



*The Department of Internal Medicine
Brody School of Medicine
East Carolina University*

presents the
**29th Annual
Yash P. Kataria
Internal Medicine
Research Day
April 1, 2015**



***29th Annual Yash P. Kataria
Internal Medicine Research Day
2015***

***Wednesday, April 1st, 2015
9:45 AM – 3:30 PM
East Carolina Heart Institute***

Paul Bolin, Jr., MD
*Professor and Chair
Department of Internal Medicine*

Research Day Advisory Committee
*Mark Bowling, MD, Chair
Cindy Kukoly
Cathy Munson
Nan Leffler
MeShall Hills
Bobbie Harris*



The Brody School
of Medicine

Department of Internal Medicine

29th Annual Yash P. Kataria Internal Medicine Research Day

Wednesday, April 1st, 2015

9:00am	Refreshments - ECHI Atrium	<i>Poster Presentations available for viewing</i>
9:50am	Welcome - ECHI Auditorium	<i>Paul Bolin, Jr., MD Department of Internal Medicine</i>
9:55am	Administrative Comments - ECHI Auditorium	<i>Mark Bowling, MD, Chair Internal Medicine Research Day Committee</i>
10:00am	First Oral Session, ECHI Auditorium Moderator: Robert J. Tanenberg, MD	
10:00am	OP1 THE EFFICACY AND SAFETY OF THE COMBINATION OF ANTI-BRAF AGENT AND MEK INHIBITOR IN ADVANCED MELANOMA PATIENTS WITH BRAF V600E MUTATION: A META- ANALYSIS	<i>C Xie, P Chae, P Attluri</i>
10:15am	OP2 THE USE OF CEFTAROLINE IN TREATMENT FOR SKIN AND SOFT TISSUE INFECTIONS IN OBESE PATIENTS	<i>H Nguyen, K Shah, M Gooch, M Dhillion, D Siraj, M Ashraf, P Cook</i>
10:30am	OP3 RETROSPECTIVE ANALYSIS OF ECHOCARDIOGRAPHY IN THOSE SUSPECTED OF PULMONARY HYPERTENTION WITH COMPARISON TO RIGHT HEART CATHETERIZATION	<i>TB Eason, L Alapati, S Mehra, JM Cahill, SM George</i>
10:45am	OP4 REDUCTION IN CENTRAL LINE ASSOCIATED BLOOD STREAM INFECTIONS IN THE MEDICAL INTERMEDIATE UNIT	<i>S Brown, M Woodard, D Best, B Smallwod, D Collins, H Freeman, R Barnes, D English, W Stevens N, C Linton, K Jackson, D Harper, R Shaw</i>

11:00 am

Keynote Address: ECHI Auditorium

“Evolving Molecular Therapy for Neuromuscular Disease”

Jerry R. Mendell, MD
*Curran-Peters Chair in Pediatric Research
Professor of Pediatrics and Neurology
Director Gene Therapy Center
Director of Paul D. Wellstone Center
Nationwide Children’s Hospital and The Ohio State University*

12:00pm

ECHI Conference Room
Lunch followed by
Poster Session (12:00 – 1:30pm)

Second Oral Session, ECHI Auditorium
Moderator: Keith Ramsey, MD

- 1:30pm **OP5** **ADHERENCE WITH QUALITY CARE INDICATORS IMPROVED BY INFECTIOUS DISEASES CONSULTATION IN STAPHYLOCOCCUS AUREUS BACTEREMIA AT UNIVERSITY AND COMMUNITY HOSPITALS** M Dhillon, KB Shah, MS Ashraf, HH Nguyen, PP Cook
- 1:45pm **OP6** **MICRORNAS TARGETING PPAR γ PATHWAYS ARE ELEVATED IN BRONCHOALVEOLAR LAVAGE (BAL) CELLS FROM SARCOIDOSIS PATIENTS AND FROM MICE BEARING CARBON NANOTUBE INDUCED GRANULOMAS** M McPeck, A Malur, BP Barna, M Fessler, C Wingard, Y Kataria, MJ Thomassen
- 2:00pm **OP7** **THE EFFICACY OF DPP4 INHIBITORS COMPARED TO SULFONYLUREAS AS ADD-ON THERAPY TO METFORMIN IN TYPE 2 DIABETES: A META-ANALYSIS** BM Mishriky, DM Cummings, RJ Tanenberg
- 2:15pm **OP8** **IS NURSING HOME SPECIFIC ANTIBIOGRAM NECESSARY FOR ALL NURSING HOMES?** K Shah, P Cook, TJ Lee, JD Christie, X Fang, MS Ashraf
- 2:30pm **OP9** **CRITICAL EVENT SIMULATION TEAM: A NEW EDUCATIONAL, PATIENT SAFETY, AND QUALITY IMPROVEMENT INITIATIVE** T Pancoast, M Ritchie, A Hidalgo, J Ford, S Ingram, P Ouellette, W Robey
- 2:45pm **OP10** **EVALUATION OF TRANSITION-AGED PATIENT OUTCOMES ON ADULT HOSPITAL WARD** H Lai, C Christiano, G Hidalgo
- 3:00pm **OP11** **RELEVANCE OF THE EXTRAPITUITARY PROLACTIN PROMOTER POLYMORPHISM AT -1149G/T ON PROLACTIN AND DEHYDROEPIANDROSTERONE IN AMERICAN AFRICAN AND EUROPEAN WOMEN WITH SYSTEMIC LUPUS ERYTHEMATOSUS.** EL Treadwell, C Stevens, K Wiley, B word, W Melchior, W Tolleson, N Gopee, G Hammons, BD Lyn-Cook

3:15pm

Closing Remarks and Award Presentations
Paul Bolin, Jr., MD, Chair Department of Internal Medicine

*Poster Presentations, ECHI Conference Room
Research*

- | | | |
|-------------|---|--|
| PR1 | GENETIC CONTROLS OF GERM CELL FATE SPECIFICATION IN CAENORHABDITIS ELEGANS | <u>SS Mamillapalli</u> , YC Kwon, MH Lee |
| PR2 | THE ROLE OF MICRORNA ON GERM CELL FATE SPECIFICATION IN CAENORHABDITIS ELEGANS | <u>RP Parekh</u> , MH Lee |
| PR3 | ACIDIC TUMOR MICROENVIRONMENT ACTIVATES ENDOPLASMIC RETICULUM STRESS PATHWAYS THROUGH THE GPR4 RECEPTOR IN ENDOTHELIAL CELLS | <u>L Dong</u> , EA Krewson, LV Yang |
| PR4 | THE MULTIFARIOUS EFFECTS OF ACIDOSIS ON LYMPHOMA CELL METABOLISM AND THE TUMOR SUPPRESSIVE FUNCTION OF THE PROTON SENSING G-PROTEIN COUPLED RECEPTOR TDAG8 | <u>EJ Sanderlin</u> , CR Justus, Z Li, LV Yang |
| PR5 | GPR4 ACTIVATION BY ACIDOSIS HINDERS ANGIOGENIC PROCESS OF HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS | <u>EA Krewson</u> , LV Yang |
| PR6 | NOTCH SIGNALING REGULATES EPITHELIAL-MESENCHYMAL TRANSITION IN COLORECTAL CANCER | <u>AW Fender</u> , KE Vinson, DC George, G Sigounas |
| PR7 | DIETARY INTERVENTIONS IN MANAGING IRRITABLE BOWEL SYNDROME: A SYSTEMATIC LITERATURE REVIEW | <u>DA Gliga</u> , H Movahed, E Ali |
| PR8 | STRIKING OUT HYPOGLYCEMIA: A MULTIDISCIPLINARY APPROACH | <u>DA Rao</u> , ES Thoma, S Hardee, N Armistead, R Tanenberg |
| PR9 | LIVING LONGER WITH PANCREATIC CANCER: MORE THAN FOLFIRINOX? | <u>P Chae</u> , P Punja, N Koutlas, P Atluri |
| PR10 | REAL-TIME DUTY HOUR REPORTING FOR NEPHROLOGY FELLOWS THROUGH A SMARTPHONE APP | <u>WA Davila</u> ; HA Coore, TP Desai |
| PR11 | COMPARING TWO MODELS THAT REDUCE THE NUMBER OF NEPHROLOGY FELLOWSHIP POSITIONS IN THE UNITED STATES | <u>WA Davila</u> , T Desai |
| PR12 | EVALUATION OF CARDIOMYOPATHY IN THE HEMODIALYSIS PATIENTS | <u>A Bilal</u> , C Christiano. |
| PR13 | TRANSMITTED DRUG RESISTANCE IN TREATMENT NAÏVE HIV PATIENTS | <u>A Abubaker</u> , X Fang, N Fadul |

Poster Presentations, ECHI Conference Room
Vignette

PV1	PRIMARY CENTRAL NERVOUS SYSTEM LYMPHOMA IN PATIENT WITH HIV DISEASE	<u>A Abubaker</u> , D Rao, H Nguyen, A Stang, K Ramsey
PV2	A DANGEROUS CAUSE OF NECROTIZING FASCIITIS	<u>A Abubaker</u> , M Dhillon, H Nguyen, MS Ashraf
PV3	<i>VIBRIO VULNIFICUS</i> AS CAUSE OF NECROTIZING FASCIITIS AND BACTEREMIA	<u>A Abubaker</u> , M Dhillon, H Nguyen, K Roach, MS Ashraf
PV4	A RARE CAUSE OF RECURRENT PERICARDIAL EFFUSION	<u>A Abubaker</u> , K Roach, H Nguyen, MS Ashraf
PV5	RARE BUT SEVERE INFECTION IN CARDIAC TRANSPLANT PATIENT	<u>H Nguyen</u> , A Do, A Stang, A Abubaker, M Dhillon, J Christie, P Cook
PV6	IATROGENIC SUPERIOR VENACAVA SYNDROME	<u>S Chalise</u> , N Sultana, P Rice, M Bowling
PV7	CAVITARY PNEUMOCYSTIS JIROVECII PNEUMONIA (PJP), UNCOMMON PRESENTATION OF A COMMON DISEASE	<u>M Rizwan</u> , N Sultana, M Al-Janabi, T Pancoast
PV8	AEROMONAS HYDROPHILA NECROTIZING FASCIITIS IN A CIRRHOTIC PATIENT	D Willis, C Zhong, <u>K Roach</u> , A Rao
PV9	20 YEAR OLD MALE WITH DESCENDING NECROTIZING MEDISTINITIS	<u>A El-Bakush</u> , M Al-Janabi, R Dietrich, M Bowling
PV10	“TOTO, I’VE A FEELING WE’RE NOT IN KANSAS ANYMORE”: MYCOBACTERIUM KANSASII LUNG MASS MASQUERADING AS MALIGNANCY	<u>A Stang</u> , H Nguyen, M Ashraf, P Cook
PV11	ECTOPIC ADRENOCORTICOTROPIN PRODUCTION IN A PATIENT WITH ADENOCARCINOMA OF LUNG- A CASE REPORT	<u>P Prodduturvar</u> , N Bongu, S Nauman, S Mehra
PV12	GUILLIAN BARRE SYNDROME (GBS) CAUSED BY CYTOMEGALOVIRUS (CMV) INFECTION IN RENAL TRANSPLANT RECIPIENT	<u>A.Gurram</u> , P.Jawa
PV13	UP AGAINST EXTREMES: THE CHALLENGE OF TREATING THROMBOEMBOLISM IN OBESE PATIENTS AFTER GASTRIC BYPASS	SA Marco, <u>G Sangah</u> , K Parikh
PV14	AN UNUSUAL CAUSE FOR GASTROINTESTINAL BLEEDING	<u>ZJ Iqbal</u> , S Abid, E Ali
PV15	A CASE OF SEROLOGY NEGATIVE NEUROCYSTICERCOSIS IN THE UNITED STATES	<u>J Emberger</u> , H Nguyen, A Stang, MS Ashraf
PV16	PARALYZED: THE TICK BENEATH	<u>M. Christiano</u> , R Dietrich
PV17	FIRM FACIAL PAPULES	<u>EL Stewart</u> , C Phillips, K Park

PV18	EARLY ONSET OSTEOARTHRITIS IN A NOVEL KINDRED WITH AUTOSOMAL DOMINANT HYPOCALCEMIA	<u>M Javaid</u> , JA Sutter, <u>BE Ramirez</u> , FJ Cook
PV19	OMEPRAZOLE INDUCED SYMPTOMATIC HYPOCALCEMIA IN A PATIENT WITH SILENT HYPOPARATHYROIDISM	<u>M Javaid</u> , C Houston, J Giordano, RJ Tanenberg
PV20	ACTH-SECRETING PHEOCHROMOCYTOMA	<u>MS Kalia-Reynolds</u> , J Giordano, I Khanna, C Houston
PV21	SEVERE MULTIORGAN FAILURE WITHIN HOURS AFTER HIGH DOSE CARFILZOMIB THERAPY FOR REFRACTORY MULTIPLE MYELOMA	<u>A El-Bakush</u> , M Al-Janabi, A Ismail, R Dietrich
PV22	STROKE IN PATIENT WITH JANUS KINASE 2 POSITIVE ESSENTIAL THROMBOCYTHEMIA	<u>CP Craig</u> , S Jayananda, D Liles
PV23	KAPOSI'S SARCOMA IN AN HIV NEGATIVE CENTRAL AMERICAN PATIENT: A CASE REPORT	<u>P Chae</u> , L Salmon, P Atluri
PV24	INTRACRANIAL PLASMABLASTIC LYMPHOMA REVEALING ACQUIRED IMMUNE DEFICIENCY SYNDROME	<u>SA Marco</u> , LE Salmon, M Muzaffar
PV25	SPONTANEOUS REMISSION OF ACUTE MYELOID LEUKEMIA: COULD IT BE THE ESOMEPRAZOLE?	<u>ER Bonner</u> , RN Friend
PV26	ACUTE MYELOID LEUKEMIA WITH CRYPTIC <i>DEK-NUP214</i> FUSION PRESENTING AS PYODERMA GANGRENOSUM.	<u>E Levin</u> , H Lin, P Chae, P Papenhausen, B Dangott, R Juskevicius
PV27	CAN IT STILL BE STILL'S? THE DIFFICULT ROAD TO A DIAGNOSIS.	<u>E Levin</u> , V Dhingra, S DeMarco
PV28	BASAL CELL CARCINOMA MASQUERADING AS A MELANOMA MASQUERADING AS AN ACTINIC KERATOSIS	<u>KR Liner</u> , R Harris, CM Phillips
PV29	THE WRATH OF A SICKLE CELL PAIN CRISIS: IDENTIFYING MULTIORGAN DYSFUNCTION EARLY IN A SICKLE CELL PATIENT	<u>K Parikh</u> , T Nguyen, A Ismail, Z Rehman
PV30	FREE OPEN ACCESS MEDICAL SIMULATION	<u>M Ritchie</u> , A Hidalgo, T Pancoast



In 2008, the annual departmental research day program was dedicated and renamed the **Yash P. Kataria Internal Medicine Research Day** to honor the many contributions of Dr. Yash P. Kataria and to support the educational and research program in the Department of Internal Medicine at the Brody School of Medicine at ECU.

Dr. Kataria is Professor Emeritus of Medicine at BSOM and continues to contribute actively to the clinical, educational and research mission of the pulmonary and critical care division at BSOM. He was the first pulmonologist in eastern NC and helped to establish the pulmonary specialty at BSOM 30 years ago and has been an integral force since the inception of the medical school. Yash was the first division chief of pulmonary medicine at BSOM and successfully recruited and established a clinical and active laboratory research program. Yash was the section head of pulmonary at BSOM /PCMH from 1978-1995, Vice Chair of the Dept. of Medicine 1987-1992 and Interim Chair 1986-87. Yash is of course known regionally, nationally and internationally for his passion in translational research with a particular focus on sarcoidosis. He has authored over 70 publications, has received the Trudeau Award from the American Lung Association, Lifetime Achievement Award by the NC Thoracic Society, on many occasions been listed on the “Best Doctors” list, has been a reviewer and/or on editorial board for numerous specialty journals.

Over his 30 year career, he has cared for thousands of patients with sarcoidosis and he arguably has one of the largest sarcoid cohorts in the US. Yash is revered by his patients and families. Yash has literally trained hundreds of medical students and house staff and is cherished by them as a role model and outstanding teacher at the bedside and in clinics. Yash has been a fixture in the international sarcoid community and has contributed actively at a leadership level at ACCP, ATS and WASOG. Scientifically, Yash is perhaps best known for promulgating a paradigm shift in our understanding of sarcoid immunology. While it was accepted dogma in the 70s that sarcoidosis was a disease of “depressed immunity” and anergy, Yash proposed and championed the concept that it is a pro-inflammatory disease with involvement of activated T-cells, cytokines, etc. Yash and his group also proposed that the active “sarcoid factor” was localized to the cell walls of alveolar macrophages and monocytes or an “autologous kveim” model (this remains an intriguing hypothesis!).

One of the missions of the medical school is community service in which medical school faculty plunged deeply. Yash lived in and loved Greenville where he raised two lovely children.

He was actively involved in the J. H Rose Attendance Area Foundation Advisory Committee; also served as a Member Board of Academic Boosters Club, Rose High School, Greenville, NC and President, Parent Teacher Association, Greenville Middle School, Greenville, NC. He also helped to develop support groups for patients with sarcoidosis & COPD and played leadership roles in the local American Lung Association of NC. We are honoring Dr. Kataria by dedicating our annual Internal Medicine Research Day, which he started in 1987, to the **Yash P. Kataria Internal Medicine Research Day**. We will continue to build on the tradition of encouraging research by inviting leading guest speakers and facilitating scholarship and interaction by our trainees and faculty.



Keynote Address :

“Evolving Molecular Therapy for Neuromuscular Disease”

Jerry R. Mendell, MD

***Curran-Peters Chair in Pediatric Research
Professor of Pediatrics and Neurology***

Nationwide Children’s Hospital and The Ohio State University

After completing medical school at UT Southwestern and Neurology Residency at the New York Neurological Institute, Columbia University, Jerry R. Mendell’s research in muscular dystrophy was initiated during his post-doctoral fellowship in Medical Neurology, NINDS, NIH, 1969. An early important observation related to Duchenne muscular dystrophy (DMD) was that the small groups of muscle fibers undergoing necrosis and regeneration could be experimentally reproduced by a functional ischemia model using aortic ligation and serotonin (Mendell JR, et al. Duchenne muscular dystrophy: functional ischemia reproduces its characteristic lesions. *Science* 172:1143-45, 1971). Additional efforts to support this hypothesis were encouraging (Mendell, et al. *Nature* 239: 533-44, 1972; Mendell, et al. *Nature* 247:103-4, 1974) but clinical trials to promote blood flow were disappointing. However, with further confirmation of the importance of this pathway, it is of interest that the field has moved on to current clinical trials using PDE5 inhibitors including sildenafil and tadalafil.

Mendell moved to The Ohio State University in 1972 and established a neuromuscular division in the Department of Neurology. His collaborative studies extended to a network of clinician scientists, the CIDD group (Clinical Investigation of Duchenne Dystrophy), addressing basic questions in DMD. Over a 15-year period, the group introduced and standardized methods for DMD clinical trials, defined the natural history of DMD, determined sample size based on power calculations, and reinforced the importance of intra- and inter-rater reliability scores. In 1989, the CIDD group unequivocally defined corticosteroids as the first treatment to be effective for DMD (Mendell JR, et al. Randomized, Double-Blind Six-Month Trial of Prednisone in Duchenne’s muscular dystrophy. *N Engl J Med* 320:1592-97, 1989). This has been confirmed multiple times and remains the standard of care for the diseases.

The research direction for the Mendell Lab has focused for many years on attempts to find ways of understanding and correcting the basic molecular defect in DMD and other dystrophies. Seminal studies include myoblast transfer (Mendell, et al. *N Engl J Med* 1995; 333:832-38), the first DMD gene therapy clinical trial (Mendell, et al *N Engl J Med* 2010; 363:1429-37), and the first successful gene therapy trial for muscular dystrophy showing prolonged alpha-sarcoglycan gene expression in limb girdle muscular dystrophy, type 2D (Mendell JR, et al *Ann Neurol* 2010; 68:629-97). In addition the first, randomized controlled trial of exon skipping was done by the Mendell Research group and is the first therapeutic agent to show increased dystrophin expression in DMD. This work was just published on August 1, 2012 (Mendell et al. Eteplirsen for the Treatment of Duchenne Muscular Dystrophy. *Ann Neurol* Aug 1 Epub). An additional important contribution was the development of the two-tier system for detection of DMD in the newborn. This was a seminal study of nearly 40,000 newborn males that now sets the stage for the implementation of newborn screening for DMD (Mendell et al. Evidence Based Path to Newborn Screening for Duchenne Muscular Dystrophy. *Ann Neurol* 2012; 71:304-13).

Much of the recent efforts of the Mendell Lab have been directed toward gene delivery through the circulation in both mouse and non-human primate. Clinical trials will begin later this year for delivery of the alpha-SG and microdystrophin genes to the lower limbs in AAV. These will be groundbreaking studies for the field with gene delivery through the vasculature that could prolong ambulation. Currently an on-going and very promising gene therapy trial utilizes follistatin to increase the size and strength of muscle mass in Becker muscular dystrophy and sporadic inclusion body myositis patients.

All in all, the learning curve has been steep, but research is beginning to pay great dividends for muscular dystrophy patients. Studies from the Mendell Lab and Research Group are contributing significantly to this field including 315 Journal publications and 3 Neuromuscular Books.

The Keynote Address - Celebrating 29 Years

- | | |
|--|---|
| <p>1987 Morris Reichlin, MD
 <i>Professor of Medicine
 University of Oklahoma, School of Medicine</i></p> | <p>2002 William Roper, MD, MPH
 <i>Dean, School of Public Health
 University of North Carolina at Chapel Hill.</i></p> |
| <p>1988 Jesse Roth, MD
 <i>Director, Intramural Research
 National Institute of Diabetes and Digestive and
 Kidney Diseases, NIH</i></p> | <p>2003 Jeffrey P. Engel, MD
 <i>Division Head, General Communicable Disease Control
 State Epidemiologist, Division of Public Health
 NC Department of Health and Human Services</i></p> |
| <p>1989 Roy Patterson, MD
 <i>Professor and Chair
 Department of Medicine
 Northwestern University Medical School</i></p> | <p>2004 Helen Burstin, MD, MPH
 <i>Director of the Center for Primary Care, Prevention and
 Clinical Partnerships, Agency for Healthcare Research
 and Quality</i></p> |
| <p>1990 Edward W. Hook, MD
 <i>Professor and Chair
 Department of Medicine
 University of Virginia, Health Sciences Center</i></p> | <p>2005 Marschall S. Runge, MD, PhD
 <i>Chair, Department of Medicine
 University of North Carolina at Chapel Hill
 President, UNC Physicians</i></p> |
| <p>1991 Albert F. LoBuglio, MD
 <i>Director, Comprehensive Cancer Center
 Director, Division of Hematology/Oncology
 University of Alabama at Birmingham</i></p> | <p>2006 Jose Caro, MD
 <i>Vice President, Endocrine Research and Clinical
 Investigation
 Lilly Corporate Center, Indianapolis</i></p> |
| <p>1992 Raj K. Goyal, MD
 <i>Harvard Medical School
 Chief Gastroenterology Division
 Beth Israel Hospital</i></p> | <p>2007 William Stratford May, MD, PhD
 <i>Chair, Hematology and Oncology
 Director, Shands Cancer Center
 University of Florida</i></p> |
| <p>1993 Richard E. Kerber, MD
 <i>Professor of Medicine
 Associate Director Cardiovascular Division
 The University of Iowa College of Medicine</i></p> | <p>2008 Phillip A. Bromberg, MD
 <i>Bonner Professor of Medicine
 Scientific Director of the Center for Environmental
 Medicine, Asthma and Lung Biology
 University of North Carolina at Chapel Hill</i></p> |
| <p>1994 James S. Louie, MD
 <i>Chief, Division of Rheumatology
 Department of Medicine
 Harbor-UCLA Medical Center</i></p> | <p>2009 Randy L. Jirtle, PhD
 <i>Professor of Radiation Oncology and Pathology
 Duke University Medical Center</i></p> |
| <p>1995 Matthew I. Gilmour, B.Sc., PhD
 <i>Center for Environmental Medicine and Lung Biology
 University of North Carolina at Chapel Hill</i></p> | <p>2010 Robert M. Lust, PhD
 <i>Interim Associate Dean, Research and Graduate Studies
 Chair, Department of Physiology
 East Carolina University, Brody School of Medicine</i></p> |
| <p>1998 O. Michael Colvin, MD
 <i>William Singleton Professor of Cancer Research
 Director, Duke Comprehensive Cancer Center</i></p> | <p>2011 David C. Goff Jr., MD, PhD
 <i>Chair, Department of Epidemiology and Prevention
 Division of Public Health Services
 Wake Forest University School of Medicine</i></p> |
| <p>1999 Jerry Palmer, MD
 <i>Professor of Medicine
 Director, Diabetes Research Center
 University of Washington</i></p> | <p>2012 Vinay Kumar, MBBS, MD, FRCPath
 <i>Donald N Pritzker Professor and Chair
 Department of Pathology
 University of Chicago</i></p> |
| <p>2000 Thomas Feldbush, PhD
 <i>Vice Chancellor for Research and Graduate Studies
 Dean, Graduate School
 East Carolina University</i></p> | <p>2013 Paul W. Nobel, MD
 <i>Chair, Department of Medicine
 Director, Women's Guild Lung Institute
 Cedars-Sinai, Los Angeles, California</i></p> |
| <p>2001 William B. Applegate, MD, MPH
 <i>Professor and Chair
 Department of Internal Medicine
 Wake Forest University School of Medicine</i></p> | <p>2014 Vishva Dixit, MD
 <i>Vice President
 Early Discovery Research
 Genentech, Inc.</i></p> |

W. James Metzger, Jr., MD Award

The W. James Metzger, Jr., M.D. award is presented to the most outstanding presentation by a junior faculty member in the Department of Internal Medicine. A peer-review process selects the winner. The recipient of the award receives a certificate and has his/her name engraved on a plaque that is displayed in the Department of Internal Medicine Library. The recipient also receives recognition on the Department of Internal Medicine web site.

Dr. Metzger, a native of Pittsburgh, Pennsylvania, was a graduate of Stanford University and Northwestern University Medical School, Chicago, Illinois. He completed his residency and research fellowship in Allergy-Clinical Immunology at Northwestern University. After serving in the United States Air Force, he came to Greenville in 1984 to join the East Carolina University School of Medicine. During his tenure at East Carolina University, Dr. Metzger rose to the rank of Professor of Medicine. He was Section Head of the Section of Allergy-Immunology and held the appointments of Vice Chairman of Research, Department of Internal Medicine; Executive Director, the Center for Asthma, Allergy, and Immunology; Assistant Vice Chancellor for Clinical Research; Assistant Dean for Clinical Research; and Director, Clinical Trials Office. He was the recipient of the East Carolina University Award for Excellence in Research and Creative Activity and the Distinguished Research Professor of Medicine. His research was published in the New England Journal of Medicine, Nature, and other journals. Dr. Metzger had mentored numerous faculty and fellows.

In August 2000 Dr. Metzger accepted a position as Professor of Allergy, Asthma and Immunology at the National Jewish Medical and Research Center and was a faculty member at the University of Colorado Medical School, Denver, Colorado. He died on November 11, 2000 at the age of 55. Dr. Metzger represented excellence in research.

2001 Recipients:

**Carlos A. Estrada, MD, MS
Paul Mehlhop, MD**

2007 Recipient:

Christopher Newton, MD

2012 Recipient:

Maria Ruiz-Echevarria, PhD

2003 Recipient:

Lisa Staton, MD

2008 Recipient:

Li Yang, PhD

2013 Recipient:

Moahad Dar, MD

2004 Recipient:

Cassandra Salgado, MD

2009 Recipient:

Li Yang, PhD

2014 Recipient:

Mark Bowling, MD

2005 Recipient:

Barbara J. Muller-Borer, PhD

2010 Recipient:

Sunil Sharma, MD

2006 Recipient:

Timothy P. Gavin, PhD

2011 Recipient:

Sunil Sharma, MD

ABSTRACTS

In Presentation Order

OP = Oral Presentation

PR = Poster Research

PV = Poster Vignette

OP1

THE EFFICACY AND SAFETY OF THE COMBINATION OF ANTI-BRAF AGENT AND MEK INHIBITOR IN ADVANCED MELANOMA PATIENTS WITH BRAF V600E MUTATION: A META- ANALYSIS

C Xie, P Chae, P Atluri

Background: The combination of BRAF inhibitor and MEK inhibitor has been shown to improve outcomes for advanced melanoma patients with BRAF V600E mutation. A meta-analysis was performed to evaluate the efficacy and safety of the combination of BRAF and MEK inhibition in these patients.

Patients and methods: Eligible trials were phase II or phase III randomized controlled trials (RCTs) comparing the combination therapy with anti-BRAF agent alone in advanced melanoma with BRAF V600E mutation reporting progression-free survival (PFS), overall survival (OS), objective response rates (ORR) and adverse effects (AEs).

Results: Four trials with five comparisons comprising 1784 patients were eligible for inclusion. BRAF inhibitors were vemurafenib and dabrafenib; MEK inhibitors were trametinib and cobimetinib. The combination therapy improved PFS [hazard ratios (HR) 0.56; 95% confidence interval (CI) 0.47–0.68], OS (HR 0.69; 95% CI 0.57–0.82), and ORR [relative risk (RR) 1.31; 95% CI 1.17–1.47]. There was no significant difference regarding overall grade 3 or higher AEs between them (RR 1.00; 95% CI 0.86–1.17). However, the risk of developing skin malignancy was significantly decreased about 80% in the combination arm (RR 0.19; 95% CI 0.10–0.36). The overall 3-grade and higher AEs in dabrafenib/trametinib group was less than vemurafenib/cobimetinib group, with 291/668 (43.6%) vs 159/254 (62.6%). Arthralgia, rash, diarrhea was relatively more frequently presented in vemurafenib/cobimetinib group.

Conclusions: Our meta-analysis suggests the benefit of combination therapy of anti-BRAF agent and MEK inhibitor for advanced melanoma patients with BRAF V600E mutation as first-line treatment, as well as decreased risk of developing skin malignancy. Trials in our meta-analysis differed in terms of BRAF inhibitor and MEK inhibitors, yet delivered comparable outcomes. However, the clinical trial with the head-to-head comparison between these different combinations for advanced melanoma needs to be addressed. Based on the AE data, dabrafenib/trametinib may be considered as first line over vemurafenib/cobimetinib despite comparable outcomes.

Notes:

OP2

THE USE OF CEFTAROLINE IN TREATMENT FOR SKIN AND SOFT TISSUE INFECTIONS IN OBESE PATIENTS

H Nguyen, K Shah, M Gooch, M Dhillon, D Siraj, M Ashraf, P Cook

Background: Ceftaroline is a broad spectrum fifth generation cephalosporin which exhibits bactericidal activity against gram-positive organisms (including MRSA), macrolide-resistant *S. pyogenes*, as well as non-extended-spectrum b-lactamase (ESBL)-producing gram negatives. It is in development for treatment of hospitalized patients with skin and skin structure infections (SSSIs). Previous published study showed that clinical cure rates were similar for ceftaroline and vancomycin plus aztreonam in patients with SSSI. However, majority of the patient population listed in the study had BMI < 30. There is no study about the efficacy of Ceftaroline on patients with BMI>30. We therefore conduct a study to compare the efficacy and safety of ceftaroline in the populations with normal BMI versus BMI > 30. We also tried to determine the non-inferiority of the clinical cure rate achieved with ceftaroline in obese patients when compared with vancomycin plus/minus gram negative coverage combination therapy.

Methods: Retrospective study that included patients who were admitted to the hospital from May to Sept of 2013 with the diagnosis of SSSI and got treated with ceftaroline. Patients' BMIs were calculated, and duration of treatment and adverse effects were noted. We also looked into the data for vancomycin use in patients with same infection from May to Sept 2012 and 2011 to compare efficacy and safety between vancomycin and ceftaroline.

Results: Of total of 244 patients, 134 received ceftaroline and 110 received vancomycin combination therapy. Among 134 patients with ceftaroline, 84 patients (62.7%) had BMI > 30. Clinical improvement or cure rates were similar between patients with BMI <30 and BMI>30 (96% versus 95.2%). The rates of adverse events or serious adverse events were minimal in both groups. Among 110 patients treated with vancomycin, there were 85 patients (77.3%) with BMI>30. When compared with vancomycin in patients with BMI>30, there was a higher rate of acute kidney injury in patients with BMI>30 who got treated with vancomycin (7.06% p=0.028).

Conclusion: Ceftaroline achieved high clinical rates in both normal BMI patient population as well as among obesity (BMI>30). The medication was well tolerated and had minimal side effect profile in both normal and obese patients. In addition, ceftaroline was as efficacious against SSSI as vancomycin therapy but has a lower profile of acute kidney injury.

Notes:

OP3

RETROSPECTIVE ANALYSIS OF ECHOCARDIOGRAPHY IN THOSE SUSPECTED OF PULMONARY HYPERTENSION WITH COMPARISON TO RIGHT HEART CATHETERIZATION

TB Eason, L Alapati, S Mehra, JM Cahill, SM George

Background: Pulmonary hypertension (PH) is a condition which can be life-threatening and proper diagnosis is essential for determining therapy for a patient. Current PH screening utilizes echocardiography to estimate pulmonary artery pressure (PAP) using tricuspid regurgitation (TR), but this method does not distinguish between Group I (arterial) and Group II (venous) PH. Patients must undergo a right heart catheterization to further classify their disease and receive the best possible treatment. **Methods:** This project was a retrospective study which identified patients who had undergone both a transthoracic Doppler echocardiogram and a RHC within 60 days of each other and did not have mitral or aortic valve pathology. Velocity data, velocity spectral density, and electrocardiogram parameters were extracted from the echo image. The flow density was calculated to determine at which velocities the majority of the fluid is traveling. Lastly, a Fast Fourier Transform (FFT) was conducted on the velocity signal to examine the frequency domain.

Results: The patient population consisted of 14 patients: 5 patients with Group I PH, 6 with Group II PH, 2 with borderline PH (20mmHg < mean PAP < 25mmHg), and 1 with no PH. Patients with Group I PH exhibited a high flow density at lower velocities with the density decreasing as the velocity increased compared to Group II PH which exhibited higher flow densities across all velocities. Strong correlation coefficients were found between the maximum TR and the mean and systolic PAP (R=0.7747, 0.7416) with better correlations for Group I (R=0.7808, 0.8086) than Group II (R=0.5957, 0.6416). There is a weaker correlation using the FFT data (R=0.5284). A t-test was conducted to find a significant difference between average flow density and FFT in Group I and Group II PH patients (p=0.0277,0.0459), with Group II PH have a significantly higher average. **Conclusions:** This study confirmed previous findings that TR velocity is a strong predictor of both mean and systolic PAP; this relationship is stronger for Group I than Group II PH. We conclude that velocity density is an effective way to distinguish between Group I and Group II PH. The FFT signal did not produce any better predictive values, but should not be neglected in further studies. This study uncovered new parameters that may be diagnostically useful in non-invasive evaluation of patients with PH and should be further investigated.

Notes:

OP4

REDUCTION IN CENTRAL LINE ASSOCIATED BLOOD STREAM INFECTIONS IN THE MEDICAL INTERMEDIATE UNIT

S Brown, M Woodard, D Best, B Smallwod, D Collins, H Freeman, R Barnes, D English, W Stevens N, C Linton, K Jackson, D Harper, R Shaw.

BACKGROUND: Central line associated blood stream infections (CLABSI) are serious safety events in hospitals and result in increased mortality, length of stay, and costs. It was noted in fiscal year 2012 that the number of CLABSI in the medical intermediate unit (MIU) at Vidant Medical Center was higher than the goal set by the hospital quality committee. The objective of this project was to implement initiatives that would result in a reduction in CLABSI in fiscal year 2013.

METHODS: This was a prospective study interventional study. At the start of fiscal year 2013 the following interventions were implemented in the MIU: 1. A unit task force completed weekly audits and started unit based education for staff. 2. Daily rounding by quality nurses to assess need for central venous lines (CVL) and assist with removal of unnecessary lines. 3. CVL bundle checks during bedside shift report and hourly rounding to insure CVL occlusive dressings. 4. CVL dressing kits were made available at the bedside. 5. CVL dressing change team was created and checked all CVL dressings daily. If the dressing was not occlusive, the team changed it. A CVL rounding book was used to monitor dressing compliance.

RESULTS: # CLABS/year Bundle Compliance CVL utilization*

2012	10	87%	0.4
2013	7	92%	0.32

* number of central lines/patient.

CONCLUSIONS: Initiating a unit based task force to address CLABSI, daily rounding to assess need for CVLs, and monitoring compliance with CVL bundles resulted in a significant reduction in the rate of CVL usage and number of CLABSI in the MIU. The reduction in CLABSI is probably related to both the reduction in CVLs and bundle compliance with occlusive dressings. We do not have data to document reduction in mortality or costs, but believe that if implemented throughout the Health System, this program would show positive outcomes.

Notes:

OP5

ADHERENCE WITH QUALITY CARE INDICATORS IMPROVED BY INFECTIOUS DISEASES CONSULTATION IN STAPHYLOCOCCUS AUREUS BACTEREMIA AT UNIVERSITY AND COMMUNITY HOSPITALS

M Dhillon, KB Shah, MS Ashraf, HH Nguyen, PP Cook

Background: Adherence to evidence-based quality of care indicators in treatment of *Staphylococcus aureus* bacteremia (SAB) has been shown to improve clinical outcomes. We studied the effect of Infectious Diseases (ID) consultation on adherence to established quality of care indicators for management of SAB.

Methods: Using a retrospective study design, we conducted chart reviews on all patients who were managed for SAB at Vidant Medical Center and affiliated community hospitals during a one-year period (November, 2012 to November, 2013). Subjects were divided into two groups: those who received ID consultations and those who did not. Information on demographics, quality-of-care indicators, and clinical outcomes were obtained. Fisher's exact test and chi square analysis were used to assess differences in the two groups with $p < 0.05$ denoting statistically significance.

Results: Of 182 patients with SAB, 84 patients (mean age 51, 59.5% male, 47.6% Caucasian) had ID consultation and 98 (mean age 56, 60.4% male, 38.7% Caucasian) did not receive ID consultation. Methicillin-sensitive *Staphylococcus aureus* (MSSA) were identified in 55.9% of patients in ID consultation and 61.2% in non-ID consultation group ($p = 0.56$). As compared to the non-ID consultation group, patients in the ID consultation group were more likely to have repeat blood cultures within 96 hours (86.9% vs. 69.3%, $p = 0.008$), have an echocardiogram (92.8% vs. 62.2%, $p < 0.0001$), and receive appropriate antibiotics in terms of duration and choice (100%, vs. 76.5%, $p < 0.0001$). In addition, patients in the ID consultation group were more likely to have early de-escalation (within 24 hours) to Nafcillin or Cefazolin (from Vancomycin or Daptomycin) in cases of MSSA bacteremia (89.3% vs. 71.6%, $p = 0.03$). Recurrent bacteremia within 90 days and all-cause mortality during the initial admission were similar in the two groups. There was a trend towards decreased mortality in ID consultation group (9.5% vs. 20.4%, $p = 0.06$).

Conclusions: ID consultation increases adherence with evidence-based quality of care indicators, leads to more appropriate antimicrobial therapy, and can improve patient outcomes during management of SAB. Clinicians should consider getting ID consultation for all patients with SAB.

Notes:

OP6

MICRORNAS TARGETING PPAR γ PATHWAYS ARE ELEVATED IN BRONCHOALVEOLAR LAVAGE (BAL) CELLS FROM SARCOIDOSIS PATIENTS AND FROM MICE BEARING CARBON NANOTUBE INDUCED GRANULOMAS

M McPeck, A Malur, BP Barna, M Fessler, C Wingard, Y Kataria, MJ Thomassen

Granuloma formation in the lung represents a complex and poorly defined response involving environmental and host factors that can culminate in persistent and chronic inflammatory disease. We established a murine model of multiwall carbon nanotube (MWCNT)-induced chronic pulmonary granulomatous disease which bears a striking resemblance to granulomas in sarcoidosis. At 60 days post instillation of MWCNT, bronchoalveolar lavage (BAL) cells exhibit increased pro-inflammatory cytokines and reduced peroxisome proliferator-activated receptor gamma (PPAR γ) - characteristics also present in sarcoidosis BAL cells. PPAR γ is a critical factor in lipid homeostasis but can also function as a negative regulator of inflammation. We hypothesized that down-regulation of PPAR γ pathways might involve microRNAs (miRs, small non-coding RNAs that post-transcriptionally repress mRNA). Investigation of BAL cells from 60-day MWCNT-treated mice indicated significant ($p < 0.05$) upregulation of both miR-33 (15-fold, $n = 7$) and miR-27b (3.4-fold, $n = 5$) compared to sham controls ($n \geq 4$). Elevated miR-33 (5-fold, $n = 4$) and miR-27b (2.5-fold, $n = 5$) were also detected in sarcoidosis BAL cells compared to healthy controls ($n \geq 3$). Because miR-27b targets PPAR γ and miR-33 targets the PPAR γ -regulated lipid transporters ABCA1 and ABCG1, (which also promote anti-inflammatory pathways) we investigated the status of ABCA1 and ABCG1 in BAL cells. In 60-day MWCNT-bearing mice ($n = 5$), both ABCA1 (-1.7-fold) and ABCG1 (-2.0-fold) were significantly ($p < 0.05$) decreased compared to sham controls ($n = 3$). Lipid transporters were also significantly ($p < 0.05$) reduced in sarcoidosis BAL cells: (ABCA1 -4.3-fold, and ABCG1 -3.4-fold, $n \geq 5$) compared to healthy controls ($n = 6$). These results from studies in progress suggest that microRNAs 27b and 33 may promote chronic inflammation in pulmonary granulomatous disease by targeting anti-inflammatory PPAR γ pathways.

Notes:

OP7

THE EFFICACY OF DPP4 INHIBITORS COMPARED TO SULFONYLUREAS AS ADD-ON THERAPY TO METFORMIN IN TYPE 2 DIABETES: A META-ANALYSIS

BM Mishriky, DM Cummings, RJ Tanenberg

Purpose: Recent guidelines recommend metformin as the best initial drug for Type 2 diabetes (T2D). However, there is no consensus for add-on therapy if metformin fails to achieve the therapeutic goal. Sulfonylurea (SU) is an older while dipeptidyl peptidase-4 inhibitors (DPP4-I) a newer antidiabetic medication. We performed this meta-analysis to determine the efficacy of DPP4-I compared to SU as add-on therapy to metformin in inadequately controlled T2D. **Methods:** We searched MEDLINE, CENTRAL, EMBASE, and CINAHL for randomized trials comparing DPP4-I to SU as add-on therapy to metformin in inadequately controlled T2D and reported a change in A1c from baseline to a minimum of 12 weeks. Number needed to harm (NNH) was calculated for statistically significant side effects. **Results:**

Sixteen studies were included. Pooled results showed a significantly greater reduction in A1c from baseline to 12 weeks favoring SU (MD[95%CI] = 0.21% [0.06, 0.35]) but no significant difference at 52 and 104 weeks between the two groups (MD[95%CI] = -0.01% [-0.07, 0.05] and -0.06% [-0.13, 0.02] respectively). There was a significantly greater weight reduction at 12, 52, and 104 weeks favoring DPP4-I (MD[95%CI] = -1.57Kg [-1.85, -1.28], -2.11Kg [-2.49, -1.72], and -2.13Kg [-2.58, -1.68] respectively). The proportion of patients achieving A1c < 7%, irrespective of hypoglycemia, showed no difference between the groups at 12, 52, and 104 weeks (RR[95%CI] = 0.93[0.78, 1.12], 1.03[0.95, 1.12] and 1.01[0.93, 1.09] respectively). However, there was a statistically significantly greater proportion of patients achieving A1c < 7% with no hypoglycemia episodes in the DPP4-I group at 52 and 104 weeks (RR[95%CI] = 1.20 [1.05, 1.37] and 1.53 [1.16, 2.02] respectively). Incidence of hypoglycemia (≥1 episode) was significantly higher at 12, 52, and 104 weeks in SU group (RR[95% CI] = 0.31 [0.22, 0.45]; NNH=8, 0.11 [0.08, 0.16]; NNH=5, and 0.14 [0.11, 0.20]; NNH=5 respectively). The percentage of patients with hypoglycemia was higher with the SU compared to DPP4-I (20% vs 6% at 12 weeks, 24% vs 3% at 52 weeks, and 27% vs 4% at 104 weeks respectively). **Conclusion:** While both SU and DPP4-I can be considered as options for add-on therapy to metformin in inadequately controlled T2D, SU results in a significantly increased risk of hypoglycemia and weight gain. By contrast; DPP4-I produce 0.4-0.6% reduction in HbA1c, lower risk of hypoglycemia, and weight loss.

Notes:

OP8

IS NURSING HOME SPECIFIC ANTIBIOGRAM NECESSARY FOR ALL NURSING HOMES?

K Shah, P Cook, TJ Lee, JD Christie, X Fang, MS Ashraf

Background: Smaller sample size in nursing homes can be one of the limitations in developing useful facility specific antibiogram. Studies looking into variability of antibiotic susceptibilities for closely located nursing homes are lacking. We examined the differences in the susceptibility results of the commonly prescribed antibiotics for the most common organism identified on urine cultures in 4 different nursing homes located within 5 miles of each other.

Methods: Using a retrospective study design, we collected susceptibility results of all positive urine cultures sent to Vidant Medical Center microbiology laboratory from 4 closely located nursing homes during the calendar year of 2012. Positive culture results lacking definitive identification or antibiotic susceptibility of microorganisms were excluded. Susceptibility results for commonly used antibiotics were compared for statistically significant differences in the most commonly identified organism among 4 nursing homes using Fisher's exact test.

Results: *Escherichia coli* was the most common microorganism from urine cultures with 99 total isolates (21, 23, 19 and 36 from nursing homes A, B, C and D, respectively). When comparing sensitivities of *Escherichia coli* in nursing homes A, B, C and D, 86%, 78%, 63% and 81% of the isolates were sensitive to trimethoprim-sulfamethoxazole, respectively ($p=0.35$); 76%, 78%, 58% and 61% to ciprofloxacin ($p=0.33$), 90%, 87%, 89% and 81% to cefazolin ($p=0.69$); 95%, 96%, 95% and 100% to nitrofurantoin ($p=0.68$). The only significant difference was in the sensitivity of ampicillin (62%, 39%, 11% and 44%, $p=0.01$). Other common organisms included *Klebsiella pneumoniae* (n=38), *Enterococcus species* (n=36), *Proteus mirabilis* (n= 34), and *Pseudomonas aeruginosa* (n=22). The number of organisms was too small for statistical analysis for all of these isolates.

Conclusions: We have demonstrated that antimicrobial susceptibilities were similar for most antibiotics in closely located nursing homes. Antimicrobial stewardship programs can consider developing regional nursing home antibiograms if multiple nursing homes are located in close proximity. This practice can be particularly helpful when developing antibiograms for smaller size nursing homes.

Notes:

OP9

CRITICAL EVENT SIMULATION TEAM: A NEW EDUCATIONAL, PATIENT SAFETY AND QUALITY IMPROVEMENT INITIATIVE

T Pancoast, M Ritchie, A Hidalgo, J Ford, S Ingram, P Ouellette, W Robey

Concept: Procedural skills deteriorate at a predictable rate requiring periodic retraining. A significant number of adverse events leading to root cause analysis are related to the performance of procedures. We created a Critical Event Simulation Team (CEST) to react immediately to adverse events related to the performance of procedures to analyze the event and take corrective action by formulating and then staging a related simulation scenario. Here we describe the first case addressed by the CEST.

Methods: CEST is run by Critical Care fellows in conjunction with the BSOM Clinical Simulation Center. A stepwise approach to the analysis and reaction to critical adverse events has been developed. Step 1: Identification of the event. Step 2: Data collection. Step 3: Data analysis. Step 4: Creation of a simulation session. Step 5: Conduction of a simulation session. Step 6: Evaluation and bidirectional feedback.

Results: The case involved a difficult airway with failed bedside surgical cricothyrotomy. The event was reported the same day, providers interviewed over the next 3 days, a written timeline completed and simulated scenario prepared by day 4. The key to the event analysis was rapid sequential assessment of the patient, treatment and intervention decisions, and, ultimately, performance of the procedure. The scenario involved a second by second step-wise reconstruction using a high fidelity simulator. Analysis of the case prior to the simulation suggested that failure to achieve airway access in a timely manner was due to inadequate refresher training for the procedure. This was confirmed by running the simulation. Post simulation education involved hands on training with faculty guided practice opportunity. Participants uniformly evaluated the session as helpful.

Conclusion: The CEST concept allowed us to quickly assess and deconstruct a critical event and perform a real time, root cause analysis with reproduction of the clinical event using a high fidelity simulator. Use of this process identified a training deficiency and led to an educational session with refresher training designed to prevent the event from occurring again. Initial assessment of the process and experience by participants was uniformly positive. A stepwise investigative and educational approach using simulation-based modalities can be used to expose healthcare individuals and teams to realistic clinical challenges surrounding actual clinical events.

Notes:

OP10

EVALUATION OF TRANSITION-AGED PATIENT OUTCOMES ON ADULT HOSPITAL WARD

H Lai, C Christiano, G Hidalgo

Background: Outcomes in adolescents and young adults (AYA) with chronic diseases of pediatric onset following transfer to adult practitioners remains poorly defined. Formal programs are developing within most tertiary care academic programs to track clinical outcomes for AYA with pediatric-onset chronic disease (POCD) entering adult services. This quality initiative was designed to examine hospital outcomes in AYA admitted to an adult ward. **Objective:** Assess hospital outcomes in AYA admitted to an adult medicine ward at a single tertiary hospital offering both adult and pediatric hospital services. Investigate nursing perspectives towards caring for AYA on an adult ward. **Methods:** Hospital length of stay (LOS), readmission rates, cost of care, mortality and events of harm were retrospectively analyzed for patients under 26 years admitted to a Medicine ward over a 3-year period from September 2008 to Oct 2012. Ward nurses were surveyed on their experience in pediatrics and their level of comfort or discomfort in managing pediatric-onset disorders in AYA. **Results:** AYA under 26 years of age accounted for 9% of patients admitted to Medicine ward. Hospital length of stay (LOS) was 0.55 days longer than the average length of stay with average cost of ~\$37,000. 30-, 60-, and 90-day readmission rates were 11%, 8%, 6%. There was no documented mortality patients under 26 years of age over this 3-year period. Events of harm in patients under age 26 were high over the first year of evaluation with significantly fewer the subsequent 2 years suggesting a difference in practice leading to improvement in care. During the last year of evaluation events of harm comprised only 0.15% of all events of harm. Although AYA in the adult ward suffered fewer events of harm and lower mortality, they are perceived by nursing staff as more 'difficult' patients. In addition they have slightly longer average length of stay 6.55 versus 6 days and represented 9% of all readmissions over a 3-year period although they comprised only 5.9% of patients.

Conclusions: Although they tend to be less ill, AYA spend a longer time in the hospital and bounce back fairly frequently within the first 30 days. This suggests a need for better aftercare service coordination along with improved understanding of transitional barriers to care in POCD. Increase nursing ratio, improved patient education of disease process, and increased clarity of physician orders were identified as areas for improvement.

Notes:

RELEVANCE OF THE EXTRAPITUITARY PROLACTIN PROMOTER POLYMORPHISM AT -1149G/T ON PROLACTIN AND DEHYDROEPIANDROSTERONE IN AMERICAN AFRICAN AND EUROPEAN WOMEN WITH SYSTEMIC LUPUS ERYTHEMATOSUS.

EL Treadwell, C Stevens, K Wiley, B Word, W Melchior, W Tolleson, N Gopee, G Hammons, BD Lyn-Cook

Background and Objectives: Systemic lupus erythematosus (SLE) is a complex, potential debilitating and fatal autoimmune disease affecting 1.4-2.0 million Americans. Hormonal, infectious, and environmental factors have been implicated in the etiology. Higher levels of prolactin, lower levels of dehydroepiandrosterone (DHEA) and induction of inflammatory cytokines have been reported in SLE patients. African American women (AAW) have SLE 2-3 times more than European American women (EAW), and with more morbidity and mortality. The purpose of this study was to determine the effect of the presence of the -1149G/T single-nucleotide polymorphism (SNP) (rs1341239) in the promoter of the extra pituitary prolactin gene on SLE.

Method: 256 patients were recruited by IRB informed consent from the Brody School of Medicine Rheumatology Clinics; 135 SLE (American College of Rheumatology Criteria) and 129 matched controls. 87 SLE were AAW, 25 EAW, 10 AA men and 5 EA men. Prolactin T→G¹¹⁴⁹ polymorphism genotyping was performed using restriction fragment polymorphism (RFLP) and confirmed by DNA sequencing using peripheral blood mononuclear cells (PMBCs). RNA was extracted using a Paxgene RNA Kit and cDNAs were synthesized by a RT-for-PCR kit. DHEA and related circulating hormones were assayed using a RIA method. Cytokines tumor necrosis factor- α (TNF- α) and IL-6 were performed by specific enzyme-linked immunoassay kits. SLE disease activity (SLEDAI) was performed using a validated 1-10 scoring disease scale. A 2-tailed Mann Whitney t-test was used for statistical comparison. **Results:** SLE patients had higher prolactin serum levels than non-SLE patients ($p=0.0026$). The -1149TT genotype was correlated with higher prolactin in serum and gene expression ($p=0.0017$) in PMBCs, lower DHEA levels in serum and with the 1149TT genotype ($p=0.001$), higher IL-6 serum levels ($p=0.0001$) and TNF- α levels ($p=0.042$) in AAW. Higher SLEDAI scores were in AAW ($p=0.001$) and EAW ($p=0.0001$) with SLE compared to non-SLE controls. **Conclusion:** This study suggests that the -1149TT SNP polymorphism genotype may be a risk factor for SLE and may predict who could possibly benefit from DHEA therapy.

Notes:

PR1

GENETIC CONTROLS OF GERM CELL FATE SPECIFICATION IN CAENORHABDITIS ELEGANS

SS Mamillapalli, YC Kwon, MH Lee

BACKGROUND: The precise regulation of germ cell fate (sperm or oocyte) lies at the heart of reproduction and fertility. The nematode *C. elegans* hermaphrodites produce a discrete number of sperm during larval development and then switch to produce oocyte during adulthood. Importantly, many of the key germ cell fate regulators control the translation of mRNAs. Therefore, translational control of sperm-oocyte gene mRNA is critical for *C. elegans* germ cell fate specification.

METHODS: We used *C. elegans* as a model system to understand the mechanism of germ cell fate specification. A number of practical advantages have made *C. elegans* an attractive model for genetic and developmental biological research. To identify the key germ cell fate regulators, we performed phenotype-based RNAi screening. Biological roles of the identified regulators were determined using gene-specific mutants or transgenic animals. In addition, germ cell fate was examined by staining dissected gonads with antibodies.

RESULTS: Our genetic analyses found that FBF-1 (a member of PUF RNA-binding proteins) and LIP-1 (an ERK phosphatase) redundantly repress oocyte fate specification. Our genetic screening has also identified several G2/M cell cycle regulators that promote oocyte fate specification. Notably, several G2/M cell cycle regulators are negatively regulated by FBF-1 and LIP-1. Therefore, we suggest that germ cell fate may be regulated by three factors: Translational regulators, Signal transduction, and cell cycle regulators.

CONCLUSION: These results suggest that FBF-1 and LIP-1 may repress oocyte fate by inhibiting G2/M cell cycle progression in *C. elegans* germline.

Notes:

PR2

THE ROLE OF microRNA ON GERM CELL FATE SPECIFICATION IN CAENORHABDITIS ELEGANS

RP Parekh, MH Lee

Background: Germ cells, the cells that are specialized to produce sperm and oocytes, maintain the potential to create an entirely new organism. Throughout most of their life, germ cells reside in the gonad and germ cell fate is tightly regulated by both extrinsic signaling pathways and intrinsic regulators. It has been reported that post-translational inhibition is one of critical mechanisms for germ cell fate specification. Recently, we reported that PUF-8 (a translational repressor) and LIP-1 (an ERK phosphatase) promote oocyte fate by inhibiting Ras-ERK MAPK signaling using the nematode *C. elegans* as a model system. Based on this finding, we performed genetic analysis to identify new translational regulators that reprogram germ cell fate in *lip-1* mutant background.

Methods: To identify key translational regulators that reprogram germ cell fate in *lip-1* mutant background, we performed a focused RNAi screening and generated compound mutants using a standard genetic approach. Germ cell fate was determined by staining dissected gonads with sperm-specific marker (SP56) and oocyte marker (RME2).

Results: Our genetic approaches have identified several RNA-binding proteins and microRNAs that normally inhibit sperm fate specification in *lip-1* mutants. Among them, we focused on the role of microRNAs in germ cell fate specification. Single mutation for the identified microRNA did not display germ cell fate defect, but this mutation significantly promoted sperm fate in *lip-1* mutants. It suggests that the microRNA and LIP-1 redundantly repress sperm fate specification. Next, we are studying whether this germ cell phenotype depends on Ras-ERK signaling pathway. This result from this study will be reported at the meeting. Notably, microRNAs play a critical role in animal development. However, the potential role of microRNAs in germ cell fate specification has not been yet studied. Therefore, our finding will provide insights into the translational regulation of germ cell fate specification in multicellular organisms, including humans.

Conclusion: Our findings conclude that *C. elegans* germ cell fate may be regulated by microRNA at a translational level. microRNAs are conserved regulators. Therefore, we suggest that similar regulatory mechanism might be conserved in other animals.

Notes:

PR3

ACIDIC TUMOR MICROENVIRONMENT ACTIVATES ENDOPLASMIC RETICULUM STRESS PATHWAYS THROUGH THE GPR4 RECEPTOR IN ENDOTHELIAL CELLS

L Dong, EA Krewson, LV Yang

Background: The tumor microenvironment is characterized by acidosis and hypoxia due to tumor heterogeneity, aerobic glycolysis (“Warburg effect”) and the defective vasculature that is inefficient to deliver oxygen and to remove metabolic acid byproduct. How the acidic microenvironment affects the function of blood vessels, however, is not well defined. GPR4, a member of the proton-sensing G protein-coupled receptors, has high expression in vascular endothelial cells. We have previously reported that acidosis induces a broad inflammatory response in human vascular endothelial cells through the GPR4 receptor. Acidosis also increases the expression of several endoplasmic reticulum (ER) stress genes such as CHOP and ATF3. In the current study, we have thoroughly examined acidosis/GPR4-induced ER stress pathways mainly in human umbilical vein endothelial cells (HUVEC).

Methods: HUVECs were stably transduced with the expression plasmids or the vector control to generate HUVEC with endogenous or overexpressed levels of GPR4. HUVECs were stably transduced with the GPR4-targeted small hairpin RNA (shRNA)-expressing construct or the scramble shRNA-expressing construct to generate HUVEC with endogenous or knockdown levels of GPR4. HUVECs were treated with pH 6.4 medium to activate GPR4 or with pH 7.4 medium as a negative control. Western Blotting and RT-PCR were carried out to detect the expression of target genes at protein and mRNA levels, respectively. **Results:** Our study showed that all three arms of the ER stress/unfolded protein response (UPR) pathways were activated by acidosis in HUVEC, as we observed an increased expression of phosphorylated eIF2 α , phosphorylated IRE1 α , and cleaved ATF6 upon acidic pH treatment. The expression of other downstream mediators of the UPR, such as ATF4, ATF3, CHOP and spliced XBP1, were also induced by acidosis, at the mRNA and/or protein levels. Moreover, we found that GPR4 plays an important role in mediating the ER stress response induced by acidotic stimulation. **Conclusion:** As ER stress/UPR can cause inflammation and cell apoptosis, acidosis/GPR4-induced ER stress pathways in endothelial cells may regulate vascular growth and inflammatory response in the acidic tumor microenvironment.

Notes:

PR4

THE MULTIFARIOUS EFFECTS OF ACIDOSIS ON LYMPHOMA CELL METABOLISM AND THE TUMOR SUPPRESSIVE FUNCTION OF THE PROTON SENSING G-PROTEIN COUPLED RECEPTOR TDAG8.

EJ Sanderlin, CR Justus, Z Li, LV Yang

Introduction: The tumor microenvironment is characterized by regional acidosis due to deregulated cancer cell metabolism and defective blood perfusion. Acidosis within the tumor microenvironment can have both tumor promoting and inhibiting effects much depending on the duration of acidotic stress. The T-cell Death Associated Gene 8 (TDAG8), a family member of the proton-sensing G-protein coupled receptors (GPCRs), has the ability to sense protons in the extracellular milieu and transduce intracellular signaling through G-proteins to modulate tumor cell function. Here we provide evidence supporting TDAG8 function in lymphoma as a tumor suppressor by sensitizing lymphoma cells to apoptosis, regulating cellular metabolism, and potentially slowing cell cycle progression. **Methods:** TDAG8 was stably overexpressed in Ramos and U937 lymphoma cells. Ramos and U937 cells were treated with medium buffered to pH 7.4 and pH 6.4 during various time points. Cell cycle analysis was performed using propidium iodide and analyzed by FACS. Several apoptosis, metabolic, and cell cycle regulators were measured for protein expression using Western blot analysis. Intracellular pH was measured using pHrodo Red AM ester dye and visualized by fluorescence microscope. Extracellular lactate was measured using the Lactate Colorimetric Assay. Intracellular ATP concentration was determined by Cell Titer Glo Cell Viability Assay. The Cyquant cell proliferation assay was used to measure cell proliferation. The tumor xenograft model was performed by subcutaneous injections with Ramos and U937 cells overexpressing TDAG8. **Results:** Acidosis decreased cell proliferation in lymphoma cells and also blocked cell cycle progression in the S phase. Moreover, extracellular lactate was decreased after acidic pH treatment coupled with a decrease in intracellular ATP levels. Whether TDAG8 is involved in these observations is currently unclear, but suspected to be the result of c-Myc down regulation in response to acidotic stress. Additionally, we observed a sharp increase of intracellular acidity as a result of treatment of media buffered to pH 6.4. This decrease in intracellular pH may contribute to DNA damage, causing cell cycle inhibition and apoptosis. Furthermore, a clear induction of cleaved PARP was observed upon acidic treatment of Ramos cells. *In vivo* we observed a clear reduction in average tumor mass and growth in tumors overexpressing TDAG8. Collectively, we propose a role for TDAG8 in Ramos and U937 cells as a tumor suppressor.

Notes:

PR5

GPR4 ACTIVATION BY ACIDOSIS HINDERS ANGIOGENIC PROCESS OF HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS

EA Krewson, LV Yang

Background: Angiogenesis is the growth of new blood vessels from existing vasculature and is critical for embryogenesis, wound healing, and tumor development, invasion, and metastasis. Angiogenesis involves the activation of endothelial cells (ECs) to proliferate, migrate, and create a tubular network. Newly lined vascular ECs recruit pericytes and smooth muscle cells (SMCs), for the promotion of vascular stability and maturation. Within various disease states, vasculature becomes torturous and disorganized partly due to acidosis and hypoxia. How the acidic microenvironment influences EC function is unclear. Proton-sensing G-protein coupled receptor 4 (GPR4) is highly expressed in ECs and is activated by protonation of histidine residues. We provide evidence supporting GPR4 activation inhibits EC migration and tubular formation. **Methods:** We stably transduced a construct that overexpresses GPR4 in human umbilical vein endothelial cells (HUVEC/GPR4). We performed a wound-healing assay and tubular formation assay on HUVEC compared with HUVEC/GPR4 under physiological pH 7.4 and acidic pH 6.4 conditions. Furthermore, we have orchestrated a co-culture system with HUVECs and primary coronary artery smooth muscle cells (PCASMC). This system allows us to observe the function of GPR4 in vascular stability by the recruitment of pericytes and SMCs. We exposed our co-culture system to physiological and acidic pH and observed tubular structure and stability. **Results:** We observed HUVEC/GPR4 cells treated under acidic pH inhibited cell migration. Within a tube formation assay, HUVEC/GPR4 cells treated under physiological pH displayed shorter-lived tubular structures. Under acidic conditions, HUVEC/GPR4 cells did not exhibit any tube formation capabilities. With respect to the physiological pH co-culture system, we have found that PCASMC promote EC tube stabilization and prolong vascular-like structure. Under acidic pH, we observed PCASMC exhibit a delayed migration to the HUVEC tubular structures. Following treatment of HUVEC/GPR4 cells with acidic media during the co-culture system, PCASMC did not rescue or prolong the vascular structure formation. **Conclusion:** Employment of EC migration, tube formation, and co-culture assays elucidated the key role for GPR4 as a component in negatively regulating angiogenesis.

Notes:

PR6

NOTCH SIGNALING REGULATES EPITHELIAL-MESENCHYMAL TRANSITION IN COLORECTAL CANCER

AW Fender, KE Vinson, DC George, G Sigounas

Background: Colorectal cancer (CRC) is the third leading cause of cancer death in the United States, resulting in an average of 50,000 deaths per year. Curative options are limited if surgery and chemotherapy are unsuccessful. Several studies have indicated that CRC aggressiveness and potential for metastatic spread are associated with the acquisition of stem cell like properties. The Notch-1 receptor and its cognate signaling pathway is well known for controlling cell fate decisions and stem-cell phenotypes. Alterations in Notch receptors and Notch signaling have been reported for some colon cancers. Furthermore, our preliminary results indicated that in CRC patient samples, Notch-1 expression was increased in colon tumor tissue as compared with normal colon tissue. Herein, we examine a potential role for Notch-1 signaling in CRC. **Methods:** The experiments described in this study were conducted with the colon cancer cell line HCT-116. These cells were transduced with an IRES-GFP retrovirus expressing human intracytoplasmic domain of Notch-1 (ICN1). Cell dividing time, colony growth, wound healing, transwell migration, anchorage independent growth, and Western blot analysis were performed using standard methodology. Purified Notch ligand studies were conducted using Jagged-1. Pharmacologic inhibition of Notch-1 signaling was induced by DAPT, an effective γ -secretase inhibitor. **Results:** Retroviral transduction of constitutively active Notch-1 (ICN1) into the colon tumor cell line HCT-116 resulted in increased expression of the EMT/stemness associated proteins CD44, Slug, Smad-3, and induction of expression of the Notch ligand Jagged-1. Meanwhile, there was a four-fold decreased expression of E-cadherin. These changes were accompanied by alterations in anchorage dependent growth, migration, and proliferation. Experiments with the pan-Notch inhibitor DAPT, and soluble Jagged-1-Fc protein provided evidence that Notch-1 signaling activates CD44, Slug and Smad-3 via a cascade of other Notch-receptors through induction of Jagged-1 expression. **Conclusions:** These data indicate a key role for Notch signaling in the phenotype of CRC and suggest that targeting of Notch signaling may be of therapeutic value in colon cancers.

Notes:

PR7

DIETARY INTERVENTIONS IN MANAGING IRRITABLE BOWEL SYNDROME: A SYSTEMATIC LITERATURE REVIEW

DA Gliga, H Movahed, E Ali

Alterations in enteric nervous system and gut function lead to symptoms collectively known as functional gut symptoms (FGS). These include pain, change in bowel habits, discomfort, distention and obstipation. The number of people affected with Irritable Bowel Syndrome (IBS) reaches 5-20% of the Western countries populations and gastrointestinal discomfort is reflected through aforementioned symptoms.

The purpose of this review is to synthesize up-to-date studies available in the Ovid Medline search engine. We focused on specific dietary changes, methods of applying aforementioned modifications and the effect of their implementation in individuals with IBS. Pilot studies have indicated that FODMAP (Fermentable Oligo-, Di- and Mono-saccharides and Polyols) restricted diets are beneficial to relieve symptoms in those with irritable bowel disease. Studies investigated the effect of these constituents on gastrointestinal symptoms alone or in combination. Questions were raised regarding whether the relief was due to restriction of these molecules specifically or whether the basis of the relief-phenomenon was restriction of poorly absorbed short-chain carbohydrates.

Based on our analysis, diets low in FODMAPs are evidence-based interventions in management of FGS. A knowledgeable credential dietitian begins with a review of current medications, GI symptoms, assessment of nutritional status and food intake, evaluation of supplement intake and the use of mind-body therapies. From there a plan is developed to train the patient to strategically eliminate and reintroduce foods containing FODMAPs. This helps decrease global symptoms in most IBS patients. With a dietitian-delivered diet instruction, the highest degree of compliance is ensured. The results allow inclusion or exclusion of fructose and/or lactose when designing the FODMAP diet. Overall this allows for minimizing dietary restrictions only when necessary. A six-to-eight week trial period is encouraged for the FODMAP limited diet, allowing for identification of diet components triggering FGS. The process of adding insoluble fibers either led to increased experiencing of IBS symptoms or had no effect. Besides dietary changes, fructose malabsorption can also be reversed with antibiotic use. This indicates that small bowel bacterial overgrowth contributes to this physiological phenomenon. Overall, avoidance of fermentable carbohydrates is an emergent therapy for IBS.

Notes:

PR8

STRIKING OUT HYPOGLYCEMIA: A MULTIDISCIPLINARY APPROACH

DA Rao, ES Thoma, S Hardee, N Armistead, R Tanenberg

Background: Severe hypoglycemia (blood glucose \leq 50mg/dL) leads to prolonged length of hospital stay, transfer to higher level of care, and increased morbidity and mortality. Our institution established the Adult Medical Service Hypoglycemia Strike Team to reducing hypoglycemic events. The team adopted a multidisciplinary approach to improving hypoglycemia, consisting of nurse managers, patient safety coordinators, pharmacists, resident and attending physicians.

The baseline incidence of hypoglycemia was 33 events/month and order set usage, a leading indicator of performance, was at 38%. The overall rate of hypoglycemic events prior to intervention was 2.0%.

Methods: The hypoglycemia committee used a systematic approach with five different strategies for intervention: resident education and feedback on quality improvement measures, hospitalist feedback on order set usage and incidence of hypoglycemia, pharmacist review of blood glucose and insulin dosing recommendations, nursing education, and reporting of events into a safety intelligence database.

Results: The results of these interventions were promising as overall diabetes order set utilization increased from 38% to 57% (a 45% improvement), hypoglycemic events decreased from an average of 33/month to 22/month (a 32% improvement), and the rate of hypoglycemia improved from 2% to 1.2% (a 40% improvement).

Conclusion: Our interventions showed that a combination of education, feedback on quality improvement data, and leveraging use of the electronic health record (EHR) led to dramatic improvement in healthcare quality and patient safety.

Notes:

PR9

LIVING LONGER WITH PANCREATIC CANCER: MORE THAN FOLFIRINOX?

P Chae, P Punja, N Koutlas, P Atluri

Background: Effective treatment for unresectable locally advanced pancreatic cancer (LAPC) or metastatic pancreatic cancer is limited and has a poor prognosis with a five year survival rate of 6.7%. FOLFIRINOX has been studied as a neoadjuvant therapy for LAPC and has a response rate of 32%. We set out to investigate which factors, if any, have a meaningful impact on time to disease progression within this cohort of patients. **Methods:** A retrospective chart review was conducted. All patients who received FOLFIRINOX at our institution from 2011-2014 were included. 16 of 19 had unresectable LAPC or metastatic disease at time of diagnosis. A few received FOLFIRINOX upon first progression after adjuvant therapy. **Results:** Of the 19 patients that received FOLFIRINOX at our institution, 3/19 (16%) were considered resectable at time of diagnosis and all 3 underwent Whipple surgery. The remaining patients had either metastatic or unresectable disease. The mean number of cycles of FOLFIRINOX received was 3.4. 5/19 (26%) received radiation alone in either the adjuvant or palliative setting. The mean time to progressive disease was 8.1 months. In the 5 patients who received radiation, the mean time to progression was 10.1 months. The 3 patients with the longest time to progression had a mean time of 18.7 months until their disease progressed. 2 of the 3 patients who received radiation therapy had metastatic disease at time of diagnosis. **Conclusions:** Novel approaches are being used to get patients to R0 resection if they have borderline resectable pancreatic cancer or unresectable LAPC consisting of combination chemotherapy followed by radiation. Metastatic patients or unresectable LAPC who do not undergo surgery have limited survival. Reviewing our data we found that the addition of radiation in the metastatic setting may impact time to progression as determined by RECIST criteria. Of the three patients who had the longest time to progression, two were metastatic and both underwent palliative RT to the primary pancreatic lesion. Incorporation of RT to the primary lesion upon completion of FOLFIRINOX therapy should be considered as an innovative approach to increase time to progression and potentially progression free survival and overall survival. Clinical trials randomizing selected patients with metastatic or unresected LAPC to FOLFIRINOX followed by RT versus no RT should be considered.

Notes:

PR10

REAL-TIME DUTY HOUR REPORTING FOR NEPHROLOGY FELLOWS THROUGH A SMARTPHONE APP

WA Davila, HA Coore, TP Desai

Duty hour monitoring is an important but time-consuming task for trainees to complete in a timely manner. Despite a number of commercial software, our trainees have had delays in reporting their hours by weeks to months. These delays prevent Program Directors (PD) from identifying overworked trainees in real-time. In this early investigation, we designed an iOS App, based on the suggestions of our trainees, to record duty hours and measured the time delay in reporting those hours.

We programmed the Nephrology On-Demand Plus iOS App to collect duty hour information. Each entry asked the fellow their 1) name, 2) unique hospital employee number, 3) time and day in and out, and 4) rotation name. The App automatically added a time-stamp to each entry. We compared the time difference between the time-out and time-stamp to calculate the delay in reporting.

A total of 305 duty hour entries were recorded from 7/1/14-10/31/14, representing a 100% completion rate. Approximately 81% of all entries were logged on the same calendar day as the clinical shift worked (77% for 1st year trainees, 85% for 2nd year trainees). The median time delay was 0.72 hours (IQR 0.20-2.2 hours). On two occasions, a trainee was placed on modified-assignment after his entries projected a greater-than 80-hour work week. The total number of duty hour violations was zero.

We report a near “real-time” duty hour monitoring system using an iOS App. The limited reporting delay has allowed the PD to anticipate duty-hour violations and take proactive measures to manage over-worked trainees.

Notes:

PR11

COMPARING TWO MODELS THAT REDUCE THE NUMBER OF NEPHROLOGY FELLOWSHIP POSITIONS IN THE UNITED STATES

WA. Davila, T Desai

Since 2002 there has been a steady decline in the number of applications to nephrology training programs. Numerous reasons have been posited to explain this decrease. A universally accepted solution is to decrease the number of available fellowship positions. Proponents believe that training programs have grown too big compared to the current demand for training positions. The method for reduction, however, has not been established. We analyzed two models that decrease the number of available training positions and compare them head-to-head to identify the least burdensome method by which this reduction should occur.

In the first model (survival of the fittest; SotFM) fellowship positions are eliminated if they were unfilled through the National Residency Match Program's (NRMP) 2013 Specialty Match. In the second model (equal proportions; EPM), a mathematical formula is used to apportion fellow positions per state. This formula calculates a priority score using statewide ESRD prevalence data from the 2013 USRDS Report & the geometric mean between a given state's current apportionment (n) & its next apportioned position (n+1). The most equitable model was defined as that which resulted in 1) the least number of states losing fellow positions, 2) the lowest percent reduction for any single state, or 3) both.

There were 416 nephrology positions offered & 47 unfilled in 2013. In the SotFM, 23 states would sacrifice these 47 positions. In the EPM, 369 positions were apportioned (=416-47); only 9 states would experience a reduction. The largest single-state reduction in fellow positions was 67% in the SotFM & 50% in the EPM.

The EPM results in less burdensome reduction of fellow positions nationwide. Fewer states experience a reduction in their total number of positions (9 v 23) & there is a lower maximum single-state burden (50 v 67%) in the EPM. The EPM, is a model that injects fairness into the painful process of reducing the total number of fellow positions across America.

Notes:

PR12

EVALUATION OF CARDIOMYOPATHY IN THE HEMODIALYSIS PATIENTS

A Bilal, C Christiano.

Background: Dialysis patients should be evaluated for the presence of cardiomyopathy (systolic or diastolic dysfunction) in the same manner as the general population, using echocardiographic testing. Echocardiograms should be performed in all patients at (1) the initiation of dialysis, (2) once patients have achieved dry weight (ideally within 1–3 months of dialysis initiation), and (3) at 3-yearly intervals thereafter. In patients at high risk for coronary artery disease (e.g., those with diabetic chronic kidney disease), coronary angiography may be appropriate, even in patients with negative stress imaging test, due to lower diagnostic accuracy of noninvasive stress imaging test in chronic kidney disease patients.

Methods: We selected 20 new hemodialysis patients from our outpatient dialysis unit, tabulated their dialysis initiation date and echocardiogram date. Some of the patients underwent noninvasive stress imaging test.

Results: Of the 20 patients in our study, almost all of them had an echocardiogram prior or at the time of dialysis initiation. Almost 50% of the patients did not have an echocardiogram 1-6 months after the dialysis initiation.

Conclusion: These findings suggest that most of the hemodialysis patients do not get a follow up echocardiogram, and those patients are at high risk of developing cardiovascular complications. Patients should be closely followed and should get echocardiograms post dialysis initiation to prevent cardiovascular complications.

Notes:

TRANSMITTED DRUG RESISTANCE IN TREATMENT NAÏVE HIV PATIENTS

A Abubaker, X Fang, N Fadul

BACKGROUND: Transmitted Drug Resistance (TDR) is uncommon in HIV. Patients with TDRs are likely to receive more complex regimens and have poor treatment outcomes. The objective of this study is to examine the prevalence of TDR in chronically infected newly diagnosed HIV patients in Eastern North Carolina and whether there are associations between demographic factors and the presence of TDR.

METHODS: This is a retrospective chart review of all newly diagnosed, treatment naïve HIV patients seen for the first time at the Brody School of Medicine HIV Program. We collected demographic information including age, sex, county of residence and HIV risk factor. We also collected laboratory information including CD4 count and HIV viral load and HIV genotype at first visit. We recorded presence or absence of TDR as well as individual mutations in the non-nucleoside reverse transcriptase inhibitors (NNRTI) nucleoside reverse transcriptase inhibitors (NRTI) protease inhibitors (PI) and integrase inhibitors (INI) drug classes. Only major mutations as defined by Stanford HIV Drug Resistance Database were included. Descriptive statistics were used to analyze the data.

RESULTS: One hundred and four patients were included in the study, 18 patient were excluded due to lack of genotypic information. The prevalence of baseline TDR was 12% for all drug classes (16 % in the NNRTI, 3% in NRTIs, and 1% in the PI drug classes). There was no TDR in the INI class. Age <25 years had a statistically significant association with presence of TDR (<25, 35%; 25-49, 12% and >50, 0%; p=0.03).

CONCLUSIONS: TDR was detected in 12% of treatment-naïve HIV patients, with resistance to the NNRTI class being the most common. There was a significant association between the presence of TDR and age <25 years.

Notes:

PV1

PRIMARY CENTRAL NERVOUS SYSTEM LYMPHOMA IN PATIENT WITH HIV DISEASE

A Abubaker, D Rao, H Nguyen, A Stang, K Ramsey

Learning Objective: To review and discuss the medical literature for the clinical manifestations and management of HIV disease with brain-occupying lesions.

Case presentation:

A 51 year old Hispanic male, previously healthy, presented with 5 days of acute left side weakness associated with on and off headache, and a weight loss of 15 pounds over few months. On the day of admission, he measured 1/5 strength of his left upper limb on exam. Further evaluation with brain MRI noted to have 2.6x2x1.9cm rim-enhancing mass in the posterior aspect of the right frontal lobe. He was also confirmed as positive for HIV on EIA/Western Blot testing with a CD4 count of 45/mm³ and viral load of 40,000 copies/ml. He was also found to have positive serology for *Toxoplasma gondii*. He was treated empirically for Toxoplasmosis with pyramethamine and sulfadiazine. The patient subsequently underwent stereotactic brain biopsy, after which the surgical pathology studies confirmed the diagnosis of primary B-cell lymphoma. The patient was treated with chemotherapy initially and planned to start antiretroviral therapy (HAART) as soon as possible.

Discussion:

Approximately 20-to-30 percent of CNS lesions in patients with the acquired immune deficiency syndrome (AIDS) are ultimately found to be primary central nervous system lymphoma (PCL), with toxoplasmosis and progressive multifocal leukoencephalopathy accounting for many of the remaining cases. This distinction can be made using a combination of cerebrospinal fluid (CSF) cytology, serologic testing, an empiric trial of antibiotics, Epstein-Barr virus (EBV) DNA in CSF/ tissue sample, and diagnostic brain biopsy. Space occupying lesions in advanced HIV patients with CD4 count less than 50/mm³ and positive serology for Toxoplasmosis have always been a diagnostic challenge for physicians. Thus, the only definite way to differentiate between the Toxoplasmosis and CNS lymphoma is to obtain a tissue biopsy if it can safely be performed. When surgical intervention is not an option, another approach is to treat empirically for the presumptive diagnosis of CNS Toxoplasmosis for within 2-4 weeks. If there is no response to therapy, then a definitive biopsy is indicated.

Notes:

PV2

A DANGEROUS CAUSE OF NECROTIZING FASCIITIS

A Abubaker, M Dhillon, H Nguyen, M S Ashraf

Learning objective: To understand the importance of early diagnosis and treatment of *Vibrio vulnificus* infection in the disease process

Case presentation:

An 86 year old M with history of HTN, CKD was in his usual state of health until two days prior to his admission at Vidant Medical Center when he developed pain and swelling in his left index finger after minor injury while he was cleaning oyster. He went to his primary care provider who prescribed hydrocodone and erythromycin for cellulitis. At that time he denied any other symptoms. On same night, he developed watery diarrhea along with nausea and vomiting. He became weak and noticed of a fever. Next day morning he noticed that the swelling getting worse and he had blister spreading along his left arm. He therefore was brought to the emergency. He went into septic shock and underwent debridement of the left index finger urgently. Blood culture grew *V. Vulnificus* as well as the culture from the tissue. He was treated with Ceftriaxone with doxycycline. Later on he underwent skin grafts of left forearm and hand, plus amputation of the left fifth finger.

Summary:

V. vulnificus should be in the differential diagnosis of necrotizing soft-tissue infections along with other microbes, such as group A *Streptococcus*, *Staph aureus*, *Pseudomonas*, and *Clostridium perfringens*. *Vibrio vulnificus* is a naturally occurring halophilic gram-negative rod that in coastal waters. *V. vulnificus*, with a case-fatality rate exceeding 50%, is the leading cause of seafood-related deaths in the United States. *V. vulnificus* causes primarily 2 distinct syndromes: 1-Wound infections. 2- Primary septicemia. *V. vulnificus* may contaminate wounds exposed to estuarine waters, shellfish, or fish. Typical examples include hand injuries related to opening oysters or leg lacerations related to entering, exiting, or launching boats. A presumptive diagnosis of *V. vulnificus* septicemia should be made in any person with history of shellfish exposure, fever, septic shock, and characteristic hemorrhagic bullous lesions. The diagnosis is confirmed by culture. *V. vulnificus* will grow without difficulty in standard media. Septicemia or serious wound infections required combination therapy with either minocycline or doxycycline plus either cefotaxime or ceftriaxone. Also surgical intervention needs to be considered as early as possible to prevent complication.

Notes:

PV3

VIBRIO VULNIFICUS AS CAUSE OF NECROTIZING FASCIITIS AND BACTEREMIA

A Abubaker, M Dhillon, H Nguyen, K Roach, M S Ashraf

Learning objective: To understand the importance of recognizing and treatment of *V.vulnificus* infection earlier in disease process

Case presentation:

86 year old M with History of HTN,CKD was in his usual state of health 2 days prior to presentation when he developed pain and swelling in his L - index finger after minor injury while he was cleaning oyster .He went to his PMD who prescribed hydrocodone and erythromycin, at that time he deny any other symptoms . On same night he developed watery diarrhea along with nausea and vomiting. He became weak and he noticed that he had fever. Next day morning he notice the swelling is getting worse and he had blister along the skin. Admitted to hospital with septic shock and underwent I&D ,Blood culture grew *V.Vulnificus* as well as the culture from the tissue .He was treated with Ceftriaxone with doxycycline .Later on underwent skin grafts of left forearm and hand, amputation of fifth finger

Summary:

V. vulnificus should be in the differential diagnosis of necrotizing soft-tissue infections along with other microbes, such as group A *Streptococcus*, *Staph aureus*, *Pseudomonas*, and *Clostridium perfringens*. *Vibrio vulnificus* is a naturally occurring halophilic gram-negative rod that in coastal waters. *V. vulnificus*, with a case-fatality rate exceeding 50%, is the leading cause of seafood-related deaths in the United States .*V. vulnificus* causes primarily 2 distinct syndromes: 1-Wound infections.2- Primary septicemia . *V. vulnificus* may contaminate wounds exposed to estuarine waters, shellfish, or fish. Typical examples include hand injuries related to opening oysters or leg lacerations related to entering, exiting, or launching boats. A presumptive diagnosis of *V. vulnificus* septicemia should be made in any person with fever, hypotension, or symptoms of septic shock, characteristic bullous skin lesions, and risk factors for infection. The diagnosis is confirmed by culture; *V. vulnificus* will grow without difficulty in standard media. septicemia or serious wound infections using combination therapy with either minocycline or doxycycline (100 mg orally twice daily) plus either cefotaxime (2 g intravenously every eight hours) or ceftriaxone (1 g intravenously daily). A possible alternative is levofloxacin (500 mg orally or intravenously once daily). Antibiotic should be started earlier with illness to prevent complication.

Notes:

PV4

A RARE CAUSE OF RECURRENT PERICARDIAL EFFUSION

A Abubaker, K Roach, H Nguyen, M S Ashraf

Learning objective: To recognize the importance of keeping Tuberculosis (TB) in the differential diagnosis when evaluating patient with pericarditis. Review and discuss up-to-date literature in clinical manifestation and management of TB constrictive pericarditis

Case presentation:

A 69 years old male with history of DM and HTN for more than 10 years and no risk factor for TB was admitted with recurrent congestive heart failure (CHF), uncontrolled atrial fibrillation and chronic pericardial effusion over the last 2 months. He was found have pericardial knock and Kussmauls sign with severe volume over load. Further evaluation with echocardiography noted to have septal bounce, hemodynamic evidence of diastolic pressure equalization, dip and plateau RV/LV filling, and discordant RV/LV pressures with end expiration, and chest CT finding of thick pericardium (12 mm). Patient underwent pericardiectomy and PPD was not obtained. He was sent home with diagnosis of CHF exacerbation. Later, culture of pericardial fluid grew 2 colonies of *Mycobacterium tuberculosis* complex. He was treated with 4 drug anti TB regimen along with tapering dose of steroid .He did very well and did not required further admission for decompensated heart failure .

Summary:

Tuberculous pericardial effusion (TPE) is still common in African and Asian countries, but with migrant populations and the spread of AIDS there are now more frequent reports of TPE from other parts of the world like Europe and the Western hemisphere. TPE usually presents as a slowly progressive febrile illness. When it presents as an acute pericarditis, which is uncommon, or as cardiac tamponade, which is frequent, the diagnosis is more likely to be delayed or missed. The diagnosis is established by detection of tubercle bacilli in smear or culture of pericardial fluid and/or by detection of tubercle bacilli or caseating granuloma on histological examination of the pericardium. Initial diagnostic evaluation consists of chest radiography, echocardiography, and AFB culture of the pericardial fluid. The approach to anti tuberculosis therapy for TB pericarditis is generally the same as for pulmonary tuberculosis plus slow tapering steroid. TB should be considered in the differential diagnosis in patients presented with recurrent pericardial effusion with unknown etiology, and PPD should be part of the work.

Notes:

PV5

RARE BUT SEVERE INFECTION IN CARDIAC TRANSPLANT PATIENT

H Nguyen, A Do, A Stang, A Abubaker, M Dhillon, J Christie, P Cook

Learning objectives: To review the literature regarding the microbiology, clinical presentation, diagnosis, and treatment of *Scedosporidium* infection.

Case report: A seventy year-old Caucasian male with a history of cardiac transplant in 2011 was admitted to the hospital because of altered mental status. There was a history of disseminated *Aspergillus* infection for which the patient was taking voriconazole. One week prior to admission, the patient had an uncomplicated macular pouching surgery. Three days later, the patient fell at his home and sustained an injury of his left knee. Two days prior to admission, he developed a fever (101 degrees F) and was found to be disoriented at his home. At the time of admission, a chest x-ray revealed patchy infiltrates of the right lung. A computed axial tomography scan of the brain showed no acute change. Shortly after admission to the hospital, the patient developed septic shock requiring vasopressors and mechanical ventilation. On day 5 of his hospitalization, admission blood cultures became positive for *Scedosporidium apiospermum*. The patient subsequently developed multi-organ failure and died despite resuscitation efforts.

Discussion: *Scedosporidium* species is a fungal opportunistic pathogen that causes infection in severely ill and immunocompromised patients. *S. apiospermum*, the asexual stage of *Pseudallescheria boydii*, and *S. prolificans* are two major human pathogens, found in soil and polluted water globally. The infections can be localized following trauma in immunocompetent hosts but can also present with pneumonia, keratitis, brain abscess, and osteomyelitis in immunocompromised hosts. Disseminated infection carries a high mortality. Histologically, the organism is a septated, non-pigmented hyphae branching at acute angle and may resemble *Aspergillus*. Culture is the definitive means to establish the diagnosis. Among the species, *S. prolificans* shows high resistance to antifungals and may require in vitro susceptibility for guide treatment. Treatment normally includes a minimum of 12 weeks of antifungal drugs. Adjunctive surgical debridement may be necessary. The optimal duration of antifungal therapy is unknown, and the overall prognosis remains poor with severe infections.

Notes:

PV6

IATROGENIC SUPERIOR VENACAVA SYNDROME

S Chalise, N Sultana, P Rice, M Bowling

LEARNING OBJECTIVES: Superior Venacava (SVC) obstruction causes signs and symptoms of SVC syndrome. Commonly caused by malignancy (extrinsic compression from lung cancer or lymphomas) while benign causes include intravascular devices causing thrombosis. We report a patient who developed SVC syndrome due to inflated tracheostomy balloon, which completely resolved after deflating the balloon.

CASE INFORMATION: A 38 year old woman with history of C4 level incomplete quadriplegia secondary to motor vehicle accident at age 9, chronic tracheostomy, tracheal stenosis and tracheobronchomalacia presented with dyspnea and uncompensated hypercarbic respiratory acidosis requiring mechanical ventilator support. She did not have fever, chest pain or increased secretions. Her tracheostomy was changed to #6 Bivona^R TTS with high-pressure cuff, inflated with 7 ml sterile water. Four days later, progressive swelling of upper chest, neck and face were noted. CT scan of neck and chest revealed focal narrowing of SVC at the level of tracheal cuff inflation site. The tracheostomy cuff was deflated with complete resolution of the swelling within 72 hrs. For permanent use, tracheostomy was changed to #7 tracho with a low-pressure air filled cuff.

SUMMARY: SVC syndrome results from obstruction of blood flow through the SVC. This causes edema of face, neck, upper chest, upper back and arms. Edema of the larynx and pharynx may cause dysphagia, stridor, hoarseness, cough and dyspnea. Headache and confusion occurs if there is elevated intracranial pressure.

The obstruction of SVC can be extrinsic or intrinsic. In the past, benign causes like syphilitic aortic aneurysm and tuberculosis used to be the commonest cause. Now, malignancy predominates the cause in more than 90% of cases. In 2007, most common malignant causes are non-small cell lung cancer (50%), small cell lung cancer (25%), lymphoma (10%) and metastatic disease (10%). Thrombosis secondary to increased use of intravascular devices is being increasingly noted. Our case involved unique etiology - inflated tracheostomy cuff. It is important to recognize the potential complication of tracheostomy tube especially in a patient with tracheomalacia, which manifested in our case as SVC syndrome.

Notes:

PV7

CAVITARY PNEUMOCYSTIS JIROVECII PNEUMONIA (PJP), UNCOMMON PRESENTATION OF A COMMON DISEASE

M Rizwan, N Sultana, M Al-Janabi, T Pancoast

LEARNING OBJECTS: PJP remains the most common serious opportunistic organism in HIV patients. PJP presenting with cavitory lesion as initial manifestation of AIDS is extremely rare. Establishing the diagnosis of cavitory lung lesion is important, because missed diagnosis will increase morbidity and mortality.

CASE PRESENTATION: 50 years old, hypertensive female, started feeling sick after dental extraction 3 months prior. She felt weak, fatigued and had lost about 22 pounds. She complained of progressive shortness of breath, cough with occasional production of whitish phlegm and chills with fevers. She had 16 pack years smoking history. Her PPD and HIV test were negative within past year. At presentation the patient was tachypneic, had 102.3F fever and was saturating 96% on room air. Chest was clear to auscultation. WBC count was 4.7k/ μ L. CXR showed bilateral mid and upper lung opacities with cavitation in left upper lobe. CT chest revealed patchy opacities involving the upper and lower lobes with multiple upper lobe cavitory lesions in both lungs (Fig). PPD and sputum for AFB were negative. Broncho alveolar lavage (BAL) was positive for PJP. Trans bronchial biopsy (TBBx) results were non diagnostic. HIV test resulted positive with CD4 count of 7/ μ L. She was started on Bactrim and antiretroviral therapy. CT chest 4 months later showed resolution of cavitory lesions, but showed soft tissue nodular opacity within left upper and right middle lobes. PET scan revealed pretracheal lymphadenopathy. Repeat BAL was positive for AFB yet lymph node biopsies were negative. Antituberculous treatment was initiated, but stopped a few days later as culture grew mycobacterium chelonae-abscessus. Repeat CXR 2 months later showed improvement of soft tissue opacity.

DISCUSSION: Differential diagnosis of cavitory lesions in HIV patients is broad and includes neoplasms, infections from bacteria, mycobacteria, fungi or parasites; vascular diseases and traumatic processes. PJP presenting with cavitation is rare and manifests as a solitary cavity, usually in the upper lobe. PJP cavitory lesions have been associated with pentamidine use and may increase the risk of pneumothorax. In cavitory PJP, BAL or TBBx are frequently negative as compared to classic PJP; performing both procedures may be necessary to establish a diagnosis. Most of the PJP cavitory lesions will resolve with treatment provided there is no concomitant infection.

Notes:

PV8

AEROMONAS HYDROPHILA NECROTIZING FASCIITIS IN A CIRRHOTIC PATIENT

D Willis, C Zhong, K Roach, A Rao

Learning Objectives: Necrotizing skin and soft tissue infections, including necrotizing fasciitis, are rapidly progressive and often fulminant infections. If not recognized and treated early they can lead to extensive tissue destruction, systemic toxicity and death. Aeromonas hydrophila is a gram negative rod that is more often isolated in aquatic environments. Diarrheal disease is the most common clinical manifestation. Immunocompromised states, including hepatic disease, can lead to more severe extraintestinal manifestations. Reports of wound infections and bacteremia, especially in the pediatric population, are documented. There are scant reported cases of Aeromonas induced necrotizing fasciitis. **Case Presentation:** Our patient was a 58 year-old male with alcoholic cirrhosis and COPD who had been developing dyspnea and worsening recurrent ascites and presented to the emergency department with increasing lethargy and confusion. He also had a slowly healing, painful left-lower extremity cellulitis treated during a recently prior admission with IV vancomycin. On arrival, he was tachypneic at a rate of 28/min. However, he was afebrile, normotensive with a normal heart rate. Initial white blood cell count was 2,000/ μ L. Spontaneous bacterial peritonitis suspected in ED and paracentesis with culture was completed. He was not given antibiotics initially, for unclear reasons. Following admission, he began to rapidly decline. His left-lower extremity lost perfusion, became discolored and cold. Laboratory evaluation showed an elevated lactate at 12.2 mmol/L. Pan-cultures were drawn and the patient was started on empiric antibiotics. Further clinical deterioration led to endotracheal intubation and transfer to intensive care. Skin discoloration migrated superiorly and included his entire left-lower extremity as well as parts of his right-lower extremity and abdomen. Surgical consultation felt that his presentation was consistent with necrotizing fasciitis but surgical intervention at that point was futile. He subsequently expired. His blood, wound and ascitic fluid cultures all grew Aeromonas hydrophila. History given by family did not include water exposure or preceding GI illness. **Summary:** Necrotizing fasciitis must be recognized and managed quickly with definitive surgical intervention. Delay in antibiotics certainly hastened clinical decline in this cirrhotic patient who presented with evidence of systemic inflammation. Although Aeromonas is a rare cause of necrotizing fasciitis, it must be considered in the setting of an immunocompromised patient with rapidly evolving soft tissue disease.

Notes:

PV9

20 YEAR OLD MALE WITH DESCENDING NECROTIZING MEDISTINITIS

A El-Bakush, M Al-Janabi, R Dietrich, M Bowling

LEARNING OBJECTIVES: Descending Necrotizing Mediastinitis DNM is an infection starting in the oropharynx which spreads through the fascial planes to the mediastinum. Predisposing factors include: Obesity, DM, drug use, immunosuppression, recent surgery, and traumatic wounds. It is rare and can be missed when it occurs in a young patient with no risk factors. Mortality is high. We report a young male with no predisposing risk factors who developed DNM after an upper respiratory tract infection.

CASE INFORMATION: 20 year old male with no PMH presented with severe sub sternal chest pain, 8/10, sharp, constant, increased by inspiration, decreased by leaning forward, associated with chills, nausea and vomiting. He had an URTI 6 days before admission and was treated with penicillin for 5 days. No history of smoking, ETOH or illicit drug use. Exam: Diaphoretic in moderate distress. Normal vital signs except for a RR of 22/min. Chest: Decreased breath sounds bilateral lower zones, and absent basally. S1 and S2 with a pericardial friction rub. Labs: WBC 32.5 k/ul, Hb 13.9 g/dl, Plts 220 k/ul, Trop and CKMB negative. ECHO: Small <1 cm pericardial effusion. EKG: Sinus tachycardia, ST elevation in leads II, III, V2-V6. Blood C/S: Negative. Initial CT scan chest: Small pericardial effusion. Diffuse deep soft tissue edema within the lower neck and mediastinum. Moderately large bilateral pleural effusions with mild bilateral pulmonary edema. He was diagnosed as bacterial pericarditis, started on antibiotics. A diagnostic thoracentesis revealed bilateral empyema. A repeat CT chest showed empyema, and deep space infection of the lower neck and mediastinum. He was treated surgically with a VATS procedure and proper antibiotics.

SUMMARY: DNM originating from deep neck infections is rare. It can be caused by: Dental infections, Ludwig's angina, pharyngitis, tonsillitis, parotitis, epiglottitis, and Lemierre syndrom. Symptoms and signs include respiratory distress, fever, chest pain, pleuritic pain, odynophagia, crepitus and edema of the neck or chest, and signs of bacteremia and sepsis. Diagnosis is by CT scan. Diagnosis requires a high index of suspicion. The treatment is adequate surgical drainage and debridement which is transthoracic or trans cervical, plus antibiotics. Our patient is young and without previous medical problems, early recognition was important for treatment and minimizing further complications.

Notes:

PV10

“TOTO, I’VE A FEELING WE’RE NOT IN KANSAS ANYMORE”: MYCOBACTERIUM KANSASII LUNG MASS MASQUERADING AS MALIGNANCY

A Stang, H Nguyen, M Ashraf, P Cook

Objectives: To increase awareness among providers of *Mycobacterium kansasii* presenting as a cavitary lung mass; to review the etiology, pathogenesis, clinical manifestations and management of *M. kansasii* pulmonary disease; and to briefly review cognitive biases in medical decision making

Case: A 51 year old Caucasian female with 7 pack-year smoking history presented to her primary care provider with complaint of night sweats and shortness of breath for the past 6 months. Chest radiography showed a cavitary pneumonia in the right upper lobe; follow-up imaging was advised to exclude an underlying neoplasm. Because both her symptoms and abnormal lung imaging findings persisted after empiric treatment with azithromycin, she was referred to Hematology-Oncology for further evaluation. The patient underwent computed tomography scan of the chest, bronchoscopy and mediastinoscopy with no malignant cells identified, another round of antibiotic therapy, and a trial of corticosteroid therapy. Due to worsening symptoms and size of the cavitary lung lesion, she underwent thoracotomy and right upper lobectomy. Pathologic examination of the resected lung mass revealed no dysplasia or malignancy, but granulomatous inflammation was found. Ultimately, cultures of the lung tissue grew *Mycobacterium kansasii*, and the patient was placed on therapy with rifampin, ethambutol, and isoniazid.

Discussion: Anchoring is a cognitive bias that can affect medical decision making and describes the tendency to rely too heavily upon an initial piece of information (“the anchor”) in making subsequent judgments. In this case, the mention of malignancy in the initial radiographic interpretation may have served as an anchor in terms of further workup. *Mycobacterium kansasii* is a nontuberculous mycobacterium endemic in the south and central United States that can produce disease clinically indistinguishable from pulmonary tuberculosis or even a cavitary malignancy, as in this case. *M. kansasii* isolated from a clinical specimen almost always warrants treatment due to its virulent nature. Untreated, it can lead to death in more than 50% of affected individuals. However, with appropriate therapy, *M. kansasii lung* disease carries a better prognosis than that of many other lung infections due to nontuberculous mycobacteria.

Notes:

PV11

ECTOPIC ADRENOCORTICOTROPIN PRODUCTION IN A PATIENT WITH ADENOCARCINOMA OF LUNG- A CASE REPORT

P Prodduturvar, N Bongu, S Nauman, S Mehra

Learning objective: Ectopic adrenocorticotrophic (ACTH) production causing paraneoplastic Cushing's syndrome has been associated with various malignancies such as small cell lung cancer, carcinoid tumors and medullary carcinoma of thyroid. We present a rare case of a 61year old male with paraneoplastic Cushing's syndrome in association with adenocarcinoma of the lung. **Case Information:** 66 year old male with past medical history significant for 40 years of tobacco abuse and chronic alcoholism presented to our institution after sustaining an accidental fall. His laboratory studies demonstrated hypokalemia with metabolic alkalosis and on further work up chest radiograph showed a lung mass. This was followed by computed tomography scan of his chest, abdomen and pelvis which showed a right lung mass, with multiple enlarged mediastinal lymph nodes with multiple lesions throughout the liver. The largest lesion was in the central aspect of the liver measuring 6.1 x 8.1 cm. Also noted were multiple bony lesions and bilateral adrenal lesions. The biopsy of his liver mass showed metastatic adenocarcinoma, consistent with lung primary, positive for CK7 and TTF1 and negative for CK20 and CDX2. The constellation of findings, including blood chemistry with severe hypokalemia and metabolic alkalosis, presence of anasarca, hypertension and hyperglycemia along with elevated serum ACTH and urine free cortisol in a patient with adenocarcinoma of the lung was consistent with ectopic ACTH Cushing's syndrome. He was started on Ketoconazole and spironolactone, which led to normalization of his serum potassium and metabolic alkalosis. **Summary:** Persistent hypokalemia and metabolic alkalosis in a patient with Lung cancer should prompt consideration for diagnosis of ectopic ACTH secretion and paraneoplastic Cushing's syndrome. Ectopic ACTH production with paraneoplastic Cushing's syndrome associated with adenocarcinoma of the lung is very rare. To the best of our knowledge, we have come across very few cases associated with non-small carcinoma of lung. The treatment of the ectopic ACTH syndrome is surgical excision of the tumor if feasible, thereby removing the source of ACTH. Hypercortisolism can be controlled with steroidogenesis inhibitors such as ketoconazole or metyrapone, which inhibit aldosterone production. Although most commonly seen with small cell lung cancer and other neuroendocrine tumors, clinicians should consider this diagnosis in patients with adenocarcinoma of lung and treat accordingly to improve the outcome of patients.

Notes:

PV12

GUILLIAN BARRE SYNDROME (GBS) CAUSED BY CYTOMEGALOVIRUS (CMV) INFECTION IN RENAL TRANSPLANT RECIPIENT

A.Gurram, P.Jawa

Introduction:

CMV infection causing GBS is not common in solid organ transplant recipients. CMV infection causing GBS is associated with poor prognosis as per anecdotal data. We report a case of GBS in a renal transplant recipient, who came in with complaint of lower extremity weakness and found to have GBS.

Case Description:

A 54 year-old African American male with history significant for renal transplantation in 2013 on immunosuppression, diabetes mellitus, hypertension presented as a transfer from outside hospital for lower extremity weakness. He had suffered from flu like upper respiratory illness about 2 wks ago that got better and 2-3 days after that he developed lower extremity weakness. Physical examination was remarkable for lower extremity weakness, strength was 2-3/5 with intact reflexes. Initial investigation showed hemoglobin 11.7 mg/dl, platelets 315 k/ul, wbc 5.3 k/ul, sodium 143 meq/l, potassium 4.2 meq/l, creatinine 2.3 mg/dl, BUN 55 mg/dl, AST, ALT, bilirubin were within normal limits, CMV viral load of 1212822 copies/ml. MRI of brain, spine was normal. Lumbar puncture showed albuminocytologic dissociation and diagnosis of GBS was made. Neurology consultation did confirm GBS. Later on they admitted to intensive care unit and intubated for a week and respiratory failure has improved. Patient was treated Intravenous immunoglobulins, plasma exchange and intra venous ganciclovir. Repeat CMV viral load was less than 500 after a month. Patient also had kidney biopsy showed focal tubular injury and no rejection. The patient continues to have lower extremity weakness which is improving very slowly.

Discussion: This case report illustrates that renal transplant recipients with lower extremity weakness should be suspected for GBS. Treatment with intravenous immunoglobulins, plasma exchange, ganciclovir may help alleviation of symptoms.

Notes:

PV13

UP AGAINST EXTREMES: THE CHALLENGE OF TREATING THROMBOEMBOLISM IN OBESE PATIENTS AFTER GASTRIC BYPASS

SA Marco, [G Sangah](#), K Parikh

Learning Objectives: With the growing obesity health epidemic, weight loss surgery is becoming more common. Obesity and peri-operative immobilization are both independent risk factors for venous thromboembolism. Despite rising use of novel anticoagulants, there is a paucity of data on the efficacy and safety of therapeutic anticoagulation in this special patient population. **Case Description:** A 36 year old Caucasian female with hypertension, super obesity, recent gastric obesity procedure and intra-operative infrarenal inferior vena cava (IVC) filter placement presented to the emergency room with shortness of breath, right groin pain, and right lower extremity swelling one month after her Roux-en-y gastric bypass. Per her history; she received 40 milligrams Enoxaparin subcutaneously twice daily for 10 days immediately following her gastric bypass as thromboembolism prophylaxis. She had no known history of thromboembolism or clotting disorder. Exam at initial presentation was significant for tachycardia and hypoxia, she had a BMI of 62. Lower extremity Doppler ultrasound revealed a large right Deep Venous Thrombosis (DVT). Computed Tomography Angiogram (CTA) of her chest showed bilateral pulmonary emboli. Echocardiogram showed no evidence of right heart strain. She was started on Rivaroxaban and discharged home on supplemental oxygen. Four days after discharge, she re-presented to our hospital for worsening shortness of breath and persistent right groin pain. She was hemodynamically stable, without increased oxygen requirement. Electrocardiogram showed sinus rhythm. Chemical profile and complete blood count profile were unremarkable. Repeat CTA was not done out of concern for unnecessary radiation. Heparin drip was initiated and a literature review was undertaken to evaluate the appropriateness of novel anticoagulants for extreme obesity and gastric obesity procedure. The decision was made to initiate Warfarin therapy with close monitoring. Patient was discharged 4 days after Warfarin initiation with therapeutic INR. At 8 week follow up, INR remained stable. **Summary:** Our case highlights a rising challenge in medicine; the need for physicians to extrapolate previously published data to extremes of weight, all in the setting of known alterations in absorption after gastric bypass. Ultimately, more studies are needed to fully determine the safety, effectiveness and pharmacokinetics of anticoagulant dosing for therapeutic anticoagulation in this special patient population.

Notes:

PV14

AN UNUSUAL CAUSE FOR GASTROINTESTINAL BLEEDING

[ZJ Iqbal](#), S Abid, E Ali

Learning Objectives: Blue Rubber Bleb Nevus Syndrome is an exceedingly rare case that was found in a patient at our institution and it does not normally present for the first time in older individuals. It is more commonly seen in younger individuals with chronic iron deficiency anemia. Ours is a case of bright red blood per rectum presenting for the first time in an elderly individual and how the case was managed. The genetics behind the syndrome will also be explored.

Case Information: A 76-year-old Caucasian man was admitted with 4 episodes of painless bright red blood per rectum in 1 day. His past medical history included stroke and atrial fibrillation. He was taking warfarin without a history of prior GI bleeding. He did not have a family history of GI bleeding or cancer. On physical examination, his pulse was irregular and his blood pressure was within normal limits. There were small, scattered blue papules present on his legs and trunk which were raised and compressible. He did not have external hemorrhoids. The remainder of his examination was unremarkable. On admission, his hemoglobin was 13.7 g/dL and his INR was 2.9. His warfarin was discontinued and 2 units of fresh frozen plasma and vitamin K were administered. His rectal bleeding improved within the next 24 hours. A colonoscopy performed the next day showed non-bleeding diverticulosis in the descending and sigmoid colon. There were several bluish vascular malformations of different sizes in the left colon, which were not actively bleeding. His cutaneous and intestinal lesions were consistent with the diagnosis of Blue Rubber Bleb Nevus Syndrome. No further investigation or treatment was planned as per the patient's decision.

Summary: This is a rare disorder consisting of gastrointestinal bleeding, cutaneous vascular nevi, and intestinal lesions. Venous malformations are located on the trunk and arms. A common presentation is a lower GI bleed and iron deficiency anemia in a younger patient. We report this syndrome discovered in an older male after an acute GI bleed. Its characteristic lesions clinically diagnose blue Rubber Bleb Nevus Syndrome. It can be inherited sporadically but autosomal dominant inheritance has been reported.

Notes:

PV15

A CASE OF SEROLOGY NEGATIVE NEUROCYSTICERCOSIS IN THE UNITED STATES

J Emberger, H Nguyen, A Stang, MS Ashraf

Objective: To review the etiology, epidemiology, clinical manifestation and management of neurocysticercosis.

Case: 23 year old Mexican male with no significant PMH who presented with sudden onset severe headache, neck pain, nausea and vomiting. Patient also reported an 8 pound weight loss in 4 months, night sweats, chills and blurry vision. Patient was a migrant farm worker from Mexico who moved to the United States 6 months prior. His mental status had declined at home prior to presenting to clinic. He was subsequently sent to the ED and was empirically started on Vancomycin. He developed hypotension and required intubation. Head CT showed mild hydrocephalus. External CSF ventricular drain was placed and the patient was started on Decadron to reduce intracranial swelling. Post procedure, MRI head revealed a cystic lesion in the fourth ventricle extending into the dorsal subarachnoid space causing obstructive hydrocephalus and punctate calcifications in the supratentorial brain suggestive of neurocysticercosis. Cysticercosis IgG serum antibody was negative. External ventricular drain was replaced with a VP shunt. Patient self-extubated and mental status improved from admission. Shortly after shunt placement, the patient underwent posterior fossa craniotomy with excision of the cystic lesion. Pathology findings were consistent with cysticerci infection from taenia solium. As there was concern for racemose neurocysticercosis with extension of the cystic lesion into the arachnoid space, patient was treated with a 4 week course of Albendazole and Prednisone. **Summary:** Neurocysticercosis is a parasitic infection that is acquired through the ingestion of eggs produced by the *Taenia solium* tapeworm. Humans can be hosts to both the adult tapeworm and intermediate hosts to the cysticerci larvae. Neurocysticercosis infection is a significant cause of epilepsy throughout the world. Previously, this infection was mostly limited to endemic countries in Latin America, southern Asia, sub-Saharan Africa and India but incidence in the United States is rising. Diagnosis is based on brain imaging and serology. However, sensitivity is 50% to 60% in patients with one intracranial cysticercus and poor in patients with calcified cysticerci. Treatment is based on the extent and location of infection in the brain. Obstructive lesions require surgery. Lesions extending into the subarachnoid space will require extended courses of treatment with anti-parasitic agents.

Notes:

PV16

PARALYZED: THE TICK BENEATH

M. Christianto, R Dietrich

LEARNING OBJECTIVES:

1. To recognize a rare case of acute complete paralysis
2. To differentiate tick paralysis from other causes of bilateral paralysis

CASE INFORMATION:

A 72-year old male presented with a chief complaint of acute weakness. The symptoms started several hours prior to presentation in the emergency department, affecting all four extremities, progressively getting worse, accompanied by brief visual hallucinations and brief near-loss of consciousness. No other symptoms noted. Vital signs were within normal limits. Physical examination showed almost complete paralysis of all four extremities, cranial nerves functions were intact, sensorium were intact, and there was no sensory deficit. Initial search of the patient's body did not show any abnormal findings.

Blood sugar, complete blood count, serum electrolytes, renal function, and liver function tests were all within normal limits.

CT scan and MRI of the head did not show any acute stroke. MRI of the cervical spine did not show significant spinal canal compromise.

Code stroke was initially called but tissue plasminogen activator (tPA) was not given due to the atypical presentation. Patient was brought to the medical ICU due to fear of respiratory compromise. Repeat body search in the medical ICU showed a finding of 1 tick beneath the scrotum and 2 ticks latching on pubic hairs. The ticks were removed carefully and patient improved significantly with 50% strength coming back within 5-10 minutes.

Patient was observed in the medical ICU overnight and on the next day his strength was approximately 90% restored and patient was discharged home.

SUMMARY:

A remarkable case of tick paralysis presenting with bilateral acute generalized progressive weakness masquerading as possible stroke, especially posterior circulation stroke, and also to differentiate this from other less common cause of bilateral acute paralysis such as spinal cord lesions and Guillain Barre Syndrome. It is important for the medical practitioners to recognize the atypical presentation to avoid giving unnecessary interventions that are high risk, such as tPA infusion, Intravenous Immunoglobulin, or plasmapheresis.

Notes:

PV17

FIRM FACIAL PAPULES

EL Stewart, C Phillips, K Park

Learning Objectives: Facial papules have a wide differential and osteoma cutis is not commonly encountered. This case highlights some salient features.

Case Information: A 63 year old African American woman with history of pulmonary and ocular sarcoidosis, presented to Dermatology clinic for evaluation of bumps on her bilateral cheeks and forehead that had been present for several years. She had multiple small, firm, papules on cheeks and forehead. A punch biopsy was performed of one papule which revealed a rock-hard central area that was difficult to remove. On pathology, areas of bony trabeculae, surrounded by fatty marrow structures were evident, consistent with osteoma cutis. Osteoma cutis can have several different presentations. One form is associated with Albright hereditary osteodystrophy, and has associated findings of pseudohypoparathyroidism, brachydactyly, short stature, obesity and round facies. Our patient did not have syndromic features and so the diagnosis of miliary osteomas of the face was made. In patients with cutaneous ossification, serum calcium, serum phosphate, parathyroid hormone (PTH) and vitamin D3 levels should be checked to evaluate for systemic metabolic derangements.

Summary: This is an interesting case of a patient with an autoimmune disease presenting with a dermatologic complaint and endocrine abnormalities.

Notes:

PV18

EARLY ONSET OSTEOARTHRITIS IN A NOVEL KINDRED WITH AUTOSOMAL DOMINANT HYPOCALCEMIA

M Javaid, JA Sutter, BE Ramirez, FJ Cook

Learning Objectives: Autosomal dominant hypocalcemia (ADH) is a rare disorder associated with variable clinical severity. This disorder is caused by activating mutations in the calcium sensing receptor (CaSR) gene. ADH has also been associated with premature osteoarthritis of the knees and short stature. We present a kindred with ADH in which the index case has a CaSR mutation previously reported in one other family. In our kindred, premature small joint osteoarthritis is also noted in multiple generations.

Case Information: Our index case (generation IV), a 15 year old female followed in pediatric endocrinology clinic, presented in her neonatal period with severe hypocalcemia resulting in seizures and tetany. She took calcium and calcitriol until age 3. She remained asymptomatic off these medications until age 11, when calcium and calcitriol were resumed due to moderate hypocalcemia. Her CaSR gene analysis revealed a missense mutation causing the amino acid change p.Glu767Lys at position 2299 in Exon 7. This mutation co-segregates with hypocalcemia in a previously reported kindred and has been associated with an increase in calcium affinity of the mutant CaSR. In the adult endocrinology clinic, the 66 year old grandmother of the index patient (generation II) presented for continued treatment of hypocalcemia, hyperphosphatemia, hypoparathyroidism, and hypercalciuria. She was diagnosed with mildly symptomatic hypocalcemia in her fourth decade, treated with calcium and calcitriol. She has short stature (1.47 meters) and striking deformities consistent with osteoarthritis of the small joints of both hands with onset in the fourth decade. Three members of generation I and at least one member of generation III (also seen in our clinic) also suffer from hypocalcemia and early onset osteoarthritis in the hands. **Summary:** More than 70 activating mutations in the CaSR have been previously reported in the literature, with clinical features ranging from asymptomatic to severe hypocalcemia with or without Bartter's syndrome. The kindred we describe adds to this body of literature, and demonstrates an apparent association of a CaSR gene mutation with premature osteoarthritis of the small joints in the hands. As the CaSR is expressed widely in various organs of the body, including cartilage and bone, more studies in patients with ADH may elucidate the role of the CaSR in the health of the skeleton and joints.

Notes:

PV19

OMEPRAZOLE INDUCED SYMPTOMATIC HYPOCALCEMIA IN A PATIENT WITH SILENT HYPOPARATHYROIDISM

M Javaid, C Houston, J Giordano, RJ Tanenberg

Introduction: Proton pump inhibitors (PPI) are associated with variable absorption of calcium supplements especially Calcium Carbonate. PPIs can affect absorption of dietary calcium by altering gastric pH. We report a patient whose hypoparathyroidism unmasked when he developed significant hypocalcemia after initiation of Omeprazole even though he was not on any Calcium supplements. **Case presentation:** A 54 y/o White Male was sent to Emergency Department from his primary care provider office for critically low Calcium level. His past medical history was significant for Type 2 Diabetes Mellitus, Hypertension, Gastro esophageal Reflux disease (GERD), Renal stones, remote history of Hyperparathyroidism and parathyroidectomy of one of the glands 15 years ago due to recurrent renal stones (35 stones). He reported taking Calcium supplement in the form of Tums for 4-6 months after his surgery and then he stopped it. He remained symptom free for all these years until last year, when he started experiencing sporadic numbness and tingling of face and arms, which responded well to Tums. He presented to his doctor's office with flu like symptoms, shortness of breath, left arm weakness, and numbness and tingling of his arms and face. His home medications were significant for Omeprazole DR 40 mg daily (added a year ago) and intermittent use of Tums. He reported taking no other medications. Physical examination was positive for Chvostek sign. His lab work was as follows: Calcium 6.6 mg/dl, ionized Calcium 3.1 mg/dl, Albumin 4.1 g/dl, Magnesium 1.8 mg/dl and inappropriately normal iPTH 16.6 pg/dl. 12- Lead EKG revealed prolong QTc interval (>500mS). He responded well to IV calcium, oral Calcium Carbonate and IV Magnesium Sulfate. During his hospital stay, Omeprazole was discontinued and he was started on Calcium Citrate 630 mg, 3 tablets three times a day along with Calcitriol 0.25 mcg every day. He remained asymptomatic with stable calcium level and off Omeprazole. **Conclusion:** We suggest that normal dietary Calcium absorption can be disturbed when PPIs are added in patients' with borderline hypoparathyroidism and may manifest with symptomatic hypocalcemia which can be detrimental if not caught timely. Calcium Citrate can be a better choice if PPI's are clinically indicated in a patient with history of Parathyroidectomy.

Notes:

PV20

ACTH-SECRETING PHEOCHROMOCYTOMA

MS Kalia-Reynolds, J Giordano, I Khanna, C Houston

Introduction: We present a case of ACTH-producing pheochromocytoma, which is an extremely rare but important cause of ectopic Cushing's syndrome.

Case Description: A 56 year old female presented with 3 years of worsening diabetes mellitus, hypertension, facial swelling and irritability. Her blood pressure was 160/94, pulse was 98, and BMI was 36. She had typical Cushingoid features of moon facies, facial plethora, hirsutism, cervicodorsal fat deposition and purple abdominal striae. Labs revealed sodium 142 mEq/L, potassium 2.7 mEq/L, random cortisol 101.9 ug/dL, ACTH 299 pg/mL and urinary free cortisol of 11,870 mcg/24 hrs. She was hospitalized and received potassium, spironolactone and ketoconazole to treat her hypercortisolemia. Pituitary MRI and other pituitary hormone levels were normal. High-dose dexamethasone suppression test resulted in a 2.7% decrease in serum cortisol. These results suggested ectopic ACTH secretion. Abdominal CT revealed a 5.6 cm heterogeneous right adrenal mass. Serum aldosterone, renin, testosterone and DHEAS were normal. Urine and plasma metanephrines were greater than 4 times the upper limit of normal. Right adrenalectomy was performed following initiation of alpha and beta blockade. Pathology confirmed pheochromocytoma with positive ACTH staining. Her Cushingoid features rapidly improved; her diabetes mellitus, hypertension and hypokalemia resolved; and her plasma metanephrines normalized. Glucocorticoid replacement was tapered with eventual recovery of her HPA axis and sustained normalization of her ACTH and cortisol.

Discussion: Pheochromocytoma is a rare source of ectopic ACTH production. This condition has protean manifestations and clinical features of catecholamine excess may be obscured. Recognition of the patient's pheochromocytoma was critical to implementation of preoperative measures to avert hypertensive crisis. Our case underscores the need for clinicians to recognize the possibility of pheochromocytoma in patients with Cushing's syndrome.

Notes:

PV21

SEVERE MULTIORGAN FAILURE WITHIN HOURS AFTER HIGH DOSE CARFILZOMIB THERAPY FOR REFRACTORY MULTIPLE MYELOMA

A El-Bakush, M Al-Janabi, A Ismail, R Dietrich

LEARNING OBJECTIVES: Carfilzomib is a new selective proteasome inhibitor that was approved in 2012 for treatment of relapsed and refractory multiple myeloma. We present a patient who presented with severe multiorgan failure less than 24 hours after a high dose Carfilzomib infusion.

CASE: A 73 year old male with Multiple Myeloma for 5 years, whose disease has progressed despite treatment with multiple chemotherapy regimens presented with severe shortness of breath within 12 hours after his last Carfilzomib infusion. Upon arrival he was hypotensive, hypoxic, with tachypnea and tachycardia. He was intubated and started on vasopressors. His labs showed: Cr elevation from 1 to 3.3 in 24 hours, Hb of 7.1, potassium 5.8, phosphorus 7.9, lactic acid of 5.1, BNP of 20000, calcium of 7.1, uric acid of 12.2, WBC 14 with 21-30% bands, Troponin of 224, ABG showed metabolic and respiratory acidosis, CXR showed diffuse bilateral infiltrates consistent with pulmonary edema. An Echo showed an EF of 25-30% which has decreased from 55% two months prior. He was diagnosed with shock, severe CHF, pulmonary edema, myocardial ischemia, acute renal failure, and tumor lysis syndrome from chemotherapy. He was treated appropriately in the MICU initially and later transferred to CICU but unfortunately he died the next day.

SUMMARY: Carfilzomib can cause: cardiac failure including congestive heart failure, pulmonary edema, and ejection fraction decrease in about 7% of the patients, renal failure in 9% of the patients, life threatening multiorgan failure in less than 1% of the patients. It is also known to cause respiratory failure, and precipitate tumor lysis syndrome. Our patient developed multiorgan failure secondary to Carfilzomib treatment and died in a short period of time despite medical treatment

Notes:

PV22

STROKE IN PATIENT WITH JANUS KINASE 2 POSITIVE ESSENTIAL THROMBOCYTHEMIA

CP Craig, S Jayananda, D Liles

Learning Objectives: Discuss major complications and diagnostic criteria of Essential Thrombocythemia (ET). Highlight the Janus Kinase 2 (JAK2) mutation and its clinical importance in ET. Discuss risk stratification of patients with ET and the decision to start platelet lowering therapy. **Case Information:** 57 year old female with hypertension presented with headache, right upper extremity weakness and right homonymous superior quadrantanopia. MRI brain showed acute left PCA infarction, right posterior inferior cerebellar artery infarction and small punctuate infarction of right occipital pole. Platelet count on admission was 792,000/uL and increased to 1.6 million/uL during her hospitalization. JAK2 mutation was positive in the setting of negative BCR-ABL1 mutation and normal erythropoetin level. Bone marrow biopsy showed hyper cellular marrow with marked thrombocytosis and loose megakaryocytic clustering. The patient was started on aspirin 81 mg and hydroxyurea 1000 mg daily. Upon chart review, the patient had documented platelet counts of 482,000/uL one year prior and 683,000/uL one month prior to her admission for stroke. World Health Organization classification for ET includes platelet count greater than 450,000/uL, bone marrow biopsy with proliferation of megakaryocytic lineage, acquired mutation or clonal marker, and no evidence for reactive thrombocytosis. High risk patients are classified as previous thrombosis or bleeding event, greater than 60 years of age, and platelet count greater than 1.5 million/uL. Treatment standard of care of high risk patients includes aspirin and platelet reducing agents like hydroxyurea or anagrelide. Whether the JAK2 mutation should be part of the risk stratification remains an area of uncertainty. **Summary:** ET is a myeloproliferative disorder characterized by persistent thrombocytosis and increased risk for thrombosis and hemorrhage. Acquired JAK2 mutation is found in 30-50% of the patients and may signify a more aggressive disease course. Prior to admission, our patient would have fallen into a low risk category. A significant thrombotic event in a low risk, JAK2 positive patient raises the question if JAK2 status should hold a more significant role in risk stratification. We posit that early recognition of thrombocytosis and determination of JAK2 status could lead more appropriate risk stratification and therefore decreased morbidity.

Notes:

PV23

KAPOSI'S SARCOMA IN AN HIV NEGATIVE CENTRAL AMERICAN PATIENT: A CASE REPORT

P Chae, L Salmon, P Atluri

Background: Kaposi's Sarcoma (KS) is a rare tumor known to affect certain populations including young African adult males, patients receiving immunosuppressive therapy, prepubescent children and elderly men of Italian Eastern European Jewish ancestry and in persons with AIDS. It is the most common AIDS associated cancer in the United States. Several variants of KS exist including Classic, Endemic, Immunosuppressive or transplant associated and Epidemic or AIDS associated. Median survival varies from months to decades depending on the variant. **Case Presentation:** A 72 YO M from Honduras visiting his family in North Carolina presents to the Emergency Room in September 2014 from a primary care clinic for worsening left hand swelling and drainage. He states he carries a known diagnosis of KS of his left hand treated in Honduras in 2013. He had been treated at a cancer center in Honduras from 8/2013 to 12/2013 with 6 treatments of paclitaxel and cisplatin every 21 days and was subsequently monitored. Pertinent reported history includes having tested negative multiple times for HIV in Honduras, no travel outside of Honduras except to the United States, heterosexual male and no prior history of immunosuppressive therapy. **Clinical Course:** The patient is admitted to the Hematology/Oncology service where he is started on IV antibiotics for presumed cellulitis. Dermatology is consulted for biopsy of the lesion for concern of recurrent KS. The shave biopsy confirms diagnosis of Kaposi's Sarcoma, nodular stage and the neoplastic cells also test positive for HHV-8 by immunohistochemical staining. Additionally, he also tests negative for HIV, has normal CD4 and CMV viral load <500. Due to extent of disease, he is initiated on paclitaxel 100mg/m² q2 weeks and continued on treatment as an outpatient at Leo Jenkins Cancer Center. He has had remarkable response with significant decrease in size of the lesions and has had some return of function in his left hand. He has tolerated chemotherapy well to date. **Discussion:** We present an interesting case of endemic KS in a patient who does not possess the usual risk factors for KS including residing in an endemic area. This is one of the only if not only known reported endemic case of a person native to Central America. Further studies are needed to investigate optimal therapy in these patients as choice systemic chemotherapy is largely based on regimens used for AIDS related KS.

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INTRACRANIAL PLASMABLASTIC LYMPHOMA REVEALING ACQUIRED IMMUNE DEFICIENCY SYNDROME

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Learning Objectives: Plasmablastic lymphoma (PBL) is an aggressive, Epstein Barr Virus (EBV) associated sub-type of diffuse large B cell lymphoma which occurs almost exclusively in the setting of immunodeficiency, and especially with Human Immunodeficiency Virus (HIV) infection. PBL has a predilection for extra nodal involvement, particularly the oral cavity and intracranial involvement at presentation is rare. We report a case of Intracranial PBL as presenting feature of AIDS in a young man.

Case Information: A 49 year old Caucasian male without medical history presented to his primary care physicians office complaining of an enlarging head mass of one month duration. The patient denied any constitutional, visual or other neurologic symptoms. Fundoscopic exam showed sharp vessels without papilledema. The patient was thought to have a well-organized scalp hematoma and Computer Tomography (CT) scan of the head was done before planned in-office percutaneous evacuation. CT head revealed a 4.1 x 6.3 x 6.0 centimeter hyper dense mass with additional lytic skull lesions. He was started on decadron and referred for outpatient biopsy. Twelve days later he was found unresponsive at home and was taken to the emergency room. Repeat CT Head showed interval growth to 5.2 x 8.7 x 6.0 centimeters causing 16 millimeter subfalcine herniation. A second intracranial mass was identified, as was a spiculated lung nodule and a vesicular rash on his right arm, upper right chest, and back. Laboratory analysis was significant for leukocytosis and hypercalcemia. He was admitted with suspected brain metastases of unknown primary and recurrent shingles. Emergent craniotomy with resection was performed. Pathology revealed an EBV+ high grade lymphoma. Tumor markers were consistent with diagnosis of intracranial Plasmablastic lymphoma. Western blot confirmed HIV positivity. CD4 count was zero with an HIV viral load of 72,606. He was started on Highly Active Antiretroviral therapy (HAART) along with an intensive chemotherapy regimen. One year later, he has completed 5 cycles of chemotherapy with excellent response and his HIV Viral load is less than 40.

Summary: This case illustrates the diagnostic and therapeutic challenges associated with PBL. PBL is associated with HIV in 80-90% of cases and may be the presenting manifestation of HIV infection as in this case. Timely recognition of this entity along with initiation of combination chemotherapy with HAART is associated with improved outcome.

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SPONTANEOUS REMISSION OF ACUTE MYELOID LEUKEMIA: COULD IT BE THE ESOMEPRAZOLE?

ER Bonner, RN Friend

Ulcer prophylaxis is initiated for many hospitalized patients without much thought concerning the effect these medications may have on a disease process. We present a case of pathologically confirmed acute myeloid leukemia (AML) that was completely changed by a daily esomeprazole tablet.

Case: A 67 year old woman in previously good health, presented to Vidant from an outside hospital in April 2014. She was admitted with complaints of fatigue and malaise. On admission, her white blood cell (WBC) count was greater than 40,000/uL. Therefore, she was transferred to our Hematology service. Upon arrival, her WBC count was confirmed to be 47,200/uL with a differential of 90% monocytes. A bone marrow biopsy and aspirate analysis revealed an increased population of blasts with monocytic immunophenotype and supported the diagnosis of AML with monocytic differentiation. The only new medication she received over the next two days was esomeprazole. Two days after admission, her WBC count started to decrease. A repeat bone marrow biopsy was performed because she was leukopenic with a WBC count of 2,700/uL. Results from the repeat biopsy revealed no significant increase in blasts, monocytes, or abnormal lymphoid population. Her prophylactic medications were discontinued at discharge. In July, she presented with complaints of dyspnea. She was found to have a leukocytosis of 45,900/uL. Flow cytometry of her peripheral blood revealed an extensive population of immature cells with myelomonocytic immunophenotype. A significant population of blasts with myelomonocytic immunophenotype consistent with AML with monocytic differentiation was confirmed in the bone marrow. This biopsy review was similar to her initial pathology. She has since been treated with induction and consolidative chemotherapy. She remains without evidence of disease recurrence. **Summary:** A short-term remission of AML is exceedingly rare. Literature review confirms that the etiology of a spontaneous remission is unclear. The leading hypothesis is associated with infection, blood transfusion or granulocyte colony-stimulating factor therapy, which triggers an immune response to exert an anti-leukemic effect. Our patient had no signs of infection, did not receive any transfusions, and the only medication she received, with any known potential for leukopenia, is esomeprazole. Exposures to new medications, even short-term prophylaxis, should always be considered when otherwise unexplainable outcomes present themselves.

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ACUTE MYELOID LEUKEMIA WITH CRYPTIC *DEK-NUP214* FUSION PRESENTING AS PYODERMA GANGRENOSUM.

E Levin, H Lin, P Chae, P Papenhausen, B Dangott, R Juskevicius

A skin lesion may at times be the key to reaching a diagnosis. PG is associated with several disease processes including IBD and AML. We present a case of PG leading to the diagnosis of a rare form of AML.

A 33-year-old AAF presented to an outside hospital with abdominal pain and was sent home with a GI cocktail. The patient then returned with continued abdominal pain and a lesion on her right arm. The lesion was initially thought to be necrotizing fasciitis, and debridement was performed. Dermatology consultation and pathology confirmed PG. IBD was initially considered given the patient's presenting complaint and a CT abdomen/pelvis showing bowel wall thickening. The peripheral blood smear review showed blasts and oncology was consulted regarding concerns for AML. BM biopsies showed increased blasts with an immature myeloid immunophenotype. The proportion of blasts did not reach 20% and the cytogenetic and molecular testing including *BCR-ABL*, *FLT3*, *t(8;21)*, *t(15;17)*, *NPM1*, *MPL* and MDS FISH panel were negative. Thus, the features were not definitively diagnostic of AML and, given the patient's acute illness, a reactive cause of increased blasts was considered as a possibility. However, further molecular cytogenetic studies using a high resolution SNP microarray analysis revealed a clonal change characterized by a small interstitial deletion which included part of the *DEK* gene at 6p22.3 and a small interstitial duplication including part of the *NUP214* gene at 9q34.13, suggesting a cryptic fusion of the genes. The *DEK-NUP214* fusion is seen in AML with *t(6;9)* and is associated MDS. Interphase FISH, using a *NUP214* targeted BAC, confirmed that the duplication was not at the site of the gene in support of a cryptic fusion. Given the clinical context and morphologic findings, the array and related FISH results were considered supportive of AML. Subsequent biopsy confirmed the diagnosis and chemotherapy was initiated.

In summary, we emphasize the need to perform a thorough hematologic investigation in a patient who presents with a lesion considered to be PG. In our case this led to the initial consideration of AML, however due to low blast counts and normal karyotype/ molecular findings, the SNP array testing was required to provide additional support for the definitive diagnosis of this rare form of AML.

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CAN IT STILL BE STILL'S? THE DIFFICULT ROAD TO A DIAGNOSIS.

E Levin, V Dhingra, S DeMarco

Adult-onset Still's disease (AOSD) is a rare systemic inflammatory disorder which is difficult to diagnose as it is a diagnosis of exclusion. The diagnosis is a clinical one with many classification criteria proposed but as of yet none definitively accepted. The road to the diagnosis is difficult as malignancy, other rheumatologic disease, and infection must be excluded. A 55 year old AAF with a history of DM, HTN, presents with complaints of joint pain and fever for 6 months. She initially noted a rash on her arm with a subjective fever and chills. She went to the ED where her temperature was noted to be 101 F and it was suspected that she was having an allergic reaction to metformin that was recently initiated. The metformin was discontinued yet the patient continued to have intermittent fever for several months and a similar rash. A couple of months after the initial rash, the patient began to experience migratory joint pain. Initially, the joint pain was in bilateral wrists then traveled to her right shoulder and bilateral ankles. While experiencing joint pain, the patient continued to have intermittent fever and rash. Patient received a course of doxycycline for 14 days. The patient was admitted for a work-up of joint pain; on the admission physical exam, patient was unable to ambulate due to pain in bilateral knees. Other significant physical exam findings included: temperature 102.9 F, swelling in bilateral wrists left more than right, tender to touch in the wrists, no warmth over joint, pre-auricular small mobile lymph node was palpated. Pertinent labs included ANA <40, Lyme negative, EBV negative, CMV IgG 4.82, CMV IgM negative, ferritin >8250, RF <10, Anti-CCP negative, WBC 13.8, AST 46, ALT 27, Alk phos 60. CT chest showed mild hilar and bilateral axillary lymphadenopathy. CT abdomen/pelvis indicated inguinal lymphadenopathy. Blood cultures and urine culture were negative. Infectious and malignant etiology was not thought likely. At this point AOSD was considered as the patient met 5 criteria including 2 major criteria per the Yamaguchi criteria. She was initiated on steroids and NSAIDs and has been followed up in clinic. In a patient with FOU it is important to consider AOSD as part of the differential. However, the work-up still remains difficult and expensive as it requires the exclusion of malignancy and infection. In our case, the typical evanescent skin rash, elevated ferritin, arthralgia, leukocytosis, lymphadenopathy, negative RF make the diagnosis still likely to be AOSD.

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BASAL CELL CARCINOMA MASQUERADING AS A MELANOMA MASQUERADING AS AN ACTINIC KERATOSIS

KR Liner, R Harris, CM Phillips

Learning Objectives: This case demonstrates the importance of a thorough physical exam in patients with a history of skin cancer, extensive sun exposure, and the presence of numerous skin lesions. While the physical exam may be challenging in the detection of new skin cancers, recognizing that basal cell carcinomas may masquerade as actinic keratosis or with melanoma-like features is necessary to prevent delay of proper treatment.

Case Information: An 82-year-old male with a history of multiple nonmelanoma skin cancers and severe sun damage presented to our clinic for a regularly scheduled skin check. On initial exam, it appeared he had a small basal cell carcinoma on his chest, numerous actinic keratoses on his face, scalp, and arms, and hundreds of seborrheic keratoses on his chest and back. As the clinician was palpating the actinic keratoses prior to cryotherapy, he removed a small portion of a crust from a suspected actinic keratosis that revealed an atypical area underneath. Complete removal of the crust exposed a nodular lesion with features concerning for melanoma including deep blue pigmentation and a blue-white veil on dermoscopy. A deep shave biopsy was performed and the lesion was pathologically consistent with a basal cell carcinoma.

Summary: A good physical exam may be challenging in patients who present with previous skin cancers, sun damaged skin, and numerous actinic and seborrheic keratoses. Basal cell carcinomas share similar features as melanomas and may also mimic more benign lesions such as actinic keratoses. Specifically, this case identified a basal cell carcinoma that had dermoscopic features of a melanoma and an initial clinical appearance mimicking actinic keratosis.

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THE WRATH OF A SICKLE CELL PAIN CRISIS: IDENTIFYING MULTIORGAN DYSFUNCTION EARLY IN A SICKLE CELL PATIENT

K Parikh, T Nguyen, A Ismail, Z Rehman

Learning objective: While a vaso-occlusive crisis and hemolysis are hallmarks of sickle cell disease, multiorgan dysfunction syndrome (MODS) is a rare but fatal complication of sickle cell management that requires prompt attention.

Case summary: A 40 year old African-American male, with known homozygous sickle cell anemia, was admitted to the hospital for a vaso-occlusive pain episode. Despite conventional management with intravenous (IV) hydration, supplemental oxygen and analgesics, MODS ensued over the next 24 hours, requiring critical care unit management. The patient had acute kidney injury, fulminant hepatic injury, nonfocal encephalopathy and type II myocardial infarction. He was emergently dialyzed for hyperkalemia and exchange transfused to reach a hemoglobin S of 19 % (goal is less than 30%). Infection and disseminated intravascular coagulation (DIC) studies were negative. The prompt initiation of dialysis and exchange transfusion resulted in dramatic improvements not only clinically but also in his kidney and liver function through serial measurements of his comprehensive metabolic profile and increase in urine output. We were able to prevent the patient from committing to permanent dialysis through our early diagnosis and prompt management of the MODS.

Conclusion: Sickle cell anemia is the most common genetic disease. During the early stages, sickle cell crisis can be managed with IV hydration, supplemental oxygen and analgesics. MODS is defined as failure of two or more organs, according to the Acute Physiology and Chronic Health Evaluation II (APACHE II) criteria; and the onset of organ failure can be associated with fever, rapid fall in hematocrit and platelet count, nonfocal encephalopathy and rhabdomyolysis. It is imperative that physicians identify it early, as it can have a high mortality of 20%. However, in order to manage our patient appropriately, other etiologies were ruled out, such as sepsis and DIC, as infection can be a contributing source to the increased morbidity and mortality of patients with sickle cell anemia. MODS is a rare and deadly complication of a vaso-occlusive crisis and it is important that physicians broaden their differential and initiate treatment immediately when assessing patients that are admitted simply for the conventional management of a "pain crisis".

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FREE OPEN ACCESS MEDICAL SIMULATION

M Ritchie, A Hidalgo, T Pancoast

As technology continues to advance, we are able to utilize it to enhance our medical education. In 2012, FOAM (Free Open Access Medical education) was coined and we have had a huge influx in FOAM content. Residents and Attendings are now using twitter and RSS feeds to further develop their knowledge. One of the best aspects of FOAM is in its name; free. FOAM is more than websites, podcasts and tweets, it is the belief that medicine can be better working together, to share our knowledge rather than sell it. It is with this in mind that we created an open-access simulation program.

Simulation is a great way to experience difficult situations and procedures. Repetition and muscle memory improves competency and decreases complications. There has been a significant increase in simulation fellowships and programs using simulation. However, it can be difficult to get access to these expensive programs and mannequins.

Pirate Sim (www.4critcare.com) is an easy-to-use simulation program that gives residents and training programs access to free simulation. Standardizing scenarios is important for consistency and can also save time and resources. These cases are designed to be run in a simulation environment, but can be viewed from personal computers by residents to assess their knowledge and review difficult cases. There are options for adult, pediatric, trauma, critical care and student cases. Participants can view the case's HPI, labs, EKGs, and images in real time on a television or computer and can toggle between them. The proctor will have access to the patient course, which will provide instructions on case flow. With each case, there is a discussion page with key points and critical actions.

Pirate Sim is a user-friendly simulation program that is free. Also, there are opportunities to be an author and create more cases. Pirate Sim can be extremely useful for residents and programs with or without access to expensive simulation mannequins and software.

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