Incidence and significance of psammoma bodies in Xp11.2 translocation renal cell carcinoma and papillary renal cell carcinoma

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Abstract. The aim of the present study was to investigate the incidence and significance of psammoma bodies (PBs) in Xp11.2 translocation renal cell carcinoma (Xp11.2 tRCC) and papillary renal cell carcinoma (PRCC). The presence of PBs, irregular calcifications, hyaline globules and nested architecture in RCC tissues, which included 47 cases of Xp11.2 tRCC and 95 cases of PRCC, was examined by two pathologists. Compared with PRCC, patients with Xp11.2 tRCC exhibited a higher frequency of PBs, hyaline globules and nested architecture. The presence of PBs in combination with the occurrence of a nested architecture achieved a specificity of 93.7% when diagnosing Xp11.2 tRCC. However, there were no significant differences in the overall survival between patients with and without PBs in both types of RCC. Therefore, the presence of PBs combined with nested architecture may provide guidance for the diagnosis of Xp11.2 tRCC; however, PBs cannot predict tumor behavior in Xp11.2 tRCC or PRCC.

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Abbreviations: AJCC, American Joint Committee on Cancer; FISH, fluorescence *in situ* hybridization; OS, overall survival; PBs, psammoma bodies; PRCC, papillary renal cell carcinoma; PSF, progression-free survival; PSM, propensity score matching; PTC, papillary thyroid carcinoma; RCC, renal cell carcinoma; Xp11.2 tRCC, Xp11.2 translocation renal cell carcinoma

Key words: psammoma bodies, hyaline globules, renal cell carcinoma, Xp11.2 translocation, propensity score matching

Introduction

Psammoma bodies (PBs) are basophilic structures with laminated concretions, with a diameter generally ranging from 20-100 μm (1). PBs are composed of calcium apatite and are most commonly observed in papillary thyroid carcinoma (PTC), meningioma, and papillary serous cystadenocarcinoma of the ovary (2). Studies on serous cystadenocarcinoma of the ovary and meningioma demonstrated that collagen production by neoplastic cells and subsequent calcification led to the formation of PBs (2,3). However, an ultrastructural study of PTC revealed that tumor cell necrosis was responsible for the formation of PBs (2,4). Although various theories have been published to explain the origin of PBs, the underlying biochemical mechanism is poorly understood (2-4).

PBs have been reported in ~50% of PTC cases, which makes it an important marker for the pathological diagnosis of PTC (5). In addition, PBs are of great significance in the diagnosis of meningiomas (6) and serous cystadenocarcinomas of ovary (2), with incidence rates of 45 and 33%, respectively. Although PBs from various types of neoplasms share identical light-microscopic characteristics, their significance in diverse types of tumors should be further investigated. Cai *et al* (7) found PBs to be useful predictors of aggressive tumor behavior and patients with PTC with PBs had a poorer prognosis compared with those without PBs (7). However, Das (2) reported that PBs not only lead to degeneration or death of tumor cells, but serve as a barrier against the spread of the neoplasm.

In renal cell carcinoma (RCC), PBs are most commonly observed in Xp11.2 translocation RCC (Xp11.2 tRCC) (8) and

papillary (P)RCC (9). Xp11.2 tRCC, a rare and aggressive type of cancer, harbors a chromosomal translocation involving the Xp11.2 breakpoint and transcription factor binding to IGHM enhancer 3 (*TFE3)* gene fusion (10,11). Microscopically, Xp11.2 tRCC mimics clear cell and PRCC (10,11). The typical morphology of Xp11.2 tRCC includes PBs, irregular calcifications, hyaline globules and nested architecture, particularly for the subtype with the UBX domain containing tether for SLC2A4 (*ASPL)-TFE3* gene fusion (10,12). Furthermore, hyaline globules are considered as precursors of PBs and irregular calcifications in PCT (13). However, to the best of our knowledge, the incidence and significance of PBs, irregular calcifications, hyaline globules and nested architecture in Xp11.2 tRCC have only been described in reports with low patient numbers. Therefore, the aim of the present study was to investigate the association between the presence of these specific morphologies and clinicopathological characteristics. A group of PRCC cases were further enrolled due to the high incidence of PBs (9).

Materials and methods

Ethics approval. All procedures were approved by the Medical Ethics Committee for Human Experiments of Nanjing Drum Tower Hospital (Nanjing, China).

Diagnostic methods. A total of 47 patients with Xp11.2 tRCC were selected between January 2007 and September 2017 at the Nanjing Drum Tower Hospital. Patients were diagnosed by applying self-designed break-apart *TFE3* fluorescence *in situ* hybridization (FISH) probes. Briefly, *ASPL-TFE3* dual-fusion FISH probes were used for diagnosing *ASPL-TFE3* tRCC. The break-apart and the dual-fusion FISH probe results were validated by reverse transcription-PCR analysis (13). The inclusion criteria comprised patients with RCC who were positive for TFE3 by immunohistochemistry and FISH. Exclusion criteria comprise patients without integrated clinical and pathological data.

Clinicopathological characteristics. According to the 2016 World Health Organization classification of renal cell carcinoma, patients with PRCC were divided into type 1 (31 cases) and type 2 (64 cases) (14). Of the 95 cases of PRCC, 31 were type 1 and 64 were type 2. Immediately after excision, tumor samples were fixed in buffered 10% formalin for at least 24 h, embedded in paraffin, cut into 4-μm thick sections and stained by hematoxylin and eosin (0.5-1%) for 5-20 min at room temperature. The hematoxylin and eosin-stained sections of all 142 cases were retrospectively reviewed by two experienced pathologists. To further investigate the role of PBs in Xp11.2 tRCC and PRCC, each group was divided into two subgroups according to the presence of PBs. Besides, sensitivity, specificity and Youden's index of PB in the diagnosis of Xp11.2 RCC was measured by FISH. Clinical data including patient age at onset, sex, maximum tumor diameter, American Joint Committee on Cancer (AJCC) stage (15) and surgical procedure were obtained from reviewing medical records.

Survival data. All patients were followed every 3 months during the first year, every 6 months during the following 4 years and annually after 5 years until the time of death or the loss of follow-up. The survival data were obtained from latest follow-up information. Progression-free survival (PFS) was defined as the time interval between the date of surgery and the date of disease-progression or censoring at the time of the last follow-up. Overall survival (OS) was defined from the date of surgery to the mortality date or the last follow-up.

Statistical analysis. Continuous variables are presented as mean ± standard deviation and were compared using Student's t-test or one way analysis of variance followed by Student-Newman-Keuls post-hoc test in case of multiple group comparisons. Categorical variables were expressed as counts and percentages, and χ^2 or Fisher exact test were used for comparisons. Due to selection bias between subgroups, propensity score matching (PSM) was preformed to balance the distribution of covariates. A non-parsimonious logistic regression model encompassing patients' age, sex, tumor diameter, AJCC stage, surgical procedure and tumor subtype was used to calculate a propensity score for each patient. A 1:1 matching ratio was used in the propensity analyses. Following PSM, matched covariates were re-compared between the two groups with statistical tests. Subsequently, PFS and OS curves were obtained by Kaplan-Meier analysis and statistic comparisons were undertaken using the log-rank test.

In all tests, a two-sided $P<0.05$ was considered to indicate statistically significant differences. Statistical analysis was performed with SPSS software version 23.0 (IBM Corp.). GraphPad Prism software version 5.0 (GraphPad Software, Inc.) was used for graphics of survival curves.

Results

Frequency of PBs in different tumor types. Among the 47 cases of Xp11.2 tRCC, 13 (27.7%) were identified as *ASPL-TFE3* tRCC. The incidence rates of PBs in *ASPL-TFE3* tRCC and type 1 PRCC were 69.2 and 41.9%, respectively. The presence of PBs, irregular calcifications, hyaline globules and nested architecture was determined and recorded (Fig. 1). Table I presents the baseline clinical characteristics and frequency of PBs, irregular calcifications, hyaline globules and nested architecture in all the patients. In the Xp11.2 tRCC group, the incidence rate of PBs, hyaline globules and nested architecture was significantly higher compared with that in PRCCs (all $P<0.05$).

Association of other morphological characteristics with PBs. Hyaline globules were present in 22 cases (88.0%) of Xp11.2 tRCC with PBs, as opposed to 13 (59.1%) cases without PBs ($P=0.053$). In addition, 91.7% (33/36) cases of Xp11.2 tRCC displayed PBs or irregular calcifications, as opposed to 18.2% (2/11) cases without PBs ($P<0.001$). Similarly, a significantly higher number of PRCC cases with PBs or irregular calcifications contained hyaline globules, as opposed to cases without PBs or irregular calcifications [45/54 (83.3%) vs. 8/41 (19.5%), respectively; $P<0.001$]. In addition, the sensitivity, specificity and Youden's index of PBs in diagnosing Xp11.2 tRCC were 53.2, 69.5 and 22.7% respectively. For nested architecture, another histopathological characteristic of Xp11.2 tRCC, the sensitivity, specificity and Youden's index were

Table I. Clinicopathological characteristics of Xp11.2 tRCC and PRCC.

Characteristic	Xp11.2 tRCC (n=47)	PRCC (n=95)	P-value
Age (years)	31.32±15.66	54.47±14.02	<0.001
Sex			0.050
Male	20 (42.6)	25 (26.3)	
Female	27 (57.4)	70 (73.7)	
Tumor size (cm)	5.43±2.39	5.01±2.60	0.360
AJCC stage			0.608
1	29 (61.7)	63 (66.3)	
2	4 (8.5)	8 (8.4)	
3	11 (23.4)	18 (18.9)	
4	3 (6.4)	6 (6.3)	
Psammoma body	25 (53.2)	29 (30.5)	0.009
Irregular calcification	29 (61.7)	42 (44.2)	0.050
Hyaline globule	35 (74.5)	53 (55.8)	0.031
Nested architecture	16 (34.0)	14 (14.7)	0.008

Data are presented as n (%) or the mean ± standard deviation. AJCC, American Joint Committee on Cancer; PRCC, papillary renal cell carcinoma; Xp11.2 tRCC, Xp11.2 translocation renal cell carcinoma.

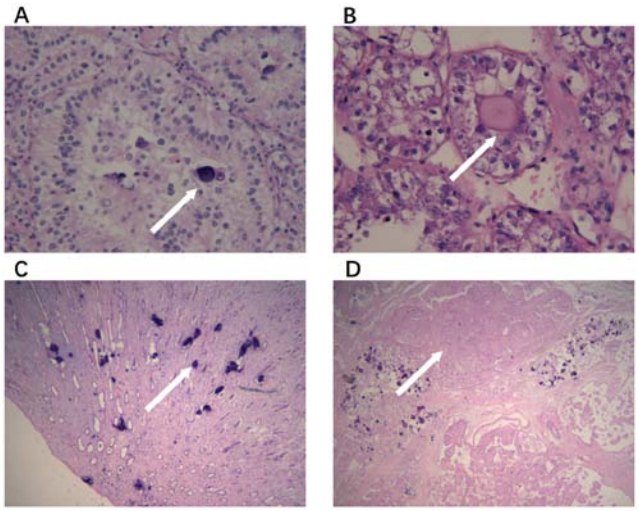

Figure 1. PBs, irregular calcifications, hyaline globules and nested architecture in tissue samples from Xp11.2 translocation renal cell carcinoma. Typical appearance of (A) PB with laminated a structure (white arrow; magnification, x400), (B) hyaline globules surrounded by a layer of neoplastic cells (white arrow; magnification. x400), (C) irregular calcifications (highlighted using white arrow; magnification, x100) and (D) irregular calcification deposits outside the nested formations (highlighted using white arrow; magnification, x100). PBs, psammoma bodies.

34.0, 85.3 and 19.3%, respectively. When PBs were combined with nested architecture, the specificity in diagnosing Xp11.2 tRCC increased to 93.7%; however, sensitivity dropped to 17.0% (Table II).

The clinicopathological characteristics of patients with Xp11.2 tRCC and PRCC before and after PSM are summarized in Tables III and IV, respectively. Prior to PSM, only the AJCC stages in the PRCC group were proven to differ significantly between the groups with and without PB (P=0.015). After PSM, 44 patients with Xp11.2 tRCC and 58 with PRCC were selected and no significant differences in clinicopathological characteristics, including patient age, sex distribution, tumor size, AJCC stage and subtype was associated with the presence of PBs.

Follow-up and survival data. Except for two patients, no patients were lost to follow-up. The mean follow-up duration was 44.69±31.56 months (range 5-121 months) in the Xp11.2 tRCC group and 27.23±21.40 months (range 1-89 months) in the PRCC group. During follow-up, 14 patients with Xp11.2 tRCC (29.8%) and 22 patients with PRCC (23.2%) showed disease progression. The 5-year OS rate for patients with Xp11.2 tRCC and PRCC was 81.8 and 67.1%, respectively (P=0.196). After adjusting the covariates using PSM, there were no statistical differences in the survival curves for PFS and OS based on PB occurrence (Fig. 2).

Discussion

Since PBs were first described, various mechanisms underlying PB morphogenesis have been suggested and the hypothesis of dystrophic calcification appears to be the most likely explanation. During the process of dystrophic calcification, events such as thickening of the basal lamina, vascular thrombosis, calcification and tumor cell necrosis occur sequentially (4). The formation of papillae, where the precursors of PBs usually originate, is an important morphological characteristic associated with PBs (2). After analyzing the IgG distribution of blood vessels, whorls and PBs of meningiomas, Tabuchi *et al* (16) hypothesized that humoral immunity participates in the formation of whorls and PBs. Another important contributor of the formation of PBs is type IV collagen (3). Described by a nested formation around the basement membrane, type IV collagen may be involved in anchoring PBs to the site of development of meningiomas (3). Additionally, type IV collagen was found

Table II. The role of psammoma bodies and nested architecture in diagnosing Xp11.2 tRCC.

Characteristic	Sensitivity (%)	Specificity (%)	Youden's index (%)
Psammoma body	53.2	69.5	22.7
Nested architecture	34.0	85.3	19.3
Psammoma body + Nested architecture	93.7	17.0	10.7

Table III. Comparisons of clinicopathological characteristics of Xp11.2 tRCC based on the presence of PB.

		PB			
Characteristic	No PB (n=22)	All (n=25)	P-value[a]	PSM-adjusted (n=22)	P-value[a]
Age (years)[b]	29.32±18.41	32.84±13.51	0.465	32.68±13.64	0.500
Sex[c]			0.831		1
Female	13 (59.09)	14 (56.00)		13 (59.09)	
Male	9 (40.91)	11 (44.00)		9 (40.91)	
Tumor size (cm)[b]	6.00±2.55	4.92±2.17	0.166	5.03±2.28	0.173
AJCC stage[c]			0.458		0.666
1	12 (54.55)	17 (86.00)		14 (63.64)	
2	3 (13.64)	1 (4.00)		1 (4.55)	
3	5 (22.72)	6 (24.00)		6 (27.26)	
4	2 (9.09)	1 (4.00)		1 (4.55)	
Subtype[c]			0.173		0.173
ASPL	4 (18.18)	9 (36.00)		8 (36.36)	
Non-*ASPL*	18 (81.82)	16 (64.00)		14 (63.64)	

Data are presented as n (%) or the mean ± standard deviation. [a]P-value vs. no PB determined using [b]analysis of variance or [c]χ^2 test. AJCC, American Joint Committee on Cancer; Xp11.2, Xp11.2 tRCC translocation renal cell carcinoma; *ASPL*, UBX domain containing tether for SLC2A4; PBs, psammoma bodies; PSM, propensity score matching.

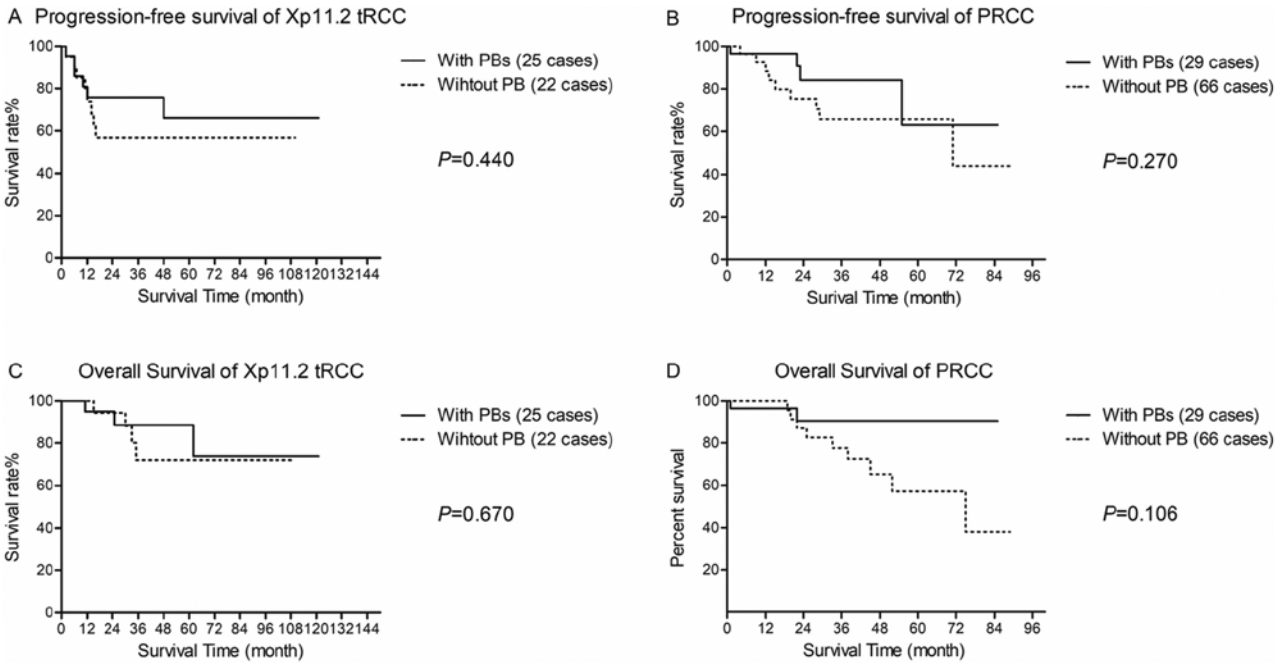

Figure 2. Survival analysis of patients with and without PB. Progression-free survival for patients with (A) Xp11.2 tRCC and (B) PRCC. Overall survival of patients with (C) Xp11.2 tRCC and (D) PRCC. PBs, psammoma bodies; RCC, renal cell carcinoma; PRCC, papillary RCC; Xp11.2 tRCC, Xp11.2 translocation RCC.

Table IV. Comparisons of clinicopathological characteristics of PRCC based on the presence of PB.

		No PB			
Characteristic	PB (n=29)	Original (n=66)	P-value[a]	PSM-adjusted (n=29)	P-value[a]
Age (years)[b]	52.07±15.67	55.53±13.22	0.265	59.17±13.42	0.053
Sex[c]			0.409		1
Male	6 (20.69)	19 (28.79)		6 (20.69)	
Female	23 (79.31)	47 (71.21)		23 (79.31)	
Tumor size (cm)[b]	4.36±2.24	5.30±2.72	0.115	5.21±2.96	0.224
AJCC stage[c]			0.015		0.058
1	24 (82.76)	39 (59.09)		18 (62.07)	
2	3 (10.34)	5 (7.57)		4 (13.79)	
3	1 (3.45)	17 (25.76)		6 (20.69)	
4	1 (3.45)	5 (7.57)		1 (3.45)	
Subtype[c]			0.093		0.097
1	13 (44.83)	18 (27.27)		7 (24.14)	
2	16 (55.17)	48 (72.73)		22 (75.86)	

Data are presented as n (%) or the mean ± standard deviation. [a]P-value vs. PB determined using [b]analysis of variance or [c]χ^2 test. PBs, psammoma bodies; AJCC, American Joint Committee on Cancer; PRCC, papillary renal cell carcinoma; PSM, propensity score matching.

Table V. Literature described cases of Xp11.2 tRCC involving psammoma bodies.

Author, year	Patients (n)	Diagnosis methods	Incident rate of PB (%)	(Refs.)
Qu *et al*, 2016	30	IHC+FISH	26.7	(20)
Rao *et al*, 2011	19	IHC+CK	94.7	(21)
Zou *et al*, 2014	9	IHC	77.8	(22)
He *et al*, 2014	6	IHC	83.3	(23)
Komai *et al*, 2009	7	IHC+CK	42.9	(24)
Argani *et al*, 2007	28	IHC+PCR	50.0	(10)
Camparo *et al*, 2008	31	IHC+CK	62.0	(25)

CK, cytogenetic karyotype; FISH, fluorescence *in situ* hybridization; IHC, immunohistochemical; Xp11.2 tRCC, Xp11.2 translocation renal cell carcinoma.

to be selectively expressed in PB-forming ovarian cancer cell lines, which suggested that type IV collagen slows down the formation of PBs during the growth of ovarian cancer cells (17).

In RCC, PBs have mainly been reported for Xp11.2 tRCC and PRCC (8,9), and occasionally for chromophobe RCC (18) and renal oncocytoma (19). Unlike the aforementioned studies, which were largely supported by immunohistochemistry and molecular techniques, the present study was a morphological study to summarize the incidence of PBs in PRCC and Xp11.2 tRCC. Published reports of Xp11.2 tRCC with identified PBs are summarized in Table V, to reflect that the incidence rate of PB in Xp11.2 tRCC varies greatly among different reports (10,20-25). To the best of our knowledge, there has been no previous report about the clinical significance of PBs in Xp11.2 tRCC.

Compared with PRCC, Xp11.2 tRCC shares a similar histopathological structure, but exhibits a more aggressive course (10,11). In the diagnosis of Xp11.2 tRCC, *TFE3* immunohistochemical staining serves as the basic method, with high false-positive rates and low predictive values (26). Therefore, FISH, PCR or karyotype analysis is applied for a definitive diagnosis (12). Although frequently present in Xp11.2 tRCC and PRCC, the diagnostic and predictive role of PBs has not yet been fully elucidated (8,9). The present study included 47 cases of Xp11.2 tRCC and 95 cases of PRCC, and demonstrated that the incidence of PBs in Xp11.2 tRCC was significantly higher compared with that in PRCC. The specificity of PBs combined with nested architecture in diagnosing Xp11.2 tRCC reached 93.7%, suggesting a potential diagnostic role in Xp11.2 tRCC screening.

Numerous studies have reported the association between hyaline globules and PBs/irregular calcifications (3,13,27). In a PTC case diagnosed by fine-needle aspiration,

Haji *et al* (27) found only few PBs, but numerous laminated hyaline globules. Immunohistochemical staining using paraffin-embedded sections revealed that these globules were positive for von Kossa staining, but negative for thyroglobulin, suggesting an early or precursor form of PBs. In the study from Das *et al* (13), the presence of hyaline degeneration among cases with and without PBs/irregular calcifications was 80.0% vs. 22.7%, respectively. Similarly, the statistical analysis of the present study favored the hypothesis that hyaline globules may be precursors of PBs in Xp11.2 tRCC and PRCC.

The predictive role of PBs has been associated with tumor types (2,7). In the PRCC group, type 1 tumors contained more PBs and displayed a more favorable behavior (9). In the Xp11.2 tRCC group, *ASPL-TFE3* tRCC exhibited a higher incidence of PBs compared with other fusion types. However, *ASPL-TFE3* tRCC has been reported to be more likely associated with lymph node metastasis and disease progression (10,11,28). Based on this, it is crucial to investigate whether PBs exert opposite effects in different tumor types. However, the PSM data obtained in the present study suggested that the presence of PBs was not associated with any of the observed clinicopathological characteristics, including tumor size, AJCC stage or survival, in PRCC and Xp11.2 tRCC.

To the best of our knowledge, the present study was the first to systematically investigate the incidence and significance of PBs, irregular calcifications, hyaline globules and nested architecture in Xp11.2 tRCC and PRCC. However, there were certain limitations. This was a retrospective study and the sample size of Xp11.2 tRCC was low due to its scarcity; however, it is believed to be one of the largest single-center clinical reports on Xp11.2 tRCC. Additionally, as patients received various adjuvant therapies following surgery, selection bias persisted even after PSM was performed. Furthermore, other subtypes of RCC, including chromophobe RCC and renal oncocytoma, were not included in the present study due to the small number of cases and their favorable prognosis. Further studies are required to elucidate the biochemical mechanisms underlying the development of PBs in RCC.

The incidence rate of PBs in Xp11.2 tRCC was found to be significantly higher compared with that in PRCC, and the presence of hyaline globules was associated with PBs and irregular calcifications. However, the presence of PBs was not found to be associated with tumor subtype or prognosis in Xp11.2 tRCC or PRCC. When PBs and nested architecture were present simultaneously in RCC, additional methods may be used to investigate the potential of Xp11.2 tRCC occurrence.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

WG, DL and HG provided the idea and acquired funding for the studies. NL and FQ made substantial contributions to the conception or design of the work. NL, KW, WZ, ZW, SA and WM analyzed and interpreted the patient data regarding psammoma bodies in Xp11.2 translocation renal cell carcinoma and papillary renal cell carcinoma. FQ, LX, JY and MC performed the histological examinations of the renal cell carcinomas and were major contributors to the writing of the manuscript. WG, LX and HG were involved in drafting and revising the manuscript. All the authors have read and approved the final manuscript.

Ethics approval and consent to participate

All procedures were approved by the Medical Ethics Committee for Human Experiments of Nanjing Drum Tower Hospital (Nanjing, China). At time of hospitalization, patients provided written informed consent for the collection and use of their tissues.

Patient consent for publication

The patients, or parents, guardians or next of kin provided written informed consent for the publication.

Competing interests

The authors declare that they have no competing interests.

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