Vicious vessels: A large pelvic arteriovenous malformation causing gross hematuria and hydronephrosis

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INTRODUCTION

Gross hematuria typically raises concern for urothelial carcinoma. Standard evaluation includes CT urogram, cystoscopy, and urine cytology; however, rare vascular anomalies, such as pelvic arteriovenous malformations (AVMs), can manifest as hypervascular bladder lesions that resemble neoplastic processes on these modalities. This case report illustrates the benefit of integrated urology and interventional radiology in diagnosing and treating a pelvic AVM.

CASE REPORT

A 40-year-old G0P0 woman with type 2 diabetes mellitus and no prior surgeries presented to an emergency department (ED) with acute urinary retention and gross hematuria. Non-contrast CT KUB revealed moderate left hydroureteronephrosis, an 8.0 ×5.5 cm hyperattenuating bladder mass, and other nodules in the left pelvis abutting and indenting the bladder and left ureter (Figure 1). She was managed with continuous bladder irrigation, required three packed red blood cell transfusions for symptomatic anemia, and admitted for further evaluation.

She was brought to the operating room for cystoscopy, clot evacuation, possible transurethral resection of bladder tumor, left ureteroscopy with retrograde pyelogram under anesthesia. The bladder mass seen on CT KUB was blood clot. The trigone was displaced anteriorly by extrinsic compression. No obvious sessile, papillary or solid tumors were seen, however there was significant hypervascularity at the level of the bladder trigone involving both ureteric orifices with large and engorged blood vessels with mild nodularity and diffusely erythematous mucosa. On ureteroscopy the left distal ureter showed the same engorged vessels,

extending proximally up to 10 cm from the ureteric orifice, with mild hydroureteronephrosis.[9] Transurethral resection of trigonal mucosa and the right ureteric orifice to detrusor muscle was performed due to concern about possible underlying malignancy, thus a 6 Fr×24 cm double-J ureteric stent was placed.

CT urography post-resection confirmed resolution of the bladder lesion and identified pelvic venous congestion. Histopathology demonstrated cystitis cystica without dysplasia; detrusor muscle was present, and urine cytology was negative for malignancy.
An infused MRI pelvis and body was completed demonstrating left pelvic varices concerning for an underlying arteriovenous malformation involving branches of the left internal iliac artery, prompting further angiographic evaluation (Figure 2).

Approximately two months after the index presentation, the patient underwent *diagnostic* pelvic digital subtraction angiography (DSA). Right common femoral arterial access was obtained under ultrasound guidance with a 5 Fr sheath. DSA of the internal iliac arteries identified a high-flow nidus in the left pelvic plexus arising from the left ovarian vein, as well as multiple branches from the internal iliac artery and external iliac artery.

Shortly thereafter, the patient underwent *therapeutic* pelvic DSA. Access was gained through the right internal jugular vein, common femoral artery and vein with 5 French long sheaths placed in all three sites. The gonadal vein was first embolized via coil and STS foam. Then, the left internal iliac vein nidus was coil embolized. A superior nidus was also seen and management of the same deferred for clinical follow up, with the thought that if symptoms remained this too could be coiled in a staged fashion (as a possible stage 2 embolization of the uterine vein).

Six months after the index presentation, follow-up MRI showed the anteroinferior component of the AVM was occluded while the posterosuperior component remained patent, with complete resolution of the left hydroureteronephrosis. At fourteen months, follow-up via MRA again demonstrated near-complete nidus thrombosis with marked reduction in pelvic venous engorgement with occlusion of the left gonadal vein and the same residual superior nidus (Figure 3).

The ureteric stent was removed 6 weeks after embolization, she has voided without obstructive symptoms since. From embolization to final follow-up at fourteen months, she had no recurrence of gross hematuria, required no further transfusions, remained free of pelvic pain, and the left hydroureteronephrosis resolved. Stage 2 embolization of the uterine vein was therefore not undertaken.

DISCUSSION

Pelvic arteriovenous malformations (AVMs) are exceedingly rare vascular lesions that can masquerade as urothelial malignancy, clot-retaining masses, or benign venous disorders such as pelvic congestion syndrome. This case is notable for severe, transfusion-dependent hematuria

and ureteral involvement. Of the ureteral AVMs reported in the literature, most presented with intermittent hematuria or flank pain,[1,3-5] few, if any, required ongoing transfusions.

The ureteral and periureteral involvement in our patient contributed not only to bleeding but also to obstructive uropathy. Several reports have described ureteral AVMs mimicking more common pathologies, including fibroepithelial polyps and urothelial carcinoma.[3-5,7] Our case reinforces that vascular malformations should be considered in the differential of "lateralizing" hematuria, as described by Rowbotham & Anson,[2] particularly when bleeding is refractory, recurrent, or disproportionate to cystoscopy findings.

The diagnostic pathway reflects the complementary roles of modern imaging modalities. Non-contrast CT initially suggested a bladder tumor, but subsequent CT urography revealed resolution of the intravesical clot and pelvic varices. MRI with angiographic sequences strengthened the suspicion of a high-flow vascular malformation, while digital subtraction angiography confirmed a large, complex AVM nidus encasing the distal ureter (Figure 2). Historically, such uncertainty led many patients to nephroureterectomy,[3,4] however the integration of CTU, MRI, and angiography now permits precise diagnosis and minimally invasive treatment[8].

Unique case contributions

Most notably, the patient presented with transfusion-dependent hematuria secondary to a pelvic AVM encasing the distal ureter, a severity rarely reported in the literature. Furthermore, the successful use of endovascular embolization underscores the capacity of minimally invasive therapy to achieve durable hemorrhage control and preserve renal and bladder integrity, in contrast to historical cases managed with extirpative surgery[8].

Importantly, angiography enables clear distinction between high-flow AVM and low-flow pelvic congestion syndrome. Park et al. highlighted that PCS is generally managed with venous embolization or stenting, whereas AVMs require targeted occlusion of the nidus itself, often with coils, particles, or liquid embolics.[6,10] The angiographic finding of early arterialized venous drainage and a well-defined nidus in our case was pathognomonic for AVM (Figure 2). Moreover, transfusion dependence argues strongly against PCS, which rarely, produces such hemodynamic instability.

CONCLUSIONS

Pelvic AVMs may masquerade as bladder neoplasms on CT, US, and cystoscopy. A coordinated approach combining urologic intervention for diagnosis and IR-guided embolization, supported by arteriography and MRA, enables precise diagnosis, targeted therapy, and superior patient outcomes.

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FIGURES AND TABLES

Figure 1. Index non-contrast computed tomography (CT) at presentation**.** (a) Axial and (b, c) coronal images obtained at the emergency department presentation demonstrate an 8 × 5.5 cm hyperattenuating bladder mass later found to be blood clot (arrow), with moderate left hydroureteronephrosis (curved arrows). There were additional nodules abutting and indenting the left ureter and bladder (arrowheads).

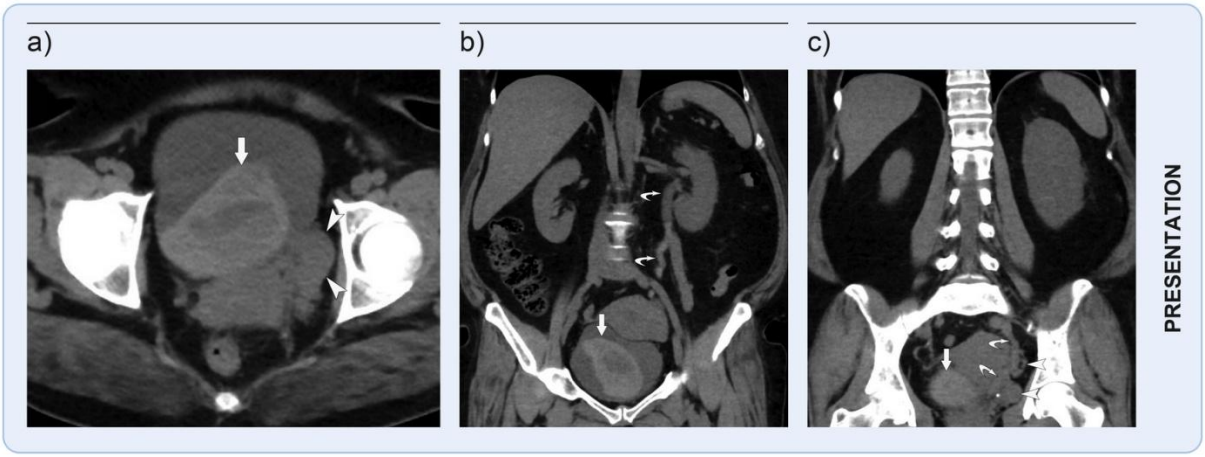

Figure 2. Pre-embolization. (a, b) Coronal T1-weighted post-contrast 3D VIBE magnetic resonance images demonstrate serpiginous dilated vascular channels in the left hemipelvis adjacent to the distal ureter (arrowheads). (c) Venographic imaging demonstrates a high-flow pelvic AVM. The long arrow (1) identifies the AVM nidus. The short arrow (2) indicates the left gonadal vein, which is patent pre-embolization. The arrow (3) highlights an inferior feeding vessel arising from the internal iliac system.

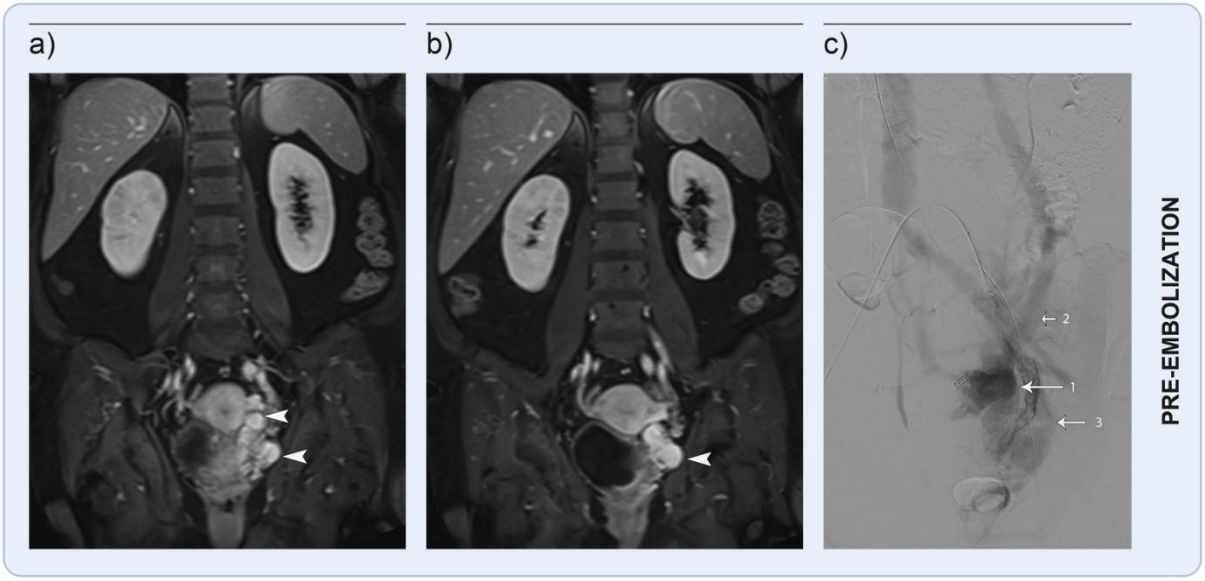

Figure 3. Post-embolization. (a, b) Followup T1-weighted post-contrast 3D VIBE magnetic resonance images show interval caliber reduction of abnormal pelvic vessels (arrowheads). (c) Corresponding venography demonstrates successful embolization, with coil occlusion of the left gonadal vein (2) and the inferior internal iliac feeder (3), resulting in marked reduction of flow through the AVM nidus (1). The internal iliac artery remains patent, while the gonadal vein is no longer opacified, consistent with complete occlusion.