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CASE REPORT *RADIOLOGY*

A rare confluence: Intratumoral arteriovenous malformation in renal cell carcinoma

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ABSTRACT

Renal cell carcinoma (RCC) is the most common renal tumor, accounting for approximately 2% of adult malignancies. However, there are also rare presentations of RCC with metastatic sites, paraneoplastic disease, and abnormal findings that may mislead to diagnosis, including bleeding or vascular malformation like arteriovenous malformation (AVM). Here we present the case of a woman complaining of hematuria, weakness, and fatigue. Ultrasound examination revealed a large mixed echogenic lesion in the right kidney, and further examination revealed a large renal lesion with intratumoral AVM.

Keywords: Arteriovenous malformations, Hematuria, Kidney, Renal malignancy, Vascular malformation

INTRODUCTION

Renal cell carcinoma (RCC), the eighth most common malignant tumor in adults, constitutes 80%90% of adult kidney cancers. The risk of RCC development increases with conditions like acquired polycystic kidney disease and inherited disorders such as Von Hippel-Lindau disease and tuberous sclerosis. Metastasis typically occurs first in the lungs, followed by the bones. While the classic triad of RCC includes hematuria (55%), abdominal pain (40%), and palpable abdominal mass (35%), it manifests only in less than 10% of patients. Presently, over 50% of RCCs are incidentally diagnosed through cross-sectional imaging studies.[1] Among malignant renal tumors, RCCs account for 90%, with clear cell RCCs comprising 75%, papillary RCCs 7%–15%, and chromophobe RCCs 5%. Collecting duct and medullary carcinomas are rare, representing less than 1% of renal tumors.

Metanephric, nephroblastic, and mesenchymal tumors account for the other 10%. AVMs are abnormal vascular shunts between arteries and veins occurring due to the absence of a capillary bed. The prevalence of AVMs in the population is 0.04%[2], and there are two types: congenital or acquired (after biopsy, injury, and malignancy). RCC increases proangiogenic factors like VEGF, which in turn increases the vascularity and expansion of AVMs.[1] AVM in association with RCC is sometimes known to cause reversible hypertension and heart failure.[3]

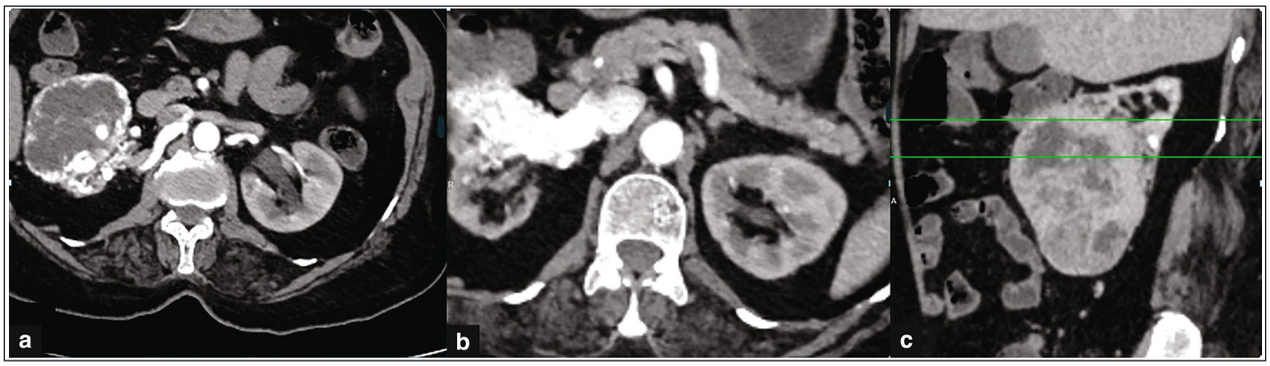

Figure 1: Large exophytic heterogeneously enhancing lesion in the corticomedullary phase. (a) Early draining dilated right renal vein in the arterial phase. (b) Prominent right main renal artery with multiple intratumoral feeding arterial branches in the arterial phase. (c) Large exophytic heterogeneously enhancing lesion in the corticomedullary phase.

CASE REPORT

A 78-year-old hypertensive female presented to the emergency department (ED) for complaints of hematuria. She had similar complaints in the past and was treated conservatively in a nearby hospital and referred to a higher center for further management but failed to follow up. She later developed generalized weakness, fatigue, and similar episodes of hematuria with clots. She had no history of fever, weight loss or abdominal pain. She is not a known case of diabetes but has been hypertensive for the past six years and is on medication.

On clinical examination, she was conscious and coherent. Physical examination was positive for pallor and mild right flank tenderness. Additionally, normal bowel sounds were present.

Initial vital signs on presentation were as follows: blood pressure of 170/90 mmHg, pulse rate of 90 bpm, temperature of 98.6 °F, respiratory rate of 17 breaths per minute, and 100% oxygenation on room air. The CBC showed HB of 10.8%, TLC of 10,600, and normal platelet count and normal RFT values (serum creatinine, 1.19 mg/dL).

Imaging Findings

On ultrasound imaging, an exophytic, heterogeneously hypoechoic lesion was noted in the inferior pole of the right kidney, which is more likely suggestive of a renal mass lesion—malignant lesion.

On further workup with triphasic CT, it showed reduced right kidney size with thinned-out parenchyma and relatively increased hilar fat and E/O. A large, well-defined, exophytic soft tissue density lesion is noted arising from the lower pole cortex, causing splaying up of lower pole calyces. Postcontrast, the lesion shows heterogeneous enhancement with large areas of necrosis [Figure 1]. During the corticomedullary phase, there is markedly dilated ipsilateral patent renal veins and a tortuous network of collateral venous circulation, and early and rapid filling of the main renal vein and inferior vena cava. These findings are consistent with arteriovenous fistula. Entire lesion measuring ~70 × 77 × 88 mm (AP × TR × CC).

Anteriorly, the lesion is in close proximity to the ascending colon with focal loss of intervening fat planes.

Posteriorly, the lesion is showing claw sign with renal cortex and causing compression and posterior displacement of the calyceal system.

Medially, the lesion is closely abutting the lower branch of the renal vein. Note made of short segment faintly enhancing filling defect in the adjacent renal vein measures 5.8 mm in length and 3.6 mm in width, causing partial luminal stenosis.

Coarse calcification noted in lower pole calyx measuring ~10 mm.

Also, there is mild circumferential wall thickening of the lower pole calyceal system and renal pelvis with mild wall enhancement (better demonstrated in delayed images).

DISCUSSION

Differentiating between AVMs and RCC on cross-sectional imaging poses a challenge.

Renal mass CT protocols usually have three phases: corticomedullary, nephrogenic, and delayed excretory. Preoperative information regarding hilar anatomy can be seen in the arterial phase and can also highlight a potential tumor-related AVM.[4]

AVMs typically present with enlarged and tortuous vessels, hypertrophic draining veins, and early arterial enhancement, often accompanied by collateral circulations. Symptoms associated with AVMs include congestive heart failure, abdominal bruits, and renal hypertension. These findings are crucial to identify, as the presence of an AVM can lead to severe hemorrhage during renal manipulation, potentially complicating surgical procedures like partial nephrectomy. Moreover, AVMs carry a risk of spontaneous rupture, particularly during renal mobilization and hilar dissection. The close differential diagnosis could be metastasis.

Radical and partial nephrectomy are established treatments for suspicious renal masses. Surgeons must carefully review dedicated, high-quality, cross-sectional renal imaging preoperatively to optimize oncologic outcomes and minimize intraoperative complications. In the case discussed at a multidisciplinary committee, considering the patient's good general condition, a right radical laparoscopic nephrectomy was performed. Pathologic evaluation revealed clear cell RCC, staged as pT2b, Grade 2.

CONCLUSION

Recognizing the rare association between RCC and AVMs is crucial for patient safety and surgical planning. Given their similar presentations, a high level of suspicion is necessary to identify these potentially coexisting lesions. The safe option and definitive treatment in cases of RCC presenting with an AVM is embolization followed by radical nephrectomy.

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