8<sup>th</sup> hourly and inj. Paracetamol 1g IV SOS. On 2<sup>nd</sup> post-operative day, patient was shifted to ward.

## DISCUSSION

Perioperative hypertension is one of the common complications of anaesthesia and is most common with GA. This can occur during preoperative period due to anxiety<sup>1,4</sup>, during intra-operative period due to intubation response, due to pain induced by surgical stimulation and during extubation due to sympathetic response<sup>1,4</sup>. In the post anaesthesia period, hypertension can be associated with pain-induced sympathetic stimulation, hypothermia, hypoxia, hypercarbia, bladder distension and intravascular volume overload from excessive intraoperative intravenous fluid therapy<sup>3</sup>. Blood pressure can also rise due to discontinuation of blood pressure medications postoperatively.

Postoperative hypertension has been defined as SBP of above 190 mmHg and/or DBP of 100 mmHg on two consecutive readings<sup>1</sup>. These acute episodes occur in the first 20 minutes of the postoperative period, although its resolution can require up to 3 hours<sup>3-5</sup>. If left untreated, postoperative hypertension increases the risk of myocardial ischaemia, myocardial infarction, cerebro vascular accidents, and bleeding<sup>6</sup>. It has a variable incidence ranging from 4% to 30%<sup>2</sup>.

In our case, VAS (visual analog score) was 0/10, patient was awake, maintained saturation of 98-100% in room air and RR was 12-14/min, bladder distension was ruled out by emptying the bladder. Hence, we reached to a conclusion that post-operative hypertension was due to the single dose of inj. hydrocortisone.

Retrospectively, the literature of glucocorticoid induced hypertension was reviewed and some of the possible mechanisms and treatment of steroid induced hypertension were understood. In 1949, the glucocorticoid cortisone was developed, giving physicians the ability to effectively treat adrenal insufficiency<sup>7</sup>. Corticosteroids with strong mineral ocorticoid effects, such as fludrocortisone and hydrocortisone, produce the greatest amount of fluid retention<sup>8</sup>. Corticosteroid-induced fluid retention can be severe enough to cause hypertension. This mechanism revealed that hypertension secondary to glucocorticoid was due to its action over the mineralocorticoid receptor preferably than the glucocorticoid receptor<sup>8</sup>.

Goodwin et al, studied the glucocorticoid activity over the glucocorticoid receptor in vivo in the mice. GC induce number of effects on vascular smooth muscle (VSM) in vitro that may be pertinent to hypertension<sup>9</sup>. The glucocorticoid receptor is widely expressed in many organ systems relevant to blood pressure regulation, including the kidney, the brain and the vasculature<sup>10</sup>.

Saruta et al, described that multiple factors were involved in glucocorticoid-induced hypertension in humans and animals: 1) activation of the renin-angiotensin (R-A) system due to an increase in plasma renin substrate (PRS); 2) reduced activity of depressor systems, including the kallikrein-kinin (K-K) system, prostaglandins (PGs), and the endothelium-derived relaxing factor nitric oxide (NO); and 3) increased pressor responses to angiotensin II (Ang II) and norepinephrine<sup>8</sup>.

They have also found that the glucocorticoid receptor antagonist RU38486 prevented and improved glucocorticoid receptor agonist RU26988-induced hypertension without altering

body weight, urinary water and sodium excretion<sup>12</sup>.

Corticosteroid-induced hypertension responds better to diuretic therapy as compared to other anti-hypertensives<sup>13</sup>. All these evidences have been found to be related with the chronic administration of steroids whereas, our patient developed hypertension acutely after a single dose of hydrocortisone.

Spironolactone being an aldosterone antagonist, acts to decrease the mineral ocorticoid activity and reduces the hypertension<sup>13</sup>.

Furosemide, a loop diuretic acts at proximal and distal tubules to prevent the resorption of sodium and chloride and can be given intravenously when the patient is kept nil oral, thus reducing the hypertension, like in our case scenario.

## CONCLUSION

Perioperative hypertension is common. Hence vigilant monitoring in the postoperative period also is mandatory. Before instituting pharmacological therapy, the routine causes of postoperative hypertension should be ruled out. Although rare, steroid if used in the perioperative period, can induce hypertension and can be managed with diuretics.

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