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ORIGINAL ARTICLE

Renal pelvic and ureteral wall thickening in renal cell carcinoma: prevalence, cause, and clinical significance

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Abstract

Purpose This study was performed to characterize renal pelvic and ureteral wall thickening (PUWT) in renal cell carcinoma (RCC) patients, including prevalence, cause, and detailed radiological findings based on contrast-enhanced CT, and to correlate these features with corresponding pathological findings.

Materials and methods 152 patients pathologically diagnosed with RCC by surgery were included. Two radiologists retrospectively evaluated the presence of PUWT and the radiological characteristics based on CT. Relationships among clinical characteristics, CT findings, and PUWT were evaluated. Pathological findings of PUWT were also investigated.

Results The prevalence of RCC-related PUWT was 10% ($N=15$). PUWT was frequently observed in cases with advanced TNM stage. Tumor thrombus, sinus extent, and peritumoral neovascularity were significantly more frequent in cases with PUWT. PUWT was observed at the pelvis or from the pelvis to the upper ureter. Fourteen of 15 cases in which the pelvic/ureteral wall was pathologically investigated did not show any pathological abnormalities.

Conclusion The prevalence of PUWT was 10% and most cases were thought to be caused by temporary vascular dilatation in the pelvis and ureter, which is a secondary condition caused by hypervascular RCC, and not a direct result of tumor invasion.

Keywords Renal pelvic wall thickening · Ureteral wall thickening · Renal cell carcinoma · CT

Abbreviations

RCC	Renal cell carcinoma
UCSI	Urinary collecting system invasion
PUWT	Renal pelvic and ureteral wall thickening

Introduction

Renal cell carcinoma (RCC) is the most frequent renal malignancy and is commonly detected by ultrasonography or CT. Contrast-enhanced CT is an essential imaging modality to determine tumor characteristics, differentiate RCCs from benign renal tumors including angiomyolipoma and oncocytoma, and to evaluate tumor extent and pre-operative clinical TNM staging [1, 2].

Renal pelvic and ureteral wall thickening (PUWT), based on CT results, is caused by several types of diseases or conditions, both benign and malignant. Urothelial carcinoma is one common malignancy that causes PUWT, and this disease can manifest as a variety of characteristics including a papillary lesion, focal wall thickening, focal enhancement, or as an infiltrative lesion [3]. Moreover, various benign conditions, such as suburothelial hemorrhage, artifact due to breathing motion or ureteral peristalsis, postoperative state of cystectomy, inflammation related to reflux, urinary stasis, or intravesical treatment for a bladder tumor, can also cause PUWT [4, 5]. On pathological investigation in the patients with RCC, on the other hand, the urinary collecting system invasion (UCSI) of RCC is reported, ranging from 8 to 14% based and that UCSI serves as an independent prognostic parameter of adverse outcome [6, 7]. However, UCSI is not considered as a criterion in the current TNM staging.

In routine practice, PUWT is sometimes observed in RCC patients based on contrast-enhanced CT; however, a detailed description of PUWT with RCC has not been provided, and

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the prevalence, clinical characteristics, and detailed CT findings associated with this are still unclear. If this PUWT represents ureteral invasion of RCC or other urothelial malignancies, the surgical strategy or intervention could be altered. To address this issue, the pathological confirmation of pelvic and ureteral wall is thus indispensable. The purpose of this study was to clarify the prevalence, cause, and detailed radiological findings of PUWT based on contrast-enhanced CT in RCC patients and assess correlations among pathological findings.

Materials and methods

Patient selection

This study had institutional review board approval and informed consent was waived. We retrospectively performed a pathological database search and identified the records of 310 patients who underwent radical or partial nephrectomy for renal cell tumors at our institution between January 2011 and August 2016. Exclusion criteria included the diagnosis of benign tumors (angiomyolipoma, nephritis, oncocytoma, and benign cystic lesion) or malignant tumors other than RCC (metastasis, lymphoma, and urothelial cancer), cases with urinary tract infection, and cases for which adequate contrast-enhanced CT images were not acquired (as indicated in the following section). Finally, 152 patients were included in this study. As two patients had bilateral RCC and four patients had unilateral double RCC, 154 renal samples from 152 patients were included for radiological and pathological investigations. The following clinical information of these cases was retrospectively collected from hospital records: sex, age, laterality of RCC, tumor size based on pathological diagnosis (cm), method of diagnosis (radical nephrectomy, partial nephrectomy, or tumor biopsy), CT protocol. In cases with PUWT, the presence of hematuria and a history of anticoagulation therapy were recorded.

Imaging details

Contrast-enhanced dynamic CT images were obtained with CT systems (VCT64; GE Healthcare, Waukesha, Wis or Somatom Definition Flash; Siemens, Erlangen, Germany) with the following parameters: voltage, 120 kVP; amperage, 300–350 mA; rotation period, 0.5 s; detector collimation, 0.6–0.625 mm; pitch, 0.516–1.0; and section thickness, 2.5–3.0 mm. Further, 100–150 mL of contrast material with 600 mg of iodine per kilogram was injected for 30–35 s after pre-contrast scanning in our hospital. Scan protocols were different depending on the purpose of the examination ordered. The main scanning protocol for renal tumors consisted of a four-phase study for 93 patients. During four-phase studies, patients underwent an unenhanced scan of the abdomen to the iliac crest. Then, a corticomedullary phase image was acquired 31 s after the bolus tracking algorithm. For bolus tracking, a region of interest was set in the thoracoabdominal aorta junction with a trigger set to begin at 100 HU. Nephrographic phase imaging was performed 90 s after initiating the injection of contrast material. The excretory phase was obtained after a scanning delay of 5 min. Twenty-seven patients underwent other four-phase studies for general upper abdominal detailed examination. Patients underwent an unenhanced scan of the abdomen to the iliac crest; then, an arterial (corticomedullary) phase image was acquired 37 s after the start of injection or 17 s after the bolus tracking algorithm. For bolus tracking, a region of interest was set in the thoracoabdominal aorta junction with a trigger set to begin at 200 HU. Portal (nephrographic) phase imaging was performed 70 s after initiating the injection of contrast material. The equilibrium (excretory) phase was obtained after a scanning delay of 3 min. Twenty-two patients underwent three-phase studies including unenhanced CT, corticomedullary phase imaging performed 35 s after the start of injection, and acquisition of the excretory phase after a scanning delay of 3 min. Twelve patients who underwent CTs at outside institutions lacked records regarding the amount and injection rate of iodinated contrast material and scanning delay time. Based upon the appearance of normal kidney parenchyma, images were classified as pre-contrast phase if contrast was absent, corticomedullary phase if the renal cortex but not medulla was enhanced, nephrographic phase if the cortex and medulla were enhanced in a uniform manner, or excretory phase if the contrast material was excreted from the renal pelvis and ureters after the prior phases.

Imaging analysis

Two radiologists, with 16 years and 6 years of experience in genitourinary imaging, retrospectively interpreted all CT images using a picture archiving and communications system. The radiologists evaluated the CT findings without any knowledge of the results of other examinations. Each reader evaluated the presence of PUWT in all phases. PUWT was defined as a thickened renal pelvic and/or ureteral wall of more than 2 mm with noticeable distinction based on contrast-enhanced CT at any phase as compared to that of the ipsilateral pelvic/ureteral wall. Each reader evaluated the renal masses based on contrast-enhanced dynamic CT for the pattern of RCC enhancement, the presence of sinus extent, venous thrombus, and peritumoral neovascularity. When the decision of the two radiologists differed, the final evaluation was made by consensus.

The pattern of enhancement of the renal tumor was classified as hypervascular if the solid area within the tumor was

enhanced to a similar extent or more than the renal cortex at the corticomedullary phase or hypovascular if areas within the tumor were less enhanced than renal cortex. Sinus extent was considered positive if the tumor attached to the adjacent sinus fat tissue at the renal sinus. Venous thrombus was defined as positive if visually the tumor extended to the renal vein or inferior vena cava. Peritumoral neovascularity was defined as positive if dilated vessels at the perirenal space were visualized. The presence of PUWT was evaluated in parallel with RCC characterization. For cases with the presence of PUWT, the following detailed variables of PUWT were also evaluated: (1) type of abnormality (focal, longitudinal, or infiltrative mass) (2) lesion area (renal pelvis, renal pelvis-ureter, or ureter) (3) wall thickness (mm) based on axial images (4) longitudinal length (mm) (5) shape (circular, semicircle, or partial) (6) CT density on unenhanced CT (hypodense, isodense, hyperdense) (7) enhancement pattern (early enhancement/washout, persistent, gradual enhancement, or faint enhancement) (8) intra-ureteral protuberance (presence or absence) (9) hydronephrosis (presence or absence).

Pathological findings

The following pathological information was collected from hospital records: morphological classification (clear cell carcinoma, papillary cell carcinoma, chromophobe cell carcinoma, or others), nuclear grade (low or high grade), presence of sarcomatoid features, tumor necrosis, lymphovascular invasion, pelvicalyceal invasion, and sinus invasion. The nuclear grade description was not found in 12 cases. The pathological TNM stage was also recorded. When the pathological TNM stage was not investigated, the clinical TNM stage was applied. Tumor stage and grade were determined according to the 7th AJCC TNM staging (Grade 1, 2: low, Grade 3: high). The histological subtypes of RCC were assigned according to the 2016 World Health Organization classification system. For patients who underwent tumor biopsy, morphological classification and nuclear grade were evaluated. For cases with PUWT based on CT, the resected affected pelvic/ureteral wall was investigated pathologically and intraoperative findings were collected.

Statistical analyses

Statistical analyses were evaluated using JMP 14.0.0 software (SAS Institute, Cary, NC). The chi-square test, Fisher's exact test, and Mann–Whitney U test were used to analyze the relationship between the presence of ureteral wall thickening and clinical, radiological, and pathological features. P-values less than 0.05 were considered statistically significant. Interreader agreement was assessed for qualitative radiological findings by calculating the kappa value, which was interpreted as follows; poor agreement, <0.20; fair agreement, 0.21–0.40; moderate agreement, 0.41–0.60; good agreement, 0.61–0.81; excellent agreement, 0.81–1.00.

Results

Of the 152 patients included in this study, 113 were males and 39 were females. The mean age at the time of nephrectomy or biopsy was 61.4 years (range 23–83 years). Based on the imaging evaluation of contrast-enhanced dynamic CT, PUWT was observed in 15 of 154 kidneys among 152 cases (10%, $\kappa=0.926$). Table 1 shows the characteristics of renal tumors in the two groups (presence or absence of PUWT). The age of patients with PUWT was higher than that of patients without PUWT (66.7 vs 60.8 years; $p<0.05$), and the tumor size was significantly larger in patients with PUWT (8.4 vs 3.1 cm; $p<0.01$). The affected kidneys of the PUWT group did not have lateralization.

All 15 cases with PUWT were pathologically diagnosed as clear cell carcinoma (Table 2). Nuclear grade was high in four cases and low in 11 cases in patients with PUWT, whereas grade was high for seven cases and low for 127 cases without PUWT ($p<0.01$). Other pathological findings of tumor necrosis, lymphovascular invasion, pelvicalyceal invasion, and sinus invasion were more frequently observed in cases with PUWT than in those without PUWT (in seven, 15, four, and 12 cases among 15 cases of PUWT, respectively). The cases with PUWT were significantly more advanced based on pathological T and N stage than those without PUWT.

Based on CT findings, RCCs in all 15 cases with PUWT were hypervascular (100%), whereas 117 of 139 cases without PUWT (84.2%) were hypervascular (Table 3). Sinus extent, venous thrombus, and peritumoral neovascularity were more frequently observed in cases with PUWT than in cases without PUWT. The agreement between the two readers was excellent for RCC vascularity ($\kappa=0.898$), venous thrombus ($\kappa=0.969$, and peritumoral neovascularity ($\kappa=0.811$), and good for sinus extent ($\kappa=0.716$). None of the PUWT cases had hematuria or received anticoagulant therapy. Table 4 shows the detail findings of PUWT based on dynamic CT. All cases of PUWT were categorized as longitudinal shape, and the PUWT was located at the pelvis in eight cases and from the pelvis to the upper ureter in seven cases. The thickness of the ureter was 3.7 mm (range, 2–7 mm) and mean longitudinal length was 51 mm (range, 15–115 mm). The lesion was circular in all cases. PUWT was isodense on unenhanced CT in all cases. The pattern of enhancement for PUWT was early enhancement/washout in ten and gradual enhancement in five cases (Figs. 1, 2). Ureteral wall protuberance into the ureteral lumen was observed

Table 1 Demographic and characteristics data of patients with renal cell carcinoma

	All ($n=154$)	Presence of PUWT ($n=15$)	Absence of PUWT ($n=139$)	p value
Gender[#]				0.60
Male	74.3% (113)	80.0% (12)	73.7% (101)	
Female	25.7% (39)	20.0% (3)	26.3% (36)	
Mean age (years) *	61.4 (23–83)	66.7 (45–81)	60.8 (23–83)	0.03
Location[#]				0.99
Right	52.0% (79)	53.3% (8)	51.8% (71)	
Left	46.7% (71)	46.7% (7)	42.1% (64)	
Bilateral	1.3% (2)	0% (0)	1.3% (2)	
Mean tumor size (cm) *	3.6 (0.7–12.0)	8.4 (4.5–12)	3.1 (0.7–10.0)	<0.01
Method of diagnosis				
Radical nephrectomy	49.4% (76)	100% (15)	43.9% (61)	
Partial nephrectomy	50.6% (78)	0% (0)	56.1% (78)	
CT scan prptocol				
Renal protocol	60.4% (93)	73.3% (11)	59.0% (82)	
Four phase scan	17.5% (27)	13.3% (2)	18.0% (25)	
Three phase scan	14.3% (22)	0% (0)	15.8% (22)	
Outside data	7.8% (12)	13.3% (2)	7.2% (10)	

Data in parentheses are number of renal units

PUWT renal pelvic and ureteral wall thickening

[#]Data was calculated under number of patients ($n=152$)

[*]Data in parentheses are the ranges

as a filling defect based on the excretory phase for one case. Hydronephrosis was not observed in any of the cases.

In 15 surgically-confirmed cases, the specimens of renal pelvis and upper ureter revealed no definitive pathologically thickened pelvic/ureter wall, and tumor cell infiltration was not observed (Fig. 2c). Acute or chronic inflammatory change was also not detected. No abnormal findings from pelvis to ureter were noted during operation. In one case, a tumor thrombus in the dilated vein at the intraureteral wall (5 mm in diameter) was detected at the proximal ureter, that showed polypoid lesion macroscopically. The protuberance of the ureteral wall was observed on CT in this case (Fig. 3), and it was corresponded with the tumor thrombus pathologically.

Discussion

The present study identified PUWT in 10% of RCC cases. For RCC with PUWT, tumors were larger than those of RCC without PUWT and all were diagnosed as clear cell carcinoma. Pathologically, high nuclear grade and several invasive factors such as tumor necrosis, lymphovascular invasion, pelvicalyceal invasion, and sinus invasion were more frequently observed in cases with PUWT than in cases without PUWT.

Surgical removal of the primary tumor is the fundamental treatment strategy for T1–T3 tumors. Further, in some Stage IV patients with potentially surgically-resectable primary tumors with a solitary metastatic site, nephrectomy and surgical metastasectomy are also considered. Recently, cytoreductive nephrectomy was recommended for patients with good performance status even when immunotherapy is indicated [8, 9]. Thus, determining the exact tumor extent is important for the management of RCC.

RCC-related PUWT was associated with several characteristics in this study. First, PUWT occurred at the renal pelvis or from the renal pelvis to the ureter. Some of the thickened pelvic/ureteral wall was attached to the RCC itself extending to the sinus. Second, most lesions were circular but did not cause hydronephrosis. Third, the thickened urinary tract tended to show early enhancement and subsequent washout based on the delayed phase. Interestingly, none of the pathological specimens of the urinary tract obtained from 15 cases showed pathological wall thickening or tumor cell and inflammatory cell infiltration. Abnormal PUWT based on CT was not identified pathologically.

Considering these findings, this PUWT was suggested to be caused by temporary non-morphological changes such as arterial/venous dilatation due to hyperemic or congestive change. In cases of RCC with venous thrombus, the original pathway of venous flow was blocked and there was the formation of compensatory dilated underlying vasculature

Table 2 Pathological findings of renal tumors

	All ($n=154$)	Presence of PUWT ($n=15$)	Absence of PUWT ($n=139$)	p value
Morphological classification				0.80
Clear cell carcinoma	85.1% (131)	100% (15)	83.5% (116)	
Papillary cacinoma	7.1% (11)	0% (0)	7.9% (11)	
Chromphobe cell carcinoma	3.2% (5)	0% (0)	3.6% (5)	
Other	4.6% (7)	0% (0)	5.0% (7)	
Nuclear grade	(142)	(15)	(127)	<0.01
Low	93.0% (131)	73.3%(11)	94.4% (120)	
High	7.0% (11)	26.7%(4)	5.6% (7)	
Sarcomatoid features	(154)	(15)	(139)	0.30
Present	2.6% (4)	6.7% (1)	2.2% (3)	
Tumor necrosis	(154)	(15)	(139)	<0.01
Present	11.7% (18)	46.7% (7)	7.9% (11)	
Lymphovascular invasion	(154)	(15)	(139)	<0.01
Present	44.8% (69)	100% (15)	38.8% (54)	
Pelvicalyceal invasion	(154)	(15)	(139)	<0.01
Present	3.2% (5)	26.7% (4)	0.7% (1)	
Sinus invasion	(154)	(15)	(139)	<0.01
Present	13.6% (21)	80.0% (12)	6.5% (9)	
T stage				
T1	74.7% (115)	0% (0)	82.7% (115)	<0.01
T2	1.9% (3)	0% (0)	2.2% (3)	
T3	22.1% (34)	93.3% (14)	14.4% (20)	
T4	1.3% (2)	6.7% (1)	0.7% (1)	
N stage				0.74
N0	99.4% (153)	100% (15)	99.3% (138)	
N1	0.6% (1)	0% (0)	0.7% (1)	

Data in parentheses are number of renal units

PUWT renal pelvic and ureteral wall thickening

Table 3 CT findings of renal tumors

	All ($n=154$)	Presence of PUWT ($n=15$)	Absence of PUWT ($n=139$)	p value
Vascularity				0.09
Hypervascular	85.7% (132)	100% (15)	84.2% (117)	
Hypovascular	14.3% (22)	0% (0)	15.8% (22)	
Sinus extend	43.5% (67)	100% (15)	37.4% (52)	<0.01
Venous thrombus	12.3% (19)	93.3% (14)	3.6% (5)	<0.01
Peritumoral neovascularity	22.7% (35)	100% (15)	14.4% (20)	<0.01

PUWT renal pelvic and ureteral wall thickening

at the perirenal fat and periureteric vein. Here, this hemodynamic change due to renal vein thrombus caused PUWT, and thus represents an alternative vascular pathway. The possible reason why pathological analysis did not clarify these findings was that the dilated intraureteral artery/vein was temporary and this collateral vein could shrink and disappear during the operative procedure including renal artery or renal vein ligation.

Varicosis or varices of the renal pelvis and ureter were reported in the late 1900s [10–12]. Most of these reports were based on observations of intravenous pyelography or retrograde pyelography. Varicosis and varices of the renal pelvis and ureter are speculated to be caused by congenital venous wall weakness, venous valve incompletion, or secondary venous pressure increases due to renal vein obstruction. Further, it was demonstrated that drip infusion

Table 4 CT characters of renal pelvic and ureteral wall thickening

Case number	Lesion area	Thickness (mm)	Length (mm)	Shape	CT attenuation	Enhancement pattern	Protuberance	Hydronephrosis	Pathologic finding
1	Pelvis	5	50	Circular	iso	Early enhancement/washout	No	No	No findings
2	Pelvis-upper	2	48	Circular	iso	Early enhancement/washout	No	No	No findings
3	Pelvis-upper	5	93	Circular	iso	Gradual enhancement	No	No	No findings
4	Pelvis	2	20	Circular	iso	Early enhancement/washout	No	No	No findings
5	Pelvis	3	15	Circular	iso	Gradual enhancement	No	No	No findings
6	Pelvis-upper	6	106	Circular	iso	Early enhancement/washout	Yes	No	Tumor thrombus
7	Pelvis-upper	2	40	Circular	iso	Early enhancement/washout	No	No	No findings
8	Pelvis	3	27	Circular	iso	Early enhancement/washout	No	No	No findings
9	Pelvis-upper	7	115	Circular	iso	Gradual enhancement	No	No	No findings
10	Pelvis	2	50	Circular	iso	Gradual enhancement	No	No	No findings
11	Pelvis	2	26	Circular	iso	Early enhancement/washout	No	No	No findings
12	Pelvis	6	40	Circular	iso	Early enhancement/washout	No	No	No findings
13	Pelvis	2	16	Circular	iso	Early enhancement/washout	No	No	No findings
14	Pelvis-upper	4	60	Circular	iso	Gradual enhancement	No	No	No findings
15	Pelvis-upper	4	72	Circular	iso	Early enhancement/washout	No	No	No findings

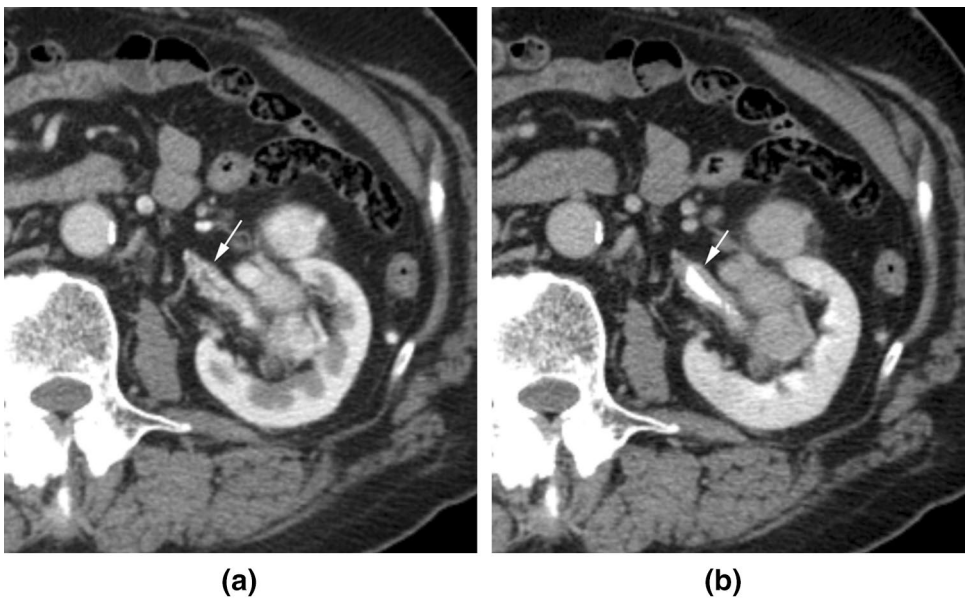

Fig. 1 80-year-old male with clear cell carcinoma of left kidney (case 15). **a** Contrast-enhanced CT shows well-enhanced irregular wall thickening at the renal pelvis to the upper ureter at the corticomedullary phase (arrow). **b** The thickened wall is isodense at the excretory phase without hydronephrosis (arrow)

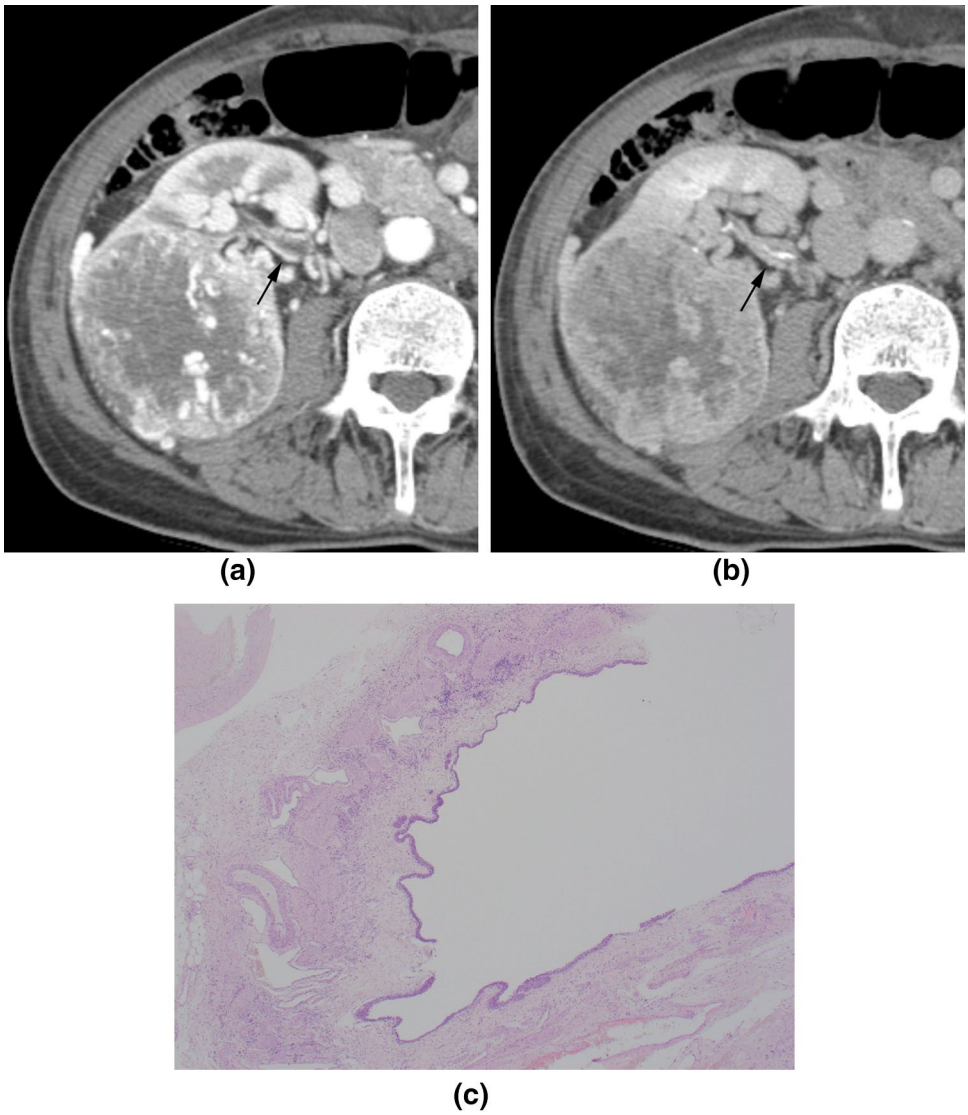

Fig. 2 73-year-old male with clear cell carcinoma of right kidney (case 13). **a** Contrast-enhanced CT shows well-enhanced ureter wall thickening from the renal pelvis to the upper ureter at the corticomedullary phase (arrow). **b** The thickened wall is isodense at the excretory phase without hydronephrosis (arrow). **c** The specimens of renal pelvis revealed no definitive pathologically thickened pelvic wall, and no tumor cell infiltration (H&E, 20 ×)

pyelography or retrograde pyelography shows pyelocaliceal deformity or scalloping of the ureter, suggesting extrinsic compression. At surgery, tortuous engorgement of the veins of the ureteral mucosa is observed [10, 11]. Although varicosis and varices of the renal pelvis and ureter are related to renal vein thrombosis or portal hypertension, the relationship between varicosis/varices and RCC has not been well documented.

Generally, upper urinary tract wall thickening is caused by both tumorous and non-neoplastic pathology. Renal pelvic carcinoma and ureteral carcinoma are the most common urinary tract tumors, and pathologically, most are urothelial carcinomas [6]. Further, non-neoplastic pathology of the urinary tract varies based on normal enhancement after cystectomy, inflammatory changes originating from the recent use of instrumentation, the presence of a ureteral stent, radiation therapy, endometriosis, breathing motion, or ureteral peristalsis. Moreover, cases with ureteral carcinoma often result in hydronephrosis. In our cases of PUWT, the renal pelvic/ureteral wall showed circular thickening without hydronephrosis. Suburothelial hemorrhage is one of the causes of the pelvic/ureteral wall thickening observed in patients receiving anticoagulation therapy. Patients with suburothelial hemorrhage often complain of hematuria, and suburothelial hemorrhage is identified as a hyperdense ureteral thickening at the renal pelvic urothelium on unenhanced CT [9]. In our cases of PUWT, no patients had hematuria and a history of anticoagulation therapy, and PUWT was not hyperdense.

RCC frequently tends to extend along the local anatomical structure including the perinephric tissue, renal sinus, Gerota's fascia, and pelvicalyceal system. Recent studies have found that the frequency of UCSI in patients with RCC ranges from 8 to 14% based on pathological investigation and that UCSI serves as an independent prognostic parameter of adverse outcome [5, 6]. Karlo et al. reported that UCSI was identified in 5% of patients

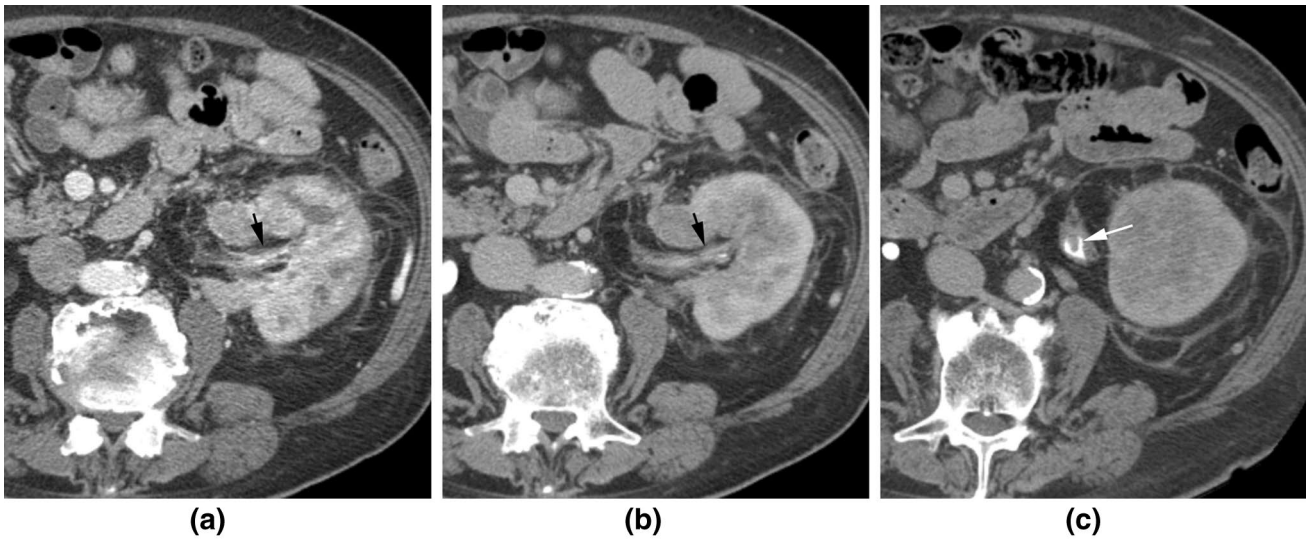

Fig. 3 59-year-old male with clear cell carcinoma of left kidney (case 7). **a** Contrast-enhanced CT shows well-enhanced wall thickening from the renal pelvis to the upper ureter at the corticomedullary phase (arrow). **b** The thickened wall is isodense at the excretory phase (arrow). **c** At the upper ureter, protuberance of the ureter wall is identified (arrow)

with RCC and was visualized as a filling defect within the collecting system based on excretory phase CT images [13]. In our study, pathological pelvicalyceal system invasion was detected at a rate of 3%. Whereas approximately 27% of cases with PUWT were positive for pelvicalyceal invasion pathologically, the remaining cases did not have pathological pelvicalyceal system invasion (Table 2), and radiologically-detected PUWT was determined to be non-tumorous in etiology.

There are several limitations to this study. First, biopsy-proven cases and clinically diagnosed cases were not included in this study. Therefore, the prevalence of PUWT might be underestimated, as PUWT was frequently observed in cases with advanced TNM stage. Second, whereas most PUWT comprised non-tumorous lesions, one case presented with co-existing tumor thrombus in the dilated intraureteral vein. As the protuberance into the ureteral lumen was observed only in this case, the protuberance on CT may be a sign of tumor thrombus in the dilated intraureteral vein.

In conclusion, the prevalence of PUWT was observed clinically, based on preoperative CT in RCC at a rate of 11%. Most PUWTs based on CT are thought to be caused by temporary vascular dilatation in the renal pelvis and ureter, which is a secondary condition caused by hypervascular RCC and not a direct result of tumor invasion.

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Compliance with ethical standards

Conflict of interest None of the authors have any relevant conflict of interest or industry support related to this project.

Ethical statement This study had institutional review board approval and informed consent was waived.

References

1. Vikram R, Beland MD, MD, Blaufox CC, Moreno JL, Gore HJ, Harvin ME et al. ACR appropriateness criteria renal cell carcinoma staging. J Am Coll Radiol. 2016;3(5):518–25.
2. Hallscheidt PJ, Bock M, Riedasch G, Zuna I, Schoenberg SO, Autschbach F, et al. Diagnostic accuracy of staging renal cell carcinomas using multidetector-row computed tomography and magnetic resonance imaging: a prospective study with histopathologic correlation. J Comput Assist Tomogr. 2004;28(3):333–9.
3. Froemming A, Potretzke T, Takahashi N, Kim B. Upper tract urothelial cancer. Eur J Radiol. 2018;98:50–60.
4. Takahashi N, Kawashima A, Glockner JF, Hartman RP, Kim B, King BF. MR urography for suspected upper tract urothelial carcinoma. Eur Radiol. 2009;19(4):912–23.
5. Gayer G, Desser TS, Hertz M, Osadchy A, Daniel BL, Zissin R. Spontaneous suburothelial hemorrhage in coagulopathic patients: CT diagnosis. AJR Am J Roentgenol. 2011;197(5):W887–W89090.
6. Anderson CB, Clark PE, Morgan TM, Stratton KL, Herrell SD, Davis R, et al. Urinary collecting system invasion is a predictor for overall and disease-specific survival in locally invasive renal cell carcinoma. Urology. 2011;78(1):99–104.

7. Verhoest G, Avakian R, Bensalah K, Thuret R, Ficarra V, Artibani W, et al. Urinary collecting system invasion is an independent prognostic factor of organ confined renal cell carcinoma. J Urol. 2009;182(3):854–9.
8. Flanigan RC, Salmon SE, Blumenstein BA, Bearman SI, Roy V, McGrath PC, et al. Nephrectomy followed by interferon alfa-2b compared with interferon alfa-2b alone for metastatic renal-cell cancer. N Engl J Med. 2001;345(23):1655–9.
9. Mickisch GH, Garin A, van Poppel H, de Prijck L, Sylvester R, European Organisation for R, et al. Radical nephrectomy plus interferon-alfa-based immunotherapy compared with interferon alfa alone in metastatic renal-cell carcinoma: a randomised trial. Lancet. 2001;358(9286):966–70.
10. Pearson JC, Tanagho EA, Palubinskas AJ. Nonoperative diagnosis of pyelocalyceal deformity due to venous impressions. Urology. 1979;13(2):207–10.
11. Beckmann CF, Abrams HL. Idiopathic renal vein varices: incidence and significance. Radiology. 1982;143(3):649–52.
12. Camuzzi FA, Kleinman SZ, Politano VA. Ureteral varices. Urology. 1980;15(6):588–9.
13. Karlo CA, Di Paolo PL, Hricak H, Tickoo SK, Russo P, Akin O. CT of renal cell carcinoma: assessment of collecting system invasion. AJR Am J Roentgenol. 2013;201(6):W821–W827827.