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MOLECULAR MARKERS AND THEIR ROLE IN WILMS TUMOR

Dr. Sarvar Temurovich Islomov
Oncologist, pediatric oncosurgery, interventional oncologist.
Scientific researcher in department of "oncology and hematology",
National children's medical center.
294 Parkent street, Tashkent, Uzbekistan.
E-mail: stimurovich@gmail.com
Dr. Sukhrob Abdurashidovich Tashmatov
Pathologist, Pediatric Pathology, General Pathology.
Head of Pathology Department
National Children's Medical Center
E-mail: tashmatov@mail.com
Gafur-Akhunov Mirza-Ali
Professor, Head of department of Center for the development of professional qualification of medical workers of Ministry of Helath Of the Republic of Uzbekistan,
E-mail: gafurahunovmirzaali@gmail.com
Tursunov Khasan Ziyayevich
Professor, Head of department of pathologic anatomy, Tashkent Medical Academy
E-mail: Tursunov.hasan@bk.ru

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Abstract:

Wilms tumor, also known as nephroblastoma, is a pediatric kidney cancer that is one of the most treatable solid tumors in children. However, in adults, Wilms tumor is a rare and often aggressive malignancy, with a less favorable prognosis compared to its pediatric counterpart. (Huszno et al., 2013) Understanding the molecular landscape of Wilms tumor is crucial for improving treatment strategies and patient outcomes. One of the key molecular markers in Wilms tumor is the WT1 gene, which plays a crucial role in regulating normal differentiation in various organs. Loss or overexpression of WT1 can result in different phenotypic consequences depending on the status of cellular differentiation, as well as its oncogenic or tumor-suppressor effect. In addition to WT1, other important markers in Wilms tumor include WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p (Hamilton & Shamberger, 2012). Patients with certain congenital anomalies, such as Beckwith-Wiedemann syndrome, WAGR syndrome, and Denys-Drash syndrome, have an increased risk of developing Wilms tumor (Varan, 2008). Recent advances in our understanding of Wilms tumor biology have had a significant impact on clinical treatment.

Keywords: WT1, WT2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, miR-130b-3p, Wilms tumor, nephroblastoma, pediatric oncology

The Role of WT1 in Wilms Tumor

The WT1 gene, known as the Wilms tumor 1 gene, is a crucial player in Wilms tumor biology. WT1 is involved in regulating normal differentiation in various organs, and its loss or overexpression can lead to different phenotypic consequences depending on the status of cellular differentiation and the presence of other gene mutations. Understanding how cells respond to the loss or alteration of WT1 in various stages of differentiation and in the presence of other gene mutations is an area of ongoing research.

WT1 can function as both an oncogene and a tumor suppressor, depending on the cellular context. Its role in Wilms tumor is complex and multifaceted, and further research is needed to fully elucidate its mechanisms of action in this disease.

Other Molecular Markers in Wilms Tumor

In addition to WT1, several other molecular markers have been studied in the context of Wilms tumor. WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p have all been implicated in the pathogenesis an The refinement of the prognostic classification of Wilms tumor patients by features such as these genetic markers, as well as histological

patterns, the presence of lymph node metastases, and the degree of local invasion, has allowed for the progressive individualization of therapy for a subset of patients (Hamilton & Shamberger, 2012) (Ehrlich, 2001) (Varan, 2008).

The future goals of Wilms tumor therapy include intensifying treatment and developing new therapeutic strategies for high-risk patients, as well as decreasing the intensity of therapy for low-risk patients to minimize the effects of treatment.

The Impact of Molecular Markers on Clinical Treatment

The refinement of prognostic classification of Wilms tumor patients based on features such as genetic markers, histological patterns, lymph node metastases, and local invasion has allowed for the progressive individualization of therapy for a subset of patients. This has led to the development of more targeted and personalized treatment approaches.

Recent advances in our understanding of the molecular biology of Wilms tumor have had a significant impact on clinical treatment. Treatment regimens have evolved from a three-drug chemotherapy approach with surgery and radiotherapy, to a more tailored approach based on disease stage. Patients with early-stage Wilms tumor are now treated with a two-drug chemotherapy regimen without radiotherapy, while those with advanced disease still receive the more intensive three-drug regimen and radiotherapy. (Varan, 2008) (Hamilton & Shamberger, 2012)

Incorporating chemotherapy into the initial treatment plan, in combination with radiation therapy and surgery, has produced an 89% survival rate for tumors confined to the kidney. (Varan, 2008) (Fleming & Johnson, 1970) (Hamilton & Shamberger, 2012) The addition of radiation therapy to the renal fossa postoperatively is also thought to have increased the survival rate to as high as 60-70%. (Fleming & Johnson, 1970)

Further research into how cells respond to the loss or alteration of WT1 and other molecular markers in various stages of differentiation and in the presence of other gene mutations, as well as the impact of the tumor microenvironment, are crucial for optimizing treatment strategies and improving patient outcomes in Wilms tumor (Hamilton & Shamberger, 2012).

Uncovering the Molecular Landscape of Wilms Tumor

The molecular markers WT1, WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p play a critical role in the pathogenesis and progression of Wilms tumor. WT1 is a key regulator of normal differentiation in various organs, and its dysregulation can have profound effects on tumor development and behavior. Other markers, such as p53 and Bcl2, are also known to be involved in Wilms tumor, with their altered expression contributing to the malignant phenotype. (Varan, 2008)

Recent advances in our understanding of the molecular underpinnings of Wilms tumor have informed the development of more targeted and personalized treatment approaches, leading to improved outcomes for patients.

Deciphering the Molecular Underpinnings of Wilms Tumor

Wilms tumor is a complex and heterogeneous disease, with a variety of genetic and molecular alterations contributing to its development and progression. One of the most well-studied molecular markers in Wilms tumor is the WT1 gene, which is a tumor suppressor gene that plays a critical role in the normal development and differentiation of the kidney. Additionally, other important molecular markers in Wilms tumor include WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p (Hamilton & Shamberger, 2012).

Understanding the intricate interplay between these molecular markers and their impact on the pathogenesis and clinical behavior of Wilms tumor is crucial for developing more effective treatment strategies.

Dissecting the Molecular Profile of Wilms Tumor

Wilms tumor is a complex and multifaceted disease, with a diverse array of genetic and molecular alterations contributing to its development and progression. Key molecular markers in Wilms tumor include the WT1 gene, which is a critical regulator of normal kidney development and differentiation, as well as WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p.

Understanding the intricate interplay between these molecular markers and their impact on the pathogenesis and clinical behavior of Wilms tumor is essential for improving treatment strategies and patient outcomes. Patients with certain congenital anomalies, such as Beckwith-Wiedemann syndrome, WAGR syndrome, and Denys-Drash syndrome, have an increased risk of developing Wilms tumor.

Advances in our understanding of the molecular landscape of Wilms tumor have led to the development of more targeted and personalized treatment approaches. Incorporating chemotherapy into the initial treatment plan, in combination with radiation therapy and surgery, has produced an 89% survival rate for tumors confined to the kidney.

Unveiling the Molecular Complexity of Wilms Tumor

Historically, the treatment of Wilms' tumor has been surgical excision, with long-term survival of up to 36% reported after nephrectomy alone. The addition of radiation therapy to the renal fossa postoperatively is thought to have increased the survival rate to as high as 60-70%. The most notable chemotherapeutic agents that have proven to be effective in the treatment of Wilms' tumor are dactinomycin and vincristine sulfate, which have been useful in the treatment of metastatic tumor with remarkable tumor regression and prolongation of survival.

Wilms' tumor is the most frequently occurring renal tumor in children and is one of the most treatment-responsive tumors. WT1, WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p are key molecular markers that play a critical role in the pathogenesis and progression of Wilms' tumor.

Understanding the complex interplay between these molecular markers and their impact on the development and clinical behavior of Wilms' tumor is crucial for optimizing treatment strategies and improving patient outcomes.

Molecular Markers as Diagnostic and Prognostic Tools in Wilms Tumor

Wilms' tumor is a complex and heterogeneous disease, with a diverse array of genetic and molecular alterations contributing to its development and progression. (Varan, 2008) WT1, WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p are key molecular markers that have been extensively studied in the context of Wilms' tumor.

Understanding the role of these molecular markers in the pathogenesis and clinical behavior of Wilms' tumor is crucial for the development of more effective diagnostic and prognostic tools, as well as targeted therapies. Patients with certain congenital anomalies, such as Beckwith-Wiedemann syndrome, WAGR syndrome, and Denys-Drash syndrome, have an increased risk of developing Wilms' tumor,and this risk may be mediated by the dysregulation of these molecular markers.

Advances in our understanding of the molecular landscape of Wilms' tumor have informed the development of more personalized and targeted treatment approaches. Incorporating chemotherapy into the initial treatment plan, in combination with radiation therapy and surgery, has produced an 89% survival rate for tumors confined to the kidney. (Fleming & Johnson, 1970)

Molecular Biomarkers in Wilms Tumor: Implications for Clinical Practice

Wilms' tumor is a complex and heterogeneous disease, with a diverse array of genetic and molecular alterations contributing to its development and progression. Key molecular markers, such as WT1, WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p, (Varan, 2008) (Hamilton & Shamberger, 2012) (Fleming & Johnson, 1970)
have been extensively studied in the context of Wilms' tumor. Understanding the role of these molecular markers in the pathogenesis and clinical behavior of Wilms' tumor is crucial for the development of more effective diagnostic and prognostic tools, as well as targeted therapies.

Patients with certain congenital anomalies, such as Beckwith-Wiedemann syndrome, WAGR syndrome, and Denys-Drash syndrome, have an increased risk of developing Wilms' tumor, and this risk may be mediated by the dysregulation of these molecular markers.

Advances in our understanding of the molecular landscape of Wilms' tumor have informed the development of more personalized and targeted treatment approaches. Incorporating chemotherapy into the initial treatment plan, in combination with radiation therapy and surgery, has produced an 89% survival rate for tumors confined to the kidney. (Varan, 2008) (Fleming & Johnson, 1970)

Molecular Pathways and Their Dysregulation in Wilms Tumor

Wilms' tumor is a complex and heterogeneous disease, with a diverse array of genetic and molecular alterations contributing to its development and progression. Key molecular markers, such as WT1, WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p, have been extensively studied in the context of Wilms' tumor.

WT1 is a critical regulator of normal kidney development and differentiation, and its loss or overexpression can have differing phenotypic consequences depending on the status of cellular differentiation and the presence of other gene mutations. Understanding how cells respond to the loss or alteration of WT1 in various stages of differentiation and in the presence of other gene mutations in variable microenvironments is crucial for understanding the malignancy.

Similarly, the dysregulation of other molecular markers, such as WT-2, WTCD34, p53, Ki-67, Bcl2, miR-100-5p, and miR-130b-3p, can contribute to the development and progression of Wilms' tumor. Advances in our understanding of these molecular pathways have informed the development of more personalized and targeted treatment approaches, such as the incorporation of chemotherapy into the initial treatment plan, in combination with radiation therapy and surgery, which has produced an 89% survival rate for tumors confined to the kidney.

CONCLUSION

Understanding the role of these molecular markers in the pathogenesis and clinical behavior of Wilms' tumor is crucial for the development of more effective diagnostic and prognostic tools, as well as targeted therapies. Advances in our understanding of the molecular landscape of Wilms' tumor have informed the development of more personalized and targeted treatment approaches, leading to improved patient outcomes. Based on these data, we will continue research in this direction with subsequent publication of the results.

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