Cortex sparing laparoscopic adrenalectomy in a patient with Conn’s syndrome

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ABSTRACT

Conn’s syndrome, an aldosterone producing adenoma, is a surgically curable cause of primary aldosteronism, classically treated by unilateral adrenalectomy. With the advent of laparoscopic surgery in the recent decade, laparoscopic adrenalectomy is currently accepted as the gold standard of treatment for Conn’s syndrome. Cortical sparing adrenalectomy is especially an ideal operation for patients with bilateral pheochromocytoma. This case report describes a successful laparoscopic adrenal cortex sparing surgery on the left side and anesthetic approach in a patient with Conn’s syndrome, who had a history of previous right surrenalectomy. Laparoscopic surgery without dividing the central adrenal vein can also be performed successfully in patients with Conn’s syndrome.

Key Words: Laparoscopy, adrenal gland, Conn’s syndrome

INTRODUCTION

Primary aldosteronism, is characterized by hypertension induced by elevated plasma aldosterone and suppressed renin activity. Conn syndrome is an aldosterone-producing adenoma and was first described in 1955 by Jerome Conn (1).

In recent years, with advances in laparoscopic surgery, laparoscopic adrenal cortex-sparing surgery has gained acceptance. Adrenal cortex-sparing surgery is a commonly practiced surgical technique especially in patients with bilateral pheochromocytoma. The current gold standard technique in patients with Conn’s syndrome is laparoscopic adrenalectomy on the side of the adenoma (2).

CASE PRESENTATION

A 30-year-old female presented with fatigue, headache, palpitations and high blood pressure. Her medical history revealed an atrophic right nephrectomy performed for a diagnosis of kidney stones on August 2008. She had hypokalemia (K+=2.3-2.4 mmol/dL) and a previous renal artery doppler ultrasonography in May 2010 was found to be normal. In the Endocrinology Department, the recumbent and ambulant saline infusion test showed elevated aldosterone levels, and the patient was planned for abdominal CT and MRI.

The abdominal CT showed absence of the right kidney, normal functioning left kidney with regular contours, and a slight thickness increase in the body of the left adrenal gland (Figure 1). In the abdominal MRI, the right kidney and right adrenal gland were not visualized, an approximately 9 mm, regular bordered nodular lesion was observed medial to the left adrenal gland, showing slight contrast enhancement following injection of IV Gadolinium, with slight signal suppression in opposite phase images (Figure 2). She was also being followed up for euthyroid multinodular goiter, under valsartan 320 mg, amlodipin10 mg, spironolactone 50 mg, metoprolol 25 mg, and oral potassium replacement therapies. In the preoperative period, complete blood count, electrolytes and biochemistry and coagulation values were all within normal limits. Since the right adrenal gland has been previously removed, in order to prevent adrenal insufficiency, a left cortex sparing adrenalectomy was decided in the endocrinology council meeting.

The patient was premedicated with 0.02 mg/kg iv midazolam, and a continous ECG, SpO₂ and non-invasive arterial blood pressure and et-CO₂ monitoring was performed. Anesthesia was induced with 6 mg/kg thiopental Na and fentanyl 1 mcg/kg. Muscle relaxation was achieved with atracurium 0.5 mg/kg and the patient was intubated endotracheally with No. 8 cuffed tube. The pneumoperitoneum was achieved with CO₂ insufflation. Intra-abdominal pressure was 12-14 mmHg. The patient’s ventilation was adjusted to attain an et-CO₂ value of 30-35 mmHg. Maintenance of anesthesia was provided with 1 MAC sevoflurane in a mixture of 50% O₂-N₂O. The operation lasted for about 2.5 hours, and the patient’s vital signs remained within normal limits. The patient received 1500 cc crystalloid infusion during surgery, and 75 mg of intramuscular diclofenac was applied for postoperative pain control at the end of the operation and the patient was extubated uneventfully.
The patient was kept in the right lateral decubitus position. The abdomen was accessed with four 10-mm ports. Adhesions due to the past operation were released. The splenic flexure and the spleen were observed as a single structure. The spleen was mobilized medially. Using a surgical plane created between the tail of the pancreas and Gerota's fascia, the pancreas was dissected medially. Inferior phrenic veins were visualized at the superior edge of the adrenal gland and were maintained. The left inferior and middle adrenal arteries were dissected (Figure 3). The insertion of the left adrenal vein to the renal vein was visualized. After these dissections, both adrenal arteries and the main adrenal vein were clipped and cut (Figure 4). Then, by using ligasure the adenoma was excised with some surrounding healthy adrenal tissue. During this excision, approximately 1/3 of the adrenal cortex was left behind. During this process, special attention was paid not to dissect the remaining adrenal gland from the surrounding tissues. Pathological analysis of the removed specimen revealed a 3.5x2x1.5 cm adrenal adenoma (Figure 5).

The patient was discharged on the 4th postoperative day, with stable vital signs and electrolyte values, after consulting with endocrinology. Laboratory values on the 20th postoperative day were as follows; Na=139 mmol/L, K=4.7 mmol/L, renin=0.17 ng/mL/h (0.7-3.3), aldosterone = 38 pg/mL (70-300), cortisol=21.51 mcg/dL (3.8-17.0). On her 3rd month follow-up, she did not require any medications, her vital signs and electrolyte values were stable. Blood cortisol, aldosterone and renin levels were also within normal limits. Since a portion of the cortex of the adrenal gland was protected during surgery, our patient did not require steroid replacement therapy. With extremely good early postoperative follow-up results, the patient is under follow-up for long-term evaluation.

DISCUSSION

JF Conn first described primary hyperaldosteronism in 1955 (3). It is characterized by hypertension and hypokalemia induced by excess release of aldosterone due to adrenal gland hyperplasia or adenoma (3, 4). Conn's syndrome is a rare cause of hypertension, which may be resistant to medical treatment. Primary hyperaldosteronism affects 5 to 13% of hypertensive patients and is the most common cause of endocrine system induced hypertension (5).

The open and laparoscopic surgical techniques have been described in adrenal gland surgery. Laparoscopic technique can be applied either transperitoneally or retroperitoneally. In this
and hypotension may occur. In our case, we did not observe such a situation. Hypokalemia and metabolic alkalosis may prolong the effect of non-depolarizing muscle relaxants (3). In our case, the patient was operated after her K+ value was brought to a normal level with oral supplements. In their case series of 59 patients, Finch et al. (14) stated that hypertension developed only in 7 patients during tumor resection, due to secretion of catecholamine by the adrenal medulla caused by manipulation of the gland, and that vasodilator therapy was needed only in two of these patients. In our case, vasodilator therapy was not required during the operation.

In this patient with Conn’s syndrome, cortex-sparing surgery has been applied with success like in patients with bilateral pheochromocytoma. In Conn syndrome that is a benign disease, although the small number of patients and lack of long-term results precludes a generalization, resection of the adenoma by protecting the healthy portion of the adrenal gland should be preferred.

Cortex-sparing adrenalectomy in this case of Conn’s syndrome, avoided Addison’s crisis risk and the need for steroid replacement. Long-term follow-up should be done meticulously in terms of tumor recurrence.

CONCLUSION
We believe that laparoscopic adrenal cortex-sparing surgery can be successfully applied in patients with Conn syndrome, with a meticulous preoperative evaluation, preparation, and perioperative monitoring.

Peer-review: Externally peer-reviewed.

Author Contributions: Study concept and design - F.Y., A.E.S.; Acquisition of data - F.Y., A.O., M.T., B.C., L.K.; Analysis and interpretation of data - F.Y., A.E.S., M.K.; Preparation of the manuscript - F.Y., A.E.S.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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