Primary small cell carcinoma of the ureter
Case report and review of the literature

Fabiola Farci, MD[a,], Francesca Manassero, MD[b], Ramona Baldesi, MD[c], Annamaria Bartolucci, MD[a], Laura Boldrini, PhD[a,], Cesare Sellì, MD[c], Pinuccia Faviana, MD[c]

Abstract
Rationale: Primary small cell carcinoma of the ureter is extremely rare, in this case report is meticulously described its aggressive clinical course and the pathological clues that help with the diagnosis. Also, a detailed table with the clinico-pathological features of analogous case reports in literature is provided.

Patient concerns: A 79-year-old female presented with gross hematuria and flank pain.

Diagnoses: Small cell carcinoma of the ureter. The surgical specimen showed a mixed histology of small cell carcinoma and transitional cell carcinoma; the common neuroendocrine markers (chromogranin A, synaptophysin, CD56) were positive, and vimentin and thyroid transcription factor 1 were negative. The patient had an advanced stage at presentation with regional nodes involvement (pT3N1).

Interventions: Segmental ureterectomy was performed but it was only possible to administer 1 cycle of platinum-based adjuvant chemotherapy due to the rapid decline of her clinical parameters.

Outcomes: The disease rapidly spread locally and metastasized.

Lesson: The clinicians must be aware of this aggressive tumor with silent clinical course and advanced stages at presentation.

Abbreviations: CT = computed tomography, CK = cytokeratin, SCC = small cell carcinoma, TTF-1 = thyroid transcription factor 1.

Keywords: carcinoma, case report, mixed histotype, neuroendocrine, pathology, small cell, urology

1. Introduction

Primary small cell carcinoma (SCC) of the urinary tract is a rare cancer, accounting for less than 0.5% of urinary tract tumors,[1] mostly localized in the bladder and prostate, while its localization in the renal pelvis or in the ureter is extremely rare. Smoking exposure causes reactive and genetic damage to the tissues, and is a main risk factor for urothelial carcinoma and small cell neuroendocrine carcinoma.[2,3] Being such a rare disease, the pathogenesis is still unclear, and 2 theories have been postulated. The first claims its origin from a neuroendocrine cell population derived from the neural crest (enterochromaffin cells) migrated in the genitourinary tract during embryogenesis; the second theorizes its genesis from the pluripotent epithelial cells of the genitourinary tract. The latter could explain the common finding of a mixed histologic profile (transitional cell carcinoma, squamous cell carcinoma, adenocarcinoma, sarcomatoid carcinoma, and sarcoma), often described as a gradual transition from 1 subtype to the other.

Small cell carcinoma of the ureter has been described in about 40 patients so far,[4–34] with similarities in symptoms, management, and outcome, as outlined in Table 1.

2. Case report

A 79-year-old female presented with right-sided back pain and gross hematuria. Her clinical history was significant for atrial fibrillation treated with oral anticoagulant, which was suspended due to hematuria. She had a smoking history of more than 20 cigarettes per day for nearly 60 years. Physical examination revealed pain at the right costovertebral angle extended to the right groin over the location of the ureter. Creatinine levels at the admission were 1.37mg/dL. Abdominal ultrasound was immediately performed, revealing a grade 3 right hydronephrosis without lithiasis and hematic material in the bladder. A functional scintigraphy study with 99mTc-diethylene-triamine-penta-acetate revealed a decreased function of the right kidney, and the calculated glomerular filtration rate (GFR, Gates method) was 27mL/min for the right kidney and 40mL/min for the left kidney. At cystoscopic examination, the bladder wall was irregular in the right emitrigon, and the right ureteral orifice was swollen and bleeding. Urine cytology showed atypical morphological features, classified as suspicious for high-grade urothelial carcinoma (the Paris System for reporting urinary cytology). The following abdominal computed tomography
| A: References | B: Age/sex | C: Smoking | D: Symptom | E: TNM, size | F: Surgical treatment | G: Adjuvant therapy | H: Immunohistochemistry | I: Associated histotypes | J: Follow-up (post-surgery) |
|--------------|-------------|------------|------------|--------------|----------------------|---------------------|------------------------|-------------------------|--------------------------|
| Current report | 79, F ca    | Heavy smoker | FP, GH       | pT3N1Mx (2 cm) | U + LA | Et (1 cyc) | chrA+, syn+, CD56+, TF1+, vim- | SCC, UC | 3 mos local recurrence, DOD |
| Zhong et al, 2017 | 62, M as | No | None | T3N0M0 (10.5 cm) | NJ | Yes | NSE+, chrA+, syn+ | SCC, UC, S, Ly | 4 mos DOD |
| 64, F as | No | GH | T3N1M0 (2 cm) | NJ + LA | Yes | NSE+, chrA+, syn+ | SCC | 9 mos NOD |
| 62, F as | No | GH | T3N0M0 (3 cm) | NJ + LA | None | NSE+, chrA+, syn+ | SCC | 6 mos NOD |
| 56, M as | No | GH | T3N0M0 (2.5 cm) | NJ | None | NSE+, chrA+, syn+ | SCC, UC, S | 6 mos NOD |
| Hensley et al, 2017 | 89, M ca | Yes | FP, GH | Not stated | Biopsy | None | CAM 3.2+ | SCC | 2 mos, local recurrence |
| 67, F ca | No | FP, GH | cT4N1M1 | NJ + L | cisPt + (4 cyc), RT | syn+, chrA+, panok+ | SCC | Metastatic (adrenal glands, pelvis), 7 mos DOD |
| Alevizopoulos et al, 2016 | 78, M ca | Not stated | GH | cT3N1M1 (4.3 cm) | Refused | Refused | CD56+, CK7-, CK20- | SCC | 13 mos DOD |
| Sood et al, 2016 | 55, F | No | FP, GH | cT3N2M0 | Planned | cisPt + (neoAd) | syn+ | SCC | 3 mos NED |
| Ueda et al, 2016 | 63, M as | Not stated | GH | T3N0M1 | NJ | cisPt + (3 cyc) | Not stated | SCC | UC | 12 mos, Local recurrence (bladder) at 3 mos. |
| Wang et al, 2016 | 69, M as | Not stated | FP, GH, HUN | cT3N0M1 (3.5 cm) | NJ | Refused | CD56+, syn+, EMA+, CK7+, chrA+, syn+, K67 20% | SCC | Metastatic; 12 mos DOD |
| Acosta et al, 2015 | 71, F ca | Not stated | FP | pT3N1Mx (4.3 cm) | NJ | cPt + (neoAd) | syn+, TF1+, chrA+, syn+, CD56+ | SCC | 6 mos DOD |
| Osaka et al, 2015 | 70, M as | Yes | FP | cT3N0M0 (before neoAd) | cT2N0M0 (after neoAd) | NeoAd | AE1/3+, CK7+, syn+, CD56+ | SCC | 38 mos NED |
| Ahzaini et al, 2013 | 54, M, af | Yes | GH | T1N0M0 | NeoAd | (I, II, III, IV, cisPt) | syn+, chrA+, syn+, CD56+, NSE+ | SCC | 24 mos NED |
| Yang et al, 2013 | 59, M as | Heavy smoker | HUN | T3N0Mx (3.5 cm) | NJ | cisPt + (4 cyc), RT (180cGyX30) | chrA+, syn+, CD56+, NSE+ | SCC, HGUC | 10 mos NED |
| Ping et al, 2013 | 65, F as | Not stated | FP | NA (5 cm) | NJ | cisPt + (4 cyc) | chrA+, syn+, CD56+, K67 67% | SCC | 4 mos NED |
| Zhao et al, 2012 | 70, F as | Not stated | FP, HUN | cT3N0M1 (1.6 cm) | NJ | Not stated | OK7+, chrA+, syn+, NSE+ | SCC | Metastatic (liver, lungs, lymph) at 9 mos. Died at 10 mos (MDF) |
| Miller et al, 2011 | 80, F | Not stated | GH | T3NnxM1 | None | None | chrA+, syn+, CD56+ | SCC | Died at 4 mos (M) |
| 73, F | Not stated | GH | T3N0M0 | None | None | chrA+, syn+, CD56+, TF1+ | SCC | 5 mos DOD |
| Patil et al, 2011 | 75, M | Yes | FP | cT3N1M1 | NJ | Referred | chrA+, syn+, CD56+, TF1+ | SCC | 10 mos DOD |
| Kho and Chan, 2010 | 77, M as | Heavy smoker | FP | T3N0M0 (2 cm) | NJ | cisPt + (3 cyc) | chrA+, syn+, CD56+, NSE+ | SCC | 2 mos DOD |
| Kozyrakis et al, 2009 | 78, M as | Heavy smoker | GH | pT2N3M0 (7.7 cm) | NJ | None | chrA+, syn+, CD56+, K67 70% | SCC, UC, Sq | Metastatic lung, 6 mos DOD |
| Kuroda et al, 2009 | 79, M as | Not stated | GH | T2N0Mx (3.7 cm) | NJ | None | chrA+, syn+, CK7-, graminelus, CD56+, S100 | SCC, UC | 36 mos NED |
| Terada, 2009 | 48, M as | Not stated | FP | cT3N0M0 (1.5 cm) | U | None | OK7+, CD56+, PDGFRa+, chrA-, syn-, NSE- | SCC | 24 mos DOD |
| Ito et al, 2009 | 84, F as | Not stated | NA | Not stated | NA | None | NA | SCC | Not stated |
| Banerji et al, 2009 | 55, M | Not stated | FP | pT3N0M0 | NA | cisPt + (6 cyc) | chrA+, syn+ | SCC | Not stated |
| Maizoi et al, 2009 | 69, M | Not stated | NA | Not stated | NA | CT, RT | NA | SCC | 14 mos NED |
| Ryu et al, 2008 | 78, M as | Not stated | FP, GH, HUN | pT3N1M0 (5 cm) | U, LA | None | AE1/3+, EMA+, N-CAM+, chrA+; syn+, S100 | SCC, UC | 3 mos |

(continued)
| A: References       | B: Age/sex | C: Smoking | D: Symptom       | E: TNM, size | F: Surgical treatment       | G: Adjuvant therapy   | H: Immunohistochemistry | I: Associated histotypes | J: Follow-up (postsurgery) |
|---------------------|------------|------------|------------------|-------------|-----------------------------|----------------------|------------------------|-------------------------|--------------------------|
| Sakuma et al, 2008  | 73, F as   | Not stated | GH, HUN         | pT4NxMx     | NU                          | Senile for chemo     | chrA+, syn+, grimmelius+ | SCC, UC                 | 9 mos DOD                |
| Martin et al, 2007  | 77, M      | Not stated | GH, HUN         | T3NxM0 (1 cm)| NU                          | cPt+ Et (4 cycl), RT: (50.4 Gy) |                          | SCC                     | 13 mos, NED              |
| Busby et al, 2006   | NA         | NA         | NA              | pT3NxM0     | NU                          | adj                   | NA                     | SCC                     | 11 mos DOD, Metastatic (her) |
| Chang et al, 2007   | 67, M as   | NA         | NA              | NA          | NA                          | NA                   | NA                     | SCC                     | 5 mos DOD                |
| Ishikawa et al, 2004| 53, M as   | NA         | HUN, SIAD       | T3N1        | NU                          | Mtx + cisPt + Et     | NSE+, vim+, EMA+        | SCC                     | 17 mos DOD               |
| Chuang and Liao, 2003| 57, M as | Not stated | GH, FP          | T3N1Mx      | NU                          | None                  | NSE+, vim+, EMA+, St100+ | SCC                     | 16 mos, metastatic (bone) |
| Kim et al, 2001     | 60, M as   | Yes        | FP              | pT2NxM0 (3.8 cm)| NU                          | None                  | NSE+, vim+, EMA+        | SCC, UC                  | >55 mos NED              |
| Gupta et al, 1999   | 72, M      | Not stated | GH              | NA          | NA                          | NA                   | NA                     | SCC, UC, Sq             | 36 mos NED               |
| Tsutsumi et al, 1993| 60, M as   | Not stated | GH              | T2NxM0 (<8 cm)| NU + pC                     | cPt+ Et (4 cycl), RT (50 Gy) |                          | SCC, UC, Sq, S           | 16 mos, metastatic (bone) |
| Sakamoto et al, 1993| 64, F as   | Not stated | FP              | pT4NxM0 (1.8 cm)| NU + pC + LA               | Cic, Ad, cisPt       | Not stated              | SCC, UC                 | 8 mos NED                |
| Sawai et al, 1999   | 62, M as   | Not stated | GH              | T2NxM0 (1.8 cm)| NU + pC + LA               | cisPt + (2 cycl)     | NSE+                   | SCC, UC                 | 8 mos NED                |

Clinical–pathological features of all known cases of small cell carcinoma reported in literature since 1990. For each patient are briefly specified the significant clinical and pathological factors (columns A–J). A: references; B: age, sex, and ethnicity of the patient (when available); C: smoking history (classified as heavy smoker if the daily consumption is more than 20 cigarettes); D: symptoms at presentation; E: TNM staging at the time of resection with tumor size (when available); F: type of surgical resection; G: adjuvant (or neoadjuvant) chemotherapy; H: immunohistochemical phenotype; I: the associated histotypes; J: follow-up post-surgery, including the duration and the recurrence/metastatic data. AC = adenocarcinoma, af = African, as = Asian, ca = Caucasian, CAM = cytokeratin CAM 5.2, chrA = chromogranin-A, CK = cytokeratin, CT = chemotherapy, DOD = died of disease, EMA = epithelial membrane antigen, F = female, FP = flank pain, GH = gross hematuria, HGUC = high-grade urothelial carcinoma in situ, HUN = hydro-ureteronephrosis, If = ifosfamide, If = ifosfamide, LA = lymphadenectomy, Ly = lymphoma, M = male, MI = myocardial infarction, MDF = multiorgan failure, mos = months, Mix = methotrexate, NA = data not available, NED = no evidence of disease, neoad = neoadjuvant therapy, NSE = nonspecific enolase, NU = nephroureterectomy, PC = partial cystectomy, POGRA = platelet-derived growth factor receptor A, RT = radiotherapy, S = sarcoma, SCC = small cell carcinoma, SIADH = inappropriate ADH secretion syndrome, Sq = squamous carcinoma, syn = synaptophysin, TC = total cystectomy, TTF1 = thyroid transcription factor 1, U = ureterectomy, UC = urothelial carcinoma, vim = vimentin.
(CT) scan found thickened walls in the distal part of the right ureter in the absence of lithiasis. A nephroureterectomy was planned, but had to be suspended due to the patient’s clinical condition, and a segmental ureterectomy was performed instead. The right distal ureter was resected together with some enlarged regional nodes; its macroscopic inspection showed a thickened and hemorrhagic wall, with a nodular neoplasm of 2 × 1.5 cm obstructing the lumen and infiltrating the surrounding adipose tissue. The tumor was composed of small cell carcinoma admixed with infiltrating transitional cell carcinoma (Fig. 1). The neuroendocrine markers (synaptophysin, chromogranin-A, CD56) were positive in the SCC part of the tumor, the mitotic count was high, and the proliferative index counted by Ki-67 was more than 90%; the tumor was negative for vimentin and TTF-1. The final diagnosis was small cell neuroendocrine carcinoma invading 80% of the surgical specimen associated with high-grade urothelial carcinoma, both infiltrating the ureteral wall, the perineural spaces, and the perivisceral adipose tissue. The ureteral resection margins were negative. The right external iliac and presacral nodes were both metastatic for small cell neuroendocrine carcinoma. The pathological stage at the diagnosis was pT3N1 and the patient underwent a 2-week cycle of etoposide chemotherapy that had to be suspended for renal failure. The disease rapidly progressed: at 2 and a half months after admission and less than a month after segmental ureterectomy, an abdominal CT scan showed a contrast-enhanced 19 × 10 cm mass surrounding and obstructing the ureter, extended without clear margins to the aortocaval space, displacing the iliac vessels and infiltrating the surrounding tissues with diffuse pelvic lymphadenopathy. Chest and cranial CT scan did not detect any other lesions. The patient died for the progression of disease 5 months after admission.

3. Discussion

The usual presentation is flank homolateral pain due to hydroureter (with or without irradiation to the groin) and gross hematuria due to the vascular invasion; less frequently the patient laments weight loss, dysuria, and urinary tract infection. When those symptoms appear, the disease might be already at late stages, also because the pain gradually increases over time, becoming chronic, allowing a differential diagnosis from lithiasis that presents as acute pain. Ultrasonography is the initial choice for imaging in a suspected obstruction of the urinary tract, helping the clinician to differentiate between lithiasis and other causes of obstruction; urography and CT scan are second-level procedures that allow to establish the presence of a mass, confirm an associated hydroureter due to the obstruction, and the extension of the tumor and the regional node status. Urine cytology can be a precious diagnostic tool, but it requires an
expert cytolgist to get the right diagnosis, considering the paucity of the neoplastic cells in the smear, the mixed histotypes, and the infrequency of small cell carcinoma. A CT-guided biopsy or a nephro-ureteral resection are the usual choices for the urologist, allowing a pathological diagnosis.

At gross examination, SCC is usually described as a firm greyish tumor, often with hemorrhagic areas, protruding and occluding the ureteral lumen, with ill-defined borders and with peritumoral wall thickening. Small cell carcinoma has an architecture similar to the neuroendocrine tumors of other sites, composed by solid sheets or different pattern such as rosette or nests, often associated with a desmoplastic reaction. Crush artefact (“Azzopardi effect”) is common. The cells are small to medium-sized with scant cytoplasm and prominent nucleus with granular (classically described as “salt and pepper”) chromatin. Mitosis and necrosis are frequent, and also vascular invasion. As previously mentioned, there may coexist other histological types such as squamous carcinoma, transitional carcinoma, adenocarcinoma, and sarcomatoid carcinoma. The differential diagnoses include poorly differentiated urothelial carcinoma, primitive neuroectodermal tumor, malignant lymphoma, lymphoepithelioma-like carcinoma, and plasmacytoid carcinoma, from which this tumor can be differentiated by immunohistochemistry.[12][13][14][15][16][17][18][19][20][21][22][23][24][25][26][27][28][29][30][31][32][33][34][35][36] The neuroendocrine stains are usually positive (chromogranin A, synaptophysin, CD56, neuron-specific enolase) and it may show positivity for keratins such as cytokeratin (CK)7, epithelial membrane antigen, and pan-CK. Uroplakin III is usually negative, and this may help the differential diagnosis with transitional cell carcinoma. Vimentin expression has been previously related to a higher metastatic potential and a poor outcome in a clinical report.[30] The tumor is frequently diagnosed at late stages, when it has already invaded the periurethral tissues and the surrounding structures. The overall median survival is 17 months, with a 51.9% of 1-year survival rate.[10] The primary treatment is surgical resection of the tumor by ureterectomy or nephroureterectomy depending on the clinical status of the patient, stage, and localization of the tumor. Due to the aggressive course of SCC, the surgeon must address the radicality of the excision as early as possible, considering that in most cases the resection alone does not seem to stop the progression of the disease. If the clinical parameters of the patient are still stable, the surgical treatment is usually followed by adjuvant chemotherapy and radiotherapy. It is important to note that the patient might be amenable for the long-term hematuria, and the obstruction caused by the tumor often causes hydroureteronephrosis and impaired kidney function, so the clinical status of the patient might hinder a radical excision and/or an effective course of a systemic chemotherapy. There are promising reports about neoadjuvant chemotherapy, which has been used successfully to downstage the disease and lengthen the overall survival[10][11][36]

4. Conclusions
Small cell carcinoma of the ureter is an aggressive disease, and the factors that seem correlate with its prognosis are smoking history, age, male sex, tumor size, nodes, metastasis grading and size at diagnosis, the expression of vimentin, and incomplete resection. Although this is a rare tumor, effective targeted therapies are expected to be applied to improve the overall survival.

Author contributions
Conceptualization: Fabiola Farci.

Data curation: Fabiola Farci, Annamaria Bartolucci, Laura Boldrini.

Formal analysis: Fabiola Farci.

Investigation: Francesca Manassero, Ramona Baldesi, Annamaria Bartolucci, Laura Boldrini.

Resources: Cesare Selli.

Supervision: Cesare Selli, Pinuccia Faviana.

Writing – original draft: Fabiola Farci.

Writing – review & editing: Pinuccia Faviana.

References
[1] Acosta AM, Kayaczy-Balla A. Primary neuroendocrine tumors of the ureter: a short review. Arch Pathol Lab Med 2016;140:714–7.
[2] Church DN, Bahil A. Clinical review: small cell carcinoma of the bladder. Cancer Treat Rev 2006;32:588–93.
[3] Zhong W, Lin R, Zhang L, et al. Clinicopathologic characteristics, therapy and outcomes of patients with primary ureteral small cell carcinoma: a case series and systematic review of the literature. Onco Targets Ther 2017;10:4105–11.
[4] Hensley PJ, Bhalodi AA, Gupta S. Primary upper urinary tract small cell carcinoma: a case series and literature review. J Endourol Case Rep 2017;3:165–8.
[5] Alevizopoulos A, Yuen HS, Soumadri S, et al. Small cell carcinoma of the upper urinary tract: a case. report and review of existing cases. J Clin Case Rep 2016;6:811.
[6] Sood A, Williamson SR, Leavitt DA. Neuroendocrine tumor of the ureter: a zebra among horses. J Endourol Case Rep 2016;2:204–8.
[7] Ueda N, Kobayashi Y, Arai H, et al. Intravesical recurrence of small cell carcinoma of the ureter: a case report. Hinyokika Kyio 2016;62:93–7.
[8] Wang W, Liu G, Li Y, et al. Neuroendocrine carcinoma of the ureter: a case report and literature review. Oncol Lett 2016;11:257–60.
[9] Acosta AM, Hamedani FS, Meeks JJ, et al. Primary ureteral thyroid transcription factor 1-positive small cell neuroendocrine carcinoma: case report and review of the literature. Int J Surg Pathol 2015;23:3472–7.
[10] Osaka K, Kobayashi K, Sakai N, et al. Successful neoadjuvant chemotherapy for primary invasive small-cell carcinoma of the ureter. Can Urol Assoc J 2015;9:E393–6.
[11] Absaini M, Riyach O, Tazi MF, et al. Small cell neuroendocrine carcinoma of the urinary tract successfully managed with neoadjuvant chemotherapy. Case Rep Urol 2013;2013:598325.
[12] Yang J, Zhao Z, Ni J, et al. Urography and CT features of primary small cell carcinoma of the ureter: a case report. Iran J Radiol 2013;10:160–3.
[13] Ping JH, Chen ZX, Jiong Q, et al. Small cell neuroendocrine carcinoma of the ureter: a case report and literature review. Oncol Lett 2014;7:278–30.
[14] Zhao Z, Wang B, Yang J, et al. Primary small cell carcinoma of the ureter with hydroureteronephrosis. BJU Int 2012.
[15] Miller RJ, Holmang S, Johansson SL, et al. Small cell carcinoma of the renal pelvis and ureter: clinicopathologic and immunohistochemical features. Arch Pathol Lab Med 2011;135:1565–9.
[16] Patil S, Kaza RC, Kakkar AK, et al. Small cell carcinoma of the renal pelvis: a case report and review of the literature. ISRN Urol 2011;2011:786505.
[17] Kho VKS, Chan PH. Primary small cell carcinoma of the upper urinary tract. J Chin Med Assoc 2010;73:173–6.
[18] Kozyrakis D, Papadapril P, Stefanaki S, et al. Small cell carcinoma of the urinary tract: a case report. Cases J 2009;2:7743.
[19] Kuroda N, Kato K, Tamura M, et al. Ureteral small cell carcinoma. Med Mol Morphol 2009;42:55–7.
[20] Terada T. Primary small cell carcinoma of the ureter: a case report involving immunohistochemical and molecular genetic analyses of KIT and PDGFRα genes. Pathology 2009;42:101–2.
[21] Ito H, Kodaira K, Kosugi M, et al. Primary small cell carcinoma of the ureter: a case report. Hinyokika Kyio 2009;55:5417–20.
[22] Banerjee JS, Korula A, Pancker JB. Multicentric small cell neuroendocrine neoplasm of the renal pelvis and ureter with concomitant focal high-grade urothelial carcinoma of the ureter: a case report. Indian J Urol 2008;24:571–4.
[23] Masui K, Kamba T, Watanabe J, et al. A case of small cell carcinoma of the ureter. Hinyokika Kyio 2008;54:411–3.
[24] Ryu Y, Kinoshita N, Abe K, et al. Small cell carcinoma of the ureter with malignant lymphoma: case report and literature review. Acta Med Nagasaki 2008;53:29–32.
[25] Sakuma T, Ujike T, Yoshida T, et al. Urothelial carcinoma of ureter with neuroendocrine differentiation: a case report. Hinyokika Kyio 2008;54:123–6.
[26] Martin SM, Gonzales JR, Lagarto EG, et al. Primary small cell carcinoma of the ureter. Int J Urol 2007;14:771–3.
[27] Busby JE, Brown GA, Tamboli P, et al. Upper urinary tract tumors with non-transitional histology: a single-center experience. Urology 2006;67:318–23.
[28] Chang CY, Reddy K, Chorneyko K, et al. Primary small cell carcinoma of the ureter. Can J Urol 2005;12:2603–6.
[29] Ishikawa S, Koyama T, Kumagai A, et al. A case of small cell carcinoma of the ureter with SIADH-like symptoms. Nihon Hinyokika Gakkai Zasshi 2004;95:725–8.
[30] Chuang CK, Liao SK. A retrospective immunohistochemical and clinicopathological study of small cell carcinomas of the urinary tract. Chang Gung Med J 2003;26:26–33.
[31] Kim TS, Seong DH, Ro JY. Small cell carcinoma of the ureter with squamous cell and transitional cell carcinomatous components associated with ureteral stone. J Korean Med Sci 2001;16:796–800.
[32] Gupta M, Xia JR, Priminger B, et al. Small cell carcinoma of the ureter arising in an adult polycystic kidney disease: a case report. Appl Immunohistochem Mol Morphol 1999;7:164–8.
[33] Tsutsumi M, Kamiya M, Sakamoto M, et al. A ureteral small cell carcinoma mixed with malignant mesodermal and ectodermal elements: a clinicopathological, morphological and immunohistochemical study. Jpn J Clin Oncol 1999;29:325–9.
[34] Sakamoto N, Hasegawa Y, Nakamura M, et al. A case of small cell carcinoma of the ureter. Rinsho Hinyokika 1990;47:764–7.
[35] Sakai N, Ogawa T, Ishibashi Y, et al. A case of small cell carcinoma of the ureter. Hinyokika Kiyo 1990;36:1455–8.
[36] Lynch SP, Shen Y, Kamat A, et al. Neoadjuvant chemotherapy in small cell urothelial cancer improves pathologic downstaging and long-term outcomes: results from a retrospective study at the MD Anderson Cancer Center. Eur Urol 2013;64:307–13.