Secondary Hypertension: Diagnosis and Management of an Adrenal Adenoma

Case Study and Commentary, Debbie L. Cohen, MD, and Raymond R. Townsend, MD

INSTRUCTIONS

The following article, “Secondary Hypertension: Diagnosis and Management of an Adrenal Adenoma,” is a continuing medical education (CME) article. To earn credit, read the article and complete the CME evaluation form on page 532.

OBJECTIVES

After participating in the continuing education activity, primary care physicians should be able to:
1. Know the renal and adrenal causes of secondary hypertension
2. Know the components of the clinical examination of a hypertensive patient
3. Be familiar with laboratory tests used to assess an incidentally discovered adrenal mass
4. Know the usual findings in primary aldosterone excess (Conn’s syndrome)
5. Understand the approach to follow-up in patients with secondary hypertension

Approximately 42.3 million adults in the United States have hypertension, according to the National Health and Nutrition Examination Surveys III (NHANES). The NHANES survey defined hypertensive patients as those with a systolic blood pressure of 140 mm Hg or greater, a diastolic blood pressure of 90 mm Hg or greater, or those taking hypertension medication [1]. Because an estimated 7.7 million adults with hypertension had adopted lifestyle changes that reduced their blood pressure to normal levels prior to the NHANES survey, it can be assumed that there are approximately 50 million adults with hypertension in the United States [2]. Although 3 out of 4 patients with hypertension are aware of their diagnosis of hypertension, blood pressure control remains poor. Many patients are untreated or inadequately treated. Only 29% of patients have their blood pressure controlled to below 140/90 mm Hg [2].

Intensive efforts have been made to improve blood pressure control by increasing both physician and patient education. Despite these efforts, blood pressure control and awareness have remained low [2]. Poor outcomes have been blamed on patient noncompliance with medications [3], limited access to medical care, financial barriers to obtaining medications, and, more recently, an insufficiently aggressive approach to blood pressure lowering by health care providers [4,5].

Hypertension is an expensive disease to treat. It has been estimated that the cost for hypertension care in the United States in 1995 was $18.7 billion; 20% of this amount represented the costs of antihypertensive drugs [6]. Given these costs, it is important to rule out potentially treatable causes of hypertension in appropriately selected patients.

CASE STUDY

Initial Presentation

A 35-year-old white woman presents to a nephrology practice for enrollment in a hypertension study testing a new medication for high blood pressure. She feels well and when untreated has a blood pressure of 154/108 mm Hg, confirmed by several measurements.

• What is the etiology of hypertension?

Etiology of Hypertension

More than 90% of patients with hypertension have essential (primary) hypertension [7]; the remaining 10% have hypertension attributed to secondary causes. The prevalence of essential and secondary hypertension is shown in Table 1.

There are 2 common types of secondary hypertension: renal and adrenal (Table 2). These 2 causes account for more than 90% of all cases of secondary hypertension. Renal causes are the most common. Hypertension ensues from renal parenchymal diseases (eg, acute or chronic glomerulonephritis), renovascular diseases (eg, renal artery stenosis), and, rarely, renin-producing tumors or primary sodium

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retention disorders (eg, Liddle’s syndrome) [8]. Adrenal causes are divided into cortical and medullary types. Cortical adrenal disease can result in increased production of aldosterone as seen in primary aldosteronism (Conn’s syndrome) or increased production of glucocorticoid as seen in Cushing’s syndrome. Medullary adrenal disease can result in increased production of norepinephrine and epinephrine as seen in a pheochromocytoma. Although less common, coarctation of the aorta can also cause secondary hypertension by causing downstream renal ischemia, resulting in increased renin production.

A number of other phenomena are associated with increased blood pressure. Exogenous hormones (including estrogen in hormone replacement therapy, glucocorticoids, mineralocorticoids), sympathomimetic agents (particularly those used in over-the-counter cold preparations), and erythropoietin (administered to patients with anemia of chronic renal failure, HIV infection, and cancer) can cause increased blood pressure. Hypertension can also accompany pregnancy, sleep apnea, acute stress situations (eg, hypoglycemia and burns), increased intravascular volume secondary to alcohol and nicotine use, or an increased cardiac output due to thyrotoxicosis, arteriovenous fistulae, or Paget’s disease of bone.

Table 1. Frequency of Diagnoses in Patients Attending a Hypertension Clinic

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Frequency</th>
</tr>
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<tr>
<td>Essential hypertension</td>
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<tr>
<td>Chronic renal disease</td>
<td>5.6</td>
</tr>
<tr>
<td>Renovascular disease</td>
<td>0.7</td>
</tr>
<tr>
<td>Primary aldosteronism</td>
<td>0.3</td>
</tr>
<tr>
<td>Cushing’s syndrome</td>
<td>0.1</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>0.1</td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
<td>0</td>
</tr>
<tr>
<td>Oral contraceptive use</td>
<td>1.0</td>
</tr>
</tbody>
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Table 2. Types and Causes of Hypertension

- Essential hypertension
- Endocrine: primary aldosteronism, Cushing’s syndrome, congenital adrenal hyperplasia, pheochromocytoma, hyperthyroidism, hypothyroidism, acromegaly, hypercalcemia, carcinoid tumors
- Renal: acute glomerulonephritis, chronic glomerulonephritis, polycystic kidney disease, diabetic nephropathy, interstitial renal diseases, renal artery stenosis, vasculitis, renin-producing tumors, primary renal sodium retentive states (Gordon’s syndrome, Liddle’s syndrome)
- Coarctation of the aorta
- Pregnancy-induced hypertension
- Foods containing tyramine and monoamine oxidase inhibitors
- Exogenous hormones: estrogen, mineralocorticoids, glucocorticoids, sympathomimetics, erythropoietin
- Acute stress situations: surgery, hypoglycemia, burns, alcohol withdrawal
- Increased intravascular volume
- Drugs: cocaine, nicotine, cyclosporine, tacrolimus
- Increased cardiac output states: thyrotoxicosis, Paget’s disease, beriberi, anemia, arteriovenous fistula, aortic valve regurgitation


When should one suspect secondary hypertension?

When treating patients with hypertension, it is important to keep in mind that the likelihood of finding secondary hypertension is low. Doing so can help one avoid a costly workup in patients with essential hypertension. However, this caveat must be balanced by understanding the possibility of finding a treatable underlying cause of hypertension. Because patients with secondary hypertension are often young, it may be possible to cure the hypertension if it is detected early enough.

One should suspect secondary hypertension in patients who present with a negative family history of hypertension; unprovoked hypokalemia; severe drug-resistant hypertension; recent worsening of or difficulty in controlling previously well-controlled essential hypertension; a triad of headache, sweating, and palpitations; an abdominal bruit; or new onset hypertension in a young patient (less than 20 years) or in a patient older than 50 years (Table 3). In patients who present with refractory hypertension, however, only 10% will have a secondary cause for their hypertension. Other causes of refractory hypertension are shown in Table 4.

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- What are the minimal diagnostic tests for evaluating a new hypertensive?

Evaluation of the Hypertensive Patient

The clinical examination should include an examination of the fundi to assess for changes seen with hypertensive retinopathy. The peripheral pulses and radiofemoral delay should be assessed to exclude coarctation of the aorta and presence of an epigastric bruit. The latter finding is highly specific, although not sensitive, for renal artery stenosis. Electrocardiography should be performed to assess for left
Ventricular hypertrophy (LVH). It is important to document the presence of LVH because it is an independent risk factor for increased cardiovascular morbidity and mortality [9,10]. An echocardiogram is the procedure of choice, however, since the sensitivity of the electrocardiographic criteria for detecting LVH may be as low as 7% to 35% with mild LVH and 10% to 50% with moderate to severe LVH [11]. Electrocardiographic evidence of reversal of LVH is associated with decreased cardiovascular morbidity [12].

Laboratory tests should include urinalysis, a chemistry panel including measurement of serum creatinine, potassium, and glucose, and a lipid panel [1]. Urinalysis is done to assess whether proteinuria or hematuria are present, since either of these may indicate a renal cause for hypertension. Measurement of glucose in the urine is also helpful to screen for diabetes. A chemistry panel is used to assess renal function; a high serum potassium concentration may indicate renal failure or renal tubular acidosis, and a low concentration may indicate a state of aldosterone excess. A lipid panel is important to assess for hyperlipidemia. Hyperlipidemia increases the patient’s cardiovascular risk profile and should be treated according to the National Cholesterol Education Program (NCEP) guidelines [13].

### History and Physical Examination

The patient was first noted to be hypertensive after the birth of her second child approximately 6 years ago. Her family history is positive for hypertension in her mother. Current medications are amiloride/hydrochlorothiazide 5/50 mg once daily, verapamil SR 240 mg daily, and a potassium supplement 20 mEq twice daily. Her height is 5’3” and her weight is 114 lb. Examination of her fundi reveals evidence of arteriolar narrowing (grade 1 hypertensive changes). Heart and lung examinations are normal. The abdomen is without bruits. Examination of the extremities shows good peripheral pulses.

### Laboratory Testing

A 12-lead electrocardiogram is normal. Results of urinalysis are normal. Her blood glucose level is 78 mg/dL. Serum concentrations of electrolytes are as follows: sodium, 141 mEq/L (normal, 136–144); potassium, 4.4 mEq/L (normal, 3.5–5.3); chloride, 102 mEq/L (normal, 96–108); and bicarbonate, 29 mEq/L (normal, 24–29). The creatinine concentration is 0.9 mg/dL (normal, 0.7–1.3 mg/dL).

- Which diagnoses related to high blood pressure have been excluded at this point?
- Is there reason to be concerned about a secondary cause or component to the patient’s elevated blood pressure?

The patient has normal pedal pulses and no unusual murmurs, which excludes coarctation of the aorta. The patient is not overweight, which excludes obesity-associated hypertension. The normal blood glucose level excludes diabetes-associated hypertension. The absence of abdominal bruits decreases the likelihood of renovascular disease. Because of the young age and normal weight of the patient at the time of diagnosis and the potassium supplement usage, a secondary form of hypertension should at least be considered.

### Further History

On further questioning, the patient reports that she had developed right sided flank/back pain, which was thought to be job-related. However, the urinalysis ordered by her primary care physician revealed microscopic hematuria, which was a new finding. It is not known whether she was menstruating at the time the urine sample was obtained. Her primary care physician ordered an ultrasonographic evaluation of her right upper quadrant to exclude the possibility of renal stones or a renal tumor. The kidney was found to be normal, but an incidental right adrenal mass was detected. Computed
Tomography (CT) confirmed the presence of a right adrenal mass.

- How frequently do adrenal masses incidentally occur?
- What tests are included in the workup of an adrenal mass?

### Workup of Adrenal Mass

Adrenal masses, or incidentalomas, occur in approximately 2% to 3% of the general population, and many are nonfunctional [14]. All patients with an incidentally discovered adrenal mass must be evaluated for malignancy and subtle hormonal overproduction and to determine whether surgical treatment is needed [15]. Because the case patient has hypertension, an adrenal workup should be done to exclude an aldosterone-secreting adenoma and pheochromocytoma as secondary causes.

To pursue a diagnosis of primary aldosterone excess, one needs to demonstrate suppressed plasma renin activity and show that aldosterone excretion is elevated, either by plasma levels or by a 24-hour urine collection [16–18]. Urine aldosterone levels need to be interpreted in the setting of a normal plasma volume and a urine sodium level of greater than 50 mEq/day [19]. The plasma aldosterone-to-renin ratio can also be suggestive of primary aldosterone excess. This ratio should be greater than 30:1 in patients with primary aldosteronism, and the plasma aldosterone level should be greater than 15 ng/dL [18].

Testing of a 24-hour urine sample for metanephrines and catecholamines is used as a screening test for a pheochromocytoma. A workup is not necessary to evaluate cortisol function because a patient with hypertension and cortisol excess would have clinical manifestations of Cushing’s syndrome.

### Testing and Diagnosis

Testing of a 24-hour urine sample for metanephrines reveals normal excretion: 97 µg of metanephrines per 24 hours (normal < 150) and 176 µg of normetanephrines per 24 hours (normal < 450). The plasma renin activity (off medications) is < 0.1 ng/mL/hr (normal, 2–4). The 24-hour urinary aldosterone excretion is 52 µg (normal, 4–19). The physician makes a diagnosis of primary aldosteronism (Conn’s syndrome). Therapy is initiated with spironolactone, which is and titrated over 2 months to 200 mg daily.

- What are the usual findings in Conn’s syndrome?

### Conn’s Syndrome

Conn described a syndrome of hypertension with hypokalemia and tumor of the adrenal cortex that secretes aldosterone. Metabolic alkalosis is frequently present. Conn’s syndrome is thought to cause less than 1% of cases of hypertension [20], although newer series suggest that the incidence may be higher, especially in drug refractory hypertension [21].

In summary, primary aldosterone excess should be suspected if there is evidence of the following: a low potassium level in blood, suppression of plasma renin activity with evidence of aldosterone excess either by elevated plasma aldosterone blood levels or an increase in 24-hour urinary excretion of aldosterone [20], elevated plasma aldosterone-to-renin ratio [18], and an increase in serum bicarbonate level due to loss of hydrogen (and potassium) ions in urine. It is also reasonable to consider aldosterone excess in a hypertensive patient with an incidentally discovered adrenal mass, in a patient with a need for potassium supplements on modest doses of diuretics, and in patients taking 3 or 4 antihypertensive agents who still have difficulty in controlling their blood pressure.

- Was there anything in the patient’s history to raise suspicion of an aldosterone problem?

The need for 40 mEq of potassium supplement is unusual in a patient on a potassium-sparing diuretic such as amiloride.

### Patient Response to Spironolactone

The patient’s blood pressure 3 months after starting spironolactone is 116/78 mm Hg.

- What are the management options in this patient?

There are 2 options: surgery in the form of a right adrenalectomy [22] or medical management of the patient [23] controlling her blood pressure as needed.

- What factors predict a good response to surgery?
- Do any factors suggest that surgery might not “cure” her high blood pressure?

The patient’s young age suggests that she should have a good response to surgery; however, the length of time she
Adrenalectomy and Follow-up

The physician discusses the treatment options with the patient, but she does not want to undergo a lateralizing procedure and declines the option of surgery as well. The physician follows her clinically on the spironolactone.

Three months later the patient reports to the physician with complaints of amenorrhea and nausea, which are side effects of the spironolactone. The physician recommends that she have adrenal surgery despite not having a lateralizing procedure. She undergoes an uneventful right adrenalectomy and recovers. Two months postoperatively, her blood pressure untreated is 118/80 mm Hg.

- How specific is a blood pressure response to spironolactone as a diagnostic tool for aldosterone excess?

Spironolactone is a competitive inhibitor of the binding of aldosterone to the mineralocorticoid receptor. Spironolactone is very effective at reducing blood pressure and restoring potassium balance to normal in states of aldosterone excess [23,28]; however, it is also an effective antihypertensive drug in patients with suppressed renin activity whose aldosterone production is normal. Therefore, the blood pressure response is suggestive but nonspecific for primary aldosteronism. The case patient’s clear response to the spironolactone led the primary care physician to refer the patient for surgery. In general, despite such a response to spironolactone, we still recommend a second localizing procedure before recommending surgery.

- What potential electrolyte problem can occur postoperatively following adrenalectomy for Conn’s syndrome?

Hyperkalemia can occur postoperatively. Occasionally, unilateral aldosterone release suppresses the aldosterone synthesis on the contralateral side, and the loss of aldosterone production following adrenalectomy can produce a state of “hypoaldosteronism” characterized by hyperkalemia and possibly some salt wasting with low blood pressures. It can take several months to recover full activity in the remaining adrenal gland.

- What is the approach to follow-up of patients with secondary hypertension?

Monitoring Hypertensive Patients

In patients whose blood pressure is restored to normal (without the need for medication) by an intervention, current guidelines suggest yearly blood pressure checks [7]. In the long-term management of secondary hypertension, when patients are not or cannot be cured by surgery or another intervention, follow-up depends on the severity of the hypertension and presence of comorbidities. In general, we recommend follow-up visits every 6 to 12 months for monitoring of stable patients. We typically do annual routine blood testing (measurement of potassium, creatinine, glucose, lipids), unless medication adjustments are made or progression (eg, further loss of renal function) is suspected. Managing hypertension in general prompts one to check for potassium abnormalities that would occur with diuretic and angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker therapy or intercurrent drug use such as nonsteroidal anti-inflammatory agents or steroids. Routine glucose testing and measurement of lipid fractions should be done to screen for diabetes and determine concurrent cardiovascular risk. Creatinine should be measured to monitor kidney function.

Repeat angiograms after a renal angioplasty have not been shown, to our knowledge, to be of value outside of research protocols; therefore, we follow patients clinically and do not routinely schedule follow-up renovascular studies unless blood pressure increases or creatinine changes prompt reevaluation. It could be argued that renal Doppler evaluations are a relatively inexpensive and noninvasive maneuver to monitor renal artery status after renal vessel angioplasty. However, they are also observer/technician
SECONDARY HYPERTENSION

dependent and time-consuming for patients, and in our experience they have a low yield. However, the experience of others has been much more gratifying [29].

In patients with diabetes and those with renal failure with or without renovascular disease, progressive loss of renal function is an unfortunate fact of medical life. The question of when to refer these patients to a nephrologist often arises.

A recent study shows that late referral to a nephrologist (late defined as 3 months before the first dialysis session) greatly compromises the nephrologist-patient relationship, and in diabetic patients leads to greater mortality in the first year on dialysis [30]. Consequently we prefer to see male patients when the creatinine level is 2 mg/dL or greater in diabetics or 3 mg/dL in nondiabetics, with corresponding values of 1.7 mg/dL and 2.5 mg/dL for women. In patients with proteinuria ++ or greater by dipstick or greater than 1 g/day by 24-hour urine collection, the data on proteinuria reduction as a therapeutic endpoint suggest that at least consultation with nephrology should be done if the proteinuria persists on subsequent repeat testing [31].

Last, it is important to recognize that secondary hypertension does not equal curable hypertension. It may be possible to reduce or even eliminate medications in some patients with hypertension when a remediable cause is found. However, some patients have essential hypertension, which is very common, with a superimposed renal artery stenosis or an intercurrent productive adrenal lesion whose relief or removal occurs on a background of essential hypertension. In our, experience factors that help identify those with a higher likelihood of remaining hypertensive after an intervention include older age, duration of hypertension greater than 5 years, and positive family history of hypertension in first-degree relatives.

Summary

One should suspect and pursue a workup for secondary hypertension in patients who appear to be more susceptible from their demographics, history, and physical findings. Conn’s syndrome accounts for 1% of all causes of hypertension and for about 5% of secondary hypertension cases. Patients with Conn’s syndrome can have a normal serum potassium level. It is important to identify patients with secondary hypertension because they may have a curable form of hypertension.

References


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<table>
<thead>
<tr>
<th>Strongly Agree</th>
<th>Strongly Disagree</th>
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I was provided with new information pertinent to my practice.
I reaffirmed a specific skill or knowledge.
This article will help with clinical decision making.
Relevant clinical outcomes are addressed.
The case is communicated in a manner that kept my interest.
The case presentation is realistic and effective.
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As a result of reading this case study, I . . .

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