Human Pathology Reports 46 (2026) 300857

Contents lists available at ScienceDirect

Human Pathology Reports

journal homepage: www.journals.elsevier.com/human-pathology-reports

Renal vascular malformation with features suggestive of arteriovenous shunting presenting as hemorrhagic medullary cyst-like lesions: a clinicopathologic diagnostic pitfall

Binyam Mohammedbirhan Berhe [a,*], Mohamedaman Mohamedsied Ibrahim [b], Temesgen Gebremariam Weldeslassie [b], Mizan Kidanu Weldemariam [b], Aregawi Kassa Abera [b]

[a] Mekelle University College of Health Sciences, Department of Pathology, Mekelle, Ethiopia
[b] Mekelle University College of Health Sciences, Department of Surgery, Mekelle, Ethiopia

ARTICLE INFO

Keywords:
Renal arteriovenous malformation
Hematuria
Nephrectomy
Diagnostic pitfall
Renal vascular lesion
Medullary lesion
Persistent hematuria
Vascular malformation
Histopathology
Kidney

ABSTRACT

Renal arteriovenous malformations (AVMs) are uncommon vascular anomalies that may present with persistent hematuria and pose significant diagnostic challenges due to their variable clinical and radiologic manifestations. Although imaging plays a central role in diagnosis, some lesions remain unrecognized until surgical excision, particularly when they lack a discrete mass and mimic other renal lesions.

We report the case of a 70-year-old male who presented with intermittent gross hematuria of three weeks' duration, occurring every 2–3 days, approximately two months before definitive diagnosis. The bleeding was initially attributed to benign prostatic hyperplasia based on ultrasonographic evidence of marked prostatic enlargement (>150 g). Baseline evaluation revealed mild anemia (hemoglobin 11 g/dL) with stable vital signs. The patient underwent transvesical prostatectomy; however, hematuria persisted postoperatively. Subsequently, he developed acute urinary retention requiring cystoscopic clot evacuation, and cystoscopy demonstrated no active bleeding from the prostatic fossa, making a lower urinary tract source less likely. Contrast-enhanced computed tomography demonstrated a non-excreting left kidney with an obstructing ureteral clot and blood within the pelvicalyceal system, suggesting a renal source of hemorrhage; however, no definitive vascular lesion was identified preoperatively. The patient developed profound anemia with a hemoglobin level of 4 g/dL and required transfusion of six units of whole blood. Due to persistent hemorrhage and suspicion of an underlying renal pathology, total nephrectomy was performed.

Gross examination revealed blood clots within the calyceal system and multiple small hemorrhagic spaces localized to the medullary papillary region without a discrete renal mass. Histopathologic examination demonstrated extensive replacement of the renal medulla by a disorganized network of tortuous, variably sized vascular channels embedded within delicate fibrocollagenous stroma. The abnormal vessels showed marked heterogeneity in wall thickness, including thin-walled dilated vascular channels and larger muscular vessels demonstrating irregular asymmetric mural thickening suggestive of arterial-type differentiation, accompanied by ectatic dilatation, papillary-like luminal outpouchings, and focal thrombus formation.

Overall, the lesion was interpreted as a renal vascular malformation with features suggestive of arteriovenous shunting; however, definitive classification as a fully characterized arteriovenous malformation was limited by the absence of angiographic correlation and elastic fiber staining.

This case highlights a distinctive gross and histopathologic presentation of renal AVM and emphasizes the importance of considering vascular malformations in patients with persistent hematuria despite treatment for more common urologic causes.

* Corresponding author.
E-mail address: benjmoha17@gmail.com (B.M. Berhe).

https://doi.org/10.1016/j.hpr.2026.300857
Received 22 July 2026; Received in revised form 22 September 2026; Accepted 18 October 2026
Available online 24 October 2026
2772-736X/© 2026 The Author(s). Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

Hematuria is a common clinical presentation that requires systematic evaluation to identify both benign and potentially life-threatening causes. In elderly patients, lower urinary tract conditions such as benign prostatic hyperplasia are frequent explanations; however, persistent hematuria despite appropriate treatment should prompt evaluation for alternative sources, including upper urinary tract pathology [1,2].

Renal vascular abnormalities represent an uncommon but clinically important cause of hematuria. Among these, renal arteriovenous malformations (AVMs) are rare vascular lesions characterized by abnormal communications between arterial and venous systems without an intervening normal capillary bed [2,3]. These lesions may be congenital or acquired and can present with variable clinical manifestations, most commonly hematuria, which may range from intermittent episodes to severe, life-threatening hemorrhage [3].

Although advances in imaging have improved recognition of renal AVMs, diagnosis remains challenging because their clinical and radiologic appearances may overlap with other renal lesions. In particular, lesions without a prominent vascular nidus or obvious mass may be overlooked or misinterpreted, resulting in delayed diagnosis or unnecessary surgical intervention [3,4].

Definitive classification of renal vascular lesions requires correlation of histopathologic findings with imaging studies and, when available, ancillary techniques such as elastic fiber staining, which may not be feasible in resource-limited settings.

In this report, we describe a renal vascular malformation with features suggestive of arteriovenous shunting presenting as persistent hematuria following treatment for presumed lower urinary tract bleeding.

2. Case presentation

2.1. Clinical History

A 70-year-old male presented with intermittent gross hematuria of three weeks' duration, occurring every 2–3 days, which had first been noted approximately two months before definitive diagnosis. Initial evaluation revealed mild anemia (hemoglobin 11 g/dL) with stable vital signs. Ultrasonography demonstrated marked prostatic enlargement (prostate volume $>$ 150 g), and the bleeding was initially attributed to benign prostatic hyperplasia. The patient subsequently underwent transvesical prostatectomy.

Following surgery, the patient was initially stable, the catheter was removed, and he was discharged home. However, hematuria persisted despite conservative advice, including increased fluid intake and outpatient follow-up.

2.2. Diagnostic assessment

At one-week follow-up, the patient showed significant clinical deterioration with a drop in hemoglobin to 4 g/dL, requiring transfusion of six units of whole blood. He also developed acute urinary retention, prompting emergency cystoscopic evaluation and evacuation of a bladder hematoma. Importantly, cystoscopy demonstrated no active bleeding from the prostatic fossa, making a prostatic source of hemorrhage unlikely.

Contrast-enhanced computed tomography (CT) was subsequently performed and demonstrated a non-excreting left kidney with an obstructing ureteral blood clot and extensive blood products within the renal pelvicalyceal system, suggesting an upper urinary tract source of hemorrhage. The right kidney was unremarkable. No definite renal mass, vascular nidus, or other specific vascular abnormality was identified on the available imaging. CT angiography was not performed due to limited availability.

Given persistent hematuria, severe anemia, and the inability to exclude an underlying malignant renal lesion, surgical intervention was planned. A left radical nephrectomy was performed for definitive diagnosis and control of ongoing hemorrhage.

2.3. Gross examination

The specimen consisted of a left radical nephrectomy specimen measuring 9.5 $\times$ 6.8 $\times$ 5 cm, with an intact renal capsule and attached perinephric adipose tissue. The external surface was smooth and unremarkable, without evidence of an exophytic lesion, surface irregularity, or gross tumor. The ureter was identified and was grossly unremarkable. On serial sectioning, the renal parenchyma demonstrated preserved cortical architecture; however, the renal sinus and pelvicalyceal system contained multiple dark red-brown blood clots partially filling the calyces and renal pelvis (Fig. 1).

A solitary, well-circumscribed unilocular cyst measuring 3 $\times$ 3 cm was identified at the lower pole of the kidney. The cyst contained clear fluid and had a thin, smooth wall without mural nodularity, solid components, or internal hemorrhage, consistent with a simple renal cyst.

No discrete solid renal mass was identified within the renal parenchyma. However, the medullary region, particularly involving the renal papillary tips, demonstrated multiple small irregular hemorrhagic spaces filled with blood and clotted material. These hemorrhagic spaces imparted a spongy appearance to the affected medullary tissue. The transition between the relatively preserved cortex and the abnormal hemorrhagic medullary region was sharply demarcated.

2.4. Microscopic examination

Histologic examination (Fig. 2) demonstrated renal parenchyma with preserved capsule and relatively preserved cortical architecture at the periphery, including intact glomeruli and tubules. The renal medullary region showed extensive replacement of the normal renal parenchyma by a complex network of abnormal vascular channels embedded within delicate fibrocollagenous stroma.

The lesion was composed of numerous irregularly arranged, tortuous

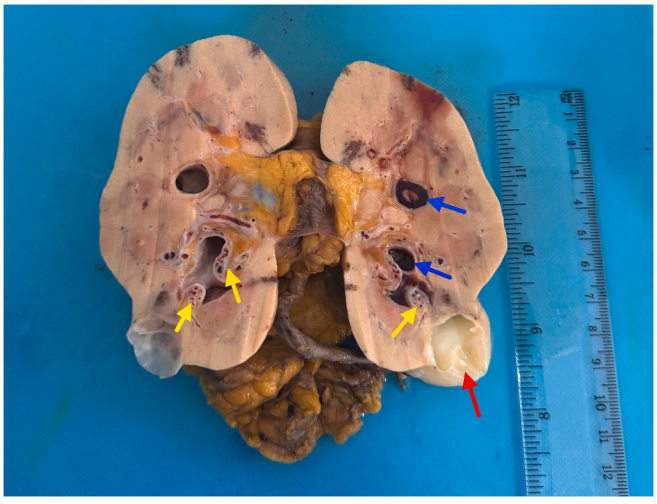


Fig. 1. Gross Examination. Gross photograph of the nephrectomy specimen demonstrating multiple blood clots within the renal pelvicalyceal system (blue arrow), correlating with the patient's persistent hematuria. A well-circumscribed unilocular simple cyst measuring 3 $\times$ 3 cm is identified at the lower pole (red arrow). The renal medullary region, particularly involving the papillary tips, shows multiple irregular hemorrhagic cyst-like spaces filled with blood and clot material (yellow arrow), producing a spongy appearance. No discrete solid renal mass is identified. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

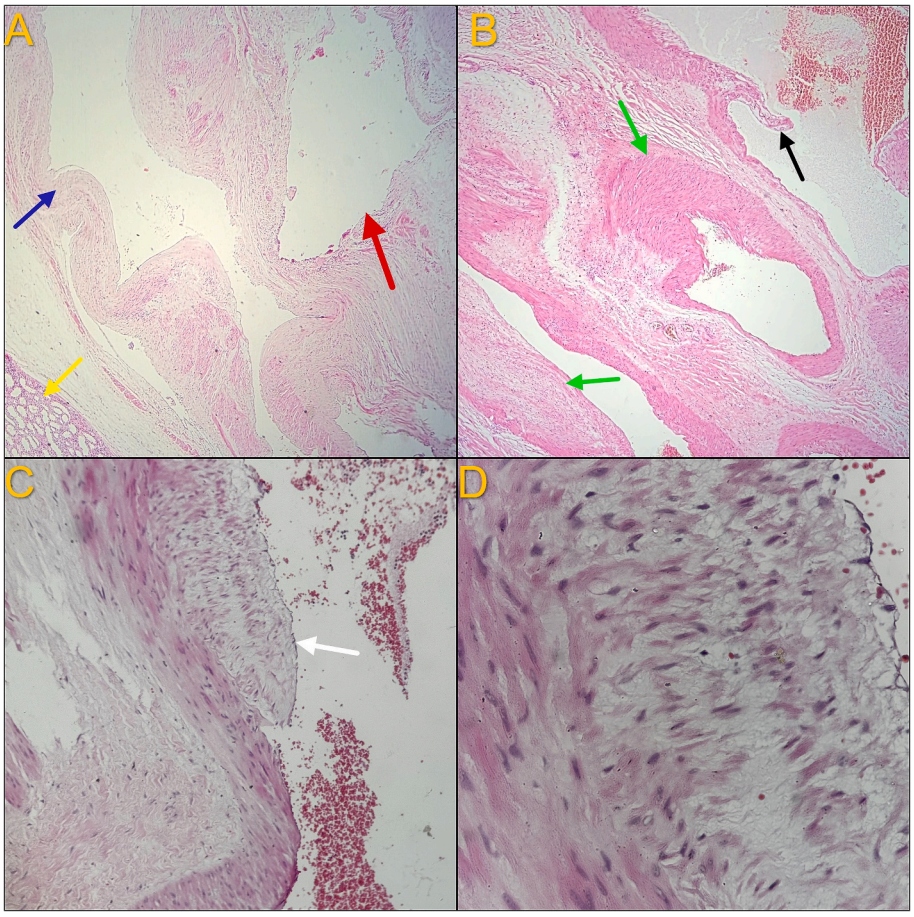


Fig. 2. Microscopic Examination. (A) Low-power photomicrograph (H&E, 4×) demonstrating extensive replacement of the renal medulla by a disorganized network of variably sized, tortuous vascular channels embedded within delicate fibrocollagenous stroma. The lesion shows a sharp interface with relatively preserved renal cortex (yellow arrow). The abnormal vascular network includes thin-walled dilated vascular channels (red arrow) and larger muscular vascular channels with prominent mural thickening (blue arrow). (B) Low-power photomicrograph (H&E, 4×) showing abnormal vascular channels with marked architectural heterogeneity, including ectatic dilatation and focal pseudopapillary luminal outpouchings (black arrow). Prominent thick-walled muscular vascular channels with irregular and asymmetric mural thickening are highlighted (green arrow). The abnormal vascular proliferation involves the renal medulla and demonstrates associated hemorrhagic changes, supporting the diagnosis of renal arteriovenous malformation. (C) Intermediate-power photomicrograph (H&E, 10×) highlighting a muscular vascular channel with irregular and asymmetric mural thickening (white arrow), emphasizing variation in vessel wall architecture. (D) High-power photomicrograph (H&E, 40×) of the area shown in (C), further demonstrating the asymmetric mural thickening and structural heterogeneity of the vascular wall. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

vascular channels of variable caliber, including thin-walled dilated channels and larger muscular vessels. The vascular channels showed marked heterogeneity in wall thickness, with some vessels exhibiting relatively attenuated walls and others demonstrating prominent smooth muscle hypertrophy and irregular asymmetric mural thickening.

Several of the larger vascular channels demonstrated tortuous configurations with focal ectatic dilatation and papillary-like luminal outpouchings, consistent with secondary vascular remodeling changes associated with abnormal hemodynamic stress. The abnormal vascular channels were arranged in close proximity with apparent communication between different vascular profiles and absence of an identifiable intervening normal capillary network.

The vascular lesion extended toward the renal collecting system, with preservation of the urothelial lining of the calyceal system. Focal hemorrhage and organizing thrombus formation were identified within and adjacent to the abnormal vascular channels, providing a pathologic correlate for the patient's persistent hematuria.

At the periphery, a sharp transition was observed between the involved medullary vascular lesion and the relatively preserved renal cortex, supporting extensive replacement of the renal medulla by the vascular malformation.

No epithelial neoplasm, atypical vascular proliferation, or features of angiomyolipoma were identified. Overall, the lesion was interpreted as a renal vascular malformation with features suggestive of arteriovenous shunting rather than a definitively classified arteriovenous malformation, given the absence of elastic staining and angiographic correlation.

The lower pole 3 × 3 cm cyst represented an incidental simple renal cyst unrelated to the hemorrhagic process.

2.5. *Intraoperative findings and surgical management*

Preoperative hemoglobin remained critically low at 4 g/dL despite transfusion support, reflecting ongoing blood loss. The patient underwent total nephrectomy under general anesthesia. Intraoperatively, the kidney appeared grossly unremarkable externally, with an intact capsule and no evidence of an exophytic mass, surface irregularity, or perinephric extension. Careful inspection and palpation did not reveal a discrete tumor or grossly apparent vascular abnormality.

The renal hilum was controlled in a standard fashion, and no enlarged feeding vessels, abnormal hilar vascular structures, or other gross features suggestive of a high-flow vascular lesion were identified intraoperatively. The procedure was completed without intraoperative complications, and adequate hemostasis was achieved.

The nephrectomy specimen was submitted for comprehensive

histopathologic evaluation to determine the source of persistent hemorrhage, particularly given the preoperative concern for an occult renal malignancy or other structural renal lesion causing ongoing bleeding.

2.6. Postoperative course and follow-up

Following nephrectomy, the patient demonstrated significant clinical improvement. Hemodynamic stability was achieved postoperatively, with complete resolution of gross hematuria and no further episodes of urinary tract bleeding. Hemoglobin increased to 8 g/dL after surgery.

The postoperative course was uneventful, with no evidence of surgical complications, including postoperative hemorrhage, infection, or urinary leakage. The patient was discharged with appropriate follow-up arrangements.

At the one-week postoperative follow-up, the patient remained clinically stable, with complete resolution of hematuria and satisfactory wound healing. At the one-month follow-up visit, recovery remained uneventful, with continued absence of urinary symptoms and restoration of functional status. The most recent laboratory evaluation demonstrated a hemoglobin level of 13.4 g/dL, indicating complete hematologic recovery following control of the bleeding source.

2.7. Timeline

The chronological sequence of symptom onset, clinical deterioration, diagnostic work-up, surgical intervention, and final histopathologic diagnosis is summarized in Table 1.

3. Discussion

Renal arteriovenous malformations (AVMs) are uncommon vascular abnormalities that constitute an important but often underrecognized cause of hematuria. Although most published reports have focused on radiologic diagnosis, angiographic characterization, and endovascular treatment strategies, detailed descriptions of nephrectomy specimens and their corresponding histopathologic findings remain relatively scarce [1–3,5,6]. The present case provides a distinctive clinicopathologic perspective by demonstrating a renal AVM presenting as hemorrhagic cyst-like spaces within the renal medulla rather than as a discrete vascular mass or a clearly identifiable radiologic lesion.

Hematuria represents the predominant clinical manifestation of renal AVMs and may vary from intermittent episodes to severe, life-threatening hemorrhage requiring transfusion and urgent intervention [1,7]. The mechanism of bleeding is thought to be related to abnormal high-flow vascular channels, increased intravascular pressure, vascular wall remodeling, and the close anatomical relationship between the malformative vessels and the collecting system, allowing rupture and communication with the pelvicalyceal system [1,7,8]. In the present case, the patient initially presented with intermittent gross hematuria and mild anemia. However, the presence of marked prostatic enlargement (>150 g) led to an initial attribution of bleeding to benign prostatic hyperplasia, a common condition in elderly men. Persistence of hematuria despite transvesical prostatectomy prompted reconsideration of the initial diagnosis and further evaluation of an alternative bleeding source.

This case illustrates a clinically important diagnostic challenge in patients presenting with hematuria: the presence of a common benign urologic condition may obscure a rare but significant underlying lesion. Following prostatectomy, the patient continued to have gross hematuria and subsequently developed acute urinary retention due to clot formation. Cystoscopic evaluation demonstrated no active bleeding from the prostatic fossa, effectively excluding the treated lower urinary tract lesion as the source of hemorrhage. Subsequent contrast-enhanced CT demonstrated a non-excreting left kidney with extensive clot within the pelvicalyceal system and ureteral obstruction, confirming an upper

Table 1
Timeline summarizing symptom onset, initial misattribution of hematuria to benign prostatic hyperplasia, subsequent diagnostic investigations, clinical deterioration, surgical management, and final histopathologic diagnosis of renal arteriovenous malformation.

Time point	Clinical event/ Investigation	Findings and clinical significance
Approximately 2 months before definitive diagnosis	Symptom onset	The patient developed intermittent gross hematuria occurring every 2–3 days for approximately 3 weeks.
Initial clinical evaluation	Laboratory and clinical assessment	Mild anemia was identified (hemoglobin 11 g/dL), with stable vital signs.
Initial imaging evaluation	Renal and pelvic ultrasonography	Marked prostatic enlargement (>150 g) was identified, leading to an initial diagnosis of benign prostatic hyperplasia as the presumed source of bleeding.
Initial management	Transvesical prostatectomy	The patient underwent surgical treatment for presumed lower urinary tract bleeding secondary to benign prostatic hyperplasia.
Early postoperative period	Clinical follow-up	The patient was initially stable; the urinary catheter was removed, and he was discharged. However, gross hematuria persisted despite conservative management and increased fluid intake.
One-week postoperative follow-up	Clinical deterioration and laboratory reassessment	The patient developed severe anemia with a hemoglobin level of 4 g/dL, indicating ongoing significant blood loss.
Emergency management	Blood transfusion and cystoscopic evaluation	The patient received six units of whole blood. Acute urinary retention developed due to bladder clot formation, requiring cystoscopic hematoma evacuation. Cystoscopy demonstrated no active bleeding from the prostatic fossa, making a lower urinary tract source unlikely.
Further diagnostic evaluation	Contrast-enhanced computed tomography (CT) of abdomen and pelvis	CT demonstrated a non-excreting left kidney with an obstructing ureteral blood clot and blood products within the renal pelvicalyceal system, indicating an upper urinary tract source of hemorrhage. No definite vascular lesion or renal mass was identified preoperatively.
Preoperative status	Clinical decision-making	Because of persistent hematuria, severe anemia, and inability to exclude an occult renal malignancy, nephrectomy was planned.
Definitive treatment	Total nephrectomy	The patient underwent total nephrectomy for definitive control of hemorrhage and pathologic evaluation of the underlying renal lesion.
Gross pathologic examination	Macroscopic evaluation of nephrectomy specimen	Blood clots were identified within the pelvicalyceal system. Multiple hemorrhagic cyst-like

(continued on next page)

Table 1 (*continued*)

Time point	Clinical event/ Investigation	Findings and clinical significance
		spaces were present within the medullary papillary region, producing a spongy appearance. A separate 3 × 3 cm simple renal cyst was identified at the lower pole. No discrete renal mass was identified.
Histopathologic examination	Microscopic diagnosis	The renal medulla was extensively replaced by a disorganized network of tortuous vascular channels, associated thrombus formation, and hemorrhagic changes, consistent with renal vascular malformation with features suggestive of arteriovenous shunting.
Immediate postoperative period	Clinical outcome	Hemoglobin improved to 8 g/dL after nephrectomy, with resolution of gross hematuria and stabilization of the patient's clinical condition.
One-month postoperative follow-up	Clinical outcome and laboratory assessment	The patient remained asymptomatic, with complete resolution of hematuria and restoration of hemoglobin to 13.4 g/dL, indicating complete hematologic recovery.

urinary tract source. However, the absence of a definite vascular abnormality or renal mass on imaging created diagnostic uncertainty. Because an occult renal neoplasm could not be excluded, nephrectomy was undertaken.

Radiologic evaluation remains central to the diagnosis of renal AVMs, with CT angiography and digital subtraction angiography considered the most informative diagnostic techniques [1,8]. Typical imaging findings include enlarged tortuous vascular channels, early opacification of venous structures during the arterial phase, aneurysmal vascular dilatation, and evidence of arteriovenous shunting [1,3,5,8]. Nevertheless, diagnosis can be challenging when the lesion is not specifically suspected, when the vascular abnormality is subtle, or when hemorrhage and clot formation dominate the imaging appearance [2,4]. In the present case, imaging successfully localized the bleeding source to the kidney but did not identify the underlying vascular malformation, emphasizing the limitation of conventional imaging in the absence of targeted vascular evaluation.

Gross examination of renal AVMs is often nonspecific because these lesions may not form a discrete mass. Previously reported cases have described hemorrhagic areas, cyst-like changes, and abnormal vascular spaces that may mimic other renal lesions [2,5,9]. In the current case, the nephrectomy specimen demonstrated multiple hemorrhagic cyst-like spaces concentrated within the medullary papillary region, producing a spongy appearance, with a sharp transition between the abnormal medulla and preserved cortical tissue. This gross pattern corresponded closely with the underlying vascular abnormality identified microscopically and represents an important diagnostic clue when evaluating nephrectomy specimens with unexplained hemorrhage.

Histopathologic examination remains essential for establishing the diagnosis of renal AVM, particularly in cases lacking definitive preoperative imaging findings. The defining morphologic feature is the presence of an abnormal vascular network composed of arterial-type and venous-type channels with direct communication and absence of a normal intervening capillary bed [2,3,6]. Additional histologic findings may include variable vessel wall thickness, vascular ectasia, thrombus formation, hemorrhage, and secondary degenerative changes related to chronic abnormal blood flow [3,6].

In this case, microscopic examination demonstrated extensive replacement of the renal medulla by a disorganized network of tortuous vascular channels embedded within delicate fibrocollagenous stroma. The vascular structures showed marked heterogeneity, including thin-walled dilated channels, capillary-sized vascular spaces, and larger muscular vessels with prominent irregular asymmetric mural thickening. Focal ectatic dilatation, pseudopapillary luminal outpouchings, and organizing thrombus formation were identified, reflecting chronic hemodynamic stress within the malformed vascular network. Importantly, the diagnosis was based solely on routine histomorphology without ancillary studies. No elastic fiber stain (e.g., Verhoeff–Van Gieson) or vascular immunohistochemistry (CD31/CD34) was performed, and no angiographic correlation was available.

A hemorrhagic cystic lesion of the renal medulla has a broad differential diagnosis that includes vascular malformations, benign vascular tumors, and vascular-rich renal neoplasms. Careful distinction is essential because these entities may show overlapping gross, radiologic, and histologic features, particularly in nephrectomy specimens without preoperative vascular imaging [10,11]. Renal anastomosing hemangioma (AH) is a benign vascular tumor characterized by tightly packed, sinusoid-like anastomosing capillary channels lined by bland endothelial cells, often with hobnail morphology. It typically presents as a well-circumscribed, mahogany-brown hemorrhagic mass arising from the renal medulla or pelvis, and may be incidentally detected or present with hematuria [12]. In contrast, the present case showed diffuse medullary replacement by an unencapsulated, infiltrative-appearing network of variably sized vascular channels without a discrete mass lesion, arguing against a well-circumscribed neoplastic process such as AH. However, significant morphologic overlap exists, particularly in limited sampling, and both lesions may show hemorrhage and medullary involvement [10,12]. Renal cell carcinoma (RCC) may rarely present with a predominantly vascular appearance due to tumor-induced angiogenesis mediated by VEGF and PDGF signaling, resulting in complex vascular channels and early venous filling on imaging, potentially mimicking vascular malformations [11]. However, unlike RCC, the present case demonstrated no epithelial proliferation, no cytologic atypia, and no solid tumor component, and the lesion consisted exclusively of abnormal vascular channels embedded in fibrocollagenous stroma, excluding a vascular-rich carcinoma. This favors a primary vascular lesion rather than tumor-associated aberrant vasculature [11]. Renal AVMs are rare vascular anomalies characterized by abnormal direct arterial–venous communication without an intervening capillary bed, while acquired lesions are more appropriately termed arteriovenous fistulas and are commonly associated with trauma, surgery, biopsy, or malignancy [5,11]. In the present case, no prior renal intervention, biopsy, or trauma was documented, although the patient had prior urologic surgery, raising the possibility of an acquired shunt lesion. Morphologically, the lesion demonstrated a complex vascular network with variable wall thickness and hemorrhagic changes, but definitive demonstration of a vascular nidus or shunt architecture could not be confirmed due to lack of angiography and elastic staining [5,11]. Other vascular lesions, including angiomyolipoma, may also enter the differential diagnosis but demonstrate distinct morphologic characteristics. Renal angiomyolipomas contain variable proportions of adipose tissue, smooth muscle proliferation, and thick-walled vessels [13]. In contrast, the present lesion showed extensive medullary involvement by a complex vascular network without epithelial or mesenchymal tumor components, supporting the diagnosis of renal AVM.

Recent studies emphasize that renal vascular lesions represent a diagnostically challenging group with frequent overlap between AVM, hemangioma, and other vascular tumors. Brown et al. demonstrated that many lesions historically labeled as AVM or hemangioma are reclassified upon detailed clinicopathologic review, highlighting the risk of overdiagnosis based on routine histology alone [10].

Importantly, renal anastomosing hemangioma may closely mimic vascular malformations due to its sinusoidal architecture, medullary location, and hemorrhagic gross appearance, which overlaps with the findings in the present case [10,12]. In the present case, the absence of elastic fiber staining and angiographic correlation limits definitive classification, and therefore the lesion is best interpreted as a renal vascular malformation with features suggestive of arteriovenous shunting, rather than a fully characterized AVM [10].

Renal vascular malformations may be congenital or acquired. Congenital AVMs are typically identified in younger patients and often demonstrate a cirsoid architecture, whereas acquired arteriovenous shunts are more frequently associated with trauma or iatrogenic injury. In the present case, the advanced age of the patient and history of prior urologic surgery raise the possibility of an acquired lesion; however, no direct renal intervention was documented [5].

From a therapeutic perspective, renal AVMs can often be successfully managed using minimally invasive approaches, particularly selective arterial embolization, which preserves renal parenchyma and avoids the morbidity associated with nephrectomy [14,15]. However, delayed recognition may result in unnecessary radical surgery, particularly when the lesion mimics malignancy or when advanced vascular imaging is unavailable. In this patient, earlier suspicion of a renal vascular lesion and performance of dedicated vascular imaging may potentially have allowed kidney-preserving treatment. Nevertheless, nephrectomy provided definitive hemorrhage control and enabled complete characterization of an otherwise occult vascular malformation.

For practicing pathologists, this case emphasizes the importance of careful gross examination and awareness of unusual hemorrhagic changes in nephrectomy specimens. The absence of a discrete renal mass does not exclude clinically significant renal pathology, and vascular malformations should be included in the differential diagnosis of unexplained renal hemorrhage, particularly when hemorrhagic medullary

Table 2
Previously reported renal arteriovenous malformation cases compared with the present case.

Reference	Patient characteristics	Clinical presentation	Imaging findings	Management	Pathologic/Gross findings
Sountoulides et al. [2]	72-year-old male	Massive hematuria and flank pain	CT scan could not differentiate between a blood clot and a tumor. Renal arteriography demonstrated an area of tortuous, coiled vascular channels	Embolization	Limited histologic correlation
Carrafiello et al. [3]	A 48-year-old Caucasian female	Gross hematuria, left flank pain, and clot retention	Doppler sonograms showed turbulent flow with an increased flow velocity. CT scan showed the presence of tortuous blood arterial opacified vessels with thin arterial ramifications of spiral form. Digital subtraction arteriography (DSA) demonstrated the feeding artery to the AVM.	Embolization	Limited histologic correlation
Gandhi et al. [5]	A 62-year-old male	Gross hematuria	Ultrasound showed large clots in the urinary bladder and cyst-like anechoic area seen at the mid-pole of the right kidney. Focal aneurysmatic dilatation with heterogeneous fill-in on colour Doppler. CT angiography demonstrated aneurysmatic dilatation of the segmental branch and early opacification of the right renal vein on the arterial phase, suggestive of renal AVM	Embolization	Limited histologic correlation
Banthia et al. [9]	28-year-old male	Gross hematuria	CT of the abdomen showed a right renal AVM. Digital subtraction angiography of the right renal vessels showed an AVM of middle and lower pole segmental arteries with communication to a large saccular aneurysm, which was arising from the right main renal vein.	Conservative/ endovascular management	Limited histologic correlation
Wang et al. [6]	A 35-year-old female	Right-sided lumbar abdominal pain with hematuria	CT scan showed blood accumulation in the right renal pelvis, upper middle ureter and bladder. Renal arteriogram showed a tortuous right upper renal pole branch artery and multiple thickened veins communicating with it.	Embolization	Limited histologic correlation
Present case	70-year-old male	Persistent gross hematuria despite transvesical prostatectomy; severe anemia (Hb 4 g/dL)	CT showed blood clots within the renal pelvicalyceal system with a non-excreting left kidney; no definite vascular lesion identified preoperatively	Total nephrectomy	Grossly, multiple hemorrhagic cyst-like spaces were identified in the medullary papillary region without a discrete renal mass, with an incidental simple renal cyst. Histologically, the renal medulla was extensively replaced by a disorganized network of tortuous vascular channels of variable caliber embedded in delicate fibrocollagenous stroma. The vessels demonstrated heterogeneous wall thickness, including thin-walled dilated channels and larger muscular vascular channels with irregular asymmetric mural thickening, along with ectatic changes, pseudopapillary luminal outpouchings, hemorrhage, and organizing thrombus formation, consistent with renal arteriovenous malformation.

changes are identified.

In conclusion, this case highlights a significant diagnostic pitfall in the evaluation of persistent hematuria. The combination of presumed benign prostatic bleeding, persistent postoperative hemorrhage, inconclusive imaging, and progressive anemia delayed recognition of the underlying renal vascular lesion. Renal AVMs may present as hemorrhagic medullary cyst-like lesions without a discrete mass, making integration of clinical history, imaging findings, gross examination, and histopathology essential. Increased awareness among clinicians, radiologists, and pathologists may facilitate earlier diagnosis, reduce unnecessary nephrectomy, and promote kidney-preserving therapeutic strategies.

Previously reported renal AVM cases have predominantly focused on radiologic diagnosis and endovascular management, with limited description of surgical pathology findings. Table 2 summarizes representative reported cases and highlights the unique clinicopathologic features of the present case, in which renal AVM presented as hemorrhagic medullary cyst-like lesions without a discrete renal mass.

4. Limitations of the study

This report has several limitations inherent to a single-case study. First, preoperative characterization of the vascular lesion was limited because dedicated vascular imaging, such as CT angiography or digital subtraction angiography, was not performed. Consequently, a definitive preoperative diagnosis of a vascular malformation with arteriovenous shunting could not be established, and the renal arteriovenous malformation was not recognized before surgical intervention. Although these modalities may allow diagnosis and facilitate minimally invasive treatment in selected cases, the patient presented with persistent severe hemorrhage, recurrent clot formation, profound anemia requiring transfusion, and a non-excreting kidney with an uncertain underlying etiology, making definitive surgical management necessary. Second, histopathologic evaluation was based on routine hematoxylin and eosin examination without ancillary vascular studies. Elastic fiber staining (e.g., Verhoeff–Van Gieson), which is important for demonstrating arterial wall architecture and confirming arterialized vessels, was not performed. In addition, immunohistochemical markers (such as CD31 and CD34) and angiographic correlation were not available. Therefore, definitive assessment of vascular architecture and confirmation of true arteriovenous shunting could not be fully established. Nevertheless, the lesion showed characteristic morphologic features, including a disorganized network of tortuous vascular channels, marked variation in vessel caliber and wall thickness, absence of an intervening capillary bed, hemorrhagic changes, and extensive medullary involvement, which together support a vascular malformation with features suggestive of arteriovenous shunting. Finally, although short-term follow-up demonstrated complete resolution of hematuria and recovery of hemoglobin levels, longer-term clinical follow-up was not available. As an individual case report, the findings cannot be generalized to all renal arteriovenous malformations; however, the detailed gross and histopathologic correlation presented here provides valuable insight into an uncommon manifestation of this rare vascular lesion and highlights an important diagnostic consideration for clinicians and pathologists.

5. Conclusion

This case highlights a significant diagnostic pitfall in persistent hematuria, where a renal vascular lesion may be obscured by more common urologic conditions and inconclusive imaging. Renal vascular malformations can present as hemorrhagic medullary cyst-like lesions without a discrete mass, mimicking other pathologies. In the absence of angiographic correlation and elastic fiber staining, definitive classification as an arteriovenous malformation cannot be established, and the lesion is best interpreted as a renal vascular malformation with features suggestive of arteriovenous shunting. Early recognition is important to enable kidney-preserving management; however, nephrectomy remains appropriate in cases of uncontrolled hemorrhage or diagnostic uncertainty.

Statement of significance

Renal arteriovenous malformations are rare vascular lesions that commonly present with hematuria, yet their surgical pathology findings are infrequently described. This case demonstrates an unusual presentation as hemorrhagic medullary cyst-like spaces without a discrete mass, creating significant diagnostic uncertainty. Histopathologic evaluation revealed a disorganized network of tortuous vascular channels with variable wall thickness, thrombosis, and hemorrhage. In the absence of angiographic correlation and elastic fiber staining, the lesion is best interpreted as a renal vascular malformation with features suggestive of arteriovenous shunting rather than a definitively classified arteriovenous malformation. This report highlights an important diagnostic pitfall and provides practical insights for evaluating vascular lesions in resource-limited settings.

CRediT authorship contribution statement

Binyam Mohammedbirhan Berhe: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Mohamedaman Mohamedsied Ibrahim:** Writing – review & editing, Validation, Investigation, Data curation. **Temesgen Gebremariam Weldeslassie:** Writing – review & editing, Methodology, Investigation, Data curation. **Mizan Kidanu Weldemariam:** Writing – review & editing, Investigation, Data curation. **Aregawi Kassa Abera:** Writing – review & editing, Validation, Investigation.

Ethics statement

This study was conducted in accordance with institutional and international ethical standards. Formal ethical approval was not required for a single case report according to institutional guidelines. Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Funding

No funding was received.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

References

[1] A. Hatzidakis, M. Rossi, C. Mamoulakis, E. Kehagias, G. Orgera, M. Krokidis, A. Karantanas, Management of renal arteriovenous malformations: a pictorial review, Insights Imaging. 5 (4) (2014) 523–530, https://doi.org/10.1007/s13244-014-0342-4. Epub 2014 Jul 5. PMID: 24996396; PMCID: PMC4141337.

[2] P. Sountoulides, I. Zachos, K. Paschalidis, I. Asouhidou, A. Fotiadou, A. Bantis, M. Palasopoulou, T. Podimatas, Massive hematuria due to a congenital renal arteriovenous malformation mimicking a renal pelvis tumor: a case report, J. Med. Case Rep. 2 (2008) 144, https://doi.org/10.1186/1752-1947-2-144. PMID: 18457585; PMCID: PMC2390576.

[3] G. Carrafiello, D. Laganà, G. Peroni, M. Mangini, F. Fontana, D. Mariani, G. Piffaretti, C. Fugazzola, Gross hematuria caused by a congenital intrarenal arteriovenous malformation: a case report, J. Med. Case Rep. 5 (2011) 510,

https://doi.org/10.1186/1752-1947-5-510. PMID: 21982481; PMCID: PMC3206864.
[4] M. Ali, W. Aziz, F. Abbas, Renal arteriovenous malformation: an unusual cause of recurrent haematuria, BMJ Case Rep. 17 (2013) (2013) bcr2013010374, https://doi.org/10.1136/bcr-2013-010374 (PMID: 23867881; PMCID: PMC3736449).
[5] S.P. Gandhi, K. Patel, V. Pandya, M. Raval, Renal arteriovenous malformation presenting with massive hematuria, Radiol. Case Rep. 10 (1) (2015) 1068, https://doi.org/10.2484/rcr.v10i1.1068. PMID: 27408666; PMCID: PMC4921167.
[6] X. Wang, Z. Zhao, Renal arteriovenous malformation causing hematuria: case report and review of the literature, Medicine (Baltimore) 102 (34) (2023) e34547, https://doi.org/10.1097/MD.0000000000034547. PMID: 37653758. PMCID: PMC10470693.
[7] M. Maruno, H. Kiyosue, S. Tanoue, N. Hongo, S. Matsumoto, H. Mori, Y. Sagara, J. Kashiwagi, Renal arteriovenous shunts: clinical features, imaging appearance, and transcatheter embolization based on angioarchitecture, Radiographics 36 (2) (2016) 580–595, https://doi.org/10.1148/rg.2016150124.
[8] M. Van den Broecke, E. Vereecke, P. De Visschere, Renal arteriovenous fistula, J. Belg. Soc. Radiol. 104 (1) (2020) 10, https://doi.org/10.5334/jbsr.2032.
[9] R. Banthia, A. Kumar, R. Prasad, H. Lal, Congenital renal arteriovenous malformation: a rare cause of visible haematuria, BMJ Case Rep. 14 (4) (2021) e242347, https://doi.org/10.1136/bcr-2021-242347.
[10] J. Brown, A.L. Folpe, P. Rao, A.J. Lazar, G.P. Paner, R. Gupta, R. Parakh, J. C. Cheville, M.B. Amin, Primary vascular tumors and tumor-like lesions of the kidney: a clinicopathologic analysis of 25 cases, Am. J. Surg. Pathol. 34 (7) (2010) 942–949, https://doi.org/10.1097/PAS.0b013e3181e4f32a.
[11] R. Comune, F. Grassi, S.G. Picchi, F. De Simone, G. Sarti, C. Giardina, M. Galluzzo, M. Scaglione, S. Tamburrini, Gross hematuria: renal cell carcinoma mimicking a renal arteriovenous malformation, Radiol. Case Rep. 19 (6) (2024) 2130–2134, https://doi.org/10.1016/j.radcr.2024.02.034.
[12] A. Aravind, S. Ahuja, S. Malik, S. Zaheer, Anastomosing hemangioma of the kidney: a rare case report, Int. J. Surg. Case Rep. 126 (2025) 110659, https://doi.org/10.1016/j.ijscr.2024.110659.
[13] S.B. González, J.C. Busquets, R.G. Figueiras, C.V. Martín, C.S. Pose, A.M. de Alegría, J.A. Mourenza, Imaging arteriovenous fistulas, AJR Am. J. Roentgenol. 193 (5) (2009) 1425–1433, https://doi.org/10.2214/AJR.09.2631.
[14] S. Murata, S. Onozawa, K. Nakazawa, A. Akiba, T. Mine, T. Ueda, D. Yasui, F. Sugihara, Y. Kondoh, S. Kumita, Endovascular embolization strategy for renal arteriovenous malformations, Acta Radiol. 55 (1) (2014) 71–77, https://doi.org/10.1177/0284185113493085.
[15] S. Takebayashi, M. Hosaka, Y. Kubota, E. Ishizuka, A. Iwasaki, S. Matsubara, Transarterial embolization and ablation of renal arteriovenous malformations: efficacy and damages in 30 patients with long-term followup, J. Urol. 159 (3) (1998) 696–701, https://doi.org/10.1016/s0022-5347(01)63703-0.