Adrenal Cortical Adenoma Initially Presenting with Acute Aortic Dissection, Hypokalemic Paralysis, and Marked Hypertension

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Adrenal cortical adenoma is a rare cause of secondary hypertension and may present with elevated blood pressure and hypokalemia. We reported a 38-year-old female with adrenal adenoma presenting with a rare initial manifestation of severe back pain. Acute aortic dissection, marked hypertension and lower leg paralysis were noted simultaneously. Stanford type B aortic dissection and left adrenal tumor were noted by computed tomography angiography. Hypokalemia, high serum aldosterone and low renin level make the clinical suspicion of adrenal adenoma. Blood pressure was under optimal treatment after intensive medical treatment and lower leg paralysis was resolved after correction of hypokalemia. The patient underwent laparoscopic adrenalectomy later and the pathology report proved to be adrenal adenoma. Blood pressure and serum potassium level returned to normal after adrenalectomy and the patient was uneventful in the following days without any medication.

Key words: adrenal cortical adenoma, secondary hypertension, hypokalemia, aortic dissection, lower leg paralysis

Introduction

Adrenal cortical adenoma is a relatively rare etiology of secondary hypertension (less than 1%) and may sometimes coexist with hypokalemia, hyperaldosteronism, and low serum renin level¹². Its symptomatic complications of blood vessels and/or hypokalemia are even less commonly seen in clinical situations. In this present case report, we presented a patient with adrenal adenoma with initial manifestation of an extremely rare triad, aortic dissection, lower leg paralysis, and marked hypertension, which has not been reported before. In this case report, we presented the clinical history and symptoms/signs of this patient, the mechanisms of different complications, and the strategies to manage these problems.

Case Report

The 38-year-old female was rather healthy before and did not notice the onset of any underlying disease. Sudden onset of severe “tearing” back pain was noted at midnight when she was watching television. The pain persisted for 30 minutes and did not resolve in spite of taking rest. There was no chest tightness/pain or dyspnea, but it was associated with cold sweating.
and palpitation. She was sent to our emergency department for help for intractable back pain. At the time of presentation, she was acute ill-looking. She had a blood pressure of 222/112 mmHg, a heart rate of 92 beats per minute, and a respiratory rate of 18 breaths per minute. There was mild elevated body temperature (37.3°C). Heart sounds were regular without evident heart murmur noted and the breathing sounds were clear. The electrocardiogram showed non-specific ST-T changes and the chest radiograph revealed mild tortuosity of aorta.

Cardiac enzymes showed mild elevation of creatin phosphokinase, but normal CK-MB, and Troponin-I levels. Aortic dissection was suspected because of coexistent severe back pain and high blood pressure. Emergent Computed Tomography (CT) scan and CT angiography revealed Stanford type B aortic dissection from aortic arch extending beneath the bifurcation of common iliac artery (Fig. 2). A 1.4cm well-defined hypodense tumor was also incidentally noted in the left adrenal gland at the same time (Fig. 1, 3). Intensive antihypertensive drugs, including Felodipine, Labetalol, Doxalosin, and Lisinopril, were used to control the blood pressure. Under the impression of aortic dissection related to marked hypertension and left adrenal tumor with unknown character, work-up for adrenal tumor was started. Hypokalemia (2.5 mmol/dl; normal: 3.5-5.3 mmol/dl), elevated aldosterone (110.25 pg/mL; normal: 39-90 pg/mL), and low renin level (0.13 pg/mL; normal: 9.8-31.3 pg/mL) were noted and adrenal cortical adenoma was suspected. Back pain improved after intensive blood pressure control under 120/80 mmHg, but weakness and mild paralysis of lower leg was noted during admission. Physical examination showed diminished lower deep tendon reflex. Duplex studies of lower leg showed normal ankle-brachial index (ABI) without evident compromise of perfusion of lower extremities. Thyroid function tests revealed normal results. Hypokalemia-related lower leg

Fig. 1  Computed Tomography (CT) scan shows a hypodense tumor measured about 1cm x 1cm in size (thin black arrow) in left adrenal gland

Fig. 2  Computed Tomography Angiography (CTA) shows aortic dissection from aortic arch to the level beneath bifurcation of common iliac artery (thin black arrows)
paralysis was suspected and potassium replacement was performed. Lower leg paralysis improved immediately after normalization of potassium level and did not occur again later. The patient underwent laparoscopic adrenectomy 3 weeks later and the pathology report showed a 1.5 × 1.2 × 1cm adrenal cortical adenoma. After surgery, blood pressure of the patient returned to normal even without antihypertensive drugs and serum potassium level was also in normal range without potassium supplement. Back pain and lower leg paralysis did not occur again in the following months and the patient was uneventful without any medication.

**Discussion**

Adrenal cortical adenoma is responsible for less than 1% of patients with hypertension and may sometimes present with secondary hypertension, hypokalemia, polyuria, metabolic alkalosis, muscle cramping, and rarely cardiovascular complications. It occurs more often in females between 30 to 50 years old and should be suspected in patients with early onset or intractable hypertension, hypokelema, low serum renin, and/or high serum aldosterone level. A high plasma aldosterone: rennin ratio (> 20) is also a useful screening test for adrenal adenoma. Further confirmative tests include adrenal vein sampling, CT scan\(^{(3,4)}\), and magnetic resonance imaging scans\(^{(4)}\).

Hypertension is a common clinical presentation of adrenal adenoma and may be initially resulted from aldosterone-related volume overload followed by peripheral vasoconstriction. Aldosterone antagonist and other antihypertensives can be used in patients with adrenal adenoma and hypertension. Laparoscopic adrenectomy is a choice to cure hypertension secondary to adrenal adenoma. In previous studies, 33-88% (average > 60%) of patients can completely stop taking antihypertensive drugs after surgical adrenectomy\(^{(5)}\).

Hypokalemia is also not uncommon in patients with adrenal adenoma, but normal serum potassium level does not completely rule out its possibility. In patients with lower serum potassium levels, muscle cramps or paralysis of lower legs may occur and may be mistaken as myopathy, poliomyelitis, or peripheral arterial disease, etc.\(^{(6-7)}\). Major causes of hypokalemia include long-term diuretic use, dietary deficiency, alcoholic myopathy, intestinal potassium wastage, Bartter’s syndrome, licorice intoxication,
amphotericin B treatment, diabetic ketoacidosis, renal tubular acidosis, and chronic diarrhea. In our case, two important causes of hypokalemic paralysis should be considered—hypokalemic paralysis secondary to hyperaldosteronism and hereditary hypokalemic periodic paralysis. Both causes of hypokalemic paralysis respond well to potassium supplement. However, surgical adrenectomy completely restored the potassium level to normal range and cured the symptom of paralysis in this patient. This result proved the cause of hypokalemic paralysis to be resulted from adrenal adenoma.

Cardiovascular complications of adrenal adenoma are even rarer and only one case of aortic dissection had been reported before. In patients of adrenal adenoma with sustained or marked hypertension, cardiovascular complications may have higher incidence. The mechanisms involve degeneration of aortic media and chronic stress against aortic walls from long-term hypertension. An intimal tear directly exposes the underlying diseased medial layer to the driving force of the intraluminal blood and results in aortic dissection. Aggressive blood pressure control to reduce shearing force of aortic wall is the major task to stabilize the progression of aortic dissection. Surgical treatment should be performed in patients with Stanford type A dissection, progression of dissection, or major organ damage. In patients with Stanford type B dissection as our case report, conservative medical treatment with intensive antihypertensives followed by laparoscopic adrenectomy may be the best method.

In recent years, endoscopic adrenectomy replaced the role of open surgery and was used worldwide to achieve better clinical outcomes. Endoscopic adrenectomy can avoid a large skin incision compared with previous surgery, result in diminished postoperative pain intensity, allow for early immobilization, decrease wound-related complication rate, have minimal blood loss, shorten postoperative hospital stay, and reduce duration of postoperative convalescence. However, better result of endoscopic adrenectomy should be achieved after longer learning phases of well-trained surgeons.

**Conclusion**

Adrenal cortical adenoma is one of the causes of secondary hypertension and hypokalemia, and may result in cardiovascular complications, including aortic dissection or limb paralysis. In physicians familiar with adrenal adenoma and its complications, this tumor could be diagnosed and treated earlier.

**References**


腎上腺皮質腺瘤以嚴重高血壓，主動脈剝離，
及下肢癱瘓為起始表現

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腎上腺皮質腺瘤(Adrenal adenoma)為一罕見之續發性高血壓原因之一。臨床上常以高血壓及低血鉀為表現。我們報告一38歲女性以嚴重背痛之症狀求診。同時發現有嚴重高血壓，Stanford B型主動脈剝離，即下肢癱瘓之表現。經電腦斷層掃描結果有左側腎上腺腫瘤合併低renin，高aldosterone，及低血鉀。因上述表現而懷疑為腎上腺皮質腺瘤合併嚴重高血壓致主動脈剝離及低血鉀性癱瘓。下肢癱瘓於矯正高血鉀後改善。經腹腔鏡腎上腺腫瘤切除後，血壓及血鉀值不經藥物即正常。而病理報告亦證實為腎上腺皮質腺瘤。

關鍵詞：腎上腺皮質腺瘤，續發性高血壓，低血鉀，主動脈剝離，下肢癱瘓

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