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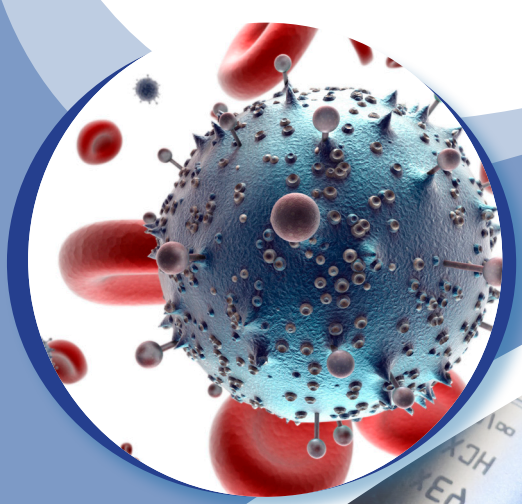
June 20-21, 2018 Paris, France



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15th European Pathology Congress
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Keynote Forum

DAY 1

Zahra Maleki, J Clin Exp Pathol 2018, Volume 8
DOI: 10.4172/2161-0681-C1-044

Zahra Maleki

The Johns Hopkins Hospital, USA

Milan salivary gland reporting system: Highlights and classification schemes

Salivary gland lesions are relatively uncommon and Fine needle aspiration (FNA) is routinely performed to evaluate these lesions. Although it is possible to reach a definitive diagnosis in some cases, there are a considerable number of remaining problematic cases. The issues precluding a definitive diagnosis on aspirated material of salivary gland lesions are as follows: scant cellularity, poorly preserved cells, cellular heterogeneity, squamous metaplasia, variable ratio of the cells and the matrix, uncommon presentation of common entities and finally rare neoplasms. Therefore, rendering a definitive diagnosis on aspirated material can be a diagnostic challenge. Moreover, the clinicians and surgeons heavily rely on diagnosis of salivary gland FNAs for their patient care and management. Milan salivary gland reporting system is introduced to provide a classification scheme for salivary gland FNA to improve the reporting diagnosis of salivary gland FNA cases. This workshop will review Milan system and its application on routine daily practices for pathologists.

Biography

Zahra Maleki is an Associate Professor of Pathology at the Johns Hopkins University School of Medicine. Her areas of clinical expertise include Surgical Pathology and Cytopathology. Her priority as a Clinician and Pathologist is to serve the patients by performing both fine needle aspirations and on site evaluations of specimen adequacy, and providing an accurate, timely diagnosis. She teaches and mentors fellows, residents, and students at the Johns Hopkins School of Medicine. She received her Medical degree from Shaheed Beheshti University of Medical Sciences and completed a residency in anatomic and clinical pathology at the Virginia Commonwealth University School of Medicine. She performed her first fellowship at the Virginia Commonwealth University School of Medicine in Pathology and a second fellowship in Cytopathology at the University of Miami.

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Qing Kay Li, J Clin Exp Pathol 2018, Volume 8
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Qing Kay Li

Sidney Kimmel Comprehensive Cancer Center Johns Hopkins, USA
The Johns Hopkins Medical Institutions, USA

Accurate sub-classification of lung cancer using small biopsy samples based on updated WHO and IASLC criteria

The new 2015 edition of the WHO classification and recommendations of IASLC (International Association of Study of Lung Cancer) emphasize the importance of accurate sub-classification of lung cancers for targeted therapy. Lung cancer is a heterogeneous group of neoplasms and accurate diagnosis and sub-classification on small biopsies can be challenging. Recent systematic reviews and meta-analyses have shown that interobserver disagreement rates on the sub-classification of non–small cell lung cancer (NSCLC) are approximately 10-20% in resected specimens and 20-30% in small biopsy specimen without immunohistochemical (IHC) stains. The morphological heterogeneity of the lung cancer is also correlated with certain molecular alterations. Therefore, it is necessary to introduce newly updated guidelines of WHO and IASLC into our daily practice to improve the accuracy of sub-classification of lung cancer for targeted therapy, particularly in small biopsy specimens.

Biography

Qing Kay Li is an Associate Professor of Pathology and Oncology at the Johns Hopkins Medical Institutions. Her areas of clinical expertise include Surgical Pathology, Cytopathology, and Oncological Pathology. She is also a Faculty and PI at Johns Hopkins Biomarker Discovery Center. She serves as Editorial Board Member for several journals; Committee Member of the American Society of Cytopathology, Papanicolaou Society of Cytopathology, NIH/NCI cancer working groups; study sections of government agents and private organizations. She has more than 100 publications, including several book chapters and books.

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Notes:

Roberto Castelli, J Clin Exp Pathol 2018, Volume 8
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Roberto Castelli

University of Milan, Italy

Personalized treatment strategies for elderly patients with myelodysplastic syndromes

Myelodysplastic syndromes (MDS) are a heterogeneous group of hematopoietic disorders characterized by ineffective hematopoiesis and peripheral cytopenia and their possible transformation into acute myeloid leukemia (AML). They typically affect the elderly but when making treatment decisions, considering chronological age may be insufficient because it poorly correlates with patient frailty. The challenge is to select the optimal treatment in these patients by balancing efficacy and toxicity. We will discuss the rationale for and methods of personalizing the treatment of elderly MDS patients. Decisions concerning treatment strategies for elderly MDS patients should be made after assessing their frailty on the basis of a geriatric assessment and an estimation of age-adjusted life expectancy. We suggest that all elderly MDS patients should undergo a timed up and go test (TUGT) as a preliminary means of identifying frail patients and that all non-frail patients should then undergo a comprehensive geriatric assessment (CGA) in order to distinguish fit and pre-frail patients. Fit patients should receive standard dose treatment, pre-frail patients should receive individualized therapy and frail patients should receive symptom-related therapy. A repeated CGA may be useful to evaluate the hematological, cognitive and socio-relational effects of MDS treatment.

Biography

Roberto Castelli has obtained his degree in Medicine from University of Milan and then specialization in Internal Medicine and Hematology from University of Milan. In addition, he obtained PhD in Clinical Methodology from University of Milan. He worked as Hematologist at Ospedale Maggiore di Milano University of Milan and at University Hospital Ospedale Luigi Sacco. He is involved in malignant and non-malignant hematological disease focusing on myelodysplastic syndromes, acute and chronic leukemias and myeloproliferative neoplasms. He is responsible of Leukemia Section at Ospedale Luigi Sacco University of Milan.

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Yener Koc, J Clin Exp Pathol 2018, Volume 8
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Yener Koc

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Toxicity, drug interactions & follow-up in hematopoietic stem cell transplantation

To establish and maintain high cure rates and low mortality in the area of hematopoietic stem cell transplantation, accesibility and availability of well-trained experts capable of interpretation of toxicity and drug interactions at 24/7 hours basis and prompt management side-effects is absolutely necessary, besides trained nurses understanding pathophysiology and clinical signs of transplant complications including graft versus host disease (GVHD), a man-made disease, threatening patients survival and quality of life which may last months or years. Recently, an independent varible called 'center effect' is being stated in international transplant meetings, corresponding to transplant center's expertise and ability that may lead to higher post-transplant survival rates above the world average. JACIE accredited transplant centers have also been shown to be associated with lower mortality and higher survival rates as satted in EBMT publications. Management of transplant complications including drug toxicity and interactions play a key role in patient outcome and development of expertise in this area requires intense training at a successful transplant center and dedication of skillfull attendings to management of transplant patients.

Biography

Yener Koc completed Hematology training at Boston University followed by Bone Marrow Transplant training between 1996-1999 at New England Medical Center, USA. He was given Outstanding Physician Award, twice at Boston University. He performed more than 1700 transplants in 24 years at centers accredited by European and International Transplant Registries (EBMT, CIBMTR) with low transplant mortality and cure rates above world average, including haploidentical allogeneic transplants. He has numerous publications in peer reviewed journals on 84 research items with 1347 citations over the past 33 years. He is working as JACIE accreditation inspector in EBMT since 2004.

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Notes:

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DAY 2

Marie-Pierre Junier, J Clin Exp Pathol 2018, Volume 8
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Marie-Pierre Junier

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A coupling between a neurotransmitter's metabolism and epigenetic regulations promotes intra-tumor heterogeneity in glioma

Cell populations with differing proliferative, stem-like and tumorigenic states co-exist in most tumors and especially malignant gliomas. Whether metabolic variations can drive this heterogeneity by controlling dynamic changes in cell states is unknown. We addressed this question by combining cell biology and neuropathological approaches. Metabolite profiling of human adult glioblastoma stem-like cells upon loss of their tumorigenicity, followed by genetic and pharmaceutical manipulations highlighted a novel signaling module that couples the catabolism of the GABA neurotransmitter and the formation of DNA epigenetic marks. This signaling module was efficient in adult glioblastoma cells with varying molecular profiles, along with cells from pediatric pontine gliomas. Importantly, we verified the relevance of all our findings in the context of the human pathology, using bioinformatics analyzes of patient-derived data at the tissue and single cell level, and immunohistochemical and metabolite analyzes of patients' tumor samples. These results highlight unexpected levels of heterogeneity among the tumor cells, and support an active participation of metabolic variations in the genesis of tumor heterogeneity.

Biography

Marie-Pierre Junier's interest in brain neoplasms developed as a natural extension of fundamental research studies aiming at deciphering the role of neuron-glia interactions during development and neurodegenerative diseases. Following studies of the role of troubles of glial cell differentiation in cancerous transformation, she created with Dr. H Chneiweiss the team "Glial plasticity and neuro-oncology" located in the Neuroscience laboratory of the Institute of Biology Paris Seine Institute (IBPS). The team project questions the dynamics of tumor cell functional heterogeneity, and its impact on malignant glioma growth.

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Notes:

Peter Stoemmer, J Clin Exp Pathol 2018, Volume 8
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Peter Stoemmer

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Tumor budding in colorectal carcinoma- difficulties in graduation and some solutions

Introduction: Infiltration of tumor cells into the peritumoral tissue is one of the most outstanding features of malignancy and, in small tumors/early tumor stages probably of eminent prognostic relevance. In colorectal carcinomas (CRCs), the graduation of the invasive potential is difficult and shows high interobserver variability, leading to several–not very convincing-attempts of standardization as a result of mixing different morphological and biochemical modes of invasion. We analysed different morphological patterns of invasion in CRCs in respect of morphogenous features and biological consequences: The expression of proliferation markers, cell adhesion molecules adherens, and regulators catenins, and invadopia-associated markers like matrix metalloproteinases Vinculin and the perinfiltrative tumorstroma.

Materials & Methods: Twenty hot spots of tumor buds in 20 cases of CRCs (400 POIs) were analysed in FFPE, 5 Ym IHC; analysis of E-Cadherin, N-Cadherin with MoAbs I5626 M0735- DAKO North America, Carpinteria California); Ki-67, CD44 with MoAb (IS2541 M0753 DAKO Denmark Glostrup) Vinculin (PoAb E18720, Spring Bioscience, California).

Results: Tumor budding (TB) in CRCs is part of the much more complex phenomenon of epithelial-mesenchymal transformation (EMT/TEM). Morphologically tumor buds were classified in four different types: (1) monocellular and oligocellular, (2) trabecular, (3) tubular, (4) irregular and sheetlike. They show different IHC results: E-Cadherin expression and membranous ß-catenin are lost in (1) and (2) and diminished in (3); it is well-expressed in (4) N-Cadherin and nuclear ß-catenin are increased in (1), (2) and (3) Vinculin and CD 44, markers of invadopodia, apparently do not play a significant role in tumor budding. Ki-67, a marker of mitotic ability is highly diminished in (1)–(3) and low in (4).

Conclusion: TB is not a simple phenomenon of tumor-cell propagation but the result of a complete change of intracellular organizations with shift of cadherins and catenins; these changes apparently block the mitotic activity of invading cells. Invasion by TB is in strict contrast to proliferation. Development of new therapies should keep in mind, that one can either block proliferation or propagation of malignant tumors.

Biography

Peter Stoemmer has completed his PhD from University Erlangen Nuremberg, Germany and Post-doctoral studies in Yale, News Haven, USA. He is the Director of Pathologie Hermanstrasse. Augsburg Germany. He has published more than 225 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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Notes:

Chandrika Gowda, J Clin Exp Pathol 2018, Volume 8
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Chandrika Gowda

Pennsylvania State University, USA

Targeting casein kinase II (CK2) for treatment of high risk leukemia

Acute lymphoblastic leukemia (ALL) is the most common cancer in children and accounts for highest death rate among children aged 10-19 years. Current treatment for relapsed high risk ALL involves augmented chemotherapy, hematopoietic stem cell transplant and radiation therapy which adds to the morbidity of already sick children. Recent genome-wide studies of leukemic blasts have detected genetic lesions such as deletions or mutations in *IKZF1*. Alterations in *IKZF1* have proven to be an indicator of inferior outcome in patients with high-risk ALL. Ikaros (*IKZF1*) functions as a master regulator of hematopoiesis and a tumor suppressor in ALL. Ikaros binds to the upstream regulatory elements of its target genes and regulates their transcription via chromatin remodeling. Casein kinase II (CK2) is a pro-oncogenic protein which is overexpressed in various cancers including leukemia. Functional experiments showed that CK2-mediated phosphorylation of Ikaros, regulates Ikaros' DNA binding affinity, subcellular localization, and protein stability. Dysregulation of several biological pathways in children with high-risk B-ALL results from CK2 overexpression and impaired Ikaros function. Targeted inhibition of CK2 restores Ikaros tumor suppressor function in high-risk B-ALL even in cases with single allele Ikaros deletion. Treatment with the selective CK2 inhibitor, CX4945 exhibits an anti-leukemic effect in primary xenograft models of high-risk B-ALL. Further studies use precision medicine approaches (targeting specific pathways and/or functional defects) to develop novel drug combinations to target these dysregulated pathways by inhibiting CK2 and restoring Ikaros tumor suppressor function as well as using a specific inhibitor of the signaling pathway.

Biography

Chandrika Gowda is a Board Certified Pediatric Hematologist-Oncologist and Physician Scientist at Penn State Children's Hospital and Penn State college of Medicine in United States. After completing her training in 2013, she continued at PSU as an Assistant Professor. Her research focus for the past seven years has been in pediatric leukemia, specifically to determine the mechanisms of tumor suppression by *IKZF1* gene and development of novel targeted therapies for treatment of high risk pediatric leukemia and neuroblastoma. She has authored several publications and presented her work in various national and international scientific forums. She leads a well-funded research program at PSU.

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