

Primary Renal Lymphoma Mimicking a Subcapsular Hematoma: A Case Report

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ABSTRACT

Primary renal lymphoma (PRL) is a rare entity with a history of controversy regarding its existence. Lymphomatous involvement of the kidney is more commonly seen secondarily to spread from an adjacent lymphomatous mass, rather than arising primarily from the kidney. PRL can mimic other renal lesions such as renal cell carcinoma, renal abscess, and metastasis; therefore, an early diagnosis is crucial to guide treatment and properly assess prognosis. We present a rare case of a 77 year-old male who presented with hematuria and PRL mimicking a subcapsular hematoma.

CASE REPORT

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Our patient, a 77 year-old male, first presented to the family practice clinic for a routine follow-up visit for hypertension and hyperlipidemia, at which time he reported having one episode of gross hematuria. The patient's physical exam findings were noncontributory. Medications taken at that time included Lisinopril 40mg daily, Clonidine 0.3mg/24hr, and Simvastatin 10mg QHS. A CECT of the abdomen/pelvis was then performed for further evaluation. This showed a crescentic hyperdensity along the posterolateral aspect of the inferior left renal pole, thought to be a spontaneous subcapsular hematoma, and the cause of the patient's gross hematuria (Figure 1). Although, a subcapsular hematoma was the leading differential; peri-renal abscess and subcapsular hematoma caused by a small renal cell carcinoma or metastasis were also considered. The initial white blood cell count was 5.6×10^3 (normal range of $3.5 - 10.5 \times 10^3$) and the creatinine was 0.7 mg/dL (normal range of 0.6-1.2). After consultation with radiology and urology, the patient was scheduled for a follow-up CECT three months later, to evaluate for the anticipated interval resolution of the subcapsular finding.

Three months later, the follow-up CECT demonstrated that the crescentic hyperdensity was still present and had slightly increased in size. It also demonstrated mild

enhancement (Figure 2). Both the growth and enhancement were not consistent with a diagnosis of a subcapsular hematoma, which should have resolved by this time. In addition, the patient manifested no lab abnormalities or infectious symptoms to suggest an abscess. Therefore hematoma and abscess were excluded. The interpreting radiologist included lymphoma, atypical renal cell carcinoma, and metastatic disease as differential considerations on this follow-up CT, and recommended an MR for further evaluation. A contrast-enhanced MR (Figure 3) performed six weeks later confirmed a T1 isointense, T2 hypointense, mildly enhancing renal mass without change in the prior differential.

Shortly after the MRI, a CT-guided biopsy (Figure 3) was performed and the patient was diagnosed with Stage I AE Extranodal Marginal Zone B-cell Renal Lymphoma arising from the left kidney (Figures 4 and 5). At the time of the biopsy, the white blood cell count was 4.9×10^3 (normal range of $3.5 - 10.5 \times 10^3$) and the creatinine was 0.8 mg/dL (normal range of 0.6-1.2). The 18FDG-PET CT scan (Figure 6a and 6c) performed immediately after the biopsy showed a thin curvilinear area of moderately increased metabolic activity (max SUV of 6.0) inferior and posterolateral to the subcapsular left renal mass. This curvilinear activity was attributed to a small post biopsy urine leak, and resolved on the follow-up PET scan, 10 months later. Only mild activity

(max SUV of 3.0), similar or slightly less than blood pool, was seen in the subcapsular renal mass on the initial PET scan immediately following the biopsy. No contralateral or extra-renal involvement was visualized on either PET scan (Figure 6).

A 99mTc-MAG3 Renal Flow and Function demonstrated that only 1/3rd of the diseased left kidney contributed to the patient's overall renal function, and he chose radiation therapy over chemotherapy or nephrectomy. He received a total of 30 Gray (15 treatments) over a three week period. The PET-CT scan four months following XRT demonstrated no anatomic or metabolic evidence of active or recurrent disease, respectively.

Annual IV CECT of kidneys has demonstrated no evidence of primary recurrence, and no development of extra-renal disease. At the patient's most recent follow-up, three years after biopsy, his white blood cell count remained normal at 5.1×10^3 (normal range of $3.5 - 10.5 \times 10^3$) and his creatinine had slightly increased to 1.1 mg/dL (normal range of 0.6-1.2).

DISCUSSION

Primary renal lymphoma accounts for < 5% of all cases of renal lymphoma, which largely accounts for its previously debated and controversial existence. This controversy stems from differentiating the more common secondary renal involvement due to an adjacent lymphomatous mass from true primary renal lymphoma. Additionally the kidney is an extranodal organ that typically does not contain lymphoid tissue [3]. One theory suggests that lymphatics in the renal capsule may be the source of lymphoma which then invades the renal parenchyma. Another explanation is that chronic inflammation attracts lymphoid cells into the kidney which can subsequently develop into lymphoma [4].

PRL typically occurs in adults without gender predilection. It is most commonly a non-Hodgkin's lymphoma of predominantly large-cell type with a B-cell immunophenotype [5]. Patients may present with flank pain, hematuria, fever, weight loss and renal failure. A diagnosis of PRL requires lymphomatous renal infiltration, non-obstructive unilateral or bilateral kidney enlargement, and no extrarenal localization at the time of diagnosis [6]. A typical diagnostic workup should include CT of the chest, abdomen, and pelvis; as well as renal and bone marrow biopsy, although workups may vary depending upon a healthcare institutions capabilities [4].

The diagnostic workup of our patient included a CECT of the chest (following the biopsy and diagnosis); 1 PET-CT exam, two CECTs of the abdomen/pelvis and 1 contrast enhanced kidney MRI leading up to the biopsy; and CT-guidance for the renal biopsy. Additionally, a bone marrow biopsy was performed shortly after the renal biopsy confirming no lymphomatous involvement. Labs at time of initial presentation to the family practice clinic and in all follow-ups were normal (values listed in the case presentation).

In our patient, the top differential consideration after the first CECT was a subcapsular hematoma which would explain the patient's hematuria; however, other considerations included were a subcapsular abscess, renal cell carcinoma, metastasis and lymphoma. The patient had no infectious symptoms or lab abnormalities at the time of the scan or in the weeks to follow; therefore an abscess was excluded. When the follow-up CECT scan revealed mild enhancement and mild interval growth despite no recurrence of the hematuria; subcapsular hematoma was excluded because it would not have enhanced and should not have grown in size. When a contrast-enhanced kidney MRI confirmed mild enhancement, a biopsy was recommended, confirming primary renal lymphoma. No contra-lateral renal or extra-renal involvement was ever visualized on any of the cross-sectional imaging, to include two 18FDG-PET CT scans, one immediately following the biopsy; and the other scan four months after the completion of radiation.

On a non-contrast CT, PRL generally presents as a mass usually slightly hyperdense to the non-enhanced renal parenchyma. On contrast-enhanced CT sequences, PRL demonstrates mild enhancement to a lesser degree than the adjacent renal parenchyma, making it appear relatively less dense than the avidly enhancing renal parenchyma. On MRI, PRL is hypointense on T2WI and hypointense to isointense on T1WI, with minimal contrast enhancement. On ultrasound, PRL appears as a hypovascular, hypoechoic-to-anechoic mass without posterior acoustic enhancement [2-5].

The overall prognosis of PRL is poor with studies reporting a range of median survival times ranging from eight months to three years, with a 40-50% 5 year survival rate [7]. PRL can mimic other primary renal malignancies such as renal cell carcinoma (RCC) and transitional cell carcinoma, or benign entities such as a subcapsular hematoma as presented in this case [8]. PRL is usually treated with systemic chemotherapy +/- radiation. Resection may be considered in cases where the lymphomatous mass is small or disease is isolated to one kidney [9].

TEACHING POINT

Although rare, inclusion of PRL in the differential of any potential renal neoplasm mimic is important to obtaining an early diagnosis, reduce adverse effects on patient management, and potentially improve overall prognosis. This is particularly important in cases of presumed benign entities such as a subcapsular hematoma where short interval follow-up imaging or other imaging modality considerations are warranted to exclude malignancy.

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FIGURES



Figure 1: This is the CT scan of a 77 year-old male with primary renal lymphoma who initially presented to the family practice clinic with gross hematuria. Axial coned-down, non-contrast CT (a - left; supine, GE LightSpeed Plus 4 slice scanner, kVp 120, mA 214, ST 5) image of the left kidney demonstrates a subcapsular, crescent-shaped area at the posterolateral aspect of the inferior renal pole (white arrow) that is slightly denser than the adjacent, non-enhanced renal parenchyma. Axial contrast-enhanced CT (b - center; supine, GE LightSpeed Plus 4 slice scanner, kVp 120, mA 131, ST 5, 100 cc visipaque, 90s delay) image at the same level as the previous image; and coned-down coronal CECT (c - right; supine, GE LightSpeed Plus 4 slice scanner, kVp 120, mA 131, ST 1, 100 cc visipaque, 90s delay) images show that the crescent-shaped subcapsular area is less dense than the enhancing renal cortex (white arrowheads).

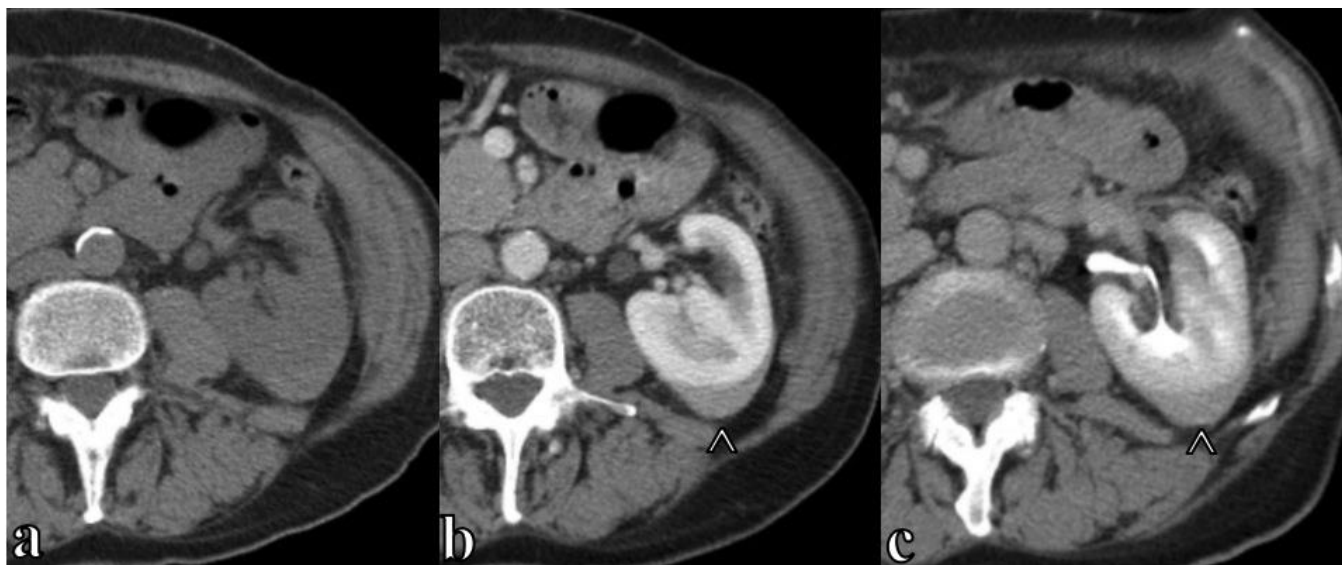


Figure 2: This is the follow-up CT scan of a 77 year-old male with primary renal lymphoma, acquired three months after he initially presented to the family practice clinic with gross hematuria. Coned-down, axial, NCCT (a - left; supine, GE LightSpeed Pro 16 slice scanner, kVp 120, mA 113, ST 5), CECT in corticomedullary phase (b - middle; supine, GE LightSpeed Pro 16 slice scanner, kVp 120, mA 80, ST 5, 100 cc visipaque, 90s delay), and CECT in early pyelographic phase (c - right; supine, GE LightSpeed Pro 16 slice scanner, kVp 120, mA 80, ST 5, 100 cc visipaque, 160s delay) images better demonstrate the mild enhancement (white arrowheads) of the crescentic subcapsular mass at the inferior left renal pole from an average HU of 33 on the NCCT to 55 in the corticomedullary phase on subimage b. There was also several millimeters of interval growth.

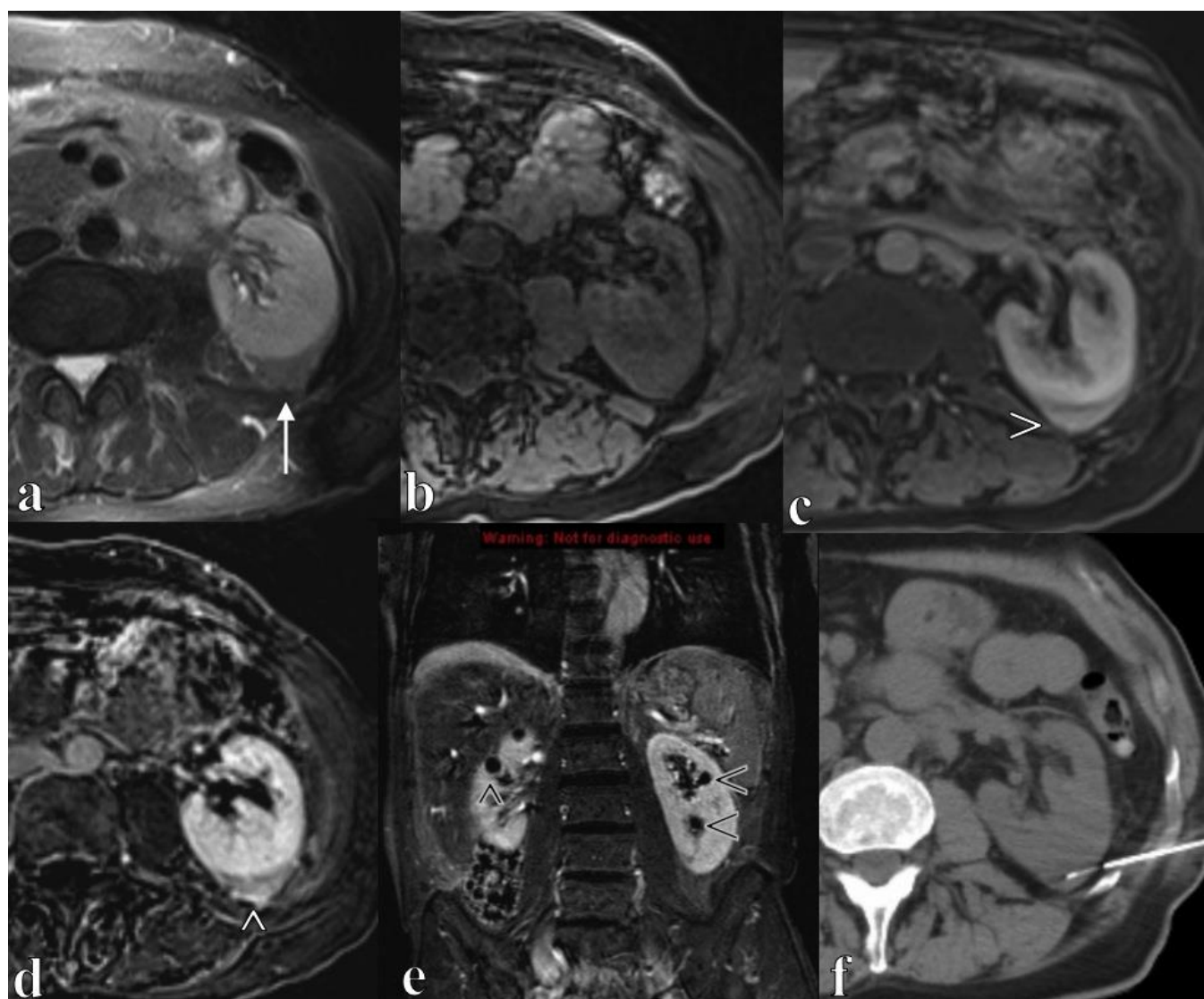


Figure 3: This is a 77 year-old male who was diagnosed with primary renal lymphoma following this MRI and CT-guided biopsy. The MRI was acquired six weeks after the follow-up CT scan, and the biopsy occurred shortly thereafter. The crescentic subcapsular mass (white arrow) is hypointense to the adjacent renal cortex on the axial T2 fat-saturated MR image (a - top left; Siemens 3 Tesla, TR 3500, TE 102, ST 6). The mass demonstrates mild enhancement (black arrowhead) from the axial T1 pre-contrast (b - top middle; Siemens 3 Tesla, TR 3.26, TE 1.18, ST 2) to post-contrast (c - top right; Siemens 3 Tesla, TR 3.26, TE 1.18, ST 2, 15 cc Magnevist contrast) MR images. This enhancement is better (white arrowheads in c and d) seen on the axial (d - bottom left; Siemens 3 Tesla, TR 3.26, TE 1.18, ST 2, 15 cc Magnevist contrast) and coronal (e - bottom middle) subtracted MR images. For comparison, multiple cysts and calyceal diverticula (black arrowheads) are seen in both kidneys as round intraparenchymal hypointensities (e - bottom middle image; Siemens 3 Tesla, TR 100, TE 2.46, ST 4, 15 cc Magnevist contrast) which are black because they do not enhance; and are therefore completely subtracted from the pre- to post-contrast images. Axial NCCT image (f - bottom right; supine, GE LightSpeed Pro 16 slice scanner, kVp 120, mA 164, ST 5) demonstrates the biopsy trochar centered within the subcapsular mass.

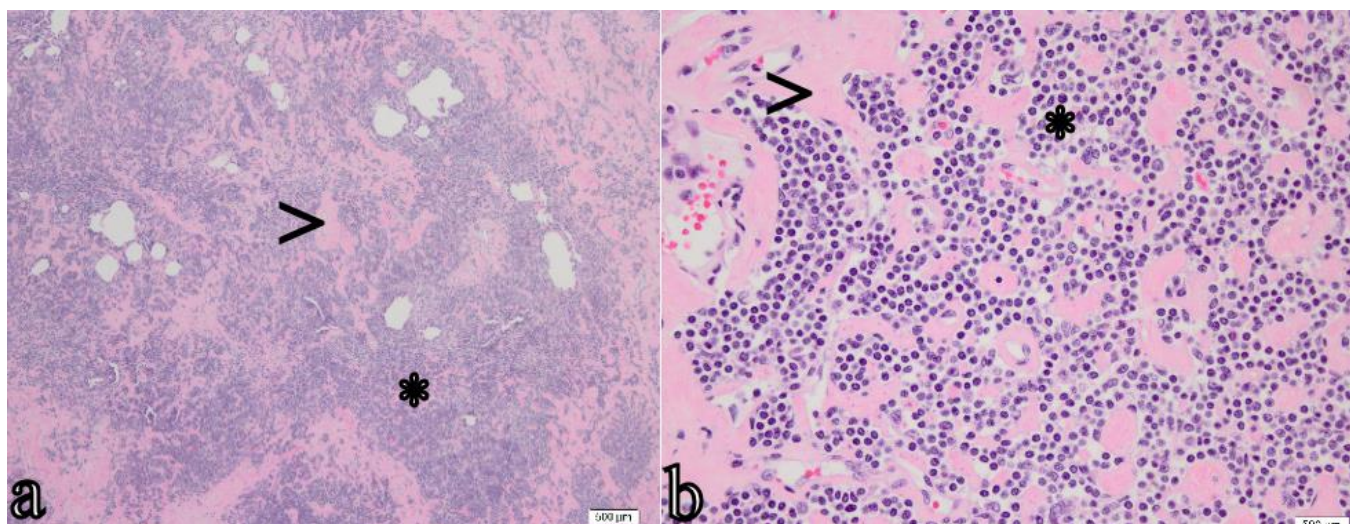


Figure 4: These are pathology images from a hematoxylin and eosin staining of a subcapsular renal mass in a 77 year-old male diagnosed with primary renal lymphoma. Low-power (4x) magnification (a - left) demonstrates small blue cells (asterisk) on a background of pink sclerotic stroma (arrowhead). Higher-power (40x) magnification (b - right) demonstrates small round lymphocytes (asterisk) infiltrating the pink sclerotic stroma (arrowhead).

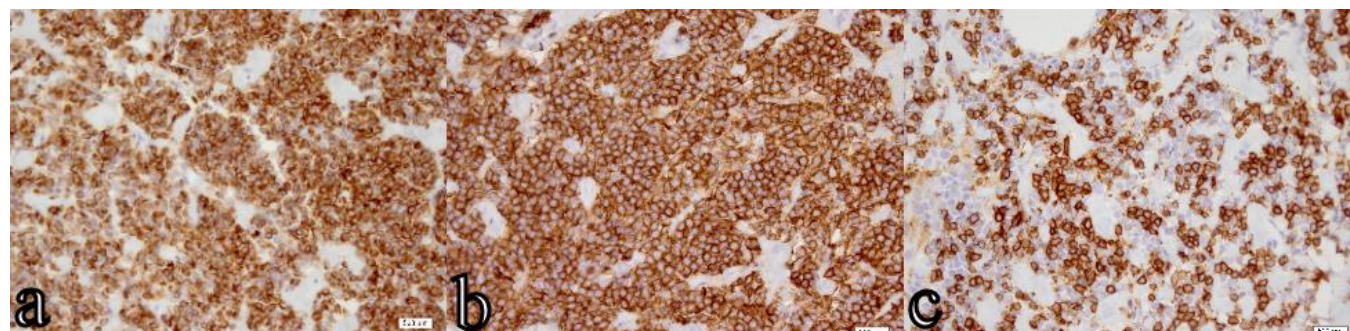


Figure 5: These are immunohistochemistry images of a 77 year-old male diagnosed with primary renal lymphoma. At 40x magnification, the cells of interest stain positive (reddish-brown) for bcl2 (a - left) and CD20 (b - middle), but negative (blue-purple) on CD5 (c - right). The cells taking up the CD5 stain (reddish brown) on sub-image (c) are immunophenotypically normal T cells. The other markers for the lymphoma were negative (bcl6, CD10, cyclin D1, CD3).

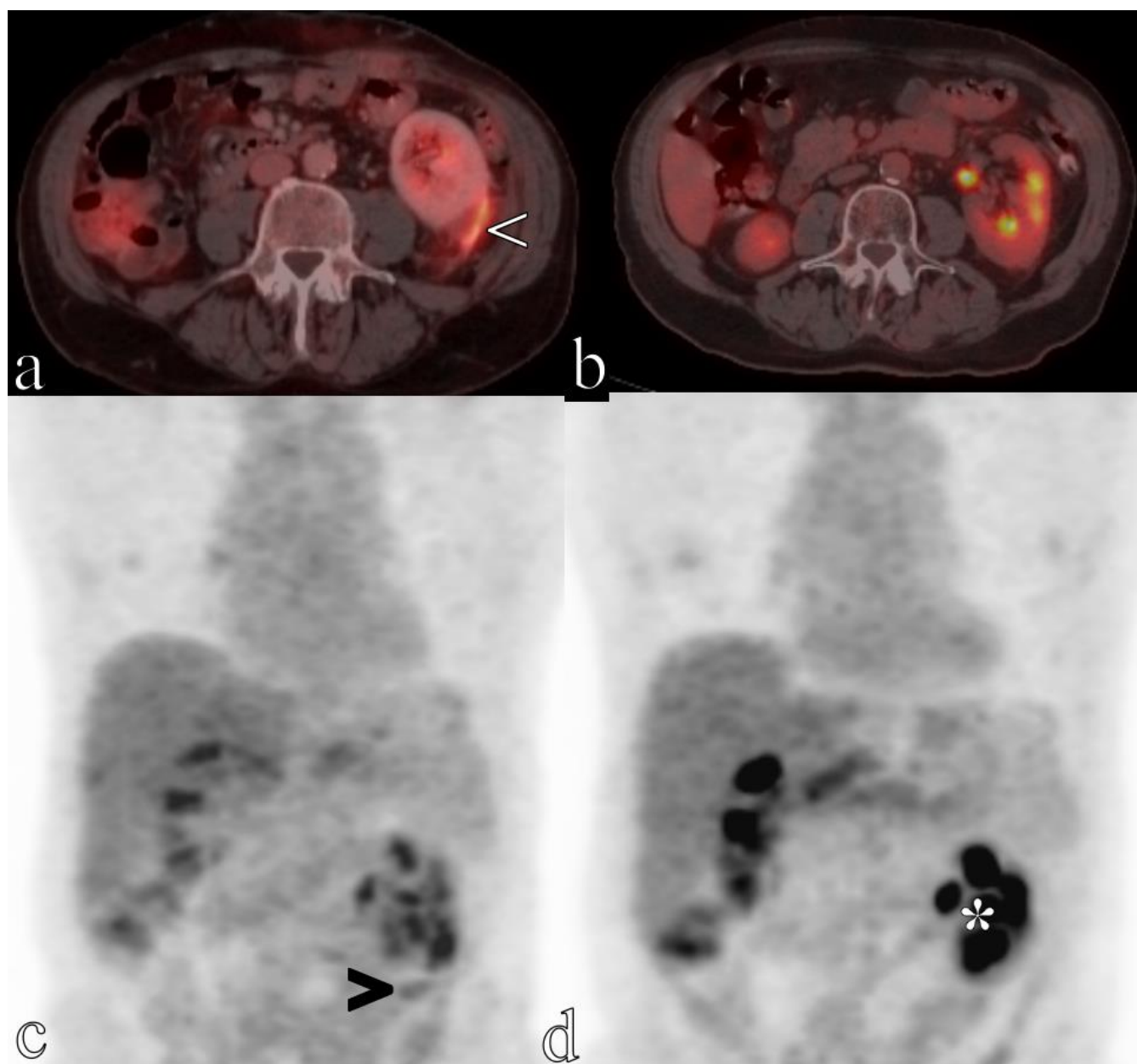


Figure 6: These are images of an 18FDG PET-CT scan immediately following the biopsy (a and c) and four months after completion of radiation in a 77 year-old male diagnosed with primary renal lymphoma. Fused-axial (a) and planar MIP (c) images (non-contrast CT - kVp 140, mA 80, ST 3.75mm; 19.7 mCi of 18FDG, and imaging 60 minutes post-injection) immediately following the biopsy demonstrate no extrarenal involvement. A thin curvilinear area of moderately increased activity (max SUV of 6.0) seen inferior and posterolateral to the mass (arrowheads) was attributed to a small post biopsy urine leak, and this resolved on the follow-up PET scan (b and d). Only mild activity (max SUV of 3.0), similar or slightly less than blood pool, is visualized within the subcapsular left renal mass. Follow-up, fused-axial (b) and planar MIP (d) 18FDG PET-CT images (non-contrast CT - kVp 140, mA 80, ST 3.75mm; 21.2 mCi of 18FDG, and imaging 60 minutes post-injection) 4 months after completion of radiation, demonstrates normal collecting system uptake (asterisk) and resolution of the thin curvilinear activity previously seen inferior and posterolateral to the subcapsular mass which was correctly attributed to a small post biopsy urine leak. No contralateral or extra-renal involvement was visualized on either PET scan.

Etiology	Autoimmune response, lymphomatous transformation of lymphoid cells in the renal capsule, and/or chronic renal inflammation recruits lymph cells into the kidney which then have the potential for lymphomatous transformation
Incidence	Primary renal lymphoma accounts for < 5% of all renal lymphoma and is otherwise very rare. Perirenal involvement occurs less than 10% of the time.
Gender Ratio	No gender predilection
Age	Middle-aged to elderly
Risk Factors	Immunosuppression, viral infections (such as EBV)
Treatment	Chemotherapy +/- radiation; or may consider resection if small and isolated to one kidney
Prognosis	~40-50% 5 year survival rate
Imaging Findings	On ultrasound, primary renal lymphoma is most often a hypovascular, hypoechoic-to-anechoic mass usually without posterior acoustic enhancement. On CT it appears as a homogenous, soft-tissue mass slightly more hyperdense than the adjacent renal parenchyma before contrast, and minimally enhances with contrast. On MR, it is hypointense on T2WI; hypo- to isointense on T1WI; and minimally enhances with contrast.

Table 1: Summary table of the key aspects of and imaging findings associated with primary renal lymphoma [5, 7, 9]

Differential	Ultrasound findings	CT findings	MR findings
Primary Renal Lymphoma	Hypovascular, hypoechoic-to-anechoic mass usually without posterior acoustic enhancement	Homogenous, soft-tissue mass slightly more hyperdense than the adjacent renal parenchyma before contrast, and minimally enhances with contrast	Hypointense on T2WI Hypo- to isointense on T1WI. Minimal contrast enhancement
Renal Cell Carcinoma	Round, iso- to hyperechoic, hypervascular mass becoming more heterogeneous in appearance with increasing size.	Hypervascular mass with enhancement less than the surrounding renal tissue best visualized on the nephrographic phase. RCC becomes more heterogeneous with increasing size	Usually isointense on T1WI. Iso- to hyperintense on T2WI. Enhances less than surrounding renal parenchyma and is best seen on post-contrast subtracted images
Renal or peri-renal/subcapsular abscess	Anechoic/hypoechoic complex cystic mass often with internal debris and occasionally “comet-tail” artifact when gas is present.	Low-attenuation mass with peripheral enhancement and possible gas collections	Peripherally enhancing mass possible signal voids representing gas collections.
Renal or peri-renal/subcapsular hematoma	Avascular, subcapsular mass with possible hematocrit-fluid level. Echogenicity will depend on age of blood products.	Subcapsular fluid collection with Hounsfield Units (HU) between 30-50. A sentinel clot or active extravasation may be seen. It is important to perform delayed (~10 min) images to evaluate for urine extravasation	Non-enhancing subcapsular mass with variable signal characteristics depending upon the age of blood products. Generally acute blood products will be isointense on T1WI and hypointense on T2WI

Table 2: Differential diagnosis table for primary renal lymphoma in a subcapsular location. This includes renal cell carcinoma, peri-renal hematoma, and peri-renal abscess [1-3, 5, 7-9].

ABBREVIATIONS

18FDG-PET: 18-fluorodeoxyglucose positron emission tomography
CECT: Contrast-enhanced, Computed tomography
EBV: Epstein-Barr virus
HU: Hounsfield Units
MRI: Magnetic resonance imaging
NECT: Non-enhanced, Computed tomography
PRL: Primary Renal Lymphoma
T1WI: T1 weighted imaging
T2WI: T2 weighted imaging
XRT: External-beam radiation therapy

KEYWORDS

Primary renal lymphoma; lymphoma; non-Hodgkin lymphoma of the kidney

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