

CASE REPORT

Perioperative management of Conn's syndrome - a case report

Latha YS, MD*, Nidhi Bhatia, MD, DNB**, Suman Arora, MD***

Senior Resident; **Assistant Professor; *Additional Professor,
Department of Anesthesiology, Postgraduate Institute of Medical Education and Research, Chandigarh (India)*

Correspondence: Dr. Suman Arora, MD (Anesthesiology), Additional Professor, Department of Anesthesiology, Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh (India); E-mail: sumanarora@hotmail.com

ABSTRACT

Conn's syndrome, also known as primary hyperaldosteronism, is a disease of the adrenal glands characterized by autonomous and excessive aldosterone production, leading to sodium retention and a fall in serum potassium. It may be associated with long standing hypertension, and cardiac and neurological complications. A 51 year old, hypertensive, male patient presented with generalised muscle weakness and hypokalemia. The patient was diagnosed to have benign adrenal adenoma with Conn's syndrome and was scheduled for laparoscopic adrenalectomy. We used epidural analgesia followed by induction of general anesthesia. Intraoperative course was uneventful except for one episode of hypotension.

Unilateral or bilateral adrenalectomy may be performed to treat Conn's syndrome depending on the pathology. Replacement corticosteroid and mineralocorticoid therapy is required for all patients undergoing bilateral adrenalectomy and occasionally in those undergoing unilateral adrenalectomy. Following surgery, the cure rate for hyperaldosteronism may be as high as 60-77%, though it may take a year or more for hypertension to resolve.

Key words: Conn's syndrome; Anesthesia; perioperative management

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INTRODUCTION

Conn's syndrome, also known as primary hyperaldosteronism, is a disease of the adrenal glands characterized by autonomous and excessive aldosterone production, leading to sodium retention and a fall in serum potassium.¹ Conn's syndrome occurs more often in females (F:M=2.5:1) between 30 to 50 years of age and rarely in children.¹ Occasionally, it may be associated with pheochromocytoma, primary hyperparathyroidism, or acromegaly.² The prevalence of primary hyperaldosteronism in patients with hypertension is 0.5-1%.^{3,4} Conn's syndrome is important because it is a potentially curable cause of hypertension. The morbidity and mortality associated with primary hyperaldosteronism is primarily related to hypokalemia and hypertension.⁵ Hypokalemia, especially if severe, causes cardiac arrhythmias, which can be fatal.⁶ Long standing hypertension may lead to cardiac and neurologic complications with associated symptoms, making these patients highly prone to develop vascular complications like stroke, angina pectoris, myocardial ischemia, claudication and aortic dissection.^{6,7,8}

CASE REPORT

A 51 year old male patient, weighing 70 kg, presented with complaints of generalised muscle weakness for the last six months, which was insidious in onset and non-progressive in nature. On further evaluation, he was found to have low potassium levels for which oral potassium supplements were started. He was a known hypertensive for the past four months, which was controlled on a twice daily dose of tab. amlodipine besylate 10 mg and cap. prazosin 5 mg. His nerve conduction studies were normal and CT abdomen showed a smoothly circumscribed, oval nodular homogenous enhancing lesion, measuring approximately 12.3×9.9 mm within the right adrenal gland suggesting benign adrenal adenoma. The patient was scheduled for laparoscopic right adrenalectomy.

On pre-operative evaluation patient was well built with a pulse rate of 78 beats/min, and blood pressure of 130/80 mmHg. Systemic and airway examination were unremarkable, hemoglobin, coagulation profile, liver function, kidney function tests, chest x-ray, ECG and 2D Echo were normal. His serum sodium levels were 137

meq/l, serum potassium was 4.4 meq/l on potassium supplements and serum aldosterone was 1173 pg/ml (normal supine: 10-105 pg/ml, upright: 34-273 pg/ml), with elevated serum aldosterone–renin ratio. The patient had a positive saline suppression test supporting a diagnosis of primary hyperaldosteronism.

The patient was premedicated with tab. alprazolam 0.25 mg and tab. ranitidine 150 mg a night before and on the morning of surgery. Antihypertensive agents were continued on the day of surgery. In the operating room, he was monitored for heart rate (HR), non-invasive blood pressure (NIBP), electrocardiogram (ECG), oxygen saturation (SpO₂), end-tidal carbon dioxide (EtCO₂) and temperature using a multichannel monitors (Datex-Ohmeda S/5 Avance). An 18G peripheral line was secured in left upper limb and injection hydrocortisone 100 mg was given intravenously (IV). His blood sugar was checked, which was 140 mg/dl. Under local anesthesia, right radial artery was cannulated for continuous, invasive blood pressure monitoring. An epidural catheter was placed at L1-L2 level, patient was given intravenous injection fentanyl 150 µg and induction took place with slow titrating dose of propofol. Neuromuscular monitoring was initiated and injection vecuronium bromide 0.1 mg/kg IV was used to facilitate tracheal intubation. Prior to intubation injection esmolol 0.5 mg/kg was given to prevent intubation response and the airway was secured with 8.5 mm endotracheal tube when the train of four (TOF) count was zero. Anesthesia was maintained with isoflurane and top-up injections of vecuronium. Another 16G IV cannula was secured in the left arm and patient was then placed in the left lateral position. Before incision, epidural block was initiated with 6 ml of 0.25% plain bupivacaine. While creating pneumoperitoneum, blood pressure fell to 65/30 mmHg which was managed with mephentermine boluses and crystalloid infusion. Rest of the intraoperative course was uneventful, with no changes occurring in the vital parameters even during handling of adrenal mass. Total surgical duration was 150 min, with a total blood loss was about 500 ml, which was replaced with Ringer lactate and colloid solutions. At the conclusion of surgery, patient was extubated after reversal of neuromuscular blockade with neostigmine and glycopyrolate. Patient's postoperative course was uneventful and he was discharged after 5 days. His further follow up revealed no alteration in potassium levels or features suggestive of hypo or hyperaldosteronism.

DISCUSSION

Conn's syndrome is an aldosterone producing adenoma, named after Jerome W. Conn (1907–1994), an American endocrinologist, who first described the condition at the University of Michigan in 1955.⁹ A benign adenoma in one of the adrenal glands produces and

secretes an excessive level of aldosterone. This promotes sodium reabsorption at the renal tubules, which causes water to follow causing an increased total blood volume, hence the resultant hypertension. As sodium is reabsorbed, potassium and hydrogen are pumped out, accounting for the resulting hypokalemia and alkalosis.⁹ These patients often present with refractory hypertension with spontaneous hypokalemia (serum potassium < 3.5 mEq/L) and difficulty in maintaining normal serum potassium levels despite the use of oral potassium supplements. Our patient, who was a known hypertensive, presented with generalised muscle weakness and hypokalemia.

In patients with Conn's syndrome, most of the anesthetic problems are related to either potassium depletion or hypertension.¹⁰ Therefore, the main goals of preoperative preparation are regulation of blood pressure and obtaining normal potassium levels. Hypokalemia and metabolic alkalosis should be corrected preoperatively. Hypokalemia prolongs the action of non-depolarizing neuromuscular blocking agents. Low serum potassium is also known to suppress baroreceptor tone, so hypokalemia should be treated aggressively.¹⁰ Regulation of blood pressure requires combination of several drugs, with their doses depending on the arterial blood pressure values as well as comorbid conditions and complications. The first choice therapeutic agent is a potassium sparing diuretic, spironolactone, given at a dose of 100-400 mg/day.¹¹ Alternative to this agent is amiloride at a dose of 5-15 mg/day or nifedipine (30-90 mg/day). ACE inhibitors (captopril) and inhibitors of the angiotensin receptors (losartan-Na) can also be used.¹²

Unilateral or bilateral adrenalectomy may be performed to treat Conn's syndrome depending on the pathology. Manipulation of the adrenal gland during tumour removal may cause cardiovascular instability due to the secretion of catecholamine's, although this is not as severe as with pheochromocytoma.⁸ Replacement corticosteroid and mineralocorticoid therapy is required for all patients undergoing bilateral adrenalectomy and occasionally in those undergoing unilateral adrenalectomy to prevent the development of acute adrenal insufficiency.¹³ Hydrocortisone is given intravenously from the time of operation until oral medications can be administered, whereupon oral fludrocortisone and hydrocortisone are given.⁸ Our patient had an uneventful intraoperative course, except for a fall in blood pressure at the time of creation of pneumoperitoneum. This fall might have been due to a probable low central venous pressure, and hence responded to adequate fluid therapy. Isoflurane, as used in this case, has proven to be a good anesthetic agent for maintaining normal hemodynamic parameters in the presence of stress due to surgery, positioning and carbon dioxide pneumo-insufflation.¹⁴ Intraoperatively, Ringer lactate was used as maintenance and replacement fluid as it

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contains potassium and low sodium.

Following surgery, the cure rate for hyperaldosteronism may be as high as 60-77%, though it may take a year or more for hypertension to resolve.⁸ The best response to surgical treatment appears to be associated with the presence of adenoma, age younger than 44 years, duration of hypertension less than 5 years and positive pre-operative

response to spironolactone.⁸

To conclude, patients with Conn's syndrome pose a challenge for the anesthesiologist due to the presence of refractory hypertension and electrolyte disturbances. Aggressive perioperative hemodynamic monitoring along with correction of electrolyte imbalance are keys to its successful management.

REFERENCES

1. Williams GH, Dluhy RG. Disorders of the adrenal cortex. In: Fauci AS, Braunwald E, Isselbacher KJ, editors. Harrison's Principles of Internal Medicine. 17th edition. Chapter 336, page 2259-60. New York, NY, USA: McGraw-Hill Professional Publishing.
2. Stoelting RK, Stephen F, Dierdorf. Endocrine Diseases In: Stoelting Anesthesia and Co-Existing Disease, Fourth Edition, Chapter 22, page 429. Elsevier Churchill Livingstone Publishing.
3. Michael FR, Fleisher LA. Anesthetic Implications of Concurrent Diseases. In: Miller's Anesthesia, Sixth Edition, Chapter 27, page 1038. Elsevier Churchill Livingstone Publishing.
4. Mattsson C, Young WF Jr. Primary aldosteronism: diagnostic and treatment strategies. *Nat Clin Pract Nephrol* 2006;2:198-208.
5. Wheeler MH and Harris DA. Diagnosis and management of primary aldosteronism. *World J Surg* 2003;27(6):627-31.
6. Born-Frontsberg E, Reincke M, Rump LC, Hahner S, Diederich S, Lorenz R, et al. Cardiovascular and cerebrovascular comorbidities of hypokalemic and normokalemic primary aldosteronism: results of the German Conn's Registry. *J Clin Endocrinol Metab.* 2009;94:1125-30.
7. Foex P, Sear JW. Hypertension: pathophysiology and treatment. *Contin Educ Anaesth Crit Care Pain* 2004;4:71-75.
8. Winship KM, Winstanley JHR, Hunter JM. Anaesthesia for Conn's Syndrome. *Anaesthesia* 1999;54:564-574.
9. Conn JW, Louis LH. Primary aldosteronism: a new clinical entity. *Ann Intern Med* 1956;44:1-15.
10. Harde M, Gyanesh P, Tiwatane S, Dave N, Iyer H. Peri-operative management of Conn's syndrome - a case report. *The Internet Journal of Anesthesiology.* 2008;19:2.
11. Harde M, Gyanesh P, Tiwatane S, Dave N, Iyer H. Peri-operative management of Conn's syndrome - a case report. *The Internet Journal of Anesthesiology.* 2008 Volume 19 Number 2.
12. Gockel I, Heintz A, Kentner R, Werner C, Junginger T. Changing pattern of the intraoperative blood pressure during endoscopic adrenalectomy in patients with Conn's syndrome. *Surg Endosc* 2005;19:1491-97.
13. Shipton EA, Hugo JM. Primary aldosteronism and its importance to the anaesthetist. *S Afr Med J* 1982; 62:60-3.
14. De Grood PM, Harbers JB, Van Egmond J, Cruil JF. Anaesthesia for laparoscopy: A comparison of five techniques including propofol, etomidate, thiopentone and isoflurane. *Anaesthesia* 1987;42:815-23.

