

The Potassium Thief: Surgical and Medical Management of Conn Syndrome in A Young Hypertensive Patient

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Abstract

This case study presents a 37-year-old female with a 12-year history of hypertension, presenting with easy fatigability and breathlessness. Laboratory investigations revealed severe hypokalemia (K^+ 1.3 mEq/L) and metabolic alkalosis (pH 7.67). Endocrine evaluation showed an elevated Aldosterone-Renin Ratio (ARR) of 7.4. Imaging confirmed a 20×18 mm right adrenal adenoma. The patient was diagnosed with Conn Syndrome and successfully managed medically before undergoing a curative right adrenalectomy. This case underscores the critical importance of early ARR screening in young, treatment-resistant hypertensives and highlights the diagnostic challenge when endocrine symptomatology is masked by concurrent severe hematological derangements.

Keywords: Conn Syndrome, Primary Hyperaldosteronism, Adrenal Adenoma, Hypokalemia, Adrenalectomy.

Introduction

Systemic hypertension is one of the most prevalent modifiable risk factors for cardio-vascular disease worldwide. While the majority of cases in older adults are classified as essential or primary hypertension, the onset of elevated blood pressure in young adults warrants a rigorous search for secondary etiologies. Primary hyperaldosteronism (PA), initially described by Jerome W. Conn in 1955, has emerged as the most common curable cause of secondary endocrine hypertension [1]. Historically considered a rare condition, widespread application of the aldosterone-to-renin ratio (ARR) as a screening tool has revealed that PA may account for up to 10% of hypertensive patients in primary care settings, and an even higher percentage among those with drug-resistant hypertension [2].

The pathophysiology of PA revolves around the autonomous, dysregulated hypersecretion of aldosterone from the adrenal cortex. This unregulated hormone production occurs independent of normal renin-angiotensin system controls. In classic Conn syndrome, the source is a unilateral aldosterone-producing adenoma (APA). Aldosterone acts on the mineralocorticoid receptors in the distal convoluted tubule and collecting ducts of the kidneys, promoting sodium reabsorption and facilitating the reciprocal excretion of potassium and hydrogen ions. This biochemical mechanism earns the adenoma the moniker of a “potassium thief,” draining the body’s potassium stores while simultaneously driving up blood volume and systemic vascular resistance [3]. The clinical consequence is a triad of hypertension, severe hypokalemia, and metabolic alkalosis, though modern screening often detects normokalemic variants early in the disease process.

Identifying PA early is critical. Prolonged exposure to excess aldosterone induces targeted organ damage, particularly cardiovascular and renal remodeling, which occurs at a rate disproportionate to the blood pressure elevation itself [4]. Patients with PA face a significantly higher risk of stroke, myocardial infarction, and atrial fibrillation compared to age- and blood-pressure-matched controls with essential hypertension. Therefore, targeted screening is highly recommended for specific high-risk cohorts.

Table 1: Indications for Primary Hyperaldosteronism Screening

Patient Cohort	Clinical Criteria
Resistant Hypertension	Blood pressure > 140/90 mmHg on three <u>antihypertensives</u>
<u>Hypokalemic Hypertension</u>	Spontaneous or diuretic-induced <u>hypokalemia</u>
Young Onset	Hypertension in patients under 40 years of age
Adrenal <u>Incidentaloma</u>	Hypertension with an incidentally discovered adrenal mass
Family History	Early-onset hypertension or cerebrovascular accident (< 40 years)

The diagnostic workflow for a suspected functioning adrenal adenoma follows a structured algorithmic approach to prevent false positives and accurately localize the lesion (Figure 1). Initially, the serum aldosterone-to-renin ratio (ARR) is measured. If elevated, confirmatory testing—such as an intravenous saline infusion test or oral sodium loading—is performed to demonstrate the lack of aldosterone suppression.

Following biochemical confirmation, radiological imaging becomes the cornerstone of subtyping the disease. High-resolution Computed Tomography (CT) of the adrenal glands is employed to distinguish between a unilateral adenoma and bilateral adrenal hyperplasia. Functioning adenomas characteristically present as well-circumscribed, hypodense lesions due to their high lipid content. For instance, absolute contrast washout greater than 60% and relative washout greater than 40% are strongly indicative of a benign adenoma rather than a malignant process. An example of typical CT imaging for adrenal localization can be seen in Figure 2, illustrating a characteristic hypodense mass.

This paper explores the intricate clinical journey of a young female patient presenting with severe hypokalemia and systemic hypertension. By detailing the sequential diagnostic reasoning, imaging interpretation, and combined medical-surgical management, this study highlights the dramatic physiological impact of a small, unilateral adrenal adenoma. The immediate normalization of potassium levels and subsequent improvement in blood pressure post-adrenalectomy underscore the curable nature of this condition when promptly recognized.

The primary contributions of this clinical case study are threefold: (1) highlighting the diagnostic complexities encountered when severe endocrine hypertension is masked by concurrent, profound hematological derangements (acute-on-chronic anemia); (2) demonstrating the efficacy of a phased multidisciplinary approach, utilizing aggressive mineralocorticoid receptor blockade for rapid preoperative hemodynamic stabilization; and (3) providing a practical, algorithmic clinical pathway for managing the critical postoperative transition to prevent transient hypoaldosteronism.

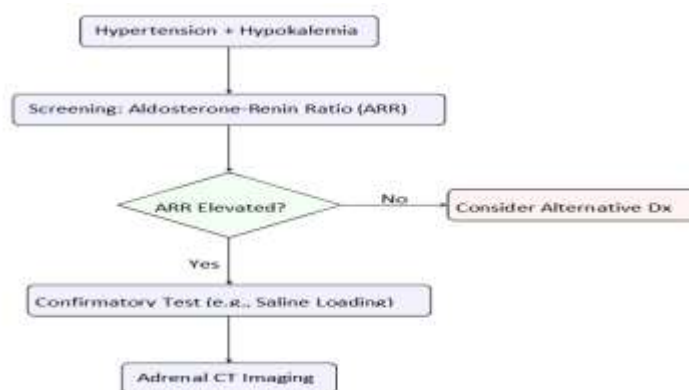


Figure 1: Standard algorithmic diagnostic workflow for suspected Primary Hyperaldosteronism.

The structure of this paper is organized to guide the reader through the complete clinical progression. Section 1 provides the pathophysiological and diagnostic introduction. Section 2 details the Case Presentation, outlining the patient's symptomatic history and initial clinical status. Section 3, Investigations, explores the laboratory and radiological findings, including the severe metabolic derangements. Section 4, Management and Outcome, discusses the preoperative medical stabilization and the definitive surgical intervention. Finally, Section 5 offers the Conclusion, summarizing the key clinical takeaways from this case.

2. Case Presentation

2.1 Demographics and Chief Complaints

A 37-year-old female, a homemaker residing in Maruvalam, was admitted to the Department of General Medicine presenting with a constellation of progressively worsening constitutional and cardiopulmonary symptoms. Her primary chief complaint was severe, unprovoked easy fatigability that had been insidious in onset but had steadily progressed over the preceding month. Accompanying this profound exhaustion was breathlessness upon exertion. According to the New York Heart Association (NYHA) functional classi-



Figure 2: Coronal view of an unenhanced CT abdomen demonstrating an adrenal mass. Such imaging is crucial for localizing functioning adenomas prior to surgical intervention.

fication [5], her dyspnea had escalated from Class II to Class III over a four-week period, significantly limiting her daily domestic activities.

Further clinical history revealed that her breathlessness was purely exertional; she explicitly denied any episodes of paroxysmal nocturnal dyspnea (PND) or orthopnea, making a primary decompensated left ventricular failure less likely. In addition to the fatigue and dyspnea, she reported recurrent episodes of giddiness over the same one-month duration. These episodes were non-rotatory, lacked any distinct postural variation, and were never associated with transient loss of consciousness, visual disturbances, or lateralizing neurological deficits. A comprehensive review of systems was otherwise unremarkable. She specifically denied any history of typical anginal chest pain, palpitations, bilateral pedal edema, reduced urine output, or infectious symptoms such as cough with expectoration and fever.

2.2 Medical, Obstetric, and Gynecological History

The patient's past medical history was highly notable for the premature onset of metabolic and cardiovascular derangements (summarized in Table 2). She was a known case of systemic hypertension, diagnosed 12 years prior when she was approximately 25 years old. The early onset of hypertension in a patient of this demographic is a classic red flag warranting a thorough investigation for secondary hypertensive disorders [6]. Her hypertension had been managed in the outpatient setting with a calcium channel blocker (Cilnidipine 10 mg once daily). Additionally, she had been diagnosed with Type 2 Diabetes Mellitus just two months prior to her current admission and was initiated on oral hypoglycemic therapy (Metformin 500 mg twice daily).

Table 2: Summary of Past Medical History and Admission Medications

Condition	Duration	Current Pharmacotherapy
Systemic Hypertension	12 Years (Onset at age 25)	Tab. Cilnidipine 10 mg OD
Type 2 Diabetes Mellitus	2 Months	Tab. Metformin 500 mg BD
Menorrhagia (AUB)	2 Months	None prior to admission

Her obstetric history consisted of two full-term normal vaginal deliveries (P2L2). Crucially, both pregnancies were complicated by severe gestational systemic hypertension, necessitating pharmacological intervention with Labetalol 100 mg twice daily during her gravid states. Her last childbirth occurred nine years ago, and postpartum screening for secondary hypertension had not been aggressively pursued at that time.

Her recent gynecological history provided a critical clue to her profound fatigability. For the past two months, her menstrual cycles had become increasingly abnormal. While she maintained a regular 30-day cycle, the duration of bleeding had prolonged to 7 to 10 days, accompanied by the passage of heavy clots and severe dysmenorrhea. Her last menstrual period (LMP) was recorded on August 7th. This abnormal uterine bleeding (AUB) was strongly suspected to be the primary driver of a secondary acute-on-chronic blood loss anemia, later confirmed by laboratory investigations.

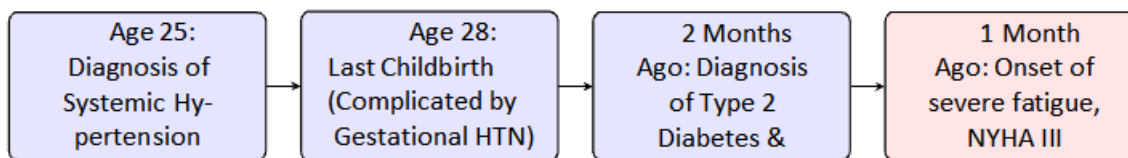


Figure 3: Timeline of the patient's major clinical milestones leading to the current acute presentation.

2.3 Physical Examination and Vital Signs

On general physical examination, the patient appeared fatigued but was conscious, alert, and oriented to time,

place, and person (Glasgow Coma Scale 15/15). She was afebrile and maintained a comfortable respiratory pattern at rest, without obvious signs of accessory muscle use. A striking physical finding was marked conjunctival pallor, directly corroborating her recent history of menorrhagia and symptoms of tissue hypoxia (exertional breathlessness). There was no clinical evidence of icterus, cyanosis, clubbing, generalized lymphadenopathy, or dependent pedal edema.

Hemodynamic assessment revealed a regular, high-volume pulse at a rate of 75 beats per minute. There were no specific bounding characters, nor was there any radio-radial or radio-femoral delay, clinically lowering the probability of aortic coarctation. However, her blood pressure was markedly elevated. To adhere to standard diagnostic protocols for young hypertensives [7], blood pressure was measured in the supine position across all four extremities to map any symmetrical discrepancies (Table 3).

Table 3: Four-Limb Blood Pressure Assessment upon Admission

Extremity	Systolic BP (mmHg)	Diastolic BP (mmHg)
Right Upper Limb	170	90
Left Upper Limb	170	90
Right Lower Limb	180	90
Left Lower Limb	180	90

The symmetrical elevation across all four limbs established the presence of uncontrolled, severe systemic hypertension without regional vascular compromise. Her respiratory rate was 15 breaths per minute with a normal thoracoabdominal pattern, and she maintained an oxygen saturation (SpO₂) of 98% on ambient room air. Systemic examination was largely unremarkable and failed to reveal signs of end-organ failure. Cardiovascular auscultation demonstrated normal first and second heart sounds (S₁, S₂) with no audible murmurs, gallops, or venous hums. The respiratory system yielded normal vesicular breath sounds bilaterally without any added crepitations or wheezes. The abdomen was soft and non-tender, with no palpable hepatosplenomegaly or auscultated renal bruits—a finding that tentatively pushed renal artery stenosis lower on the differential diagnosis list prior to sonographic evaluation. Neurological examination showed bilaterally reactive pupils (PERLA) with intact sensory and motor functions across all four limbs.

The culmination of these clinical findings—severe hypertension in a young female, coupled with symptomatic profound anemia secondary to abnormal uterine bleeding—necessitated prompt admission to the intensive care unit for stabilization, blood transfusion, and a rigorous diagnostic workup to unearth the etiology of her long-standing hypertension.

3. Investigations

The initial diagnostic strategy was tailored to address the acute cardiopulmonary symptoms while simultaneously evaluating the chronicity of the patient's hypertension and the etiology of her severe fatigability. The investigations yielded evidence of two intersecting pathologies: a severe hematological deficit driven by gynecological bleeding, and a profound metabolic derangement characteristic of autonomous endocrine hyperfunction.

3.1 Initial Laboratory Evaluation and Hematology

Upon admission, a Complete Blood Count (CBC) was performed, which immediately localized the cause of her severe pallor and exertional dyspnea. The patient exhibited profound anemia with a hemoglobin concentration of 6.2 g/dL. The red blood cell indices revealed a microcytic, hypochromic morphology (MCV 66 fL, MCH 20.1 pg), which was corroborated by the peripheral blood smear showing prominent anisocytosis. This profile was highly

consistent with chronic iron deficiency anemia secondary to the patient's reported abnormal uterine bleeding (AUB). Table 4 summarizes the hematological parameters at admission and following initial resuscitation

Table 4: Hematological Parameters Before and After Initial Transfusion

Parameter	Admission Value	Post-Transfusion Value	Reference Range
Hemoglobin (g/dL)	6.2	9.2	12.0 – 15.0
RBC Count ($\times 10^6/\mu\text{L}$)	3.10	–	4.0 – 5.0
WBC Count (cells/mm ³)	4600	–	4000 – 11000
Hematocrit (%)	20.6	–	36.0 – 46.0
MCV (fL)	66.0	–	80.0 – 100.0
Platelets ($\times 10^5/\mu\text{L}$)	3.21	2.74	1.5 – 4.0

The patient was promptly managed with the transfusion of one unit of Packed Red Blood Cells (PRBC) and intravenous Iron Sucrose (200 mg in 100 mL Normal Saline on alternate days), which successfully raised her hemoglobin to 9.2 g/dL.

3.2 Acid-Base and Electrolyte Derangements

While the anemia explained the acute fatigue, an Arterial Blood Gas (ABG) analysis uncovered a life-threatening metabolic crisis. The ABG demonstrated severe metabolic alkalosis (pH 7.67) with a compensatory retention of carbon dioxide (pCO₂ 50 mmHg)

and a markedly elevated bicarbonate level (HCO₃⁻ 57.7 mmol/L). Most alarming was the critical hypokalemia, with a serum potassium concentration plunging to 1.3 mEq/L, accompanied by borderline hyponatremia (Na⁺ 132 mEq/L) and hypocalcemia (ionized calcium 0.90 mmol/L).

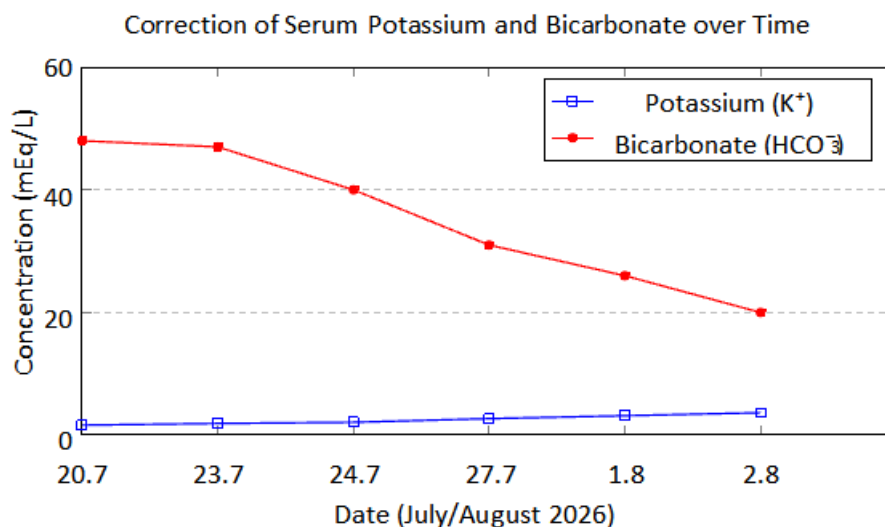


Figure 4: Trend showing the gradual correction of hypokalemia and metabolic alkalosis following aggressive medical management and mineralocorticoid receptor blockade.

Renal function tests showed a slight impairment with a serum creatinine of 1.31 mg/dL and a blood urea level of 33 mg/dL. Spot urine analysis revealed a protein-to-creatinine ratio (PCR) of 2.3, indicative of hypertensive

nephropathy. Concurrently, urinary electrolyte mapping showed a disproportionately high urinary potassium excretion (11.7 mEq/L) despite severe systemic depletion, confirming inappropriate renal potassium wasting [10].

Cardiovascular Assessment
An electrocardiogram (ECG) displayed a normal sinus rhythm with a heart rate of 75 bpm. Consistent with the profound hypokalemia, the ECG tracing exhibited characteristic T-wave flattening across the precordial leads (V1-V6) and ST-segment depression in leads V4 through V6 [11]. To evaluate the structural impact of her long-standing hypertension, a transthoracic echocardiogram (ECHO) was conducted. The imaging revealed concentric left ventricular hypertrophy (LVH) with a preserved ejection fraction of 65% and no regional wall motion abnormalities. The presence of LVH highlighted the chronicity and end-organ impact of her elevated systemic vascular resistance [9].

3.3 Endocrine and Renovascular Workup

The triad of early-onset hypertension, spontaneous profound hypokalemia, and metabolic alkalosis strongly necessitated an evaluation for secondary endocrine hypertension. A Renal Artery Doppler ultrasound was entirely normal, effectively ruling out renal artery stenosis. Consequently, a comprehensive endocrine panel was drawn (Table 5).

Table 5: Endocrine Evaluation for Secondary Hypertension

Hormonal Assay	Patient Result	Reference Range
Serum Aldosterone	12.6 ng/dL	1.8 – 16 (Supine)
Plasma Renin Activity	1.7 µIU/mL	2.8 – 39.9 (Supine)
Aldosterone-Renin Ratio (ARR)	7.4	< 3.7
Plasma Free Metanephrines	30 ng/L	7.90 – 88.70
Normetanephrines	100 ng/L	20.10 – 135.40
8 AM Serum Cortisol	10.9 µg/dL	5.0 – 25.0
Post 1mg DST Cortisol	6.24 µg/dL	Suppression normal

The hormonal evaluation excluded Pheochromocytoma (normal metanephrines) and Cushing's Syndrome (adequate, though borderline, suppression on the low-dose dexamethasone test). However, the screening test for Primary Hyperaldosteronism was starkly positive. Despite the baseline serum aldosterone falling within normal quantitative limits, the profound suppression of Plasma Renin Activity resulted in a highly elevated ARR of 7.4, indicating autonomous aldosterone secretion independent of renin control.

3.4 Radiological Localization

Following biochemical confirmation of aldosterone autonomy, cross-sectional imaging was mandated to localize the source. A multiphase contrast-enhanced computed tomography (CECT) scan of the abdomen and pelvis was performed. In addition to incidentally noting a bulky uterus with multiple fibroids (the source of the AUB) and chronic calculous cholecystitis, the CECT definitively visualized a well-defined right adrenal mass.

The lesion measured 20 × 18 mm and exhibited radiological features pathognomonic for a lipid-rich adrenal adenoma [8]. Specifically, the pre-contrast attenuation was 32 Hounsfield Units (HU). Following contrast administration, the mass demonstrated rapid enhancement to 106 HU, followed by swift washout on delayed imaging to 28 HU. Calculations revealed an absolute contrast washout greater than 60% and a relative washout greater than 40%, satisfying the stringent radiological criteria for a benign adrenal adenoma.

Table 6: Preoperative Pharmacological Regimen for Hemodynamic and Metabolic Stabilization

Medication	Dosage	Therapeutic Rationale
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Spironolactone	50 mg (1-0-0)	MR antagonism; corrects hypokalemia and
		BP.
Nifedipine (SR)	10 mg (1-0-1)	Calcium channel blockade for vasodilation.
Prazosin	5 mg (1-0-1)	Alpha-1 blockade; addresses resistant hyper-
		tension.
Clonidine	0.1 mg (1-0-0)	Central alpha-2 agonism; reduces sympa-
		thetic tone.
Telmisartan	40 mg (1-0-1)	ARB; blunts residual RAAS activity.
Metformin	500 mg (1-0-1)	Glycemic control for comorbid Type 2 DM.
Folic Acid	5 mg (0-0-1)	Nutritional support secondary to profound
		anemia.

4. Management and Outcome

The management of Primary Hyperaldosteronism (Conn Syndrome) secondary to a uni-lateral adrenal adenoma is broadly biphasic. The initial phase focuses on aggressive medical optimization to reverse the profound metabolic and hemodynamic derangements. This is followed by the definitive, curative phase: surgical extirpation of the hyperfunctioning gland. The transition between these phases requires meticulous physiological monitoring to prevent perioperative complications.

4.1 Preoperative Medical Optimization

Upon biochemical and radiological confirmation of the aldosterone-producing adenoma, the immediate therapeutic objective was to normalize the patient's severe hypokalemia and control her resistant systemic hypertension. The cornerstone of this stabilization is Mineralocorticoid Receptor (MR) blockade, which competitively inhibits the action of excess aldosterone at the distal renal tubules [2].

The patient was initiated on Spironolactone, a non-selective MR antagonist, at a dose of 50 mg once daily. Because her hypertension was severe and longstanding, monotherapy was insufficient. She required a robust, multidrug antihypertensive regimen tailored to blunt different physiological pathways of her neurohumoral axis [12]. Table 6 details the comprehensive pharmacological regimen employed to achieve preoperative hemodynamic stability.

Over the course of 14 days, this aggressive regimen successfully restored her serum potassium to a safe preoperative level (3.65 mEq/L) and resolved the metabolic alkalosis (bicarbonate normalized to 20 mEq/L), significantly reducing her perioperative arrhythmic risk.



Figure 5: Gross pathology specimen of the excised right adrenal gland. The bisected view reveals a distinct, golden-yellow, circumscribed lipid-rich adenoma (measuring approximately 20×18 mm) replacing normal adrenal cortical architecture.

4.2Surgical Intervention

Following medical optimization, the patient was scheduled for a unilateral right adrenalectomy. The surgical approach to right-sided adrenal masses requires precise anatomical navigation. Unlike the left adrenal vein, which drains into the left renal vein, the right adrenal vein is notoriously short (often just a few millimeters long) and drains directly into the posterolateral aspect of the Inferior Vena Cava (IVC) [13]. This anatomical configuration significantly elevates the risk of intraoperative hemorrhage and inadvertent IVC injury during dissection and ligation.

The surgical team successfully performed the right adrenalectomy without vascular compromise. The excised mass (Figure 5) was sent for histopathological examination. Gross inspection revealed an encapsulated, golden-yellow circumscribed nodule embedded within the adrenal cortex, a macroscopic appearance highly characteristic of a lipid-rich adenoma [14].

4.3Postoperative Care and Anticipated Complications

The immediate postoperative period is a critical phase of physiological transition. The contralateral (left) adrenal gland undergoes chronic suppression due to prolonged exposure to high systemic aldosterone and depressed renin levels. Consequently, following the removal of the adenoma, patients are at a high risk of developing transient hypoadosteronism [15].

If unrecognized, this can precipitate hyperkalemia, hyponatremia, profound dehydration, and severe hypotension. Because of this phenomenon, all preoperative antihypertensive medications—especially potassium-sparing diuretics like Spironolactone—must be discontinued immediately post-surgery. A strict postoperative monitoring algorithm (Figure 6) was implemented in the Intensive Care Unit.

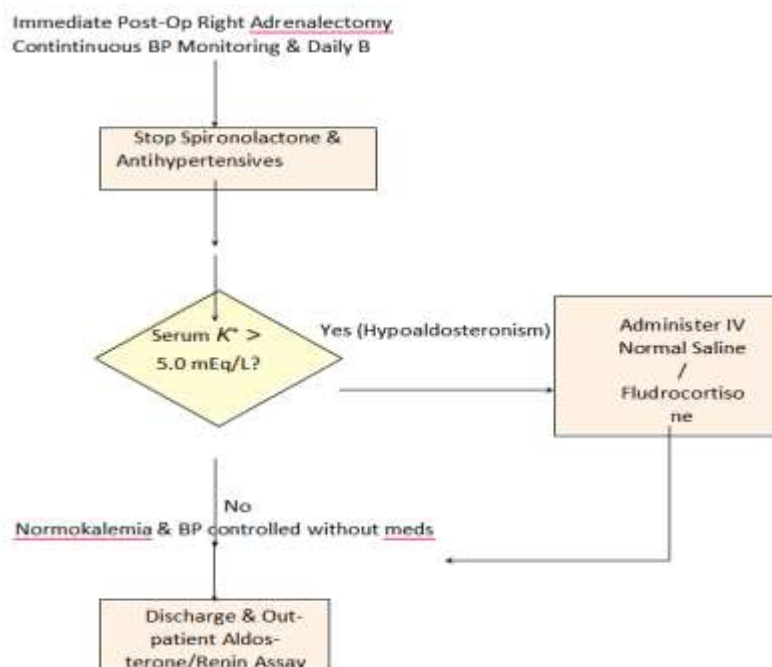


Figure 6: Postoperative clinical pathway and monitoring algorithm to mitigate the risks of transient hypoaldosteronism and hemodynamic instability.

The patient's postoperative course was relatively uneventful. Strict monitoring of her basic metabolic panel (BMP) and renal function tests (RFT) showed no evidence of acute kidney injury. Her blood pressure normalized rapidly, eliminating the need to restart her antihypertensive regimen. On postoperative day 5, her serum sodium (138 mEq/L) and potassium (4.2 mEq/L) were entirely within normal limits without supplementation. Plasma aldosterone and renin levels were drawn prior to discharge to establish a new postoperative baseline, confirming the resolution of the biochemical autonomy. She was discharged in stable condition with scheduled follow-up to monitor her long-term cardiovascular remodeling and glycemic control.

5. Conclusion

Primary hyperaldosteronism is no longer considered a rare anomaly in the landscape of systemic hypertension; rather, it is a prevalent and critically underdiagnosed pathology. This case of a 37-year-old female vividly illustrates the classical presentation of Conn Syndrome—marked by the triad of young-onset hypertension, profound hypokalemia, and metabolic alkalosis. It also highlights the clinical masking effect of concurrent co-morbidities, such as her severe anemia secondary to abnormal uterine bleeding, which initially obscured the endocrine etiology of her profound fatigability [16].

The prompt recognition of her severe metabolic derangements prevented potentially fatal arrhythmic events and triggered the appropriate diagnostic cascade. The utilization of the Aldosterone-Renin Ratio (ARR) proved

invaluable, bypassing the misleadingly “normal” absolute aldosterone levels to reveal the underlying autonomous hormone secretion. Subsequent high-resolution imaging successfully localized the lipid-rich right adrenal adenoma, paving the way for targeted therapy [17].

Surgical intervention remains the definitive gold standard for unilateral aldosterone-producing adenomas. As demonstrated in this patient’s clinical course, a successful right adrenalectomy not only eliminated the need for lifelong, high-dose antihypertensive polypharmacy but also rapidly reversed her metabolic alkalosis and hypokalemia. The careful preoperative optimization with mineralocorticoid receptor antagonists and strict postoperative vigilance for transient hypoaldosteronism were critical components of her complication-free recovery [18].

Table 7 summarizes the core clinical takeaways from this case study. Ultimately, this presentation underscores a fundamental tenet of internal medicine: clinicians must maintain a high index of suspicion for secondary endocrine causes when evaluating young patients with treatment-resistant hypertension. Early diagnosis and surgical intervention effectively abrogate the accelerated cardiovascular and renal risks associated with chronic aldosterone toxicity, offering a definitive cure rather than mere symptom management.

Table 7: Key Clinical Pearls in the Management of Conn Syndrome

Clinical Domain	Core Takeaway
Screening & Suspicion	Always calculate the <u>Aldosterone-Renin Ratio (ARR)</u> in young hypertensives (< 40 years), especially those with spontaneous <u>hypokalemia</u> .
Preoperative Care	Aggressive potassium correction and <u>mineralocorticoid</u> receptor blockade (e.g., <u>Spironolactone</u>) are mandatory to stabilize the patient.
Surgical Anatomy	Unilateral right <u>adrenalectomy</u> requires meticulous technique due to the short right adrenal vein draining <u>directly</u> into the IVC.
Postoperative Vigilance	Monitor strictly for transient <u>hypoaldosteronism</u> (hyperkalemia, <u>hypotension</u>) due to chronic suppression of the <u>contralateral</u> adrenal gland.

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