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Case Report

Nephroblastoma in Older Adult: Case Report and Review of Literature

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Keywords
Nephroblastoma · Wilms' tumor · Metanephric blastema · Nephrectomy · Renal neoplasms

Abstract
Introduction: Nephroblastoma, or Wilms' tumor, is a malignant renal neoplasm commonly found in children, is extremely rare in adults representing only 0.5% of all renal neoplasms. Adult Wilms tumor is rare, to our knowledge fewer than 300 cases have been reported in the English literature to date. However, in older adults after 60 years of age, only less than 45 cases have been reported. For this reason, treatment guidelines in adults still are lacking. Prognosis in nephroblastoma for adult patients is found to be worse than in children. ***Case Presentation:*** We report the case of a 65-year-old female with lumbar fossa mass, flank pain and hematuria, and pathologic diagnosis of Wilms tumor. We performed nephrectomy. No adjuvant treatment was given. Our patient remains asymptomatic and without evidence of recurrence 12 months after the surgery. ***Conclusion:*** Nephroblastoma in the elderly presents different clinical behavior and prognosis compared to nephroblastoma in children.

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Introduction

Wilms' tumor (WT), named after the 19th century German surgeon Carl Max Wilhelm Wilms, is the most common kidney tumor in infants and children. It is an embryonic tumor that develops from the remains of an immature kidney [1, 2]. The incidence of WT is 10.4 cases per million children under 15 years of age and 0.2 cases per 10,000 babies [3–5].

In adults, it is rare and represents 0.5% of all renal neoplasms. However, in older adults over 60 years of age, this information is unknown [6–8]. Currently, only less than 45 cases have been reported in this age group [9, 10].

There are not clinical data or radiographic investigations that can distinguish it from renal cell carcinoma. First symptoms in adults include pain and hematuria, while children experience palpation detectable, painless, rapidly increasing in size, abdominal mass [8].

Classical histopathological pattern is triphasic: blastemal, epithelial, and stromal. Additional diagnostics such as immunohistochemical staining for the presence of cytokeratin, vimentin, desmin, actin, and WT1 allows to distinguish between other rare cancer types [11].

There is no standard treatment for Wilms tumor in adults, and the therapy protocols are based on those used in children. A worse prognosis is reported for adults compared with children [12].

We report the case of a 65-year-old female with Wilms tumor. We performed nephrectomy. No adjuvant treatment was given. Our patient remains without evidence of recurrence 12 months after the surgery.

Case Report

A 65-year-old female, with a previous medical history of systemic arterial hypertension of 10 years of diagnosis, treated with fimasartan 60 mg orally every 24 h and newly diagnosed type 2 diabetes mellitus without current treatment. No hereditary family oncological history.

She went to a urology consultation in June 2023, due to presenting hematuria without clots, right thoracolumbar pain since from 2 months prior to her assessment, without weight loss or fever. On physical examination with a body mass index of 22 kg/m^2, normal blood pressure, and a palpable abdominal mass was identified in the right hemiabdomen. Laboratory studies were carried out where hemoglobin was reported to be 12.7 g/dL, hematocrit 39%, glucose 100 mg/dL, urea 43.9 mg/dL, creatinine 0.72 mg/dL, normal clotting times, and normal calcium.

Her tomography of the chest, abdomen, and pelvis with contrast, reported a right renal tumor measuring 15.4 × 10.5 cm dependent on the upper pole that crosses the interpolar line and infiltrates the renal sinus, with a hypodense center suggestive of extensive necrosis of the tumor. No evidence of lymphadenopathy suggestive of tumor activity, nor distant disease (see Fig. 1).

One month after, primary right radical nephrectomy, right adrenalectomy, perihilar and paracaval lymphadenectomy were performed. With findings of a right kidney measuring 7 × 5 × 3 cm, with a tumor dependent on the upper lobe measuring 16 × 14 × 13 cm, with multiple vessels of neoformation adhered to the duodenum and liver, with macroscopically normal renal vein, renal artery, ureter, perihilar lymph, and paracaval without data of tumor activity. Adrenal gland macroscopically without alterations and bleeding of 200 mL.

In hematoxylin and eosin staining, a classic triphasic growth pattern was reported, of blastemal cells, stromal cells, and epithelial differentiation that include tubules and primitive glomeruli. Immunohistochemistry reported: positive WT1, positive PAX8, positive AE1/AE3,

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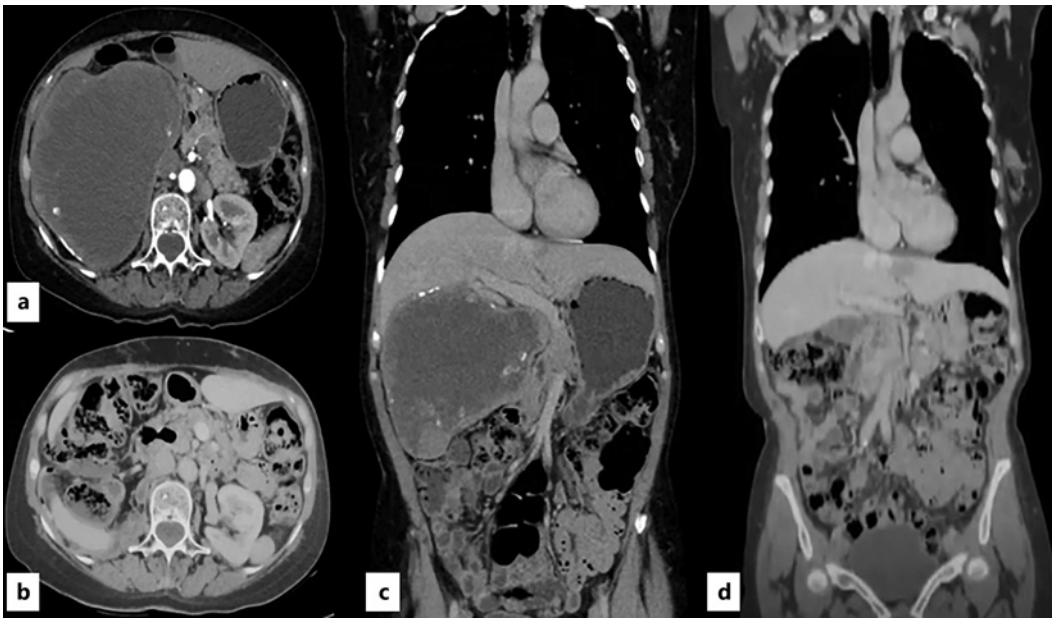

Fig. 1. Chest-abdomen-pelvis computed tomography (CT) scan. Coronal (**a**) and axial (**c**) CT projections showing a right renal tumor measuring 15.4 × 10.5 cm dependent on the upper pole that crosses the interpolar line and infiltrates the renal sinus, with a hypodense center suggestive of extensive necrosis of the tumor. No evidence of lymphadenopathy suggestive of tumor activity, nor distant disease. In control, computed tomography scan, coronal (**b**) and axial (**d**), without evidence of locoregional or distant tumor activity.

positive CD117. The pathology service reported that the tumor was completely resected and there is no evidence of tumor at or beyond the margins of resection. With penetration of the renal capsule, the tumor was not ruptured. The vessels of the renal sinus are not involved (see Fig. 2). It was concluded in partially differentiated cystic nephroblastoma, low grade (G1), mitosis <1 per 10 x high power fields, no sarcomatoid or rhabdoid component and adrenal gland without evidence of activity.

Assessed by medical oncology who concludes in an initial risk group: low (age >2 years, tumor >550 g, no lymph node (LN) disease, no pulmonary or extrapulmonary metastases, and complete surgical resection confirmed by pathology). Children's oncology group (COG) staging of Wilms tumor was stage II. Cytogenetic and molecular testing for unfavorable biomarkers, including chromosome 1q gain and/or 11p15 loss of heterozygosity (LOH) or loss of imprinting or combined LOH at 1p and 16q in chromosomes, were not performed.

Because she was a geriatric patient, with comorbidities and a poor family support network, active surveillance was decided. Currently, the patient is asymptomatic, with good functional status and with a contrast-enhanced tomography study of the chest, abdomen, and pelvis with no evidence of tumor activity (see Fig. 1).

Discussion

Renal cell carcinoma is a common kidney tumor in adults, but WT rarely affects adults over 16 years old. The true incidence of adult WT is somewhat uncertain because of confusion in terminology and difficulties in clinical and pathological differential diagnosis [13].

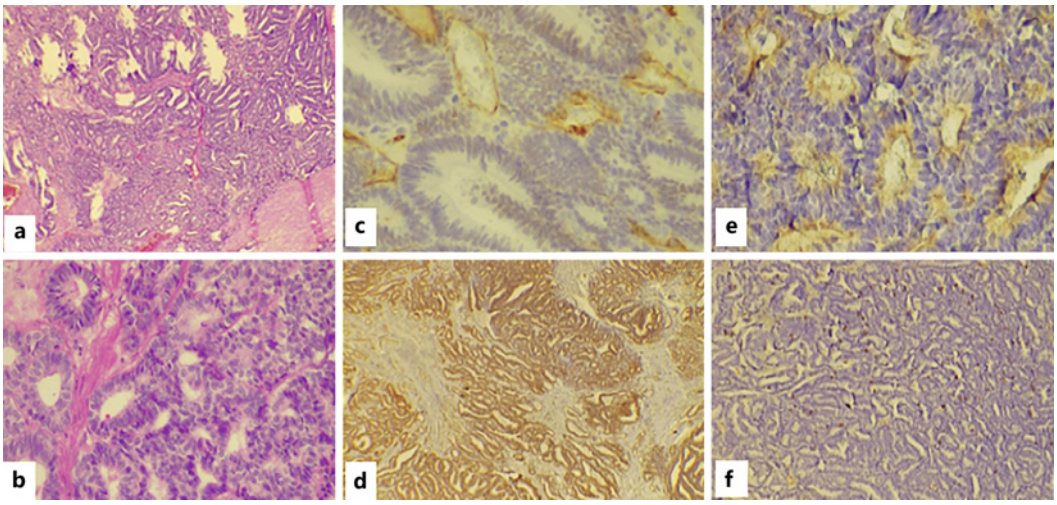

Fig. 2. The microscopic study showed: in hematoxylin and eosin staining (**a**, **b**), a classic triphasic growth pattern was reported, of blastemal cells, stromal cells, and epithelial differentiation that includes tubules and primitive glomeruli. Immunohistochemistry revealed positive staining for WT1 (**c**), PAX8 (**d**), AE1/AE3 (**e**), and CD117 (**f**).

Reported incidence in Europe and USA is 0.2 per million per year and only about 300 cases have been documented [14]. The incidence of WT is highest among African American children, followed by Caucasian children, and then Asian children.

Only 0.5–3% of WT occurs in adults and age of the patients ranges from 15 to 99 years (median age 66.5 years) [8, 15–17]. Wilms tumor occurring in adults differs from that occurring in children in several ways. Adults rarely present with bilateral disease (<1%). More adult patients had additional primary malignancies (both before and after the diagnosis of Wilms tumor) compared with their pediatric counterparts.

Mitry and et al. [8] reported 27 patients with WT aged 60–99 years, where 12 (44.4%) men and 15 (55.6%) women were reported. We carried out an exhaustive review in the English literature to date. We only found 40 patients over 60 years of age reported. However, clinical characteristics, stage, and treatment have been reported in only 15 patients, with an average age of 78.9 years, being more common in men (see Table 1) [10, 17–28].

WT arises from abnormal proliferation of metanephric blastema. Wilms' diagnostic criteria based on Kilton, Matthews, and Cohen criteria are: (1) the tumor must be the primary neoplasm of kidney, (2) the presence of primitive blastemic spindle or round cell component, (3) formation of primary or embryonal glomerulotubular structures, (4) no region of renal cell carcinoma found, (5) histological confirmation, (6) age over 15 years [14].

Clinical Presentation

First symptoms in adults include pain and hematuria, while children experience palpation detectable, painless, rapidly increasing in size, abdominal mass [1]. Weichert et al. [29] and Reinhard et al. [17] reported that the clinical presentation of adults with WT differs from that of children. The main symptom of adults is flank pain and the majority of them have a history of weight loss and a sudden drop in performance status [14–29]. Left-sided renal tumors can be confused on clinical examination with splenomegaly, and right-sided tumors with hepatomegaly. Less common symptoms include varicocele, hernia, enlarged testicle, congestive heart failure, hypoglycemia, Cushing syndrome, pleural effusion, acute abdomen and acute rupture, bleeding, and shock.

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Table 1. Nephroblastoma in older adult – general characteristics, clinical stage, therapy, and survival

Author	Age, years	Gender	Symptoms	Clinical stage	Therapy	Survival
Clay [18]	80	F	Flank pain	NC	Nephrectomy	NC
Jenkins [19]	67	M	Flank pain Hematuria Weight loss >6 months	IV	NC	Died in 6 days
Twinem [20]	75	M	Hematuria frequency	III	Nephrectomy	Died in 3 months
Hill [21]	77	M	Flank pain for 1 year Abdominal mass Hematuria for 8 weeks	I	Nephrectomy Radiotherapy	Alive 16 years
Olsen and Bischoff [22]	67	M	Hematuria for 6 weeks	I	Nephrectomy	Alive 10 years
Lacroix and Cattaneo Godio [23]	68	M	Hematuria Abdominal mass for 2 months	II	Nephrectomy	Alive 11 years
Roux et al. [24]	68	F	Abdominal mass	IV	Nephrectomy Radiotherapy	1 month
Behr and Duari [25]	77	F	Abdominal mass Flank pain Weight loss for 18 months	I	Nephrectomy	NC
Lurie et al. [26]	77	M	Hematuria Flank pain	II	Nephrectomy Chemotherapy	Died
Vogelzang et al. [27]	85	NC	NC	I	Nephrectomy	Post-surgery dead
Vogelzang et al. [27]	74	NC	NC	IV	Biopsy only	1 month
Vogelzang et al. [27]	62	NC	NC	I	Simple nephrectomy	23 months
Ali and Elnashar [28]	90	F	Right abdominal mass Abdominal pain Dyspnea Weight loss	IV (NWTS-3)	Nephrectomy Radiotherapy Chemotherapy	Died 2 months after starting chemotherapy
Biyani et al. [10]	76	M	Abdominal pain for 3 months Weight loss	NC	Left nephrectomy	Died 6 weeks after *H. influenzae* infection

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Table 1 (continued)

Author	Age, years	Gender	Symptoms	Clinical stage	Therapy	Survival
Reinhard et al. [17]	62	F	Abdominal pain	III	Chemotherapy: vincristine, dactinomycin, doxorubicin + radiotherapy with 30 Gray (Gy)	Complete remission

Of the 15 patients reported in the English literature [10, 17–28], the clinical presentation of adults with WT was hematuria (40%), flank pain (33.3%), abdominal mass (26.5%), weight loss (26.6%), and abdominal pain (20%) (see Table 1). The currently reported case presented hematuria and flank pain as the main symptom similar to what was reported in the literature.

The most common sites of hematogenous metastases include lung (81%), lung and liver (15%), and other sites (4%); spread to regional LNs also occurs. However, WT rarely metastasizes to the bone, skin, bladder, large intestine, central nervous system, and the opposite kidney [29–33].

Diagnostic Approach

Abdominal ultrasound is typically the first imaging modality utilized, because it is usually easily obtained, can be performed without sedation, and can most often quickly ascertain both the presence of a mass and organ of origin. In ultrasonography, it is a complex and large mass with large cystic components compared to renal cell carcinoma tumor, which is often a solid mass. The WT angiogram shows a hypo-vascular tumor with neovascularization within the tumor, which is called "spaghetti pattern or creeping-vine" [33, 34].

Abdominal computed tomography (CT) or magnetic resonance imaging is then often used to evaluate the extent and involvement of the renal mass. Radiographically on CT, WT may present as inhomogeneous mass with low density and less contrast enhancement than the normal parenchyma. Some are seen as complex, cystic masses, with solid components and calcification, thus mimics other renal tumors. Characteristics were presented in our reported case. Chest CT should be done to evaluate for pulmonary nodules, which is the most common site of metastatic disease [1].

Core needle biopsies, guided by interventional radiology, or open biopsy can be considered. Dykes et al. [35] reported that, in Europe percutaneous needle biopsy generally is used in children to assess the nature of massive renal tumors and if performed before nephrectomy, it has been shown to be effective in approximately 90% of cases. To avoid potential tumor spread from malignant tumors, biopsy is not routinely recommended before upfront surgery. If the patient has a resectable unilateral renal tumor (outside the setting of known WT predisposition syndromes), upfront nephrectomy is recommended when feasible. Surgery is the mandatory first treatment for adults in order to confirm histology and includes LN sampling for staging.

Histopathology and Genetic Basis

Histopathologically, there is no difference between WT occurred in adult and child. An adult Wilm's tumor with mainly epithelial differentiation should be differentiated from renal cell carcinoma and metanephric adenoma [32]. Classical histopathological pattern is triphasic: blastemal, epithelial, and stromal [36]. Hence, the histologic classification of Society of Paediatric Oncology (SIOP) provides adequate risk stratification. The three groups proposed by the revised SIOP histologic classification are as follows: (a) low risk (completely necrotic nephroblastoma or cystic partially differentiated nephroblastoma), (b) intermediate risk

(regressive, epithelial, stromal, mixed, or focal anaplastic nephroblastoma), and (c) high risk (blastemal or diffuse anaplastic nephroblastoma) [36, 37]. Renal cell carcinoma may have undifferentiated or sarcomatous areas which should be distinguished from blastemal or mesenchymal component of WT, respectively. Similarly, differential diagnosis of adult Wilm's tumor with predominantly blastemal component is small round blue cell tumors (including rhabdomyosarcoma, lymphoma, and primitive neuroectodermal tumor), metastatic small cell carcinoma from lung, immature teratoma, renal cell sarcoma, and poorly differentiated renal cell carcinoma with large sarcomatous component resembling blastema [1, 32]. Additional diagnostics such as immunohistochemical staining for the presence of desmin, actin, cytokeratin, vimentin, and WT1 allows to distinguish between other rare cancer types such as: renal sarcoma, mesoblastic nephroma, clear cell sarcoma, or rhabdoid tumor [12].

The genetic basis of WT is complicated. The most common germline variants involve WT1, which codes a transcription factor that is essential for normal kidney/genitourinary function. Familial WT gene mutations (FWT1/FWT2) are rare (1%–2% of WT) and are not associated with the WT1 mutation. For children with WT, their siblings will rarely get WT (<1%). FWT1 is on chromosome 17q; FWT2 is on chromosome 19q. *WT1* is a gene located within 11p13 is mutated in 10% of tumors and is found in WAGR syndrome, Denys-Drash syndrome, and Frasier syndrome, and is associated with bilateral WT [38].

WT2 is a gene located within 11p15 and results in overexpression of IGF2; it occurs in Beckwith-Wiedemann syndrome [39]. Wilms tumors occurring in adults have not been shown to develop in association with nephrogenic rests or be associated with developmental conditions such as WAGR, Denys-Drash, or Beckwith-Wiedemann syndromes [40].

Numerous somatic genetic variants are associated with WT; the most common are CTNNB1, DROSHA, WT1, WTX, WTX (AMER-1), DGCR8, SIX1, BCORL1, MLLT1, MYCN, SIX2; TP53 is associated with anaplastic WT. WT predisposition genes by exome sequencing include: REST, TRIM28, FBXW7, NYNRIN, KDM3B, XPO5, CHEK2, and PALB2 [41, 42]. While BRAF V600E variants are extremely uncommon in pediatric Wilms tumor, they are present in 90% of metanephric adenomas of the kidney, a typically benign condition arising almost exclusively in adults [40].

Staging

The staging of the disease is based on the Children's Oncology Group (COG) Staging of Wilms Tumor. Both the results of the imaging studies and the surgical and pathological findings at nephrectomy are used to determine the stage of disease. The stage is the same for tumors with favorable histology or anaplastic histology.

Abdominal staging can be stage I in 43% of patients (limited to renal parenchyma, completely resected. The renal capsule is intact. The tumor was not ruptured or biopsied prior to removal); stage II, demonstrating invasion into renal pelvis or renal capsule, the tumor is completely resected and there is no evidence of tumor at or beyond the margins of resection (20% of patients); or stage III in 21% of patients (with tumor outside the capsule, remaining in the abdomen, including finding of positive margins, confirmation of preoperative or intraoperative tumor spill or rupture, positive LNs, or tumor without upfront resection, tumor implants are found on the peritoneal surface). Patients with any evidence of metastatic disease (most commonly the lungs and liver, bone, brain, etc.) or LN metastases outside the abdominopelvic region seen on imaging are staged as overall stage IV (11% of patients). In stage V, Wilms tumor (5% of patients), bilateral involvement by tumor is present at diagnosis. An attempt should be made to stage each side according to the above criteria on the basis of the extent of disease [43, 44].

Treatment of Adults with Wilms Tumor

As Wilms tumor rarely occurs in adults, there is no standard treatment protocol. These patients are treated using the protocols for children developed by the National Wilms Tumor Study in North America and the Society of Paediatric Oncology (SIOP) in Europe. According to the guidelines, treatment protocols involve a combination of surgery, chemotherapy, and sometimes radiotherapy [12].

Radical nephrectomy (the removal of the tumor along with the kidney with the adrenal gland and LNs of the same side) is treatment of choice of one-sided nephroblastoma. LN status is a major long-term predictor of outcome in patients with Wilms tumor.

According to SIOP, a partial kidney resection (nephron sparing treatment) is only allowed in precisely designated cases such as in the presence of developmental disadvantages in the other kidney, genetically predisposed diseases in which the risk of nephroblastoma development is high, and in patients who only have one kidney [38, 39].

Risk stratification is used to determine the most appropriate therapy to minimize both risk of recurrence and long-term toxicity from treatment. Tumor histology, histopathologic and surgical stage, molecular markers (LOH of 1p and 16q), presence of metastatic and/or bilateral disease, and clinical factors – including age of the child, presence or absence of predisposition syndromes, and response of pulmonary lesions to neoadjuvant chemotherapy – are all used in risk stratification [45–47]. The presence of specific molecular biomarkers – such as LOH of 1p and 16q, 11p15, and 1q gain – identified in tumor tissue is associated with increased risk of relapse after initial therapy. Cytogenetic and molecular testing –for 1q gain and/or LOH of 1p and 16q – is recommended for all children with newly diagnosed FHWT [48, 49].

Initial risk assessment is based on age and clinical, radiographic, surgical, and pathologic findings. Final risk assessment is based on the initial risk factors plus presence or absence of unfavorable molecular biomarkers and the response of the lung metastases at week 6, if applicable. Factors indicating need for more intensive therapy include: older age at diagnosis, unfavorable/anaplastic histology, higher stage, larger tumor weight, unfavorable molecular biomarkers, and incomplete lung nodule response to neoadjuvant chemotherapy at week 6 [50].

Preoperative Chemotherapy

Data show that neoadjuvant and/or adjuvant chemotherapy in combination with surgery (with or without RT) improves survival for most children with WT. Preoperative chemotherapy before nephrectomy is indicated in the following situations, which have been listed previously under situations requiring a biopsy: Wilms tumor in a solitary kidney; synchronous bilateral Wilms tumor; extension of tumor thrombus in the inferior vena cava above the level of the hepatic veins. Tumor involves contiguous structures whereby the only means of removing the kidney tumor requires removal of the other structures (e.g., spleen, pancreas, or colon but excluding the adrenal gland). Inoperable Wilms tumor and pulmonary compromise resulting from extensive pulmonary metastases [51–54].

Postoperative Chemotherapy

This is to be started as soon as ileus subsides after surgery and is decided based on the postoperative histology as well as stage. The AREN0532 (NCT00352534) trial was designed to confirm the findings from NWTS-5 that adjuvant chemotherapy could be omitted for children younger than 2 years at diagnosis with stage I FH Wilms tumor that weighed less than 550 g [49, 55–59].

The most used treatment in stage I intermediate risk, stage II and III low risk, stage II-IV intermediate risk, stage I high risk is the combination with vincristine, actinomycin, and adriamycin. In stages II-IV of unfavorable high-risk histology, carboplatin/etoposide and CYCLO-ADR (cyclophosphamide-doxorubicin) should be administered.

Radiation therapy should be started 9–14 days after surgery, unless medically contraindicated. Not recommended in stage I/II with favorable histology. RT is administered to the flank, except when total abdominal irradiation (WAI) is indicated under the following conditions: diffuse tumor effusion, intraperitoneal tumor rupture, and peritoneal tumor seeding/hemorrhagic ascites or positive cytology. Other indications for radiotherapy are macroscopic residual disease after surgery, recurrent abdominal disease, pulmonary metastasis, liver metastasis, skeletal metastasis, and unresected LN metastasis [55–60]. Currently, cases of BRAF V600E-altered Wilms tumors have been reported in patients older than 30 years, and it has even been reported a male (aged 51 years) who had a relapsed metastatic Wilms tumor that harbored a BRAF V600E variant [61].

Prognosis and Prognostic Factors for Wilms Tumor

Wilms tumor is a curable disease in most affected children. Since the 1980s, the 5-year survival rate for Wilms tumor with favorable histology (FH) has been consistently greater than 90% [62–64]. The prognosis for patients with Wilms tumor depends on the following: histopathological features of the tumor (FH vs. anaplastic histology); stage of disease at diagnosis. Molecular features of the tumor such as 1q gain and LOH of 1p and 16q. 1q gain, affecting 28% of Wilms tumors, is the most powerful predictor of outcome and is associated with an adverse outcome [49, 50, 64, 65]. LOH of 11p15 and loss of imprinting of 11p15 is associated with relapse in very low-risk patients who do not receive chemotherapy [66]. Older age is associated with an adverse prognosis [67]. The EUROCARE Working Group, reported patients older than 60 years. Survival in patients of 60 years and over: 1- and 5-year survival was 58% and 42%, respectively. Five-year relative survival from WTs in adults (60–99 years) was 26.9% in males and 49.85 in females [8].

Conclusion

Nephroblastoma is extremely rare in adults. For this reason, treatment guidelines in adults still are lacking. Prognosis in nephroblastoma for adult patients is found to be worse than in children. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material, available at https://doi.org/10.1159/000540279.

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Statement of Ethics

This retrospective review of patient data did not require ethical approval in accordance with local/national guidelines. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

The authors (Ricardo Fernández-Ferreira and Jose Manuel Torres-Zazueta) contributed to the conception of the case, analysis and critical revision of the content, as well as the final approval of the version to be published. César Martínez-Medrano, Adrián Meléndez-Mendoza, María Alejandra Muñoz Rubiano, Gredel Portela-Rubio, Julieta Robles-Castro, Jorge Alberto Robles-Aviña, and José Manuel Ruíz Morales contributed to the critical revision of the content, as well as the final approval of the version to be published. Sonia Tavares-García carried out an exhaustive review of the histopathological characteristics of cancer and analysis of the article. We all agree to be responsible for all aspects of the job to ensure that questions related to the accuracy or completeness of any part of the job are properly investigated and resolved.

Data Availability Statement

Data supporting the findings of this study are openly available in the clinical file of the Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Mexico City, Mexico, with registration number: 11715241186F1959PE. Additional inquiries can be directed to the corresponding author.

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