

Anaesthetic Management of Adrenal Tumour Resection



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ABSTRACT

The adrenal glands are secretory organs that are located above each kidney and produce hormones that play a major role in metabolic processes. Tumours of the adrenal gland can cause metabolic abnormalities associated with hormonal disorders such as Cushing's syndrome, pheochromocytoma, or Conn's syndrome. To be able to provide good

anaesthetic management on resection of the adrenal tumour, it requires adequate preoperative evaluation. However, this requires many resources. Management of anaesthesia in an adrenal tumour with an unknown hormonal disorder should consider a variety of hormonal abnormalities that may present in the patient so it can be anticipated effectively.

Keywords: Incidentaloma, adrenalectomy, hormonal disorder, perioperative

Cite This Article: Widnyana, I.M.G., Putra, K.A.H., Kurniyanta, P., Suarjaya, I.P.P., Suranadi, I.W., Hartono, B. 2018. Anaesthetic Management of Adrenal Tumour Resection. *Bali Journal of Anesthesiology* 2(2): 29-32. DOI: [10.15562/bjoa.v2i2.28](https://doi.org/10.15562/bjoa.v2i2.28)

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INTRODUCTION

Incidentaloma is an adrenal tumour sized more than one centimetre in diameter and discovered accidentally during radiologic evaluation conducted on an indication other than an adrenal disorder. Along with the development of diagnostic imaging technology, its frequency is increasing. One study reported a prevalence of incidentaloma is about 4%. From research through an autopsy, the mean frequency of adrenal adenomas is about 6%. Adrenal adenoma prevalence increases with age. The possible discovery of an adrenal tumour during a abdominal CT scan in the age group 20-29 years is approximately 0.2%, while at the age of 70 years, it is about 7%.

Hormonal and metabolic disorders are common in patients with adrenal incidentaloma. Some abnormalities that may occur are subclinical Cushing's syndrome, silent pheochromocytoma, primary aldosteronism, or other hormonal disorders.

In this case report, we present a 36 year old woman with a left adrenal tumour that underwent adrenalectomy at Sanglah Hospital, Bali, Indonesia. Management of anaesthesia, in this case, is quite challenging because it requires better preparation and governance.

CASE REPORT

A 36 year old woman suffered from hypertension detected four months before surgery, accompanied by intermittent pain at her left flank. Due to her persistent hypertension, she was scheduled for an

abdominal ultrasound. The examination showed left adrenal tumour size of 9.97 × 8.29 cm.

She showed no signs or symptoms specific to adrenal hormone disorders. She has a body mass index of 29.4 kg/m² and her physical examination showed no abnormalities with the exception of hypertension (BP 160/90 mmHg).

Laboratory examination showed a normal blood count and hemostasis profile. Blood chemistry test revealed high random blood sugar (233 mg/dL), with HbA1c at 7.3%. Her electrolytes were normal and no hormonal examination was performed.

She underwent the surgery under general and epidural anaesthesia. Midazolam (2 mg) was given intravenously in the preparation room and an artery line was inserted for blood pressure (BP) monitoring. Induction was done by administering Propofol (150 mg). For intubation, 200 mcg fentanyl and 50 mg rocuronium were given. To maintain anaesthesia, propofol was continually administered with targetted controlled infusion (TCI) and rocuronium was administered intermittently. To provide good analgesia, an epidural catheter was inserted in lower thoracic level, using 10 mL of 0.25% bupivacaine and maintained by continuous bupivacaine 0.25% infusion at 5 mL/hour. Nitroglycerine and norepinephrine were prepared to anticipate the hemodynamic changes. The surgery took 2 hours 45 minutes.

Postoperatively, the patient was admitted to the Intensive Care Unit for 24 hours before being transferred to the ward. For the management of postoperative pain, she received 0.1% bupivacaine and 1 mg morphine per epidural catheter every

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12 hours, with intravenous paracetamol. Microscopic evaluation revealed lymphangioma and chronic suppurative inflammation in the fat tissue.

DISCUSSION

The adrenal gland is a retroperitoneum endocrine organ that is located on the upper pole of each kidney. Its vascularization comes from three main sources, namely the bilateral inferior phrenic artery, suprarenal arteries, and inferior suprarenal arteries, but for drainage, it usually only has a single vein leaving the glands.^{1,2} Each adrenal gland is composed of two major parts, the cortex, and medulla. Each part secretes specific hormones. The inside of the adrenal gland, called the medulla, secretes amine hormones, such as epinephrine and norepinephrine. The medulla contains sympathetic ganglion cells that the postganglionic part transformed into secretory cells called chromaffin cells or pheochromocytoma.

The adrenal cortex is a broader part consisting of three layers. The glomerulus zone is a thin layer of cells located just under the capsule and is made up of about 15% of the cortex and produces aldosterone. The fasciculate zone is located in the middle and is the largest zone making up about 75% of the adrenal cortex. This zone produces glucocorticoid cortisol, corticosterone, androgens, and estrogens. Finally, the reticular zone is the innermost part of the cortex producing androgens, dehydroepiandrosterone sulfate, androstenedione, estrogen, and glucocorticoid.³

Primary tumours of the adrenal glands often appear with specific symptoms associated with unregulated hormone secretion, non-specific symptoms related to mass effect, or the expansion of a tumour or abnormality metastatic.¹

The prevalence of adrenal adenomas was reported as 1.4 to 8.7%, and the incidence increases with age. Most adrenal adenomas are non-functional. Patients with cortisol-producing tumours will have signs and symptoms of hypercortisolism which was first described by Cushing in 1912. Pheochromocytoma is a rare malignant tumour derived from the adrenal medulla, chromaffin cells.¹

Conn's syndrome

Primary hyperaldosteronism, or Conn's syndrome, is a disorder that occurs due to excessive aldosterone production by the adrenal glands. Most cases (60%) of this syndrome are associated with unilateral adrenal adenomas, while the remaining cases (30%) are related to bilateral adenoma. The disorder is more common in women than men and is

responsible for 5-13% of secondary hypertension and 1% of essential hypertension cases.

Conn's syndrome is clinically manifested as systemic hypertension, metabolic alkalosis, hypokalemia, increased excretion of potassium through the urine, hypernatremia, fatigue, muscle cramps, and skeletal muscle weakness. Systemic hypertension, sometimes in the form of elevation of the diastolic blood pressure, may occur due to sodium and fluid retention. Hypertension is often resistant to various treatments of hypertension drugs. Fatigue, muscle cramps, and skeletal muscle weakness are related to hypokalemia. In addition to clinical manifestations, radiological and biochemical investigation plays an important role. The biochemical examination includes renin and aldosterone plasma level, and blood sodium and potassium level. Conn's syndrome generally shows decreased levels of plasma renin because of high aldosterone. The useful radiological investigation methods include an angio-CT scan and magnetic resonance imaging (MRI).

Anaesthetic management of Conn's syndrome should consider intraoperative hemodynamic changes and hypokalemia. Hypokalemia and metabolic alkalosis extend the duration of the muscle paralysis and may induce bradycardia. Hypokalemia can be even worse because of their respiratory alkalosis due to hyperventilation. Manipulation of the adrenal glands during dissection and resection triggers the release of catecholamine from the adrenal medulla resulting in hemodynamic fluctuations. Hypervolemia and hypovolemia should be treated aggressively.

Cushing Syndrome

Cushing's syndrome occurs because of elevated levels of circulating glucocorticoids. This can occur due to oversecretion or supplemental glucocorticoid therapy for a long period. Approximately 70% of Cushing's syndrome occurs due to Cushing's disease, a pituitary gland tumour that produces ACTH. Approximately 15% occurs due to ectopic ACTH production, and the remaining 15% is due to an adrenal tumour.

Clinical manifestation of Cushing's syndrome is very typical. It usually presents with triad of central obesity, moon facies, and buffalo hump. Other manifestations are proximal skeletal muscle weakness, fatigue, spontaneous fractures due to osteopenia, thinning and bruising of the skin, purple striae, sexual disorders (amenorrhea, infertility, irregular menstruation, and decreased libido), hypertension secondary to fluid retention in the body, hyperglycemia, metabolic alkalosis, and hypokalemia.

Anaesthetic management of patients with Cushing's syndrome should consider existing

disorders. Hypercortisolism can be controlled in the preoperative period with adrenal enzyme inhibitors, such as ketoconazole, metyrapone, mitotane, or aminoglutethimide. Attempts to optimize before adrenalectomy includes control of hypercortisolism, hypertension, hyperglycemia, hypokalemia, and prevention of perioperative hypercoagulability. Hypertensive drugs should be continued, except for ACE inhibitors and ARBs because of the risk of dramatic blood pressure drop during the induction. Glycemic control can be achieved by using insulin to maintain blood glucose at 120-180 mg/dL. Prevention of thromboembolism may include low molecular weight heparin (LMWH), inferior extremity compression tool, and early mobilization in the postoperative period.

Pheochromocytoma

Among the hormone-producing adrenal tumours, pheochromocytoma is the biggest challenge. The disorder is characterized by excessive production of catecholamine, causing an excessive sympathetic tone to cause severe hypertension and arrhythmias. These tumours can be derived from the adrenal or outside of the adrenal. In general, these tumours produce adrenaline, noradrenaline, and dopamine. The earliest signs of a pheochromocytoma are excessive sweating, headache, hypertension, arrhythmia, and palpitations.

Anaesthetic management in patients with pheochromocytoma must consider several things: perioperative hemodynamic control, intraoperative management, and postoperative care. Preoperative preparation requires adequate adrenergic blockade to achieve blood pressure not higher than 160/90 mmHg during the last 24 hours, no orthostatic hypotension, no changes in the ST segment and T wave on ECG, and no more than one premature ventricular contraction in five minutes. Adrenergic blockade can be achieved with a combination of α and β -blockers.

Intraoperative management requires invasive blood pressure monitoring and central venous catheters. Drugs that can stimulate the sympathetic nervous system, such as pancuronium, ketamine, halothane, and desflurane, should be avoided. Induction of anaesthesia and intubation can be done carefully to avoid an increased sympathetic tone. For maintaining the depth of anaesthesia and blunt the sympathetic response during the manipulation of the adrenal gland, total intravenous anaesthesia combination of propofol and remifentanyl can be used, combined with dexmedetomidine. After removal of the adrenal gland, patients will be at risk for hypotension due to non-selective blockade

of α -adrenergic, excessive depth of anaesthesia, or hypovolemia. This hypovolemia may occur as a result of bleeding, use of diuretics, and preoperative hypovolemia that had not been corrected. To control this problem, inotropic drugs and vasopressors may be useful.⁴

Our patient cannot be classified into one of the hormonal disorders that were described above. The diagnosis of adrenal tumour is done through abdominal ultrasound for diagnostic investigations related sustained hypertension. Most incidentalomas are not secreting and are benign. Some hormonal abnormalities that may occur in incidentaloma include subclinical Cushing's syndrome, silent pheochromocytoma, primary aldosteronism, or other hormonal disorders. Thus, preoperative evaluation should be in the more detailed way, particularly on hormonal evaluation to detect hormonal disorder related to adrenal tumour and to assess whether the tumour is malignant or not.

The examinations that should have been performed are blood and urine cortisol levels, urine 17-hydroxycorticosteroid level, plasma ACTH levels, and dexamethasone suppression test to detect the presence of subclinical Cushing's syndrome. For the evaluation of silent pheochromocytoma, it is necessary to check fractionated metanephrines and catecholamine in 24-hour urine. An increased fractionated metanephrines and catecholamine level in the urine supports the diagnosis of pheochromocytoma. Another examination needed is the ratio between the concentration of aldosterone and renin in the morning. It is necessary to detect the presence of primary hyperaldosteronism because not all tumours that produce aldosterone experiencing hypokalemia.^{5,6}

Due to the limitations of preoperative information, we cannot specifically exclude hormonal abnormalities. Therefore, the management of anaesthesia in this patient should anticipate the presence of hormonal disorders that had not been identified. Induction of anaesthesia was done with an intravenous anaesthetics agent with sufficient depth prior to manipulation, for example laryngoscopy and intubation.

Drugs such as metoclopramide, ephedrine, and chlorpromazine can trigger hypertension and thus were not given to the patient. Drugs that trigger the histamine release, such as atracurium and morphine, were also not used. For maintaining anaesthesia, N₂O gas was not used because it can cause abdominal distension. To obtain hemodynamic stability, we use continuous epidural anaesthesia. No sedative drugs were given in the ICU in order to avoid the risk of respiratory depression after surgery.^{7,8}

SUMMARY

Incidentaloma is an adrenal mass generally sized more than one centimetre in diameter discovered accidentally during radiologic evaluation on indications other than adrenal disorder. Effective anaesthetic management during the resection of adrenal incidentalomas requires adequate preoperative evaluation. However, this will take a large resource. Management of anaesthesia for an adrenal tumour with unknown hormonal disorders should account for a variety of abnormalities that may present in the patient so it can be anticipated effectively.

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