

# Peri-operative management of Conn's syndrome - a case report

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## Abstract

We present the perioperative management of a patient with Conn's Syndrome posted for laparoscopic left adrenalectomy. Primary hyperaldosteronism (Conn's Syndrome) is characterized by autonomous excessive aldosterone secretion leading to sodium retention and a fall in serum potassium. Conn's Syndrome is a surgically curable cause of secondary hypertension. Most anesthetic problems are related to hypertension and hypokalemia and high chances of perioperative bradycardia, hypotension, hypokalemia and metabolic alkalosis. The manipulation of adrenal gland during dissection and resection may lead to catecholamine release from the adrenal medulla with resultant hemodynamic fluctuations. Anesthesia for Conn's syndrome can be safely administered with the Combined Epidural General anesthesia technique with adequate preoperative control of blood pressure and normalization of electrolytes and strict perioperative monitoring.

## INTRODUCTION

Primary hyperaldosteronism (Conn's Syndrome) is characterized by autonomous excessive aldosterone secretion leading to sodium retention and a fall in serum potassium. It can be caused by adrenal tumours or less commonly from an enlarged gland due to hyperplasia. It occurs more often in females than in males (2.5:1 ratio), usually occurs between 30 to 50 years of age and rarely in children<sup>1</sup>. Occasionally, Primary hyperaldosteronism is associated with pheochromocytoma, primary hyperparathyroidism, or acromegaly<sup>2</sup>. Conn's Syndrome is a surgically curable cause of secondary hypertension. The prevalence of primary hyperaldosteronism in patients with hypertension is 0.5-1%<sup>3</sup>.

Here we present the perioperative management of a patient with Conn's Syndrome posted for laparoscopic left adrenalectomy.

## CASE HISTORY

A 29 year old female was posted for laparoscopic excision of left adrenal tumor. She gave a history of pain and cramps in both upper and lower limbs since 8 months, associated with giddiness and breathlessness on exertion. She was admitted in an ICU for 7 days and was diagnosed to be hypokalemic. She was treated conservatively with potassium supplementation and recovered fully. She was a known

hypertensive since 4 years on Tab Atenolol 25mg OD. Tab Enalapril 2.5mg OD and Tab Amlodipin 5mg OD were added for blood pressure control at the time of ICU admission. Patient was then referred to our tertiary care hospital for further evaluation.

CT-Abdomen showed a lesion 1.8 cm x 1.7 cm in size in the left adrenal gland. Serum aldosterone was 35 ng/dl (normal 1-16 ng/dl supine; 4-31 ng/dl standing) and serum aldosterone-renin ratio was elevated. The plasma aldosterone levels remained high in response to a saline load test. Urinary electrolytes were sodium 73 mmol/L and potassium 45 mmol/L. A diagnosis of primary hyperaldosteronism was made and Tab Spironolactone 100mg OD added to the anti-hypertensive regimen.

At the time of pre-operative evaluation, the patient was symptom free. She weighed 65 kg; her pulse was 84/miunte and blood pressure 146/84mmHg. Chest X-ray and ECG were normal. 2-D Echo showed degenerative aortic valve disease with normal left ventricular ejection fraction. Routine biochemical investigations were within normal limits. She had a normal Renal CT-angio, a normal renal scan and GFR in both kidneys was normal. Endocrinology consultation was done for perioperative management issues.

The patient was premedicated with Tab. Diazepam 5mg the night before surgery. Morning dose of Tab Enalapril 2.5mg

and Tab Spironolactone 100mg was given at 6 am. On arrival into the operation theatre, pulse oximeter, cardioscope, and a non-invasive blood pressure monitor was attached. An intravenous line was then secured with a 20G vein flow in the left arm. Her blood sugars were 180-216 mg/dl at that time. A 16G lumbar epidural catheter was then inserted.

The patient was premedicated with IV Glycopyrrolate 0.2mg, IV Midazolam 1mg and IV Fentanyl 150µg. General anaesthesia was induced with IV Propofol 150mg and IV Vecuronium 6mg and the patient intubated. Right Internal jugular vein and right radial artery were cannulated. Peripheral Nerve stimulator was used for monitoring the neuromuscular blockade.

The patient was then positioned in the Right lateral position with a "bend" for easy approach to the adrenals. General anaesthesia was maintained on 50% nitrous in oxygen, isoflurane (0.6-1.2%), IV Vecuronium along with intermittent positive pressure ventilation to maintain normocarbia. Blood pressure fell to 74/60 mmHg post induction and she was given IV hydrocortisone 100 mg. Epidural bolus of 4ml of 0.125% Bupivacaine was given before insufflation, after the blood pressure was stabilized, following which epidural infusion of 0.125% bupivacaine was started @ 4ml/hr. Post insufflation ( transperitoneal) haemodynamics were stable.

The duration of the surgery was 210 minutes during which the patient was given 2L of crystalloids and 500 ml colloid (Voluven). The heart rate ranged between 90-100/minute and blood pressure stabilized to 90-110 mmHg systolic. Central venous pressure at induction was 1-2 cm of H<sub>2</sub>O which increased to 6-8 cm of H<sub>2</sub>O with intravenous fluids. Intraoperative urine output measured 400ml. Serial ABGs showed no major acid-base imbalance. Serum sodium and potassium levels were within normal range intraoperatively.

Her blood sugars towards the end of procedure were 270-320mg/dl. She was reversed and extubated after return of adequate power as confirmed by peripheral nerve stimulator. The patient was then shifted to post-surgical ICU where intensive hemodynamic monitoring was continued along with blood sugar and serial potassium levels. She received Tramadol for analgesia through her epidural catheter. Her blood sugar and potassium levels were normal in serial measurements.

Her postoperative stay was uneventful. On the 3<sup>rd</sup> day and

the patient was shifted back to the ward for further management. She did not need any anti-hypertensive after surgery.

## DISCUSSION

The diagnosis of hyperaldosteronism should be suspected in non-edematous hypertensive patients presenting with spontaneous hypokalemia without taking potassium wasting diuretics<sup>4</sup>. Hypersecretion of aldosterone increases the distal tubular reabsorption of sodium for secreted potassium and hydrogen, leading to progressive hypokalemia and alkalosis. Rarely, associated nephropathy and azotemia may lead to facial puffiness and periorbital edema. Hypokalemia may present with tiredness, muscle weakness, paralysis, headaches, vomiting and decreased urinary concentrating ability leading to polyuria and nocturia and is accompanied with polydipsia.

Long standing hypertension may lead to cardiological and neurological complications with associated symptoms. Hence, these patients are prone to vascular complications like stroke, angina pectoris, myocardial ischemia, claudication and aortic dissection and these should be evaluated and watched for in the perioperative period.

Conn's syndrome is diagnosed in presence of (a) diastolic hypertension without edema, (b) low plasma renin activity that fails to rise appropriately in response to volume depletion and (c) high plasma aldosterone that does not suppress in response to volume expansion. Whereas, plasma renin activity may be low in some patients with essential hypertension too, the ratio of serum aldosterone to renin activity is a useful screening test. High urinary potassium levels in presence of low serum potassium levels also indicate towards Conn's syndrome.

Most anesthetic problems are related to hypertension and hypokalemia. The patient may be on several drugs with the potential for interaction between the drugs themselves and with the anesthetic agents used. This might lead to perioperative bradycardia and hypotension. Hypokalemia and metabolic alkalosis can theoretically prolong the action of neuromuscular blocking agents. The manipulation of adrenal gland during dissection and resection may lead to catecholamine release from the adrenal medulla with resultant hemodynamic fluctuations<sup>5</sup>.

Fortunately, our patient had none of the associated cardiovascular or renal complications apart from hypertension upon evaluation. Her blood pressure was

controlled and she had normal serum electrolytes before surgery. Intraoperative hemodynamics have been found to be better if the blood pressure has been controlled preoperatively with the aldosterone antagonist, Spironolactone <sub>6</sub>. Careful hemodynamic monitoring was required intraoperatively and was done in the form of invasive blood pressure, CVP, cardioscope and urine output measurements. Chronic hypokalemia has an antagonist action upon insulin secretion and release resulting in abnormal glucose tolerance which may manifest with the stress response to surgery and the perioperative blood sugars were raised in our patient.

IV Fentanyl was given to prevent the intubation response. The fall in blood pressure upon induction might have been an effect of inducing dose of Propofol in presence of low CVP and responded to adequate fluid therapy. IV steroid was given during the episode to stabilize the blood pressure. Isoflurane, as used in this case, has proven to be a good anaesthetic agent for induced hypotension and for maintaining normal hemodynamic parameters in the presence of stress due to surgery, positioning and carbon dioxide pneumo-insufflation <sub>7</sub>. Moreover, studies have shown that "that relevant intraoperative increases in blood pressure occur in patients with Conn's syndrome despite prior therapy with an aldosterone antagonist, necessitating specific precautionary measures during anesthesia." <sub>8</sub> Intraoperative blood pressure has been found to be significantly higher for the retroperitoneal than for the transperitoneal procedure and the transperitoneal approach was used in our patient <sub>8</sub>. Also, combined epidural anaesthesia with general anaesthesia provides more stable intra operative hemodynamics <sub>9</sub>. Carbon-dioxide pneumo-insufflation leads to adrenergic activation due to stress response and might have lead to further hemodynamic variations. We decided upon epidural anesthesia as an easy and safe aid to better intraoperative hemodynamic control and excellent postoperative analgesia.

A negative balance of potassium and sodium occurs in the first postoperative week. Even patients undergoing unilateral adrenalectomy may have temporary hypoadrenalism and may require replacement mineralocorticoid therapy <sub>10</sub>. The surgical cure rate for primary hyperaldosteronism is high although it may take more than a year for the hypertension to

resolve. The best response appears to be associated with: presence of an adenoma, with or without hyperplasia (best prognostic value), age less than 5 years and a positive response to pre-operative spironolactone <sub>5</sub>. Laparoscopic adrenalectomy is feasible and safe and has been performed as an outpatient procedure when the necessary surgical experience and optimal anesthesia were both available <sub>11</sub>.

In conclusion it can be said that anesthesia for Conn's syndrome can be safely administered with the Combined Epidural - General anesthesia technique with adequate preoperative control of blood pressure and normalization of electrolytes and strict perioperative monitoring.

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