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Resistant Hypertension Secondary to Renal Arteriovenous Malformation: A Case of Cure After Nephrectomy

Authors' Contribution:
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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 33-year-old
Final Diagnosis: Renal arterio-venous malformation
Symptoms: Hypertension
Clinical Procedure: —
Specialty: Urology

Objective: Rare coexistence of disease or pathology

Background: Evaluation of multidrug-resistant hypertension in young adults requires consideration of uncommon secondary etiologies, including renovascular disease and congenital renal vascular anomalies. Renal arteriovenous malformations (rAVMs) represent a rare cause of secondary hypertension, accounting for less than 0.1% of renal vascular abnormalities. They most commonly present with hematuria, flank pain, or, in high-flow lesions, high output cardiac failure. Hypertension as the principal manifestation is uncommon.

Case Report: We describe a 33-year-old woman with longstanding uncontrolled hypertension requiring 5 antihypertensive agents who presented for evaluation of refractory blood pressure control. Initial duplex ultrasound and angiography identified a large complex left renal lesion associated with dysmorphic parenchyma and aberrant arterial and venous architecture without a history of trauma, intervention, or prior renal surgery, consistent with a congenital vascular malformation. Diagnostic computed tomography angiography demonstrated a 6.5 × 6.5 × 5.5 cm arteriovenous malformation. Given the lesion's size and vascular complexity, endovascular embolization was deemed not technically feasible. The patient subsequently underwent elective simple nephrectomy without intraoperative complication. Histopathology confirmed a congenital high-flow rAVM with extensive parenchymal distortion. Postoperatively, the patient exhibited marked improvement in blood pressure control, eliminating the need for antihypertensive medication.

Conclusions: This case illustrates an uncommon presentation of severe drug-resistant hypertension in a young patient likely attributed to a large congenital rAVM. Congenital renal vascular malformations should be considered in the differential diagnosis of refractory hypertension. Surgical intervention remains an important therapeutic option when endovascular treatment is contraindicated or considered unlikely to preserve functional renal parenchyma.

Keywords: Congenital Abnormalities • Hypertension, Renal • Hypertension, Renovascular • Nephrectomy

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953384

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Introduction

Renal arteriovenous malformations (rAVMs) are vascular anomalies characterized by direct communication within the renal microvasculature. Although rare (prevalence less than 0.1%), rAVMs are clinically significant due to their known association with secondary hypertension, likely due to activation of the renin-angiotensin-aldosterone system (RAAS), and gross hematuria caused by vessel rupture from high intravascular pressure [1-4].

Acquired arteriovenous fistulas are most often iatrogenic, resulting from surgical interventions, renal biopsy, or malignant invasion, and account for approximately 70-80% of all renal arteriovenous abnormalities, typically arising near the collecting system, with most cases occurring in the upper pole of the kidney [5]. In contrast, congenital rAVMs occur in less than 0.04% of the population. However, the true incidence may be underestimated, as many patients remain clinically asymptomatic [6-8].

Imaging is crucial for proper diagnosis of rAVMs. Although suboptimal for fully characterizing vascular anatomy, Doppler ultrasound is an excellent first-line option for screening, as high-velocity, turbulent flow patterns are easily seen in rAVMs. Intricacies in anatomy are better understood with the use of magnetic resonance imaging (MRI) and computed tomography (CT) angiography, which provide enhanced characterization of the extent of the lesion. Digital subtraction angiography, however, remains the gold standard for diagnosis and planning of treatment given its real-time evaluation of vasculature and nidus structure.

We present the case of a 33-year-old woman with a 13-year history of worsening hypertension that was refractory to 5 antihypertensive medications. She also had a history of transient ischemic attacks (TIAs) secondary to her previous hypertensive state. The patient presented for an elective renal angiogram following suspicious findings on a prior renal artery duplex ultrasound. Initial angiography identified left renal artery stenosis and an associated large vessel defect with a dysmorphic left kidney. CT angiography subsequently identified a 6.5- × 6.5- × 5.5-cm left-sided rAVM. Owing to the lesion's complexity, endovascular embolization was considered not technically feasible. Definitive management with simple nephrectomy was therefore undertaken. This case is distinguished from other rAVM cases by several features: the dominance of hypertension rather than hematuria as the dominant clinical manifestation, the unusually large lesion size, and the degree of hypertension sustained for more than a decade prior to diagnosis. This report underscores the diagnostic and management challenges posed by symptomatic, large-volume rAVMs. Moreover, the resolution of symptoms after nephrectomy offers direct clinical evidence that an underlying renovascular mechanism was responsible for this patient›s hypertension. We emphasize the importance of including rAVM in the differential diagnoses of young patients with drug-resistant hypertension. We also include an analysis of the published literature on current therapeutic strategies.

Case Report

We present the case of a 33-year-old woman with a 13-year history of progressively worsening hypertension requiring 5 antihypertensive medications (losartan 100 mg daily, metoprolol 100 mg twice daily, spironolactone 50 mg daily, hydrochlorothiazide 12.5 mg daily, and hydralazine 50 mg twice daily). The case was complicated by multiple episodes of hypertensive urgency requiring emergency room evaluation. The patient's clinical course was further notable for 3 TIAs as well as cardiomyopathy, with a reduced ejection fraction of 25%. As part of her evaluation for refractory hypertension, she underwent cardiology assessment followed by renal artery duplex ultrasound, which demonstrated a peak systolic velocity of 452.6 cm/s with a renal-to-aorta ratio of 3.06, findings suggestive of critical left renal artery stenosis.

She subsequently underwent a renal angiogram, which demonstrated severe proximal renal artery stenosis with an approximately 6- to 8-cm defect just distal to the left renal artery takeoff, consistent with concomitant critical renal artery stenosis and likely renal vascular abnormality, with an otherwise normal right-sided renal angiogram (**Figures 1, 2**). The patient was admitted to the medical service for further workup. CT angiography of the abdomen and pelvis revealed a 6.5-cm left-sided arteriovenous malformation with dysmorphic architecture and a 3-cm nidus, along with possible wedge-shaped infarcts of the left renal parenchyma (**Figures 3, 4**). An additional, incidentally identified right pelvic arteriovenous malformation was also identified (**Figure 5**).

Given the presence of multiple arteriovenous malformations, MRI of the abdomen and brain were performed, confirming only the left rAVM without additional abnormalities. Vascular surgery consultation was requested for possible endovascular intervention; however, the patient was deemed not a candidate for embolization due to the location of the critical renal artery stenosis just distal to the renal artery take off and its close proximity to the arteriovenous malformation. After extensive discussion with the patient regarding risks and benefits, the decision was made to proceed with a simple left nephrectomy.

An open approach was elected to maintain full exposure and control of left-sided renal hilar structures. Gerota›s fascia was incised and dissected in order to better visualize the

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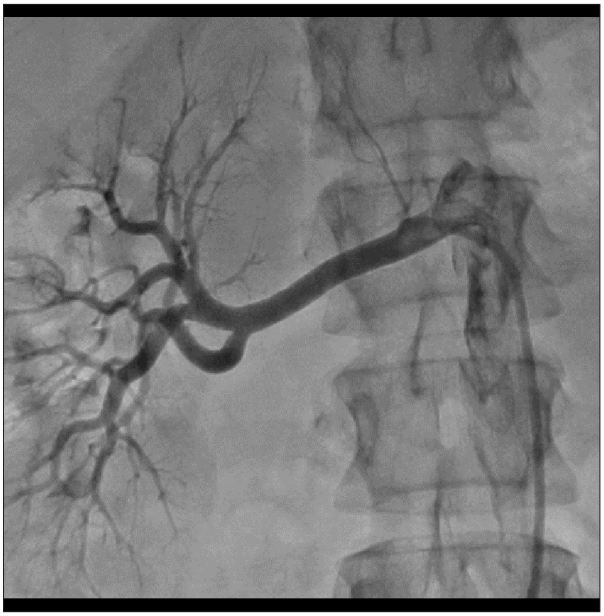

Figure 1. Angiography of the right kidney; normal angiography without pathology.

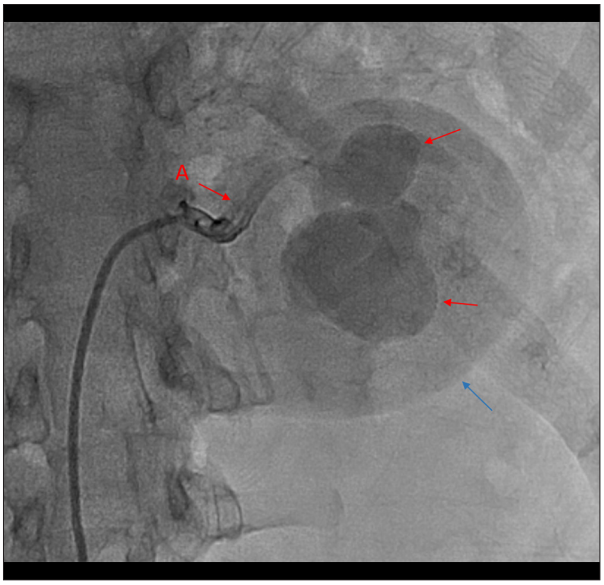

Figure 2. Angiography of the left kidney; aberrant anatomy with abnormal dilation of renal artery (red arrow A), with 3-cm nidus (red arrows) with abnormal connection to the 5.2-cm draining vein (blue).

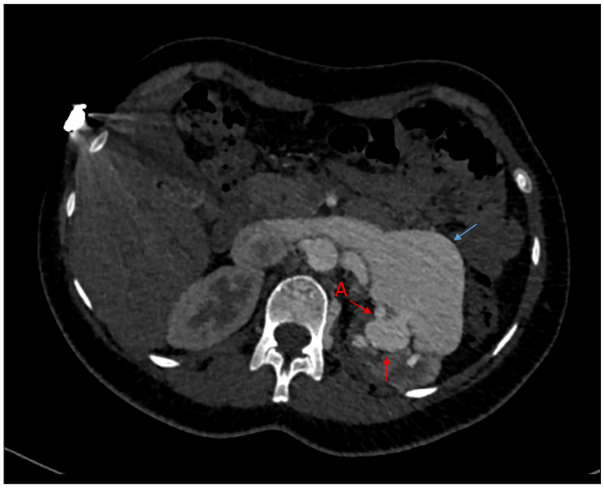

Figure 3. Computed tomography angiogram of the left kidney; axial view of the artery (red arrow A) approaching the nidus (red arrow) with abnormal connection to the large draining vein (blue arrow).

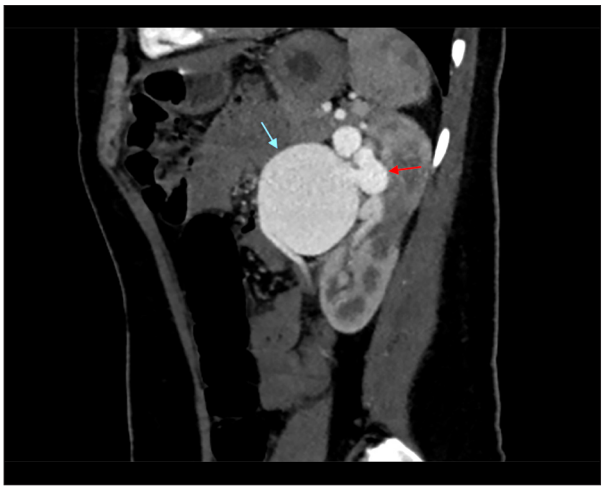

Figure 4. Computed tomography angiogram of the left kidney; sagittal view of abnormal connection between the nidus (red arrow) and the large draining vein (blue arrow).

renal vessels and arteriovenous malformation. The gonadal vein and ureter were identified inferiorly, dissection proceeded upwards toward the renal hilum, and division was accomplished using a stapling device. The large arteriovenous malformation was visualized with a palpable thrill. Given the large malformation, the renal artery and vein were easily identified and the artery was bluntly dissected proximally to its takeoff from the aorta. A small segment of normal-appearing artery was divided using a stapler with immediate loss of palpable thrill. The renal vein was dissected toward the inferior vena cava, revealing a normal caliber vein at its insertion, and was divided using a stapler. Given the abnormal hilar anatomy, the left adrenal gland was inspected and noted to have compromised vascularity, and it was subsequently excised. Finally, pathology demonstrated a left kidney with tortuous and dilated hilar vascular structures, consistent with arteriovenous malformation, and chronic nephritis within the parenchyma; the left adrenal gland demonstrated no significant abnormalities.

The patient was transferred to the intensive care unit for overnight blood pressure monitoring, and received a nicardipine drip

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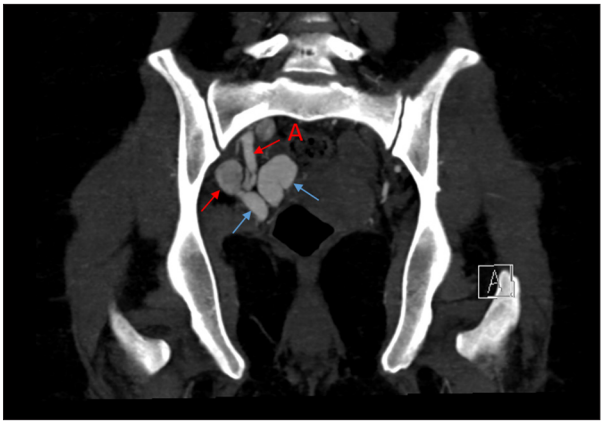

Figure 5. Computed tomography angiogram of the pelvis; coronal view of internal iliac artery (red arrow A) with nidus (red arrow) draining into multiple draining veins (blue arrow).

(stopped on postoperative day 2) that was transitioned to oral losartan 100 mg daily and metoprolol 50 mg twice daily. She was discharged on postoperative day 3 without immediate complications. However, she was readmitted on postoperative day 6 with uncontrolled left upper quadrant pain. CT of the abdomen and pelvis revealed a 6- × 5-cm wedge-shaped splenic infarct. Vascular surgery and general surgery were consulted. Given the size, the treatment of choice was conservative management with a multimodal pain regimen. The patient was discharged after 48 hours with oral pain medication. At her 2-week postoperative follow-up, there were no abnormalities on exam and resolution of left upper quadrant pain. At her 6-month follow-up, the patient underwent repeat cardiac evaluation, including stress test, echocardiography, and Holter monitoring, all of which were negative. Notably, the patient experienced resolution of hypertension and no longer required antihypertensive medications.

Discussion

Renal arteriovenous malformations are rare anatomical variants that are characterized broadly by etiology: acquired or congenital. As mentioned previously, acquired rAVMs are much more common, accounting for 70-80% of cases. These are often traumatic in origin, with disruption of normal renal architecture via renal biopsy, trauma, or malignant invasion. Patients with acquired fistulas generally present with a range of symptoms, including hematuria, flank pain, dizziness, and fever [9]. Congenital rAVMs originate during embryological development. Congenital rAVMs are further categorized by their angioarchitecture, with the cirsoid type, characterized by multiple varix-like communications, being the most common, followed by the angiomatous type, which consists of a single arterial feeder supplying distal branches, and the aneurysmal type, typically seen in older patients, which consists of an arterial aneurysm that erodes into an adjacent vein [10-12]. These lesions are believed to arise during the first trimester of gestation, when focal errors in vascular development lead to nidus formation due to failure of the intervening capillary network to develop [10,13,14]. Over time, these lesions continue to grow and become symptomatic, aligning with symptoms commonly seen in traumatic rAVMs. Congenital rAVMs demonstrate a female predominance, with women being 3 times more likely than men to be affected, and more frequently involve the right kidney than the left [6,15].

Although most rAVMs are discovered incidentally, a subset of patients present with secondary symptoms such as hematuria or uncontrolled hypertension. Hematuria is common with increased intravascular pressure, causing small venules to rupture, which leads to blood products collecting in the calyces. In the most catastrophic cases, patients may present with signs of shock due to blood loss anemia secondary to rupture of the aneurysm, which occurs more commonly in women, especially during the latter stages of pregnancy [4].

The pathophysiological mechanism directly linking rAVMs to hypertension has not been completely characterized, though it is likely driven by interrelated RAAS activation. The arteriovenous malformation creates a direct communication between the high-pressure arterial and low-pressure venous systems, producing high-velocity shunting detectable on Doppler imaging as elevated diastolic velocities [1]. The diversion of flow away from the renal parenchyma distal to the nidus toward this low-pressure venous system likely produces localized ischemia in that region. This, in turn, stimulates RAAS activation through a mechanism analogous to the 2-kidney, 1-clip Goldblatt animal model of renovascular hypertension, in which experimentally induced renal ischemia produces persistent elevation of systolic blood pressure via increased renin secretion [16]. The consequent rise in angiotensin II drives systemic vasoconstriction while aldosterone promotes sodium and water retention, collectively sustaining elevated blood pressure. Direct measurement of elevated renal vein renin concentrations on the affected side has been reported in cases of renal arteriovenous fistula with secondary hypertension, providing biochemical support for this ischemia-driven mechanism [2].

Beyond RAAS activation, one additional mechanism may contribute to hypertension. High-velocity shunting elevates venous back-pressure within the renal circulation, reducing the net pressure gradient driving glomerular filtration. Animal studies have demonstrated that even modest elevations in renal venous pressure significantly reduce glomerular filtration rate and increase plasma renin activity, compounding the ischemic stimulus already present from shunt-mediated parenchymal underperfusion [17,18]. However, this mechanism has not been directly measured in human rAVM cases and its relative contribution remains uncertain.

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The RAAS-mediated ischemic mechanism is the most directly supported by available biochemical and physiological evidence. Elevated venous back-pressure represents a plausible contributing mechanism supported by animal data, though its role in human rAVM-associated hypertension remains incompletely characterized and warrants prospective hemodynamic study. It should be noted that in cases where the rAVM involves the hilar architecture, the stenotic component is functionally inseparable from the malformation. Subsequent angiographic identification would not alter treatment when embolization is contraindicated, and nephrectomy addresses the malformation. Together these mechanisms help explain why hypertension in complex congenital rAVMs can be so severe and so resistant to multiagent therapy. Healthy young patients like our patient who require multimodal blood pressure medication to control blood pressure should have rAVMs on the differential diagnosis.

Digital subtraction angiography remains the gold standard for diagnosis, as it allows for visualization of feeding vessels, nidus architecture, and venous drainage [19]. However, several noninvasive imaging modalities are commonly used in the initial evaluation of rAVMs. Doppler ultrasound may demonstrate turbulent, high-velocity communications suggestive of arteriovenous shunting. While ultrasound is useful as an initial screening tool, its ability to characterize the extent of the arteriovenous nidus and its associated vascular anatomy is limited. CT angiography and T2-weighted MRI are superior compared with Doppler at elucidating the full extent of the malformation, allowing for visualization of the shunt itself [1]. However, the ability to visualize the arteriovenous malformation and its feeder vessels in real time through digital subtraction angiography is immensely helpful in delineating between endovascular or surgical intervention for treatment.

Currently, endovascular embolization is the preferred first-line treatment, while partial or radical nephrectomy is typically reserved for emergent cases [19]. The primary benefit of endovascular embolization is its minimally invasive nature and the prospect of preserving the renal parenchyma while maintaining renal function [20]. The embolization can be performed with gelfoam, alcohol, coils, or liquid embolics. The most common embolic material employed for rAVMs is embolization with coils, where the vessels feeding into the nidus are targeted. However, due to the nidus not being directly targeted, the treatment may not always be successful and also carries the risk of pulmonary embolism from the coil migrating. A balloon catheter or inferior vena cava filter may be employed to prevent this rare side effect. Alongside coil embolization, the other materials also have potential side effects: gelfoam embolization has been shown to be temporary, with hematuria potentially recurring; alcohol embolization can cause dyspnea and headaches for the patient; and liquid embolics carry the potential risk of the embolic material migrating downstream into the venous system [10]. Unfortunately, the size, extent of turbulence, and the material used to embolize the lesion may impact recanalization rates and could necessitate nephrectomy [5].

Otherwise, partial or radical nephrectomy is generally recommended only in patients with complicated vasculature, based on the size and location of the rAVM, where embolization is considered too technically difficult to treat or in those with life-threatening hematuria [18-21]. Although more invasive, a minimally invasive partial nephrectomy has the benefit of addressing the rAVM directly by surgically removing the nidus entirely. However, the surgery is accompanied by a low risk of developing iatrogenic vascular lesions such as pseudoaneurysms and arteriovenous fistulas. Additionally, due to the removal of parts of the kidney, renal function measured by glomerular filtration rate may decrease for the patient after the surgery [18]. Interestingly, simple nephrectomy may be chosen to avoid the formation of iatrogenic renal fistulas, which are associated with minimally invasive partial nephrectomies and often require subsequent embolization [9].

Conclusions

Congenital rAVMs are a rare and often overlooked cause of secondary hypertension, and should be considered in young patients with unexplained drug-resistant hypertension, even when hematuria is absent. This case illustrates how a large, high-flow rAVM can present primarily as severe, medication-refractory hypertension in a young patient, creating significant diagnostic and management challenges. While endovascular embolization is typically preferred, it is not universally feasible. Treatment options should be individualized based on lesion size, vascular anatomy, renal function, and technical feasibility of endovascular approaches. Nephrectomy represents a definitive treatment choice when embolization is difficult or contraindicated, especially when complex lesions involving the renal hilum are present. The complete resolution of hypertension following nephrectomy in our patient is consistent with a renovascular mechanism and emphasizes the importance of early renal vascular evaluation in young patients with unexplained, resistant hypertension. The nature of this study as a single case report limits the generalizability of this mechanism, so future study of rAVM-associated hypertension is warranted to better elucidate the significance of RAAS activation and pathophysiology in this rare, but treatable, condition.

Institution Where Work Was Done

Urology Institute at Renaissance, Doctors Hospital at Renaissance, Edinburg, TX, USA.

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Patient Consent

Signed consent was obtained from the patient.

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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