American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e952551
DOI: 10.12659/AJCR.952551

Received: 2025.12.23
Accepted: 2026.06.18
Available online: 2026.09.02
Published: 2026.XX.XX

Hyponatremic–Hypertensive Syndrome as a Rare Manifestation of Renovascular Hypertension: A Case Series

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Data Interpretation D
Manuscript Preparation E
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Financial support: None declared
Conflict of interest: None declared

Case series
Patients: Female, 4-year-old • Female, 9-year-old
Final Diagnosis: Hyponatremic-hypertensive syndrome
Symptoms: Arterial hypertension • polydipsia • polyuria
Clinical Procedure: —
Specialty: Nephrology
Objective: Unusual clinical course

Background: Hyponatremic–hypertensive syndrome is a rare but distinctive manifestation of renal ischemia caused by unilateral renal artery stenosis. It is characterized by severe arterial hypertension accompanied by hyponatremia, hypokalemia, metabolic alkalosis, and polyuria, resulting from complex interactions between the ischemic kidney and the contralateral functioning kidney. Owing to its rarity and potentially life-threatening course, early recognition and appropriate management are crucial. The purpose of this report is to highlight the clinical presentation, diagnostic challenges, and therapeutic outcomes in pediatric patients.

Case Reports: We describe the cases of 2 girls, aged 4 and 9 years, who presented with severe hypertension and electrolyte disturbances secondary to unilateral right renal artery stenosis. Both patients exhibited typical biochemical features, including hyponatremia, hypokalemia, and elevated renin-aldosterone activity. Imaging studies confirmed significant unilateral renal artery stenosis due to fibromuscular dysplasia, with renal asymmetry. In the younger patient, progressive clinical deterioration and complete renal artery occlusion necessitated nephrectomy, resulting in resolution of hypertension and electrolyte abnormalities. In the older patient, percutaneous transluminal renal angioplasty was successful, leading to normalization of blood pressure, restoration of renal size symmetry, and sustained clinical remission at 1-year follow-up.

Conclusions: Hyponatremic–hypertensive syndrome should be considered in any child with severe hypertension accompanied by electrolyte disturbances, polyuria, and polydipsia. These 2 cases of girls with fibromuscular dysplasia underscore the importance of early recognition and individualized management strategies in pediatric renovascular hypertension. Prompt diagnosis and definitive treatment of renal artery stenosis, either by revascularization or nephrectomy when appropriate, can be curative and prevent severe morbidity. It is important to note that the course of HHS can vary among patients depending on their age, the severity, and the location of the stenosis.

Keywords: Fibromuscular Dysplasia • Hypertension • Hyponatremia • Renal Artery Obstruction • Renin-Angiotensin System
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952551

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Introduction

Hyponatremic–hypertensive syndrome (HHS) is an uncommon but highly characteristic complication of renovascular disease in children, particularly when the underlying cause is unilateral renal artery stenosis due to fibromuscular dysplasia (FMD). Renovascular hypertension is responsible for 5% to 10% of all cases of pediatric hypertension, with FMD being the most common cause of renal artery stenosis in children [1-3]. HHS is rare, without good epidemiological data, but it is probably underdiagnosed and underreported. HHS manifests as the combination of severe hypertension, hyponatremia, hypokalemia, metabolic alkalosis, and polyuria. It involves a physiologically complex interplay between the ischemic kidney and the contralateral, normally-functioning kidney. Hyponatremia in the HHS is developed in the course of 2 mechanisms: most importantly, as a result of pressure-induced natriuresis in the non-stenotic kidney; and, additionally, as a result of ADH increase (Figure 1) [4-6].

The pathophysiological mechanism of HHS is rooted in unilateral activation of the RAAS. Reduced perfusion in the stenotic kidney leads to excessive renin secretion, increasing systemic angiotensin II and aldosterone levels. Angiotensin II causes systemic vasoconstriction and worsens hypertension. Additionally, angiotensin II induces efferent arteriolar constriction in the unaffected kidney, increasing intraglomerular pressure and driving natriuresis, and, in some cases, proteinuria. Simultaneously, aldosterone promotes renal loss of potassium and hydrogen ions, resulting in hypokalemia and metabolic alkalosis. The contralateral kidney becomes a site of marked sodium and water excretion. This results in extracellular volume depletion, which stimulates antidiuretic hormone (ADH) release, leading to dilutional hyponatremia despite ongoing sodium loss in the urine [4,6-9].

Case Reports

Patient 1

A 4-year-old girl presented to the Pediatric Cardiology Department due to an unspecified murmur recognized in auscultation of the heart by a family physician. The patient was born as a second child, weighing 3650 grams, and had an Apgar score of 10. Her development was normal. The family history was notable for metabolic syndrome in the father, long QT syndrome in a 9-year-old brother, and a heart murmur in a 1-year-old brother. No cases of arterial hypertension have been reported among immediate family members. The girl was admitted in good general condition. Echocardiographic examination revealed left ventricular hypertrophy with contractility at the lower limit of normal and a normal aortic arch. Chest radiography demonstrated a mild cardiomegaly (cardiothoracic ratio 0.56). Laboratory tests, including complete blood count, inflammatory markers, serum electrolyte levels (sodium 140 mmol/L, potassium 3.9 mmol/L), general urine test (specific gravity 1010), and creatinine (eGFR 138 mL/min/1.73 m^2 using the Schwartz formula [10]), were within normal limits, while the NT-proBNP concentration was elevated at 320 pg/mL (reference age-appropriate normal range: < 125 pg/mL). Screening for metabolic and adrenal disorders

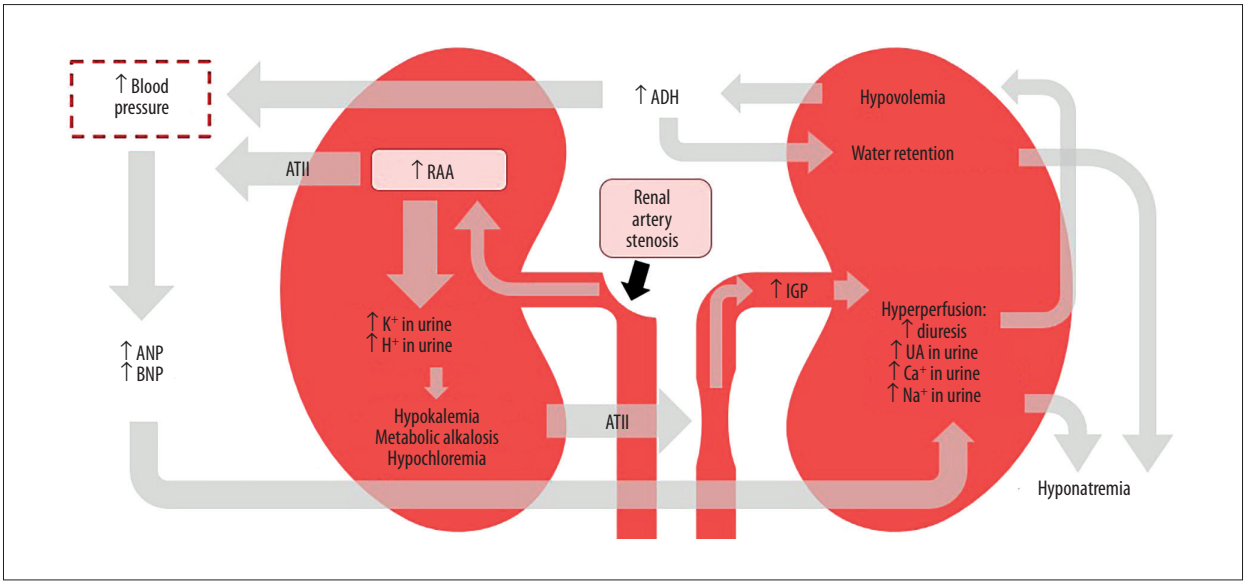

Figure 1. Pathophysiological mechanism of hyponatremic–hypertensive syndrome (HHS). ADH, antidiuretic hormone/vasopressin; AT II, angiotensin II; ANP, atrial natriuretic peptide; BNP, brain natriuretic peptide; IGP, intraglomerular pressure; RAA, renin-angiotensin-aldosterone system.

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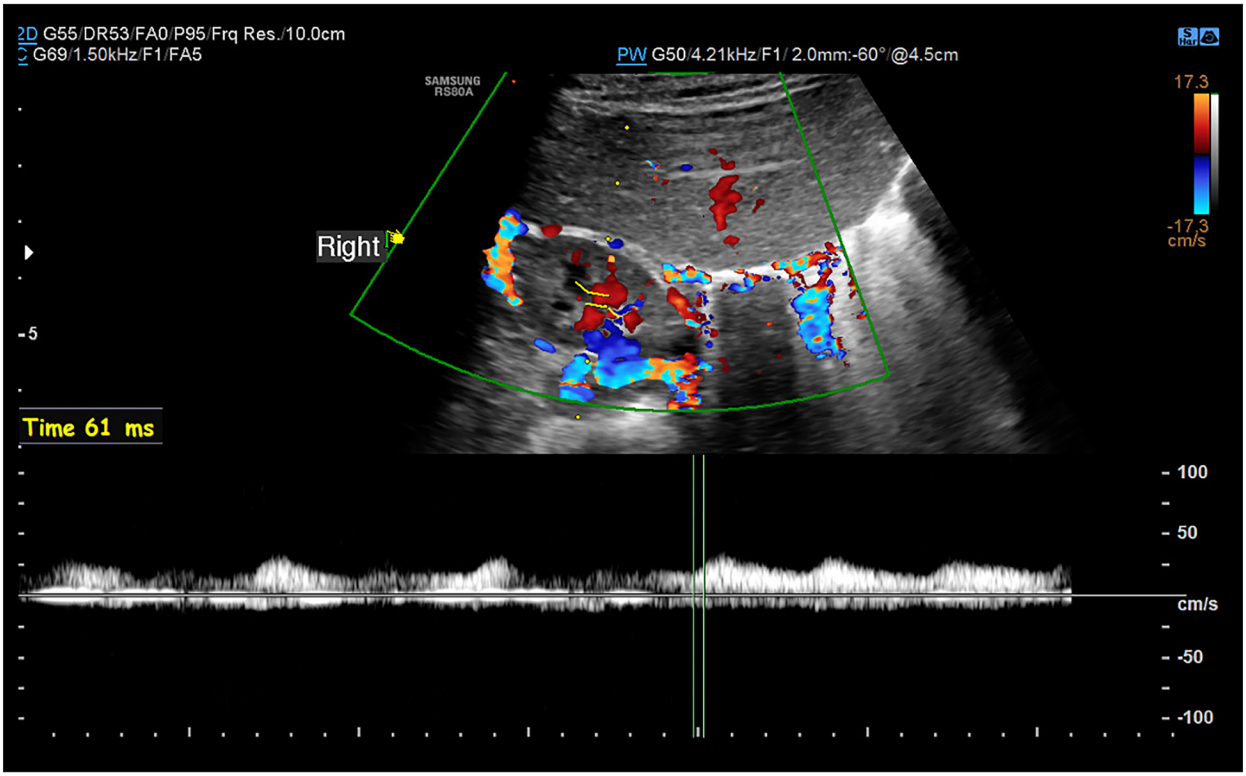

Figure 2. Doppler ultrasound of the renal arteries in a 4-year-old girl. The right renal artery was significantly narrower than the left (1.6 mm vs 2.6 mm). Doppler waveforms in the right segmental arteries demonstrated a tardus–parvus pattern, with prolonged acceleration time compared with the left kidney (61 ms vs 33 ms). The resistive index was lower on the right (0.31 vs 0.61), while peak systolic velocity was 32 cm/s on the right and 25 cm/s on the left.

(a 24-hour serum cortisol profile with adrenocorticotropic hormone (ACTH) measurement and plasma metoxycatecholamine assessment) showed no abnormalities.

During hospitalization, repeated measurements revealed substantially elevated arterial blood pressure values up to 200/140 mm Hg (in both office and bedside measurements) performed according to established guidelines [11] using a validated oscillometric device (Omron HBP-1320, Omron Corporation, Kyoto, Japan) and confirmed with an auscultatory method (Heine Gamma g7 sphygmomanometer, HEINE Optotechnik GmbH & Co. KG, Gilching, Germany). Additional laboratory evaluation demonstrated significantly increased plasma renin and aldosterone levels, with urinary albumin excretion within normal range. Renal ultrasound showed a smaller right kidney (62 mm in length) compared to the left (76 mm in length). The right renal artery measured 1.6 mm at its origin, compared with 2.6 mm on the left, and was diffusely narrowed along its entire course. Doppler ultrasound revealed a markedly prolonged acceleration time (AT) in the right renal artery—approximately twice that observed on the left—consistent with hemodynamically significant stenosis (**Figure 2**). Mild arteriolar narrowing was also recognized in an ophthalmological examination. Doppler ultrasound of the cervical arteries showed normal flow patterns. The abdominal aorta, visceral, and iliac arteries were without stenoses.

Computed tomographic angiography showed weaker contrast enhancement of the right kidney compared with the left, indicating impaired perfusion. A single right renal artery exhibited a significant stenosis (< 1 mm) before its hilar bifurcation, with post-stenotic dilatation of the distal branches and numerous collateral vessels around the right kidney. The left kidney and renal artery appeared normal. A hypotensive pharmacological therapy with amlodipine was initiated (on the 5th day of hospitalization), leading to a reduction in arterial blood pressure, although normal levels were not maintained. Renal scintigraphy (10th day of hospitalization) was also performed, confirming disproportionate renal function between the 2 kidneys.

The patient was transferred to the Nephrology Department. Repeated dynamic renal scintigraphy with technetium-99m-ethylenedicysteine (99mTc-EC) (17th day) demonstrated a considerably lower contribution of the right kidney to total renal function compared with the previous examination. The deterioration of right renal function was considered related to decreased perfusion via collateral circulation secondary to reduced systemic blood pressure. Imaging results were discussed

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Table 1. Relevant clinical parameters.

	Normal values	Patient 1		Patient 2	
		Pre-renal vascularization	Post-renal vascularization	Pre-renal vascularization	Post-renal vascularization
Serum sodium [mmol/L]	135-145	115	134	129	139
Serum potassium [mmol/L]	3.5-5.1	3.1	4.1	3.1	4.6
Urine protein [mg/dl]	< 30	175	Negative	87	Negative
Albumin-creatinine ratio (ACR) [mg/g]	< 30	1276.3	14.3	295.8	22.4
Fractional sodium excretion (FENa%)	*	1.4%	Not taken	1.3%	Not taken
pH[#]	7.35-7.45	7.577	7.433	7.509	7.406
$PaCO_2$ [mm Hg][#]	35-45	38	40	36	39
Blood bicarbonate [mEq/l][#]	19-24	30.4	25.1	28.30	24
Base excess [mEq/L][#]	-3 to 2	5.9	0.9	5.3	-0.3
Lactate [mmol/L]	0.5-1.6	1.3	1.1	1.5	1.0
Serum renin [mU/L]	4.4-46.1	276	69.73	1635	38.5
Serum aldosterone [ng/dL]	2.21-35.3	37.4	26.4	213	16.2

* In hyponatremic patients: < 1%: hypovolemia (dehydration) with preserved renal sodium retention capacity (eg, syndrome of inappropriate antidiuresis), > 1%: renal losses of sodium (eg, renal or cerebral salt wasting, diuretic use); [#] capillary blood acid-base balance; [1] the results taken 18 months after the revascularization.

with interventional radiologists, who concluded that fibromuscular dysplasia (FMD) was the most probable cause of the renal artery stenosis. Subsequently, amlodipine was replaced with metoprolol (18th day), which was well tolerated; however, intermittent elevations in arterial blood pressure persisted.

During hospitalization in the Nephrology Department, from the 20th day of hospitalization, she experienced multiple episodes of vomiting accompanied by increased thirst and polyuria of up to 5 liters per day (approximately 360 mL/kg/24h). Subsequent laboratory tests (taken on the 23rd day) revealed severe hyponatremia, hypochloremia, hypokalemia, and metabolic alkalosis, accompanied by high fractional sodium excretion, at 1.4% (**Table 1**). Serum osmolality was 235 mOsm/kg, and urine osmolality was 161 mOsm/kg. The overall clinical picture—critical renal artery stenosis accompanied by significant electrolyte disturbances—fulfilled the diagnostic criteria for hyponatremic–hypertensive syndrome (HHS). Intensive correction of electrolyte imbalances was initiated, resulting in temporary improvement of serum ion concentrations. Further ultrasound examination (24th day) showed an enlarged left kidney (87 mm in length) with a slightly heterogeneous, and blurred parenchymal structure. Plasma renin and aldosterone concentrations remained markedly elevated. Despite improvement in general condition, she continued to demonstrate electrolyte instability and intermittent episodes of severe hypertension.

Given the life-threatening symptoms, an urgent arteriography and percutaneous transluminal renal angioplasty (PTRA) were conducted on the 25th day (**Figure 3**). During an attempted angioplasty of the right renal artery, a complete occlusion of the artery was observed. After the procedure, the patient continued to experience significant electrolyte disturbances and severe hypertension requiring intravenous labetalol. Subsequently, a right nephrectomy was performed on the 27th day.

Following surgery, electrolyte disturbances gradually resolved, blood pressure slowly decreased, and the surgical wound healed appropriately. Follow-up laboratory studies demonstrated normal renal function, serum electrolytes (sodium and potassium normalized on the 29th day of hospitalization), creatinine, and urine analysis. Renal ultrasound (30th day) continued to show a heterogeneous left kidney (87 mm in length). Intravenous labetalol was discontinued (30th day), and metoprolol therapy

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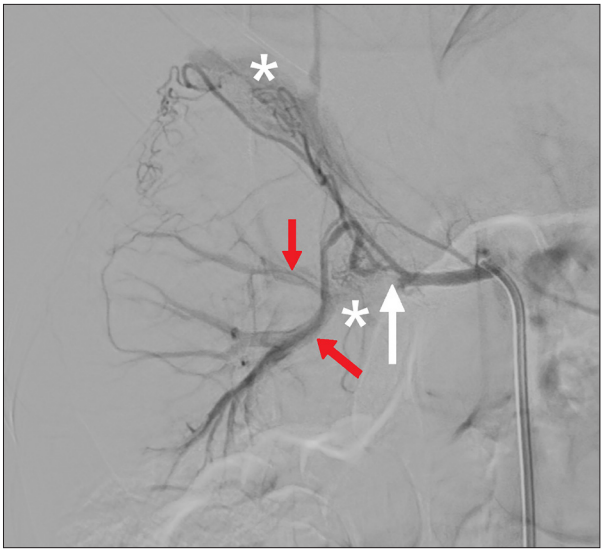

Figure 3. Angiography of the right renal artery of the 4-year-old girl. The right kidney is supplied by a single renal artery. At its division at the renal hilum, the renal artery exhibits a thread-like stenosis of > 95% on a distance of approximately 2 mm (white arrow), with post-stenotic dilatation of the distal branches (red arrows). Numerous collateral vessels are visible around the right kidney (*).

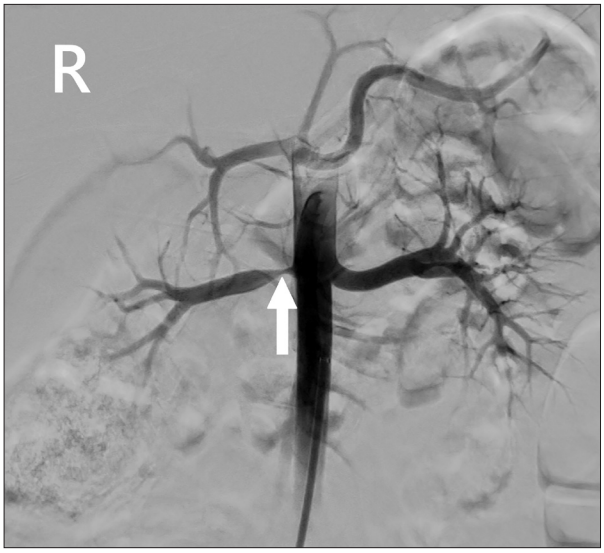

Figure 4. Arteriography of the renal arteries of the 9-year-old girl. The right renal artery shows significant narrowing near its origin from the aorta (arrow), measuring approximately 1.5 mm in diameter over a segment of about 6 mm, followed by a segment of normal caliber. The left renal artery shows no evidence of stenosis. Single renal arteries supply both kidneys.

was reinitiated due to intermittent elevations in blood pressure at a dose of 4 mg once daily (0.3 mg/kg/day). Repeated echocardiography (35th day) demonstrated persistent left ventricular hypertrophy with notable improvement in contractility. She was discharged on the 36th day of hospitalization in good general condition with appropriate follow-up recommendations.

The patient is currently 7 years old and continues to be followed in the outpatient nephrology clinic. She is not receiving any medication (metoprolol was withdrawn at a control outpatient visit 1 month after the hospital discharge) and has remained asymptomatic since hospitalization. Blood pressure values are consistently within the normal range, both at home and during follow-up visits.

Patient 2

A previously healthy 9-year-old girl was admitted to the Pediatric Department for evaluation of acute abdominal pain. Her symptoms began approximately 10 days before admission and included polydipsia, polyuria, nocturia, poor appetite, progressive general weakness, decreased muscle strength, and unintentional weight loss. The family history was negative for kidney disease, endocrine disorders, and arterial hypertension. Outpatient laboratory results showed proteinuria and slight, transient hematuria, while an outpatient abdominal ultrasound showed no abnormalities, with both kidneys appearing normal. Upon admission, she was in good general condition. Initial laboratory tests revealed hypokalemia and hyponatremia, proteinuria, and marked hematuria (dysmorphic erythrocytes), erythrocyturia (**Table 1**), and fractional sodium excretion 1.3%. Serum creatinine was within normal limits (eGFR 101 mL/min/1.73 m^2 using the Schwartz formula [10]).

Blood pressure measurements showed persistently significantly elevated values (average 150/110 mm Hg) with maximum values of 166/124 mm Hg (in both office and bedside measurements) using the above-mentioned methods and equipment. Abdominal ultrasound remained unremarkable. Given the persistent hypertension and abnormal laboratory results, she was transferred to the Pediatric Nephrology Department for specialized care.

On admission, laboratory tests revealed a low serum potassium level despite supplementation and proteinuria. The 24-hour serum cortisol profile with ACTH measurement and plasma metoxycatecholamine were within normal limits. The abdominal ultrasound revealed significant renal asymmetry, with the right kidney notably smaller (65 mm) than the left (92 mm). Doppler ultrasound demonstrated an abnormal flow spectrum with reduced resistance in the intrarenal arteries of the right kidney. Renal scintigraphy confirmed a smaller right kidney with functional deficits, including impaired perfusion and slowed, preserved excretion. Both imaging modalities yielded results were consistent with significant stenosis of the right

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renal artery. CT angiography showed severe, focal stenosis of the right renal artery. The right renal artery was significantly narrowed to approximately 1.5 mm over a 6-mm length, beginning just adjacent to the aorta. The left renal artery was normal, and each kidney was supplied with a single artery. A comprehensive survey of the remaining systemic vasculature (aorta, iliac, celiac, carotids, vertebrals, and subclavians) and a CT of the head were unremarkable for any signs of stenosis or other vascular changes. Ophthalmological examination revealed vascular changes consistent with systemic hypertension; specifically, marked arterial stenosis and increased tortuosity. The echocardiogram showed no signs of left ventricular hypertrophy.

Initial medical management involved a 3-drug antihypertensive regimen (amlodipine, doxazosin, and metoprolol) along with oral potassium supplementation, which successfully controlled the blood pressure and potassium level.

Arteriography with 3-stage angioplasty of the right renal artery was performed on the 9th day of the hospitalization using 2 × 20 mm, 3 × 20 mm, and 4 × 20 mm balloons, resulting in good morphological and hemodynamic dilation of the stenosis (**Figure 4**). The patient's blood pressure normalized, allowing the antihypertensive medications to be gradually and successfully discontinued (15th day of the hospitalization). Prophylaxis included low-molecular-weight heparin (during the procedure) and acetylsalicylic acid (for 6 months after the procedure).

Upon discharge (16th day), the patient demonstrated complete resolution of key laboratory abnormalities (**Table 1**) and excellent patency of the treated vessel (as assessed by Doppler ultrasound, with no signs of restenosis in the right renal artery).

The success of the right renal artery angioplasty was confirmed at 18-month follow-up. She remains normotensive without the need for antihypertensive medication. Her blood pressure is normal in office (110/64 mm Hg), home (95-110/55-65 mm Hg), and ambulatory blood pressure measurements (24 hour – 110/68, day – 120/74, night – 102/60 mm Hg). Renal ultrasound demonstrates normalization of renal size (right kidney 88 mm, left kidney 92 mm), resolving the prior asymmetry. Renal artery flow parameters are within normal limits, showing no evidence of restenosis or elevated velocities. Renal function, serum electrolytes, and urinalysis findings, and renin and aldosterone levels are within normal limits.

Discussion

The development of renal artery stenosis (RAS) in children is based on different pathophysiological mechanisms than in the adult population. In contrast to adults, in whom the leading cause of RAS is atherosclerosis, the etiologies in the pediatric population are primarily associated with various diseases, such as fibromuscular dysplasia, Takayasu arteritis, or syndromic presentations such as neurofibromatosis type 1 or Williams syndrome [1,12,13]. Regardless of the cause of the stenosis, this condition can lead to the development of HHS.

In our opinion, both girls had renal artery stenosis associated with FMD. FMD is a condition characterized by non-inflammatory, non-atherosclerotic narrowing of the arteries, including the renal, visceral, and central nervous system arteries. The disease can occur at any age; it most commonly affects young women, but it also can present in children as young as 1 year old. Diagnosis, in accordance with the 2019 consensus, is based on a thorough medical history, physical examination, laboratory tests, and imaging studies [14]. In both girls, we ruled out an inflammatory background (medical history, physical examination, normal inflammatory markers); the clinical examination did not indicate syndromic renal artery stenosis (as seen in genetically determined syndromes such as neurofibromatosis type 1 or Williams syndrome). Furthermore, extensive vascular studies ruled out involvement of other vascular beds and the aorta (aortic involvement is atypical for FMD) [1,14]. Additionally, the first patient underwent genetic testing (next-generation sequencing panel) to investigate causes of renal artery stenosis and elastopathy—the result was negative.

Clinically, children with HHS can present with polyuria, polydipsia, dehydration, vomiting, irritability, headaches, blurred vision, or seizures. Laboratory evaluation typically demonstrates low serum sodium, low potassium, elevated renin and aldosterone, and metabolic alkalosis [7,9,15,16]. While renin and aldosterone levels are generally elevated, normal values do not exclude renovascular disease, especially in bilateral stenosis or in cases where chronic hypertension has altered intrarenal hemodynamics.

Fractional sodium excretion can distinguish between hyponatremia with preserved sodium retention (eg, syndrome of inappropriate antidiuresis) and syndromes involving renal sodium loss. In both girls, fractional sodium excretion was > 1.0%, indicating renal loss [17]. Furthermore, they were not receiving diuretics, and their adrenal function tests were normal. Therefore, it must be concluded that in both patients, hyponatremia resulted from renal loss, with causes other than HHS ruled out. Moreover, normonatremia returned after the arterial stenosis was removed. Unfortunately, we did not subsequently assess urinary sodium excretion in either patient.

Ultrasound is a valuable tool for both general renal assessment and longitudinal monitoring. It allows evaluation of renal size and parenchymal morphology, which can reveal alterations indicative of structural changes in both the affected

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and contralateral kidneys due to hyperfiltration during periods of marked hyperreninemia. These changes can include kidney enlargement, variations in parenchymal thickness, alterations in echogenicity, and loss of corticomedullary differentiation [4]. In addition, color Doppler ultrasound facilitates evaluation of stenosis in the main renal arteries and intrarenal arterial branches [1]. Ultrasound is a dynamic and operator-dependent examination; therefore, its accuracy relies heavily on the examiner's experience. Assessments performed by less experienced operators may be prone to inconsistencies and errors, potentially impacting the diagnostic process and patient safety. Consequently, ultrasound findings obtained by inexperienced examiners should be verified by a more experienced practitioner. Diagnosis relies on a combination of clinical evaluation, laboratory assessment, and imaging. CT angiography and MR angiography provide better characterization of the renal vascular structure, but digital subtraction angiography (DSA) offers the highest spatial resolution and enables simultaneous therapeutic intervention [12,13,18].

In this patient population, the preferred pharmacological agents include calcium channel blockers, alpha-blockers, beta-blockers, direct vasodilators (eg, hydralazine), and centrally acting drugs. RAAS-blocking agents, such as ACE inhibitors and angiotensin receptor blockers, are effective, but must always be used with caution, as they can impair the function of the kidney supplied by a stenotic artery. Obviously, they are contraindicated in bilateral renal artery stenosis or stenosis of the renal artery of a solitary kidney. Diuretics can be used in renovascular hypertension but are strictly contraindicated in HHS. Furthermore, fluid resuscitation and the repletion of sodium and potassium deficits—pathognomonic features of this syndrome—are essential components of management [1].

Once blood pressure is initially controlled, either percutaneous transluminal angioplasty (PTA) or nephrectomy may be performed [4,19,20]. While some reports have speculated that HHS occurs when the stenosis of the affected artery is so severe that revascularization by PTA is impossible, this was not confirmed in one of the presented patients. Nephrectomy has been suggested as an option when an angioplasty procedure is ineffective or when the affected kidney contributes little to global renal function (< 10%) [8]. Furthermore, other sources recommend nephrectomy in patients with complete renal artery occlusion resulting in a non-functioning kidney [4].

The definitive treatment depends on the patient's age, technical considerations (type of stenosis), the capabilities of the medical center, and the team's experience. It is absolutely essential to remove the stenosis using one method or another. The younger the child, the more technically challenging the revascularization procedure becomes. In the youngest patients, nephrectomy is often the only option. In older patients (eg, 10- and 17-year-olds), a PTA should always be attempted. The general rule in children, especially in cases of FMD, is not to place stents. In cases of stenosis not of the main trunk but of the renal artery branches (common in FMD, though rarely the cause of HHS), one may attempt to occlude a segment of the artery, inducing ischemia in a small portion of the renal parenchyma [21].

The different outcomes in both of the presented cases requires discussion. In the first girl, in angiography, the renal artery had a very tight stenosis and was placed near the hilar bifurcation with extensive collaterals. The pharmacological treatment in the first patient probably led to the "treatment paradox" – lowering blood pressure with amlodipine completely prevented blood flow through the renal artery, resulting in severe hypoperfusion and ischemia, with consequent renin generation and massive hyperfiltration in the second kidney. Patient 1 presented with much more severe ion disturbances and severe polyuria that was virtually impossible to compensate for. The severity of this case is further highlighted by lesions observed in the contralateral kidney, which exhibited marked hyperfiltration. On the contrary, in Patient 2, the renal artery stenosis was located just where the renal artery branches off from the aorta and was not very tight. Therefore, ion disturbances, polyuria, and a contralateral kidney lesion were much less pronounced than in Patient 2.

Of course, this is merely a hypothesis; other factors influencing the varying clinical course in girls cannot be ruled out. Age certainly plays a role—HHS is more common in young children and boys, and is less common among adults—as do other unidentified genetic predispositions (eg, perhaps, polymorphisms in genes encoding components of the RAA system) and the timing of stenosis development (unknown in both cases) [4]. Finally, it should be remembered that not every patient, even those with critical renal artery stenosis, develops this syndrome; therefore, other factors not yet identified may contribute to its occurrence and severity. Further research is needed on the epidemiology, risk factors, and differences in clinical course and prognosis among patients with this complication of renal artery stenosis.

Conclusions

Hyponatremic–hypertensive syndrome should be considered in any child with severe hypertension accompanied by electrolyte disturbances, polyuria, and polydipsia. These 2 cases of girls with fibromuscular dysplasia underscore the importance of early recognition and individualized management strategies in pediatric renovascular hypertension. Prompt diagnosis and definitive treatment of renal artery stenosis, either by revascularization or nephrectomy when appropriate, can be curative

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and prevent severe morbidity. It is important to note that the course of HHS can vary among patients depending on their age, the severity, and the location of the stenosis.

Department and Institution Where Work Was Done

University Clinical Center of the Medical University of Warsaw, Warsaw, Poland.

Consent

The patients gave written consent to be included in the case series.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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