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Adult Wilm's tumor (Nephroblastoma), with pulmonary and liver metastasis: Case report of a rare tumor and a brief review of the literature

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Abstract

Wilms' tumor (WT) (nephroblastoma) is a childhood renal neoplasm encountered rarely in adults. Although radiological imaging is an excellent tool for diagnosis in most cases, histomorphologic diagnosis can be challenging, especially with limited sampling, which may result in the potential absence of one of the three histological components. In addition, the rare occurrence in adults makes it less likely to be included in the differential diagnosis of an adult presenting with a renal mass. We report a case of a 21-year-old woman who presented with a left abdominal palpable mass, eventually diagnosed with an adult Wilms tumor (AWT). Following the Children's Oncology Group (COG) protocol, the patient underwent total left nephrectomy. The histological features were classic triphasic, with blastemal (40%), epithelial (45%), and stromal (15%) components, and occasional foci of anaplasia, which were not sufficient to classify it as an anaplastic subtype. The patient had adjuvant chemotherapy with Dactinomycin and Doxorubicin and initially responded well to treatment.

Nine months after nephrectomy, pulmonary and liver metastases were found. Surgical treatment was not possible due to the high tumor burden in the lung and the liver. Chemotherapy, including Vincristine, Dactinomycin, and Doxorubicin, was initiated, resulting in a favorable clinical response. The patient was disease-free at the last follow-up, 26 months after treatment. This case highlights the diagnostic and therapeutic challenges associated with AWTs. We provide a brief review of the literature, including history, clinical presentation, diagnosis, and treatment. We also discuss the potential benefits of diagnosing and treating AWTs with current technological advancements.

Keywords: Wilms Tumor; Nephroblastoma; Adult; Pediatric; WHO Classification; Molecular

1. Introduction

In children, the most frequent renal tumor is WT, or nephroblastoma. This tumor represents 85–90% of all pediatric renal tumors. WT peaks in incidence at 2-3 years, and commonly occurs during the first 5 years of life. [1] The tumor was named after Max Wilms, a German surgeon who provided the first detailed account of this renal neoplasm in 1899, although the first cases were described in the mid-19th century. [2]. WT was reported bilaterally in 5-10% of cases [3], and was very rarely, also to be found in the extra-renal region. [4] [5]

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The pathogenesis of WT is thought to be due to a malformation of renal organogenesis, resulting in an overproduction of the metanephric blastema compared with normal kidney development. This embryonic origin explains the classical histologic triad of blastemal, epithelial, and stromal elements, which correspond to the various phases in the normal development of the kidney. [6] Although WT is more common among pediatric patients, it occurs as a very rare entity at adult age, accounting for less than 1% prevalence of adult renal tumors and 0.2% to 0.5% among WT series. [1] Its infrequent adult presentation presents substantial diagnostic and therapeutic challenges, as adult patients with renal masses may not suspect this tumor and it may be excluded from the differential diagnosis. AWT was historically used to label tumors found in a patient over the age of 15-16 years, although some report 18 years. [7]

The clinical presentation of AWT tends to mimic that of adult renal neoplasms usually presenting as a painful abdominal mass, with hematuria. (8) Nevertheless, diagnostic procedures and therapeutic management are challenging for this group of patients, as data on the best approach are scarce for this group of patients. The prognosis of adult Wilms' tumor seems to be less favorable than in children, with a more advanced stage at diagnosis and higher rates of relapse, thus requiring intensive multimodal treatment and long-term follow-up. [9]

2. Case presentation

A 21-year-old woman previously healthy, presented with a long history of left-sided abdominal mass, dull aching abdominal pain, hematuria, paroxysmal hypertension, and anemia. The patient had noticed the mass approximately 5 months prior to presentation, but she came to hospital due to recent increase in size. She had no significant past medical or family history and denied any known genetic syndromes or risk factors.

Abdominal ultrasound was the initial imaging technique, demonstrating a significant and heterogeneous mass involving the left kidney, with hypoechogenic and necrotic areas. Enhanced CT revealed a 13 cm well-marginated but heterogenous mass on the upper pole of the left kidney. The lesion appeared to deform the adjacent renal contour and extend into the adjacent renal parenchyma. There was no lymphadenopathy, contralateral kidney involvement, or systemic metastasis. All laboratory analyses, including renal function tests, blood count, coagulation profile, and catecholamines (to exclude neuroblastoma) were unremarkable.

The patient’s case was presented at the multidisciplinary tumor board, and the group recommended upfront surgery following the Children's Oncology Group (COG) strategy with adjuvant chemotherapy. COG advocates for primary surgery for treatment, but SIOP (International Society of Pediatric Oncology) uses preoperative chemotherapy as the first line of treatment

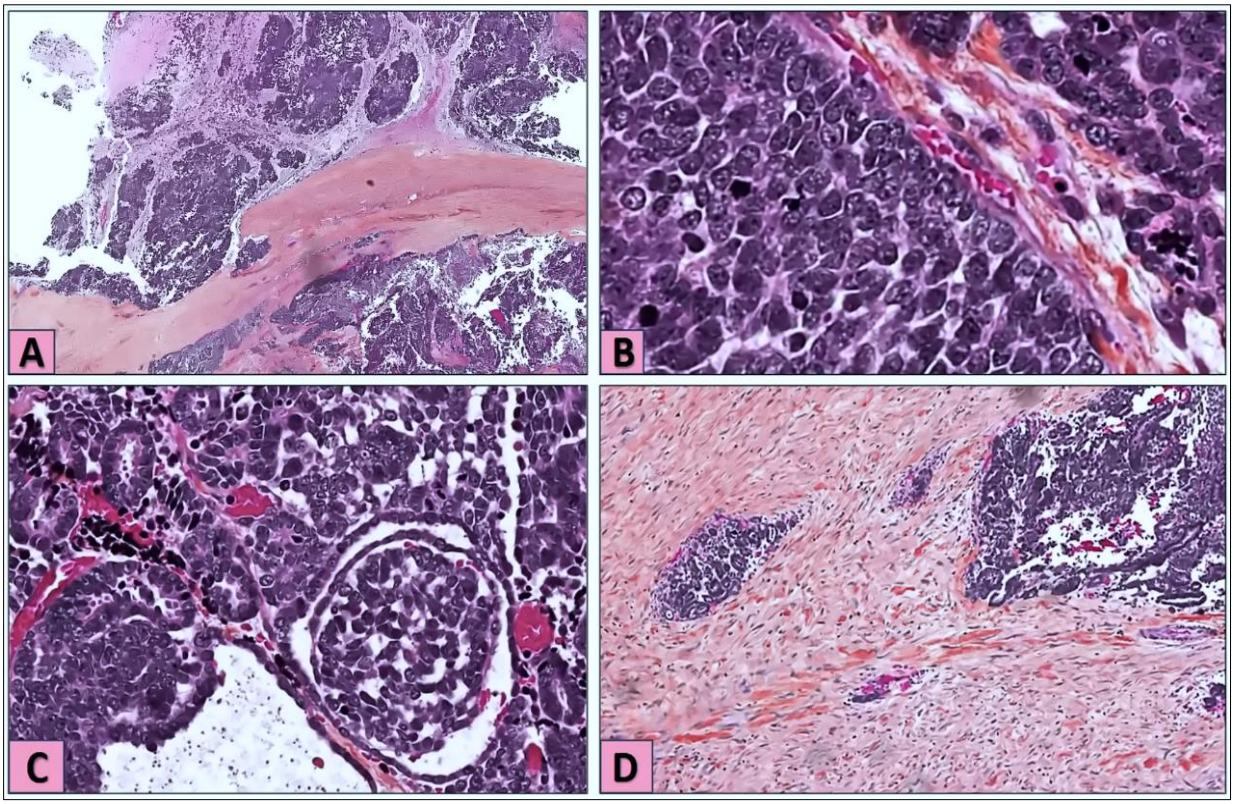
Complete left nephrectomy was performed without a prior biopsy or other tissue sampling. Intra-operatively, the tumor was completely enucleated, without any rupture in the surrounding tissue. Macroscopically, there was a single, large, well-defined 13 cm mass with a lobulated, gray-white to pink-gray cut surface, cystic areas, focal hemorrhage, and necrosis. Microscopically, the tumor showed a triphasic Wilms tumor pattern, with the blastemal element accounting for 40%, the epithelial element 45%, and the stromal element 15%. Some scattered foci of anaplasia were seen but did not meet the COG guidelines for classification of anaplastic Wilms tumor. (Figure 1 A, B, C, D) Of importance, the tumor involved the renal sinus and invaded blood and lymphatic vessels without accessing surgical resection margins. There were no positive lymph nodes in any of the five sampled lymph nodes.

Immunohistochemistry (IHC) studies supported the diagnosis. Blastemal cells were positive for WT1, PAX8, and vimentin but negative for HMB45, Melan-A, and Fli-1. Epithelial components were positive for cytokeratin, EMA, CD56, variable staining with CK7 and focal WT1. Stromal elements were positive for vimentin, BCL2, and CD34, but were negative for WT1. Molecular analysis detected a TP53 mutation (17p13) without other relevant changes. According to these results, the lesion was identified as a non-anaplastic Wilms tumor stage II (as the renal sinus and vascular invasion were present, but complete resection was performed). Postoperative adjuvant chemotherapy, including Dactinomycin and Doxorubicin was given for 24 weeks and no radiotherapy was given.

Metastasis was found in the lungs and liver 9 months later. The disease was too widely metastatic to be resected surgically, and the patient received systemic chemotherapy with Vincristine, Dactinomycin, and Doxorubicin. She responded well, and no recurrence was noted in the subsequent 26 months of follow-up. Unfortunately, the patient was subsequently lost to follow-up. Table 1 summarizes the clinical course timeline of our patient.

Table 1 Clinical Course Timeline

Time Point	Event
Month 0	Presentation with abdominal mass, hematuria, pain
Month 1	Imaging studies, tumor board evaluation
Month 2	Total left nephrectomy
Month 3–9	Adjuvant chemotherapy (Dactinomycin + Doxorubicin)
Month 9	Lung and liver metastases detected
Month 10–18	Chemotherapy (Vincristine + Dactinomycin + Doxorubicin)
Month 26	Last known follow-up, disease-free
After Month 26	Lost to follow-up



1A Low power view showing lobulated masses of mall basophilic nodules, separated by abundant fibroblastic and myxoid stroma (HandE stain X20); **1B:** High power view showing undifferentiated small round-to-oval cells with hyperchromatic nuclei and scant cytoplasm, apoptotic cells, and frequent mitosis (HandE stain X60); **1C:** High power view showing tubules of the epithelial component, glomerulus-like structures, and cystic spaces (HandE stain X40); **1D**: Intermediate power view showing prominent cellular fibrous component surrounding nodules of blastema (HandE stain X40)

Figure 1 Histomorphology of the adult Wilms tumor (AWT)

3. Discussion

3.1. History, Epidemiology, and Risk Factors

WT is one of the most common pediatric renal tumors, having an incidence of approximately 8 cases per million children in the United States.

The rate is extremely low in teens and adults, at less than 1%. [10] WT is complex because it is rare and atypical entity, with no standardized guidelines have been defined for management. [10] In adults, many cases are initially

misdiagnosed as renal cell carcinoma until histopathological diagnosis is done.[6] Most Wilms tumors (WTs) occur sporadically, but a significant number (10–15%) can present in associated with congenital syndromes such as Beckwith-Wiedemann syndrome, Denys-Drash syndrome, and WAGR syndrome. The latter is characterized by aniridia, genitourinary anomalies, mental retardation, in addition to WT. [11] This syndromic association is rare in adults. [11]

A genetic predisposition has been reported due to mutations in the tumor suppressor genes WT1 and WT2, located on chromosomes 11p13 and 11p15, respectively. [2] No predisposing factors were present and there was not any family history of malignancy in our patient, highlighting the sporadic nature of the disease in adults. Recognizing the epidemiological differences and distinct risk profiles between pediatric and adult populations is important, as this highlights the diagnostic dilemma and the need for a high index of suspicion when treating atypical renal masses in young adults. Table 2 shows a comparison between pediatric vs. adult Wilms tumor

Table 2 Comparison: Pediatric vs. Adult Wilms Tumor

Feature	**Pediatric Wilms Tumor (WT)**	**Adult/Adolescent Wilms Tumor (AWT)**
Peak Age	3–4 years	Rare >15 years
Incidence	~6% of childhood cancers	<1% of all renal tumors
Common Symptoms	Abdominal mass, hematuria, hypertension	Flank pain, mass, hematuria, systemic symptoms
Diagnosis Delay	Often early due to routine pediatric care	Frequently delayed, misdiagnosed as other tumors such as renal cell carcinoma
Histology	Often classic triphasic	May mimic other adult renal tumors
Treatment Protocols	Well-established (COG/SIOP)	Lacks standardized adult-specific protocol
Prognosis	Excellent (>90% 5-year survival)	Generally worse due to late diagnosis and advanced disease

3.2. Current WHO Classification of Wilms tumor

In the most recent edition of the World Health Organization (WHO) classification of renal tumors, 5th edition (2022), a comprehensive and well-standardized diagnostic criterion for WT was clarified. Two variants of WT were recognized, based on histological and clinical behavior: The first variant is the Favorable Histology (Non-anaplastic WT), which encompasses most of the WT cases. It does not appear to be anaplastic and seems to respond well to the currently available multimodal therapeutic strategies. Histologically, it is characterized by a combination of blastemal, stromal, and epithelial elements to varying degrees, presenting an overall triphasic pattern. This pattern strongly suggests WT, but it is not mandatory for diagnosis. [12] This was the case in our patient. The second variant is Unfavorable Histology (Anaplastic Wilms Tumor). This is based on existence of diffuse anaplasia with relatively large, hyperchromatic, pleomorphic nuclei and atypical mitoses. It is associated with treatment resistance as well as a poor prognosis. Focal anaplasia may also occur in a minority of tumors but it is not considered unfavorable unless it meets well-defined criteria validated by the COG. [12]

Additionally, the WHO is focusing on the impact of molecular alterations (e.g., TP53 mutation), which may be identified with a US FDA-approved gene-based test that can better distinguish more anaplastic tumors and those with worse prognoses. [13] Classifications are integrated with new genetic and molecular findings, but diagnosis remains defined by histopathology. [8] AWT is not classified as a distinct entity in the new WHO classification. It is considered a tumor diagnosed based on histologic and molecular features, similar to pediatric WT. [6]

3.3. Clinical Presentation and Imaging Studies

The most frequent presentation of WT in children is a painless abdominal mass detected by a caretaker. However, some other symptoms (i.e., abdominal pain, hematuria, hypertension, and fever) may also appear to indicate tumor development. [3] [8] These constitutional and local symptoms are more severe in adults. Our patient, a 21-year-old girl, came with an abdominal mass on the left side, the presence of hematuria, pain in the left flank, anemia, and hypertension, features all suggestive of renal carcinoma. WT was not considered on initial differential diagnosis due to patient age.

Imaging is a crucial part of the workup and staging of Wilms' tumor. It is also the initial imaging study in children and usually demonstrates a large, well-contained, heterogeneous mass. [10] In the present paper, the abdominal ultrasound revealed a large, heterogeneous, hypoechoic mass with necrosis, originating from the left kidney. CT imaging is the gold standard for further characterization, evaluation of tumor size, vascular involvement, lymphadenopathy, and potential metastases. [14] On CT imaging, there was a 13 cm solid renal mass with distant metastasis, preserved contralateral kidney, and no evidence of lymphatic dissemination. In adults, the "typical" imaging appearance seen in children may be absent. Histopathologic examination is often required to establish the diagnosis. [8]

3.4. Pathogenesis and Pathophysiology

Through nephrogenesis, embryonic renal cells differentiate from the undifferentiated mesenchyme within the metanephric blastema. Under normal conditions, nephrogenesis is completed at birth; however, in WT, nephrogenesis is arrested to form a triphasic tumor showing blastemal, epithelial, and stromal elements that are analogous to mature fetal kidney anatomy. [15]

The pathogenesis of Wilms' tumor is based on genetic and epigenetic changes interfering with central developmental pathways. One of the best-known genes implicated is WT1 (11p13) and WT2 region (11p15). WT1 (11p13) is a tumor suppressor gene that plays a role in the development of the kidney and gonad. WT1 mutations or deletions are identified in both sporadic and syndromic cases (such as WAGR and Denys-Drash syndrome). The WT2 region (11p15) encompasses the IGF2 gene and is associated with epigenetic regulation. Loss of imprinting and overexpression of IGF2 have been associated with tumorigenesis, especially in Beckwith-Wiedemann syndrome. [16]

Tumors with anaplastic histology also have a higher frequency of TP53 mutations, which are linked with chemoresistance and poor prognosis. In our instance, a TP53 mutation was noted despite the absence of diffuse anaplasia, suggesting possible molecular heterogeneity. [13] [15] Other genetic changes include mutations in CTNNB1 (β-catenin) and WTX, as well as abnormalities at the level of miRNA processing (i.e., DROSHA, DICER1). (17) From a pathophysiologic point of view, WT is characterized by the unlimited division of undifferentiated cells from the nephrogenic population, following their escape from regulatory influence. [13] It is likely that invasion of the renal sinus and blood vessels, as observed in our case, represents the local infiltration capacity of the tumor, without frank metastases at the time of diagnosis. The triphasic histology represents varying degrees of arrested differentiation, with the blastemal component is often considered the most aggressive and the least likely to respond to therapy. [10]

Knowledge of the molecular basis for WT has led to a greater understanding of tumor biology, as well as the opportunity to identify risk and plan therapy with targeted agents in the setting of relapsed or high-risk disease.

3.5. Pathology: Gross, Microscopic, Immunohistochemistry, and Molecular

Pathological examination of Wilms tumor remains the primary tool for diagnosis, staging, and risk assessment. Grossly, Wilms tumors are large, round masses with well-defined edges that frequently distort or replace the standard renal structure. [8] The cut section of the tumor may exhibit a variegated appearance, with gray-white and/or pink-gray areas of tissue, lobulation, necrosis, cystic degeneration, and hemorrhage. [18] In the present case, the 13 cm mass on the upper pole of the left kidney was well demarcated with a friable texture, with focal, cystic, and hemorrhagic changes were observed.

Microscopically, WT classically displays a triphasic morphology with blastemal, epithelial, and stromal components. [8] [18] In our case, 40% of the tumor was composed of the blastemal component, the most primitive and aggressive component. The epithelial component showed tubules, papillary structures, and occasional glomeruloid or rosette-like formations (comprising about 45% of the tumor). The stromal component, representing 15% of the tumor mass, revealed focal hypocellularity and hypercellular regions with spindle cells, and no heterologous differentiation was seen. These features were consistent with other reports describing WT. [10] [11] The stromal component was seen in 15% of the tumor mass. Focal areas of anaplasia were occasionally seen, though they did not satisfy COG guidelines for designation as anaplastic WT.

Pathological differential diagnosis based on histomorphology included: neuroblastoma, rhabdoid tumor, and desmoplastic small round cell tumor. IHC studies were essential for making such a differentiation in our case. The tumor cells exhibited diffuse expression of WT1, PAX8, and vimentin in the blastemal cells, with sporadic positivity for cyclin D1. The epithelial element was positive for cytokeratin, EMA, CD56, and weakly positive for WT1. Vimentin, BCL2, and CD34 were positive in stromal components, while WT1 was negative. The tumor tested negative for CK7, Fli1, HMB45, and Melan-A, thus successfully excluding other small round cell blue tumors. Our tumor IHC studies' reactions were similar to previously reported profiles. [10] [11] At the molecular level, a TP53 mutation (17p13) was detected, a finding

usually found in anaplastic WT and chemoresistance. [13] [15] In this case, a TP53 mutation in a morphologically non-anaplastic tumor could represent a more aggressive biological behavior and highlights the necessity for molecular analysis to be integrated into histopathologic evaluation. Table 3 summarizes the immunohistochemistry profile of Wilms' tumor.

Table 3 Immunohistochemistry Profile Summary of Wilms tumor (WT)

Marker	Component (s) Positive	Staining Pattern	Diagnostic Significance
WT1	Blastema (diffuse), weak in epithelium	Nuclear	Supports Wilms tumor
PAX8	Blastema	Nuclear	Renal origin
Vimentin	Blastema, Stroma	Cytoplasmic	Mesenchymal elements
Cytokeratin	Epithelium	Cytoplasmic	Epithelial differentiation
EMA	Epithelium	Membranous	Supports epithelial nature
CD56	Epithelium	Membranous	Common in WT epithelium
CD34	Stroma	Cytoplasmic	Stromal marker
Cyclin D1	Rare blastema cells	Nuclear	Not specific
HMB45, Fli1, Melan-A	Negative	–	Helps exclude RCC, sarcomas, melanomas

3.6. Management and Prognosis

Survival for Wilms tumor in children has seen significant, dramatic changes over the last few decades, and with the combined actions of clinical trial groups, such as the Children's Oncology Group (COG) and the International Society of Pediatric Oncology (SIOP), survival rates worldwide are greater than 90% for those with localized disease. [19] Because of the rare presence of WT in adolescents and adults, adult-based guidelines and treatment of WT in this age group is not well defined. [11]

In the pediatric population, multi-agent risk-adapted therapy is the standard. In general, this consists of surgical resection (nephrectomy) and subsequent chemotherapy, with radiation therapy being used in select high-risk cases, usually stage III disease or tumors with diffuse anaplasia. [20] [21] The standard chemotherapy regimen consists of vincristine, dactinomycin, and doxorubicin, depending on the histologic subtype and stage. [11] In our case, the patient was managed with primary radical nephrectomy without any preoperative chemotherapy following the COG protocol for resectable stage II tumors. She was treated with favorable-risk, non-anaplastic histology therapy with dactinomycin and doxorubicin postoperatively. Regrettably, although she had initially localized disease and responded well to the treatment, she had pulmonary and hepatic metastatic recurrence at 9 months. It is a rare complication but has been well described even with favorable histology tumors. Metastasectomy was precluded due to the bulk of metastatic disease, so she was started on a combination chemotherapy regimen with vincristine, dactinomycin, and doxorubicin, to which a complete response was achieved.

The prognosis in Wilms tumor is usually favorable in children. However, prognosis is usually poor in adults and adolescents because of late presentation, more aggressive tumor biology, and in the absence of age-specific treatment guidelines. [22] [23] The presence of TP53 mutations, particularly in histologically non-anaplastic tumors, may predict a higher recurrence rate or resistance to treatment. This was the case in our patient and is an observation of interest that should be further investigated. Regular long-term follow-up is essential to enable early detection of relapse and also to monitor patients for the late effects of chemotherapy, including cardiotoxicity and secondary neoplasms. [23] Our patient had no evidence of disease for 26 months following recurrence, and was then lost to follow-up, emphasizing the importance of structured survivorship care plans in young adults.

3.7. Nephron-Sparing Surgery for Bilateral Wilms Tumor

Most WTs are unilateral, but approximately 5-10% of children with Wilms tumor have bilateral disease. The incidence of bilateral tumors in adults is unknown due to the rarity of WT in adults and the lack of large, investigated series. Bilateral Wilms tumor (BWT) is a challenging clinical dilemma requiring a delicate balance between the cure of cancer

and the maintenance of renal function. Nephron-sparing surgery (NSS), i.e., partial nephrectomy, stands as a crucial therapeutic modality managing the delicate balance between removing tumor tissue on one hand and preserving as much healthy kidney parenchyma on the other [24] [25] Current treatment standards for bilateral Wilms tumors generally feature or preoperative chemotherapy as one of the first lines of therapy, followed by staged nephron-sparing resections.

NSS's technical aspects are highly demanding and require skilled surgery and attention to detail. Surgeons are challenged by the intricate renal anatomy coupled with the need to excise the tumor completely, as they are frequently working within small operative slots with thin margins of healthy tissue. Notwithstanding these dilemmas, the outcomes after NSS for bilateral Wilms tumors have been auspicious in the long term [26]

The importance of a multidisciplinary approach to the treatment of bilateral Wilms' tumor cannot be overstated. The successful treatment of these patients requires seamless cooperation among pediatric oncologists, urologists, anesthesiologists, pathologists, and supporting personnel, following predefined schedules that prioritize both the immediate surgical outcome and the long-term well-being of the patient [27]. As our knowledge of tumor biology grows and surgical practice continually advances, so should NSS stand as a testament to what can be achieved when clinical advances are rooted in the timeless adage "first, do no harm."

3.8. What Does the Future Hold, and What Did We Learn from This Case?

One of the greatest achievements in pediatric oncology is the successful management of WT; however, AWT is still a battleground with many uncertainties remaining. In the future, for these diagnoses and treatments, we believe the following developments will focus on molecular profiling, risk stratification, and personalized therapy.

The combination of next-generation sequencing (NGS) with molecular diagnostics is already yielding significant diagnostic and therapeutic benefits. Evaluation of genetic markers, such as TP53, WT1, IGF2, and the miRNA processing genes, is now being pursued not only for diagnosis but also for the identification of tumors at a higher risk of recurrence or resistance to chemotherapy. [13] [15] [28] As we continue to accrue and molecularly profile cases in adult and adolescent patients, we could potentially generate age-stratified prognostic models and perhaps design targeted therapies, especially for relapsed or refractory disease. Artificial intelligence (AI) and machine learning, when applied to radiology, pathology, and genomic data, also offer the potential for earlier detection and more precise classification. [29] Furthermore, the generation of minimally toxic, personalized treatment protocols could enable better quality of life and long-term results, especially for young adult patients who are often between pediatric and adult oncology.

From this case, we noted the need to maintain a broad differential diagnosis when considering a renal mass in a young adult, even if the clinical history and age suggest a more typical adult malignant process. It is important to confirm histology and immunohistochemistry when possible. Moreover, the recurrence of our patient and the known relationship to other clinical and molecular risk factors, in the absence of anaplastic histogenesis, illustrate the relevance of identifying molecular risk factors, such as TP53 mutations and in non-anaplastic tumors.

Rare presentations are a source of education. It reminds clinicians that vigilance is essential, and the multidisciplinary cooperation, the long-term follow-up and reporting of atypical cases, can promote the development of medical knowledge both in the clinic and in research.

4. Conclusion

This case is an example of an unusual presentation of Wilms' tumor in a young adult patient; it emphasizes the difficulties in diagnosis and treatment of disease in this age group. With the close correlation between clinical, radiological, histopathological, immunohistochemical, and molecular data, a final diagnosis was reached, and an appropriate therapy was introduced. Although the patients presented with and were managed effectively for localized disease, the possibility of metastatic recurrence calls for molecular risk evaluation and close long-term follow-up. It highlights the importance of recording rare occurrences and learning from atypical presentations are important to develop the most effective diagnostic approaches and improve therapeutic outcomes for rare tumors in the future.

Compliance with ethical standards

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All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Other relationships

All authors declare that no other relationships or activities could be perceived as influencing the submitted work.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

The patient was lost to follow-up, and all attempts to reach the family members were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

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