12th International Conference on

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Keynote Forum

Day I

Petr Starostik, J Clin Exp Pathol 2017, 7:1 (Suppl)
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Petr Starostik

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Implementation of NGS causes dynamic shifts in clinical molecular diagnostics

The past decade witnessed a true revolutionary change in ways how mutation detection is performed in a clinical laboratory. Single analyte tests were replaced in many labs by multianalyte next-generation sequencing (NGS). This technique significantly decreased the sequencing cost per-base, enabled labs to analyze much higher number of samples at once, and broadened the analysis scope from a single gene to gene panels or even the whole exome/genome. Many new so far unknown gene mutations were discovered by NGS. They can be used as biomarkers in diagnosis and their availability led to changes in tumor classification. They are also potential drug targets to develop targeted therapies. Several manufactures supply NGS instruments and reagents to detect both somatic and germline mutations. Many laboratories opt to develop their own laboratory-developed NGS assays which can be easily tailored to meet their needs. We developed both amplicon- and hybridization probe-based NGS assays used to detect driver and druggable mutations in different types of cancers. The assays were extensively validated, and allow for quick and sensitive detection of point mutations and indels for the most relevant therapeutic genes in several types of cancers. The complexity of NGS does not make its implementation easy. NGS wet lab workflow entails several critical steps like sample and sequencing library preparation which are critical for success. Bioinformatics is an integral part of NGS and needs to be handled by an experienced IT specialist to not only develop appropriate analysis pipeline but to also make the results available in the appropriate format in the electronic medical records. Administrative leadership is needed to secure proper reimbursement and keep track of government regulations and oversight.

Biography

Petr Starostik is an Associate Professor of Pathology and serves as the Director of Molecular Pathology in the Department of Pathology, Immunology, and Laboratory Medicine in the College of Medicine, University of Florida in Gainesville, FL. Over the years, he directed several molecular diagnostics laboratories, both in the USA and abroad. Development of Molecular Diagnostic Tests is his specialty as evidenced by his publications and the multitude of laboratory-developed tests performed in laboratories he directed. Besides clinical work, he also pursues basic research focusing on the role of FLT3 ITD in acute leukemia.

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Electron Paramagnetic Resonance (EPR) spectroscopy for diagnosis and characterization of mitochondrial dysfunction and diseases

Mitochondrial disease (MD) presents with a wide range of clinical, pathological and biochemical outcomes and is consequently very difficult to diagnose conclusively. EPR is a magnetic resonance technique that detects and characterizes unpaired electrons that are present in transition metal ions in certain oxidation states {e.g. $Fe^{(III)}$, $Cu^{(II)}$ and $Mn^{(II)}$}, clusters (e.g., $[2Fe2S]^{+}_{red}$, $[3Fe4S]^{+}_{ox}$, $[4Fe4S]^{+}_{red}$) and free radicals (e.g., $UQ^{\bullet -}$, FADH•). The mitochondrial respiratory chain complexes I-IV contain 23 potentially paramagnetic centers that exhibit distinct EPR signals depending on their redox potentials, the availability of electrons, the catalytic competence of each of the enzymatic complexes and the integrity of the electron transport chain (ETC). In addition, EPR signals may be observed from $UQ^{\bullet -}$, and from the $[3Fe4S]^{+}$ cluster of m-aconitase that arises due to oxidative stress. Key factors thought to be involved in the symptoms and pathology of MD is lowered ATP production and the production of toxic reactive oxygen species (ROS). Either or both of these can occur when electron transfer is impeded due to lowered expression, lowered activity, or structural alteration of ETC complexes, or compromised ingress or egress of reducing equivalents. EPR of rapidly-frozen fresh biopsy tissue is uniquely able to provide a snapshot of the electron distribution among the redox centers in the functioning mitochondrial ETC against a background of other biochemical and pathological assays. We recently described the first application of this methodology to a rat model of MD and will here describe progress toward translation of the approach for diagnosis and differentiation of MDs in children.

Biography

Brian Bennett has completed his BA and MA in Natural Sciences from the University of Cambridge and DPhil in Biochemistry from the University of Sussex. Currently, he is a Chair and Distinguished Professor of Physics at Marquette University, USA.

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Hong L Drum, J Clin Exp Pathol 2017, 7:1 (Suppl)
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Hong L Drum

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Chronic myelogenous leukemia with B-lymphoid blasts crisis at presentation: A case report and literature review

Chronic myelogenous leukemia (CML) with B-lymphoid blast crisis at presentation is rare. In this study, we present a case of a 52-year-old female without a prior known history of hematologic malignancy, who was presented with CML with B-lymphoid blast crisis. Review of peripheral blood smears showed moderately increased white blood cells with left-shifted granulocytosis and basophilia. Bone marrow core biopsy demonstrated markedly increased cellularity with marked, left-shifted myeloid hyperplasia. Megakaryocytes were increased with frequent small hypolobated forms. Blasts were increased, comprising 22% of the marrow. The blasts were positive for PAX-5, CD10, CD19, CD34, and TdT, and negative for MPO, consistent with B-lymphoblasts. Quantitative PCR detected BCR-ABL1 transcript (the major breakpoint, p210) at 70.5820% on the International Scale and t(9;22)(q34;q11.2) was detected by cytogenetic study. A diagnosis of CML with B-lymphoid blast crisis at presentation was rendered based on the above findings. Distinguishing a CML with B-lymphoid blast crisis at presentation from a de novo B-acute lymphoblastic leukemia (B-ALL) with t(9;22) often is not easy. The morphological features that point to a CML with B-lymphoid blast crisis rather than a de novo B-ALL with t(9;22) include concurrent presence of basophilia and left-shifted granulocytosis in the blood, and left-shifted myeloid hyperplasia and increased small atypical megakaryocytes in the bone marrow. Among these morphologic features, the presence of small atypical megakaryocytes/micromegakaryocytes in the bone marrow is considered to be most specific, although not all CML in B-lymphoid blast crises have this morphologic feature.

Biography

Hong L Drum has completed her MD from Guangdong Medical University, China. She is a senior Hematopathologist at Genoptix Medical Laboratory, a Novartis company. She has received Jaseph J. Kleiner Memorial Award from American Society for Clinical Laboratory Science, USA in 2010. She is certified in Pathology-Hematology by the American Board of Pathology, since 2007 and also certified in Anatomic and Clinical Pathology by the American Board of Pathology, since 2006. She has her publications in several reputed journals.

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Neuroblastoma pathology: An update

Neuroblastoma is often used as an omnibus term for all types of peripheral neuroblastic tumors including neuroblastoma, ganglioneuroblastoma, and ganglioneuroma. Tumors in this group are biologically diverse: Molecular/genomic properties of individual cases are closely related to their unique clinical behaviors. Biologically favorable tumors have a potential of spontaneous regression or tumor maturation and are often associated with a hyperdiploid pattern (whole chromosomal gains without structural abnormalities). Biologically favorable tumors have a potential of spontaneous regression or tumor maturation and are often associated with a hyperdiploid pattern (whole chromosomal gains without structural abnormalities). For neuroblastoma clinical trials, the children's oncology group utilizes their risk-grouping system for patient stratification and protocol assignment based on the combination of clinical stage, age at diagnosis, International Neuroblastoma Pathology Classification, *MYCN* status, DNA index, and segmental chromosomal aberrations. Estimated survival rate for the non-high-risk patients is ~90% with surgery alone (low risk) or with biopsy/surgery and moderate chemotherapy (intermediate risk). In contrast, estimated survival rate for the high-risk patients remains as low as 45~50% even after intensive treatment followed by stem-cell transplantation. Continuous efforts are being made for discovery of actionable/druggable targets in high-risk neuroblastomas. Those potential targets include: *ALK* activating mutation/amplification (dysregulating cell signaling and leading to uncontrolled proliferation of neuroblasts); *TERT* rearrangement and *ATRX/DAXX* mutation (preventing neuroblasts from telomere-mediated senescence); and MYC family protein overexpression- a new concept of highly aggressive "MYC family-driven neuroblastomas" with augmented expression of MYCN or MYC protein, also morphologically characterized by nucleolar hypertrophy (promoting MYC/MAX heterodimer formation for activating down-stream gene targets).

Biography

Hiroyuki Shimada has completed his MD and PhD from the Yokohama City University, School of Medicine and Ohio State University College of Medicine, respectively. He is a Professor of Pathology at the University of Southern California Keck School of Medicine, Founder of International Neuroblastoma Pathology Classification and Director of COG Neuroblastoma Pathology Reference Laboratory. He has been reviewing ~700 neuroblastoma cases per year from US, Canada, Australia and New Zealand and participating in various clinical and translational research activities in the field of Pediatric Oncology. He has authored/co-authored more than 180 papers.

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Cynthia Lorenzo, J Clin Exp Pathol 2017, 7:1 (Suppl)
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Cynthia Lorenzo

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Refractory anemia with ring sideroblasts, with JAK2 and SF3B1 mutations without thrombocytosis: A case report and review of literature

JAK2 mutations are rare in refractory anemia with ring sideroblasts (RARS). We present the case of an 83-year-old female who was presented with anemia and no evidence of cytosis. Blood count revealed the following: WBC 4.9 K/uL, Hgb 9.7 g/dL and platelets 174 K/uL. The bone marrow was variably cellular and showed trilineage hematopoiesis. Erythroid precursors appeared normal in number; however, numerous ring sideroblasts were identified. There were no karyotypic or FISH abnormalities. Molecular studies using next generation sequencing technology showed both JAK2 and SF3B1 mutations. These findings were most consistent with a Myelodysplastic syndrome, RARS. SF3B1 mutations are associated with a favorable prognosis in MDS, and are highly predictive for the presence of ring sideroblasts. The presence of the JAK2 mutation in this patient is unusual. JAK2 mutation usually leads to cytokine hypersensitivity and cytokine-independent growth of hematopoietic cells resulting in uncontrolled proliferation and a myeloproliferative phenotype. JAK2 mutation is rare in myelodysplastic syndromes such as RARS. However, the presence of both JAK2 and SF3B1 mutations has been seen in cases of myelodysplastic/myeloproliferative neoplasms (MDS/MPN), such as RARS associated with marked thrombocytosis (RARS-T). The current case had normal platelet count, and therefore did not satisfy the criteria for RARS-T. The possibility that this patient would later develop an MDS/MPN cannot be excluded. The reason that some MDS patients with JAK2 mutation do not develop a myeloproliferative component is unknown. Further studies are needed to determine the role of JAK2 mutation in these MDS patients.

Biography

Cynthia Lorenzo has completed her MD from Stanford University School of Medicine. She is a Senior Hematopathologist at Genoptix Medical Laboratory, USA. She has her publications in many reputed journals.

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Ismé M de Kleer

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The early origins of allergic asthma in the developing lung

Allergic diseases have their origin in early life when lungs and immune system are still developing. Sensitization to aeroallergens is an important risk factor for asthma in children and influenced by genetic polymorphisms. Ontology of asthma genes indicate the existence of two central themes conferring asthma susceptibility, (i) activation of an innate immune response leading to type-2 immunity (*IL33* and *IL1RL1*, coding for IL-33R and decoy receptor sST2) and (ii) integrity and repair of the airway epithelium (PCDH1 and CDHR3). To understand the susceptibility to allergic asthma of young children, we studied epithelial permeability and innate and type 2 immunity in response to house dust mite (HDM) exposure in neonatal mouse lungs. We found that innate lymphoid type 2 cells (ILC2) and eosinophils spontaneously accumulate in lungs during postnatal alveolarization in an IL-33 dependent manner. HDM exposure further increased IL-33, boosted cytokine production in ILC2, and promoted the migration and Th2 skewing capacity of dendritic cells leading to enhanced sensitization. We found that exposition to high dose HDM resulted in free flow of antigen through the epithelial barrier to the lung draining lymph nodes. This phenomenon was the result of an impaired alveolar barrier due to claudin-18 deficiency expression in alveolar cells. Challenges with high doses of antigen resulted in strong T cell proliferation in lung interstitium and lymph nodes and dendritic cell independent sensitization. We conclude that enhanced neonatal Th2 skewing to inhaled allergens occurs during a phase of postnatal lung remodeling, when the epithelial barrier is not completely closed and the IL-33 axis and spontaneous type 2 immunity drive immunity to allergens.

Biography

Ismé M de Kleer is a Pediatrician and Research Fellow of Pediatric Pulmonology in the Department of Pediatric Pulmonology at Erasmus Medical Center, Rotterdam, Netherlands. She completed her PhD from the Utrecht University in 2004 and Post-doctoral studies at the Flemish Institute for Biotechnology, Ghent University. She is a member of the European Respiratory Society (ERS), Netherlands Respiratory Society (NRS) and Dutch Pediatric Society and has co-authored 27 peer-reviewed articles in international journals.

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Day 2

Sarah Adelaide Crawford, J Clin Exp Pathol 2017, 7:1 (Suppl)
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Sarah Adelaide Crawford

Southern Connecticut State University, USA

Prenatal screening of maternal immune antigen biomarkers linked to microglial regulation of brain development may predict autism risk

Recent discoveries of the connections between the maternal immune system (IS) and prenatal brain development suggest that routine prenatal screening for chronic disorders associated with IS dysfunction may be useful in identifying women at heightened risk for giving birth to a child with autism. Epidemiological studies have shown that the incidence of IS disorders, including systemic lupus erythematosus (SLE), rheumatoid arthritis (RA) and chronic obesity in combination with insulin-resistant diabetes has increased significantly over the past several decades and that pregnant women with these conditions are at increased risk for having a child with autism. For this reason, physiological parameters associated with these prenatal conditions that can be detected before onset or at early stages of disease may serve as biomarkers for increased autism risk. A physiological relationship between maternal IS dysfunction and impaired embryonic/fetal brain development may be defined by critical neurodevelopmental functions of brain microglia that are responsive to both neural and immunological stimuli. Impaired regulation of the developmentally versus immunologically defined functions of brain microglia may represent a primary cause of the neurological impairments characteristic of ASD. This critical cause/effect of relationship provides the rationale for autism risk factor assessment using biomarkers associated with chronic immune conditions that impair the neurodevelopmental functions of microglia as a consequence of their inappropriate immunological activation. Moreover, the connection between abnormal IS function and impaired neural development suggests preventive approaches that can be used to decrease the overall risk for ASD in children born to mothers with these conditions.

Biography

Sarah Adelaide Crawford completed her Doctoral degree in Physicians and Surgeons Department of Biochemistry and Biophysics from Columbia University College. She completed her Master's degree in Biochemistry from the Princeton University. Her Post-doctoral research was carried out at Memorial Sloan Kettering Cancer Center in New York. She is a Professor of Genetics at Southern Connecticut State University and Director of Cancer Biology Research Laboratory. Currently, she is working on the causes and prevention of autism. She has developed a new model to explain the causes of autism and its recent dramatic increase. Applications of this model can be used in preventive approaches to screen for autism risk factors to reduce the occurrence of this disorder.

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Mary Ann G Sanders

University of Louisville, USA

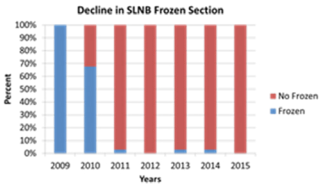
Decline in frozen section diagnosis of sentinel lymph nodes for breast cancer as a result of the ACOSOG Z0011 trial

Statement of the Problem: Results of the multicenter American College of Surgeons Oncology Group (ACOSOG) Z0011 trial published in 2011 showed that patients with early-stage breast cancer and limited sentinel lymph node (SLN) metastasis treated with breast conserving surgery and systemic therapy did not benefit from axillary lymph node dissection (ALND). The Z0011 trial was practice changing for the surgical management of breast cancer, and in turn, has proven to be equally impactful on the pathologic diagnosis of SLNBs. The purpose of this study is to demonstrate the impact of the Z0011 trial on intraoperative frozen section diagnosis of SLNBs.

Methodology & Theoretical Orientation: This is a retrospective study reviewing pathology reports from patients with primary breast cancer who met Z0011 trial clinical criteria and were initially treated with lumpectomy and SLNB from 2009 to 2015.

Findings: SLNBs sent for frozen section diagnosis ranged from 68% to 100% before Z0011 and declined to just 2% of cases after the Z0011 trial results were published in 2011. Of the post-Z0011 cases 19% had SLNs with metastasis and 97% of patients were spared ALND.

Conclusion & Significance: Following publication of the Z0011 trial results, intraoperative frozen section diagnosis of SLNs significantly decreased at our institution. Given that the vast majority of patients did not require second surgery for completion ALND, routine frozen section diagnosis for SLNB can be safely avoided in patients who meet Z0011 criteria, sparing patients the prolonged anesthesia time associated with waiting for frozen section diagnosis results and decreasing health care costs related to extra charges incurred with frozen section diagnosis.



Biography

Mary Ann G Sanders has received her Bachelor's degree from the University of Wisconsin-Madison. After she obtained her MD and PhD degrees from Washington University in St. Louis, she completed an Anatomic Pathology and Clinical Pathology Residency at the University of Louisville followed by a Breast Pathology Fellowship at the Brigham and Women's Hospital, Harvard Medical School. She joined the Department of Pathology at the University of Louisville in 2011 as an Assistant Professor and Breast Pathologist for James Graham Brown Cancer Center. She is an Associate Program Director for Pathology Residency Program at the University of Louisville.

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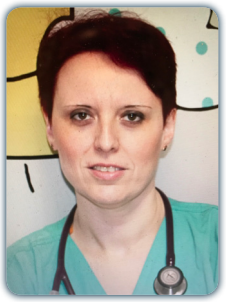

María Amparo Lopez-Ruiz

CEU Cardenal Herrera University, Spain

Neuroleptic-induced oral-facial tardive dyskinesia in a prepuberal boy with an attention-deficit hyperactivity disorder

Introduction & Aim: The use of antipsychotics, antagonists of dopamine D^2 receptors in the treatment of ADHD in children needs further revision given the incidence of side effects of unknown frequency as tardive dyskinesia and acute extrapyramidal symptoms, both induced by haloperidol and the fact that overdose can produce dangerous morbidity, sometimes even requiring intensive care treatment. Patients receiving neuroleptics such as butyrophenone haloperidol for a long period of time can develop several forms of a rare side effect included among the extrapyramidal dyskinetic syndromes, especially oral-facial involuntary movements as well as uncontrolled movements of the extremities called tardive dyskinesia. In this study, we present a case of Tardive dyskinesia, the most frequent group of involuntary movements in patient taking haloperidol.

Case Report: An 11-year-old male patient taking a high dose of haloperidol and methylphenidate in a normal dose for two years was hospitalized due to the severity of the symptoms and eventually the child developed a syndrome of Tardive dyskinesia. Upon admission, medication was stopped and the symptoms disappeared in the next 24 hours. Three days later the boy recovered completely and was discharged from the hospital to follow treatment in the outpatient clinic. There were no recurrences of the Dyskinetic reactions during that summer.

Conclusions: Given the possibility of presentation of Tardive dyskinesia and other acute extrapyramidal symptoms, we strongly recommend avoiding the prescription of haloperidol, especially associated to methylphenidate (also responsible for some cases of Tardive dyskinesia) in the treatment of attention-deficit hyperactivity disorder (ADHD).

Biography

María Amparo Lopez-Ruiz has completed her PhD from Valencia University and Post-doctoral studies from the CEU Cardenal Herrera University. She completed her Doctorate in Medicine with thesis entitled: Analysis of the use of medication in the pediatric population that visits accident and emergency department. She has been a Medical Degree Coordinator at CEU Cardenal Herrera University since 2015. She has attended international congresses of pediatrics as a Keynote Speaker and has published more than 20 papers in reputed journals.

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Michael Kalinin

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A relationship between hypoglycemia, hypothyroidism and zinc deficiency

The interaction of zinc deficiency and hypothyroidism has several reported presentations. The link between hypoglycemia and hypothyroidism is also known, but uncommon. For the last 40 years, the relationship between these two phenomena was illustrated in a handful of articles. We present a six-month-old boy with the unusual combination of these three conditions, his diagnostic evaluation and management. The case presentation is accompanied with a short literature review. According to the literature, zinc deficiency adversely affects thyroid function. Moreover, thyroid function correlates with the glucose homeostasis. Nevertheless, the association of zinc deficiency, hypothyroidism and hypoglycemia has not yet been described in the current available literature in English. Therefore, we suggest that the relationship between zinc and thyroid function should be considered in any case of severe intractable hypoglycemia and extensive skin eruption.

Biography

Michael Kalinin has completed his Medical study from the State Medical Academy, Russia. He completed his training in Pediatrics at the Tel Aviv University and Ben Gurion University, Israel. He has done his subspecialty in the field of Pediatric Intensive Care from the Hospital for Sick Children, Toronto. He has several publications in different fields of Medicine. Currently, he is the Head of Pediatric Intensive Care Unit at Barzilai Medical Center, Israel.

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Raja Alyusuf

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The immunoexpression of glucocorticoid receptors in breast carcinomas, lactational change and normal breast epithelium and its possible role in mammary carcinogenesis

Glucocorticoids (GC) are known to play a role in mammary development and differentiation, thus it is of interest to attempt to delineate it's immunoexpression across a spectrum of mammary epithelia. This study aims to delineate the distribution pattern of GRs in malignant versus non-malignant epithelium with particular emphasis on lactational change epithelium as its cells are considered the most terminally differentiated mammary cells. Immunohistochemistry (IHC) for glucocorticoid receptors (GR) was performed on archival formalin fixed paraffin embedded tissue blocks of 97 cases comprising 53 invasive carcinomas, 21 cases with lactational change and 23 cases showing normal mammary tissue histology. The results reveal an over-expression of GR receptors in mammary malignant epithelium compared to both the normal and lactational group individually and combined together as a non-malignant group. This is the first study to compare GR expression in human lactating epithelium versus malignant and benign epithelium. GR overexpression was also established in HER-2 negative cancers as compared to HER-2 positive ones, while GR immunoexpression in tumors categorized according to grade, estrogen (ER), progesterone receptor (PR) or axillary lymph node (ALN) status showed no statistical difference. It seems that GR expression in mammary epithelium promotes the development of HER-2 negative breast cancer, thus such receptors may become targets for the development of therapeutic interventions. Further studies are required to determine the level of caution that is needed if any in the use of steroid therapy in such category of patients.

Biography

Raja Alyusuf is a Fellow of Royal College of Pathologists since 1998. She is a Consultant Histopathologist with special interest in the area of Breast Treatment. She chaired the Department of Pathology at Salmaniya Medical Complex for 10 years after which she became the Deputy Chief of Medical Staff for Diagnostic Affairs at the Salmaniya Medical Complex. In addition, she is a part-time Associate Professor of Pathology at the Royal College of Surgeons, Ireland-Bahrain branch. She has over 20 publications is reputable journals and is a member of a number of international and national professional associations.

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Mir Anwar

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Wilm's tumor: past, present and future

Wilm's tumor or nephroblastoma is the most common renal tumor in children and is associated with different congenital anomalies and syndromes. Aniridia as well as hypospadias could be indices of first mutation according to Knudson and strong hypothesis i.e., PubMed. The name came from German surgeon Max Wilms who first described about child kidney tumor. Wilm's tumor (WT) is affecting one in 10,000 children in average population. 75% of all tumor occur among the normal children, remaining 25% are with other syndrome or congenital other defects. The frequency of Wilms' tumor (WT) in relatives was estimated to be less than 0.4% in sibs, 0.06% in uncles and aunts, and 0.04% in first cousins. Girls have higher risk then of boys ratio is 0.89:1. Age-WT is most common in young children; with an average age being about 3-4 years. The tumor is less common in elder children and rare in adults. Black communities are more affected than Caucasian and Asian Communities. Mostly unilateral kidney is affected but less commonly bilateral kidney also affects. 90% of WT are unilateral, 5% are of bilateral kidney involvements. Children with WAGR syndrome have about 30% to 50% chance of having a Wilm's tumor. Birth defects like aniridia, hemi-hypertrophy, cryptorchidism, hypospadias, etc., have a link of Wilm's tumor. So per research has not found any strong links between WT and environmental factors either during a mother's pregnancy or after child birth. A significant number of studies in genetics and molecular biology have improved our understanding of this malignancy discovering as well how different genes play a critical role in the organogenesis process. Surgery is obviously followed by chemotherapy. Recent studies from Europe have suggested that in some cases chemo may not be needed to continue as previously thought. It is also one of the successes of Pediatric Oncology with long term survival above 90% for localized disease and 75% for metastatic disease. Successful management of Wilms' tumor necessitates meticulous attention to correct staging of the tumor and a collaborative effort between Pediatric Oncologists, Specialist Surgeons, Radiologists, Pathologists and Radiation Oncologists. The poor outcome for patients with Wilms' tumor (WT) in developing countries has been predicated on late presentation, poverty and low rate of chemotherapeutic access.

Biography

Mir Anwar has done his graduation in Medicine from Bangladesh in 1975. He did his Post-graduation in Pediatrics from Ireland in 1982. He has done his second Post-graduation in Public Health from the University of Massachusetts, USA in 2003. He has worked as a Pediatric Consultant and Public Health Specialist in WHO/UN around the world including Asia, Middle East, Africa, Pacific Island, Ireland and USA. Since 2007, he has been working in different provinces of South Africa with the Department of Health. Presently, he is working as a Clinical and Medical Manager in Richmond Chest Hospital, South Africa. His main research interest is childhood TB with HIV in Sub-Saharan Africa. He was honored by the American Academy of Pediatrics, Royal College of Health, UK, and International College of Pediatrics, etc., for his work. He is an active member of different international and national pediatric organizations. Currently, he is one of the honorary Member of Editorial Board for the *Journal of Pediatrics & Neonatal Biology.*

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