

Case Report

Primary Renal Neuroendocrine Tumor in a Horseshoe Kidney: A Case Report of an Indolent Course

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Simple Summary

Primary renal neuroendocrine tumors (NETs) are exceptionally rare neoplasms but occur disproportionately in horseshoe kidneys. Because of their rarity and their non-specific clinical and radiological presentation, they are frequently misdiagnosed as more common renal tumors, making preoperative diagnosis particularly challenging. We report a well-differentiated primary renal neuroendocrine tumor arising in a horseshoe kidney and discuss the main diagnostic pitfalls and pathological features of this rare association. This case highlights the importance of considering primary renal neuroendocrine tumors in the differential diagnosis of renal masses arising in horseshoe kidneys to facilitate accurate diagnosis and appropriate patient management.

Abstract

Background/Objectives: Primary renal NETs are exceptionally rare neoplasms that occur disproportionately in horseshoe kidneys. Their rarity and non-specific clinical and radiological features make preoperative diagnosis particularly challenging. **Methods:** Herein, we report the case of a 60-year-old woman presenting with a painful left lumbar mass associated with dysuria and pollakiuria. **Results:** Computed tomography revealed a large heterogeneous tumor arising from the left moiety of a horseshoe kidney. Histopathological examination of the resected specimen demonstrated a 14 cm well-differentiated NET (grade 1) with diffuse chromogranin A and synaptophysin expression, a mitotic count of 1 per 2 mm², and a Ki-67 proliferation index of <1%. The histopathological and immunohistochemical findings, together with the absence of an extrarenal primary site on staging investigations, supported the diagnosis of a primary renal NET. The postoperative course was uneventful, and the patient remained disease-free after two years of follow-up. **Conclusions:** This case highlights the diagnostic challenges posed by this rare entity and underscores the importance of including primary renal neuroendocrine tumors in the differential diagnosis of renal masses arising in horseshoe kidneys.



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1. Introduction

Primary renal NETs are exceptionally rare neoplasms, accounting for less than 1% of all primary renal tumors and fewer than 0.4% of neuroendocrine tumors at all anatomical sites [1]. However, they occur disproportionately in horseshoe kidneys, with approximately

18% of reported primary renal NETs arising in this congenital anomaly [2]. To date, only about 30 cases of primary renal NETs associated with horseshoe kidney have been reported [1,2]. Herein, we report the case of a well-differentiated NET arising in a horseshoe kidney to highlight the unique features of this rare association and discuss its diagnostic, therapeutic, and prognostic challenges.

2. Observation

A 60-year-old woman with no significant past medical history presented to the Urology Department of Rabat University Hospital with urinary frequency and dysuria. An abdominal ultrasound revealed a heterogeneous abdominal mass, but its origin could not be determined, with the main differential diagnoses including a pancreatic, renal, or splenic lesion. An abdominopelvic CT scan demonstrated a large (12 cm), heterogeneous mass arising from the left moiety of a horseshoe kidney. The lesion was cortical in origin, with no involvement of the renal vascular pedicles or associated lymphadenopathy (Figure 1).

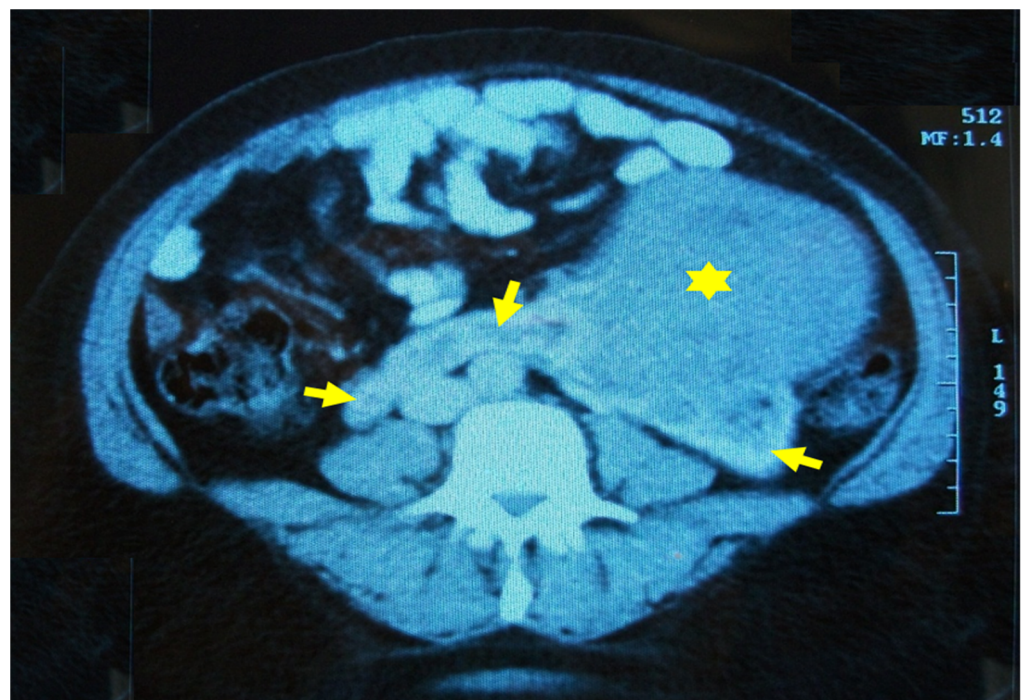


Figure 1. Axial contrast-enhanced CT scan showing a large (12 cm), heterogeneous cortical mass (yellow star) arising from the left moiety of a horseshoe kidney (yellow arrows).

Laboratory investigations were unremarkable, including normal renal function (urea, 0.3 g/L; creatinine, 8.9 mg/L). The patient underwent a left heminephrectomy. Gross pathological examination revealed a surgical specimen weighing 1350 g, containing a well-encapsulated cortical mass measuring $14 \times 13 \times 11 \text{ cm}^3$ with an exophytic component. On cut section, the tumor was friable, pinkish beige, and partially cystic, with multiple foci of hemorrhage (Figure 2).

Microscopic examination revealed a monomorphic, epithelial-like tumor proliferation with a trabecular architecture, initially suggestive of a neuroendocrine tumor. The tumor cells were medium to large in size, cuboidal in shape, and had eosinophilic cytoplasm. The nuclei were slightly oval with sparse chromatin. The mitotic index was estimated at 1 mitosis/ mm^2 . The stroma was richly vascularized with no visible tumor necrosis (Figure 3).

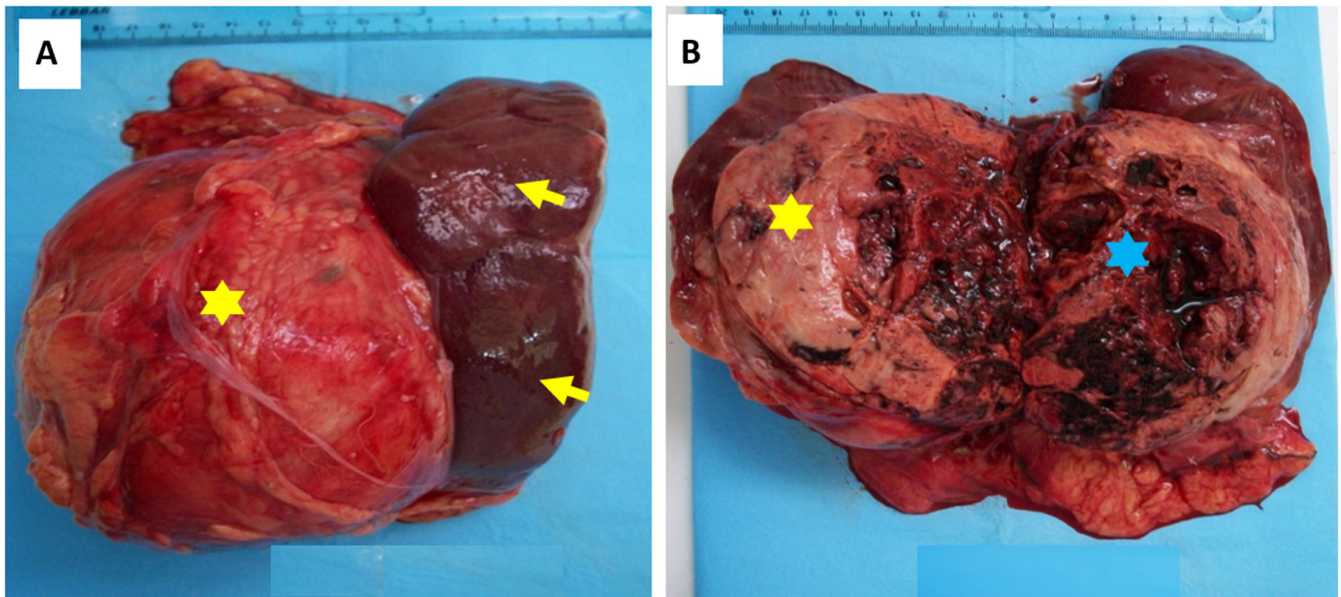


Figure 2. (A) Specimen from a left heminephrectomy showing a large mass (yellow star) attached to the kidney (yellow arrows). (B) On sectioning, the mass is a partially cystic (blue star), pinkish-beige, encapsulated tissue measuring $14 \times 13 \times 11 \text{ cm}^3$.

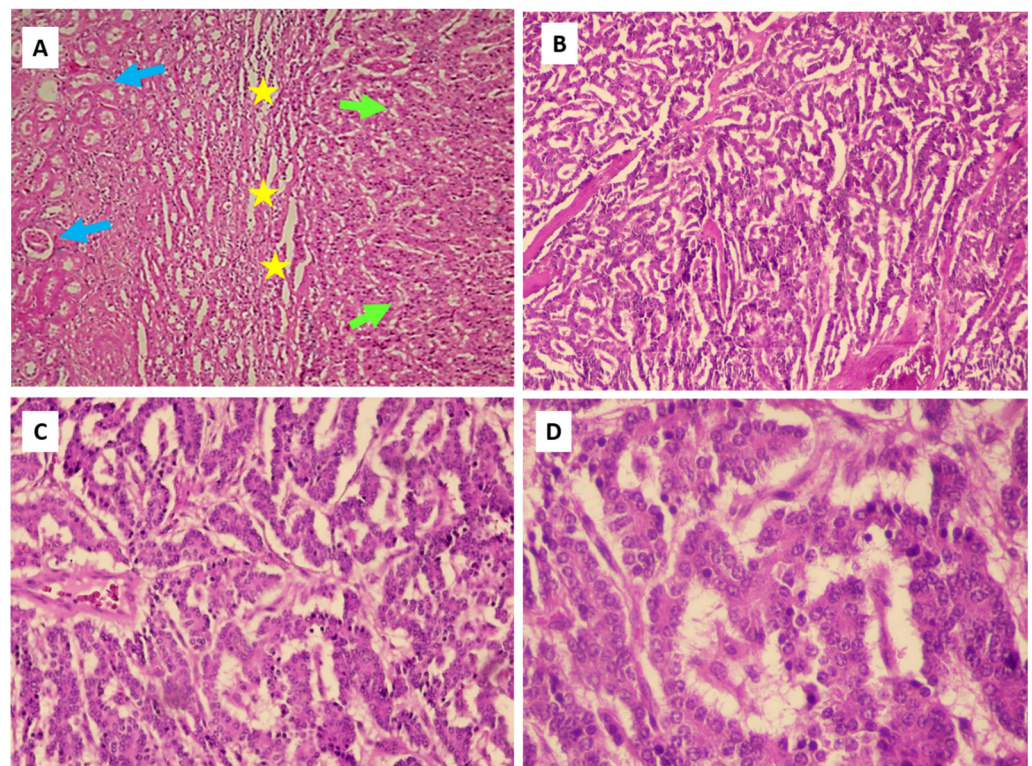


Figure 3. (A): Low-magnification microscopic image (HE $\times 40$) showing renal parenchyma (blue arrows) infiltrated by a tumor mass (green arrows) surrounded by a thin fibrous capsule (yellow stars). (B,C): Microscopic images at medium magnification (HE $\times 100$ and $\times 200$) showing the renal tumor proliferation with a cord-like architecture. The tumor cells are medium to large in size, with a cubic appearance and abundant, finely granular eosinophilic cytoplasm. The stroma is sparsely vascular. (D): High-magnification microscopic image (HE $\times 400$); the nuclei are oval and monomorphic with sparse chromatin, lacking nucleoli or mitoses.

Immunohistochemical analysis revealed diffuse positive staining of tumor cells with anti-cytokeratin, anti-chromogranin A, and anti-synaptophysin antibodies, with a Ki67

proliferation index of <1%. Staining with anti-PAX8 and anti-TTF1 antibodies was negative (Figure 4). These morphological and immunophenotypic findings, together with the negative staging work-up, supported the diagnosis of a well-differentiated primary NET arising in a horseshoe kidney. The postoperative course has been favorable with a 2-year follow-up.

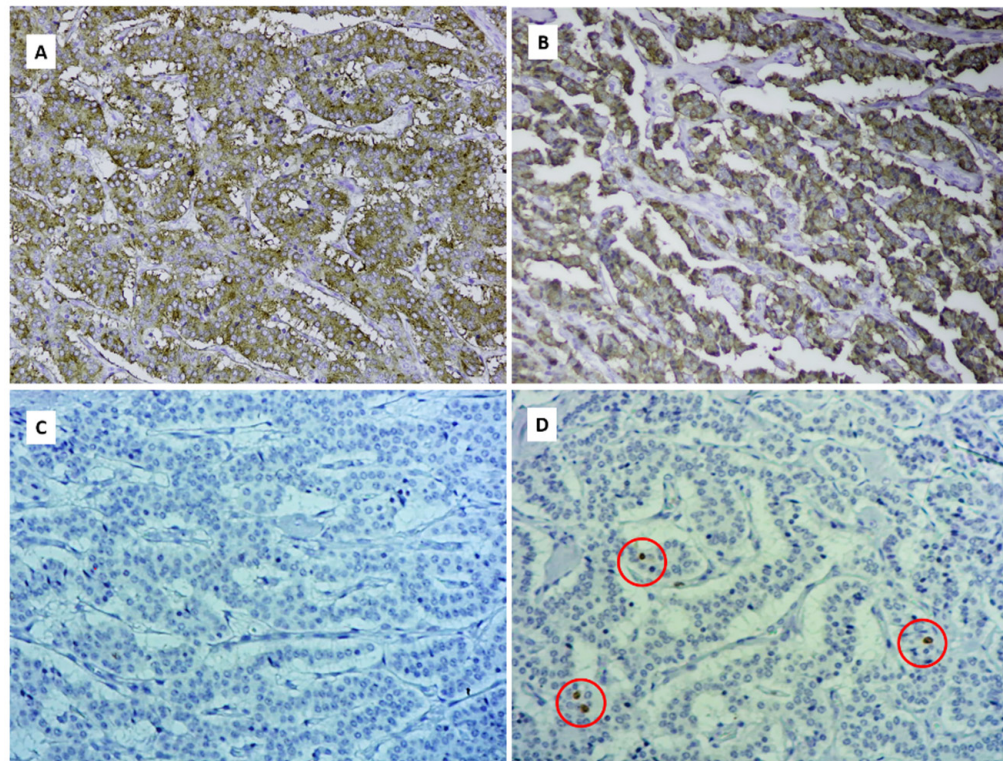


Figure 4. On immunohistochemical staining, the tumor cells show diffuse cytoplasmic positivity for anti-synaptophysin (A) and anti-chromogranin A (B). The tumor cells are negative for anti-PAX8 (C). The Ki-67 index is low (estimated at less than 1%); in this field ($\times 200$ magnification), positive nuclei are circled in red (D).

3. Discussion

3.1. Epidemiological Findings

Primary renal NETs are very rare [1,2]. The first case of this tumor was reported by Resnick in 1966 [3]. A comprehensive review of the literature from 1966 to 2020 identified approximately 100 cases of primary renal NETs, with only about 30 cases reported in horseshoe kidneys [3,4]. This renal malformation can harbor all classic renal epithelial tumors according to their usual epidemiological profile, but well-differentiated NETs have a proportionally higher incidence in this anatomical malformation than in a normal kidney [2,5].

3.2. Pathogenesis

Well-differentiated NETs belong to the group of neuroendocrine neoplasms that arise from diffuse neuroendocrine cells distributed throughout the gastrointestinal, pulmonary, thyroid, and genitourinary systems [1,2,4]. The kidney does not contain such cells under physiological conditions [4,6,7]. Therefore, the pathogenesis of these tumors is complex and remains controversial despite the various hypotheses that have been proposed [3,5]. The first theory suggests the presence of intrinsic neuroendocrine cells within the renal parenchyma derived from remnants of neural crest progenitor cells [3,4]. Another theory proposes that neuroendocrine cells may develop from areas of intestinal metaplasia in the

renal calyces following chronic infection [5,7,8]. Other hypotheses implicate embryological factors, particularly fusion and rotation abnormalities such as horseshoe kidney, in tumor development [4,5,7].

This congenital anomaly is associated with an increased risk of NETs (relative risk = 62 according to Krishnan and 82 according to Begin), as illustrated by our case [4,5,9]. The association with Wilms tumor or mature teratoma may further increase the risk of tumor development [5,9]. Finally, genetic factors have also been proposed. In particular, loss of heterozygosity at chromosome 3p21 has been suggested as a potential early event in the development of primary renal neuroendocrine tumors. However, no specific driver genes have been consistently identified, and the molecular pathogenesis of these tumors remains poorly understood [5].

3.3. Clinical and Radiological Characteristics

The mean age at diagnosis of primary renal NETs arising in horseshoe kidneys is over 45 years, with a slight male predominance, reflecting the higher prevalence of this congenital anomaly in men (male-to-female ratio, 3:1) [1,3,5]. Clinically, these tumors are often asymptomatic or present with nonspecific symptoms, including abdominal pain, hematuria, or a palpable abdominal mass, reported in 26.8% of cases [1,2,5].

Endocrine syndromes have been reported in approximately 12.6% of cases and may manifest as constipation, carcinoid syndrome (flushing), or Cushing's syndrome [1–3]. Radiological imaging plays a pivotal role in lesion characterization and preoperative assessment [6,9,10]. Computed tomography (CT) is the imaging modality of choice, providing detailed information on the position of the kidneys, the anatomy of the isthmus, tumor size, and locoregional extension [3,7,11]. Arteriography is particularly useful for surgical planning because of the frequent vascular anatomical variations associated with horseshoe kidneys [6]. Most reported tumors appear as hyperdense, hypovascular, and partially cystic masses on CT, frequently with calcifications [6,7,10,11]. Involvement of the renal isthmus and regional lymph nodes has also been frequently reported (Table 1) [1,2,8].

The clinical and radiological findings observed in our patient are consistent with those reported in the literature, except for the absence of regional lymphadenopathy despite the large tumor size.

3.4. Pathological Characteristics

Pathological examination provides the definitive diagnosis [3,12]. Macroscopically, these tumors range in size from 1.5 to 30 cm in diameter (mean, 8.4 cm) [7,12]. They are typically yellowish-white to bronze-red in color and have a solid or partially cystic appearance in approximately one-third of cases [3,5,12], consistent with the gross appearance observed in our case. Histologically, the diagnosis is suggested by a characteristic neuroendocrine architecture associated with a richly vascularized stroma and confirmed by immunohistochemistry. This combination, together with the careful exclusion of metastatic disease, supports the diagnosis of a primary renal neuroendocrine tumor (Table 1) [5,7,12].

Current guidelines from the European Association of Urology (EAU) and the National Comprehensive Cancer Network (NCCN) recommend renal tumor biopsy only in selected clinical situations, including small renal masses, indeterminate renal lesions, or metastatic disease when histological confirmation is expected to influence clinical management. In these settings, particularly when a renal mass arises in a horseshoe kidney, preoperative biopsy may provide additional value by establishing the histological diagnosis, excluding the more common possibility of metastatic neuroendocrine disease before confirming the diagnosis of a primary renal neuroendocrine tumor, and thereby guiding staging investigations and therapeutic decision-making [7,13].

Well-differentiated renal NETs share the same morphological characteristics as well-differentiated NETs described in other sites, particularly the lungs and gastrointestinal tract [12,14]. In the kidney, these tumors are classified as grade 1 or grade 2 [5,7]. Histopathological analyses reported in the literature have shown that these tumors most commonly exhibit trabecular and ribbon-like architectural patterns, a low mitotic count, and frequent hemorrhagic, necrotic, and calcific changes, particularly in larger tumors (Table 1) [5,12]. In addition to the characteristic morphology, the diagnosis is confirmed by positive immunohistochemical staining for epithelial markers (AE1/AE3) and at least two neuroendocrine markers, including chromogranin A, synaptophysin, CD56, and INSM1 [3,4,12]. In contrast, PAX8 and TTF1 are consistently negative [12]. The differential diagnosis includes morphological mimics such as metanephric adenoma, the compact variant of papillary renal cell carcinoma, and the juxtaglomerular cell tumor. These diagnoses are easily ruled out by a well-conducted immunohistochemistry panel [7,12].

The classification of NETs has undergone several revisions and refinements to address issues related to nomenclature, prognostic assessment, and organ-specific differences [12,15]. The 2021 WHO Classification of Thoracic Tumours uses the terms “typical carcinoid” and “atypical carcinoid,” whereas the WHO classification of digestive system tumors adopts the term “well-differentiated NET” and recognizes three grades (G1, G2, and G3) based on the mitotic count and Ki-67 proliferation index, using site-specific grading criteria (Table 2) [7,12,14].

Nevertheless, the 2022 WHO Classification of Urinary and Male Genital Tumours includes a separate chapter on neuroendocrine neoplasms of the genitourinary tract and recommends the term “well-differentiated NET, grade 1 or 2,” while discouraging the use of the term “carcinoid tumor.” Grade 3 well-differentiated NET is not included in this classification. Because of the rarity of these tumors, no specific grading system has been established for genitourinary NETs, and the WHO recommends applying the grading system used for gastroenteropancreatic NETs [12]. In addition to well-differentiated NETs, small-cell and large-cell neuroendocrine carcinomas constitute a high-grade category with an unfavorable prognosis and exhibit similar histopathological features regardless of the organ of origin (Table 2) [5,7,12,14].

Table 1. Clinicopathological characteristics of reported primary neuroendocrine neoplasms arising in horseshoe kidneys, adapted from the literature review by Billis et al. [1] and updated with the most recent cases reported by Kikkawa et al. [6] and Ni et al. [7].

Age/Sex	Clinical Report	Radiological Features	Histopathology	Treatment	Outcome	Reference
58 F	Incidental	Mid-lower L T: 5.5 cm Cystic-solid	Trabecular NET G1	Laparoscopic PN	NED (12 months)	Ni et al. [7]
65 F	Incidental	isthmus T: 2.7 cm Mid-inf R	Cord-like NET G2	Robotic PN	NED (6 months)	Kikkawa et al. [6]
57 F	Waist pain	T: 8.0 cm Cystic-solid	LCNEC	Laparoscopic PN	Bone metastasis (5 months)	Song et al. [16]
40 F	Hematuria	isthmus T: 14.9 cm	Trabecular NET G1	PN	LN metastasis	Gupta et al. [9]
37 F	Incidental	N.S T: 1.8 cm	NET G2	PN	NED (6 months)	Seker et al. [17]
61 F	Flank pain	R T: 5.2 cm Cystic-solid	NET	PN	NED (42 months)	Kuba et al. [18]

Table 1. Cont.

Age/Sex	Clinical Report	Radiological Features	Histopathology	Treatment	Outcome	Reference
49 F	Hematuria	L T: 5 cm	NET G1	RN	NED (18 months)	Lin et al. [19]
33 M	Incidental	Isthmus L T: 9.6 cm Cystic	WD-NET	RN	NED	El-Hawary et al. [20]
40 M	Incidental	L T: 4.5 cm	WD-NET	PN	NED (24 months)	Lamb et al. [21]
56 F	Flank pain/hematuria	R T: 5.4 cm	NET lymphovascular invasion	PN LN Dissection	NED (52 months)	Teegavarapu et al. [15]
37 M	Incidental	Isthmus T: 2.4 cm Cystic-solid	NET associated with mature teratoma	PN	LN metastasis	Sun et al. [22]
66 M	Incidental	L T: 8 cm	NET	RN	LN metastasis	Litwinowicz et al. [23]
51 M	Sciatica	R T: 3 cm	Trabecular NET	PN	NED (12 months)	de Hoog et al. [24]
50 F	Low back pain	R T: 9.7 cm	NET associated with mature teratoma + RCC	PN	NED (6 months)	Armah et al. [25]
32 M	Abdominal pain	R T: 14.5 cm	NET	PN	AWD (12 months); thyroid metastasis	Bhalla et al. [26]
68 F	Incidental	R T: 4.5 cm	WD-NET	PN	DOD (28 months)	Lane et al. [27]
51 M	Incidental	R T: 8 cm	WD-NET	RN	NED (11 months)	Lane et al. [27]
51 M	Flank Hematuria	L T: 3.5 cm	WD-NET	RN	NED (74 months)	Lane et al. [27]
33 F	Constipation	Bilateral N.S	WD-NET	Right RN With left PN	AWD (38 months); Liver metastasis	Rodríguez-Covarrubias et al. [28]
66 M	Flank pain	Isthmus T: 5 cm	WD-NET	RN	NED (3 years) LN metastasis	Briones et al. [29]
76 F	Incidental	Isthmus T: 5.5 cm Cystic	Low-grade NET	Tumorectomy	NED (12 months)	Lodding et al. [30]
23 M	Abdominal pain	Isthmus N.S	NET and mature teratoma with ossification	PN	NED	Isobe et al. [31]
58 M	Abdominal pain	Isthmus T: 4.5 cm	NET	PN	NED (12 months)	Soulié et al. [32]

Abbreviations: T, tumor size (cm); N.S, not specified; L, left; R, right; M, male; F, female; AWD, alive with disease; DOD, died of disease; NED, no evidence of disease; G1, grade 1; G2, grade 2; LCNEC, large-cell neuroendocrine carcinoma; LN, lymph node; PN, partial nephrectomy; RN, radical nephrectomy; RCC, renal cell carcinoma; WD-NET, well-differentiated neuroendocrine tumor.

Table 2. Neuroendocrine neoplasm grading of the digestive system [14].

Differentiation	Grade	Mitotic Count ^a	Ki-67 Index ^b	Tumour Necrosis
NET	G1	<2	<3	Present or absent
	G2	2–20	3–20	
	G3	>20	>20	
NEC	Poorly differentiated	n/a ^c	>20	Present ^d

NET: neuroendocrine tumour, NEC: neuroendocrine carcinoma, n/a: not applicable; ^a in areas of 2 mm². ^b Percentage of positive cells stained with Ki-67 clone MIB1 in areas of highest nuclear labelling (hotspots); in case of grade discrepancy between mitotic count and Ki-67, the highest is to be indicated. ^c NECs are high grade by definition, therefore grade is not assessed further. ^d The presence of tumour necrosis is one factor that may support the diagnosis of NEC over NET but is not definitive on its own and not mandatory when reporting NEC.

The pathological findings in our case are consistent with those reported in the literature. The tumor was classified as a well-differentiated grade 1 neuroendocrine tumor according to the recommendations of the 5th edition of the WHO Classification of Urinary and Male Genital Tumours.

3.5. Treatment and Prognosis

The standard treatment for well-differentiated neuroendocrine tumors (NETs) arising in horseshoe kidneys is conservative surgery, consisting of tumor enucleation, with or without isthmectomy, or partial nephrectomy [6,7]. No cases of secondary recurrence or multifocality have been reported that would question this conservative approach [1,2,6]. Surgery remains the treatment of choice, even in the presence of regional lymph node metastases [5,6]. Conversely, poorly differentiated neuroendocrine carcinomas require systemic chemotherapy from the outset, highlighting the importance of obtaining a pre-operative histopathological diagnosis by biopsy to guide treatment selection [6,7,14]. In patients with unresectable, recurrent, or metastatic well-differentiated NETs, long-acting somatostatin analogues (octreotide or lanreotide) constitute the mainstay of systemic therapy. However, prolonged treatment may be associated with biliary complications, particularly gallstone formation [3,33]. In patients with progressive disease, targeted therapies such as the mTOR inhibitor everolimus may be considered, while peptide receptor radionuclide therapy (PRRT) and, in selected cases, immunotherapy represent additional therapeutic options according to tumor biology and somatostatin receptor expression [16,17]. The prognosis of well-differentiated NETs after surgery is generally excellent; recent data report an overall survival rate of 85.4% [2] and a complete remission rate without recurrence in 73.2% of cases [2,6,12]. Nevertheless, late distant metastases have been described, sometimes occurring more than seven years after surgery, underscoring the need for prolonged follow-up [5,7]. Gallium-68 PET remains the gold standard for detecting recurrent or metastatic disease during follow-up [3,5]. Overall, these tumors are characterized by slow growth and low biological aggressiveness [1,2,7,12].

Compared with primary renal NETs arising in anatomically normal kidneys, primary renal NETs associated with horseshoe kidneys do not appear to exhibit major clinicopathological differences regarding clinical presentation, histological features, or immunohistochemical profile. However, they are characterized by a markedly higher frequency of association with horseshoe kidney, as previously discussed, and by a preferential location within the renal isthmus, supporting the hypothesis that embryological abnormalities may contribute to their development. Regarding prognosis, early reports suggested a more favorable outcome for primary renal NETs arising in horseshoe kidneys, even in the presence of regional lymph node metastases. However, recent reviews have not confirmed horseshoe kidney as an independent prognostic factor. Instead, prognosis appears to be more closely related to tumor characteristics, particularly tumor size, the presence of metastatic disease, and proliferative activity. Given the rarity of this entity and the limited number of reported cases, the prognostic significance of horseshoe kidney remains uncertain [4,7,12].

4. Conclusions

Well-differentiated primary renal NETs are rare neoplasms with a significantly higher incidence in horseshoe kidneys. A definitive diagnosis relies on histopathological and immunohistochemical examination, demonstrating neuroendocrine marker expression and excluding metastatic involvement from an extrarenal neuroendocrine tumor. Nephron-sparing surgery remains the treatment of choice whenever feasible. Although the prognosis is generally favorable, prolonged follow-up is recommended because late recurrences and distant metastases have been reported. Therefore, primary renal neuroen-

doocrine tumor should be included in the differential diagnosis of renal masses arising in horseshoe kidneys.

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Institutional Review Board Statement: Ethical review and approval were not required for this retrospective, non-interventional single-case report, which was based on data collected during routine clinical care without any additional diagnostic or therapeutic intervention. According to the Moroccan regulatory framework governing biomedical research, including Law No. 28–13 and its implementing Decree No. 2.20.326, this type of study does not require prior Institutional Review Board approval.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study. This is an observational study without cost or intervention for the patients. Data were collected from medical records with total respect for the confidentiality of patient information. The study was conducted in accordance with the Declaration of Helsinki. Verbal informed consent was obtained from all subjects involved in the study according to institutional guidelines.

Data Availability Statement: All data relevant to this case are contained within the article. Additional de-identified information is available from the corresponding authors upon reasonable request, subject to ethical and privacy restrictions.

Conflicts of Interest: The authors declare no conflicts of interest.

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