ACP® American College of Physicians
Leading Internal Medicine, Improving Lives | Hawaii Chapter

2024 Hawai'i Chapter

Scientific Meeting

Honolulu Country Club

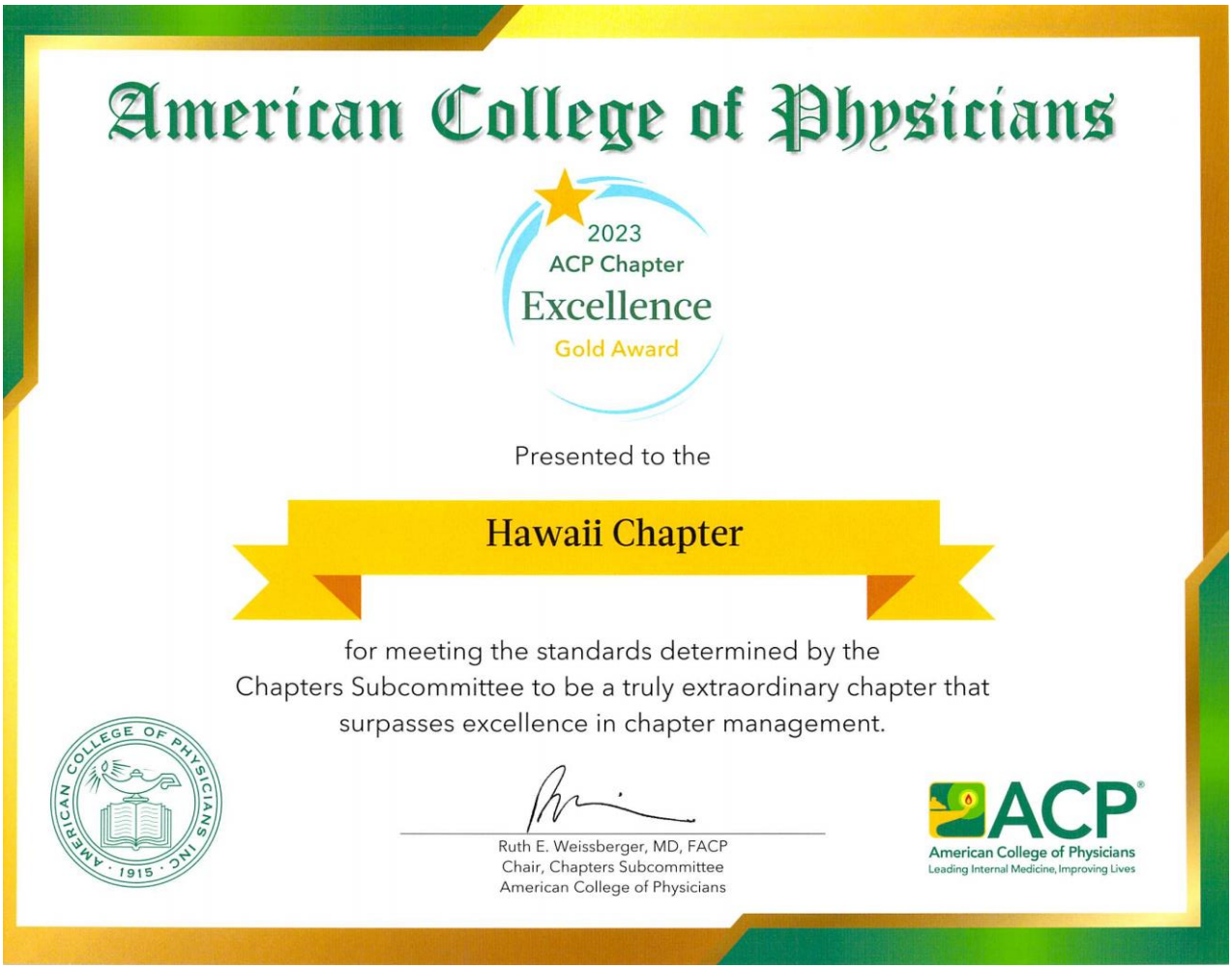
Saturday, March 9, 2024

In-person/Virtual

❖ *This activity has been designated for 6.0 CME credits and 6.0 MOC points*

Meet the New Governor-elect Designee - Hawaii

Congratulations to the Hawaii Chapter Governor-elect Designee (GED), Ryon K. Nakasone, MD, MBA, FACP. Our new GED will complete a year of training as a Governor-elect and then will start their four-year term as Governor in the Spring of 2025. As Governor, Dr. Nakasone will serve as the official representative of the College for our Chapter, providing a link between members at the local level and leadership at the national level. In the meantime, Dr. Nakasone will be working closely with Dr. Evans (the current governor) and College staff to learn about the College and their duties as Governor. To learn more about the new GED, read their bio below.



We are pleased to announce that our chapter is in receipt of the Gold Level of the 2023 Chapter Excellence Award! The award recognizes truly extraordinary chapters that surpass excellence in chapter management. We are in the company of 54 other outstanding chapters. In order to achieve the Gold Level of the Chapter Excellence Award, chapters must meet nineteen Bronze criteria, seventeen Silver criteria and multiple Gold level activities. Criteria include such activities as having a legislative action plan or agenda, holding a volunteerism/community service activity, holding multiple stand-alone meetings, having revenue sources outside of dues and meeting registration fees, implementing a strategic plan, implementing a formal recruitment and retention plan and measuring outcomes, conducting various activities for Medical Students, Residents and Early Career Physicians.

We would like to extend a special thanks to those chapter members who assisted us in all of these endeavors! For their hard work and dedication, we received this award.

2024 ACP Hawai‘i Chapter Laureate Award

It is with great pleasure and admiration that I nominate Dr. Lisa Camara for the prestigious Laureate Award. Dr. Camara exemplifies the highest standards of excellence in medical care, education, research, and community service, making her an outstanding candidate for this honor.

Dr. Camara's journey in medicine began in 1996 when she earned her Medical Degree from the John A. Burns School of Medicine at the University of Hawaii at Manoa. She completed her internship and residency training at the University of Hawaii Integrated Medical Residency Program in 1999 and went on to sever as their Chief Medical Resident. Since 2000, she has severed the community of Hawaii as a Primary Care Physician. Additionally, her expertise and compassion have earned her recognition as one of Hawaii's Best Doctors consistently over the years.

In addition to her clinical responsibilities, Dr. Camara is deeply invested in medical education. As an Assistant Clinical Professor of Medicine at the University of Hawaii, she has played a pivotal role in shaping the next generation of physicians. She served as the site coordinator and teaching attending for the University of Hawaii Internal Medicine Residency Program for 13 years and was Core Faculty for the Kaiser Permanente Hawaii Internal Medicine Residency Program. Her teaching efforts extend beyond the bedside, as she serves as a mentor for residents and medical students at various stages of their training.

Dr. Camara's commitment to research and scholarly activities is evident through her numerous presentations, publications, and involvement in academic conferences. As the Grand Rounds Chair at Kaiser Permanente Department of Medicine from 2000 to 2017, she coordinated presentations on diverse medical topics, inviting expert speakers, and ensuring the seamless execution of these sessions aimed at fostering continuous learning and professional development within the medical community. Notably, her presentation on "The Role of Social Media in Medical Education" underscores her innovative approach to advancing medical knowledge in the digital age.

Moreover, Dr. Camara's service to the medical community and the American College of Physicians (ACP) is exemplary. She joined ACP in 1996 and became a fellow of the college in 2011. She held multiple leadership positions within ACP, including Governor-elect and Governor of the Hawaii chapter (2017-2021). Her contributions include championing initiatives such as LGBTQ+ healthcare and professional education while orchestrating wellness initiatives such as the “Rock the Doc” open mic night where Physicians and Medical Students network and share their musical talents bridging the gap between teacher and learner.

In recognition of her outstanding achievements and unwavering commitment to excellence, I wholeheartedly endorse Dr. Lisa Camara for the Laureate Award. Her exemplary leadership, education to patient care, and contributions to medical education and the American College of Physicians make her a truly deserving candidate for this esteemed honor.

2024 ACP Hawai'i Scientific Meeting Schedule, March 9, 2024

7:30-7:50 am (20 minutes)
Poster Judging/Networking/Visit Exhibits

7:50-8:00 am (10 minutes)
Governor's Welcome - Samuel J. Evans, MD, FACP

Program Chair - Kuo-Chiang Lian, MD, FACP

8:00-8:45 am (45 minutes)
Session #1 - *Barnacles on the Ship of Life: Review of common Skin Growths*. - Iris Noh, MD

8:45-9:45 am (60 minutes)
Podium Presentations (4)

Podium 1 (8:45-9:00)
Deprescribing of Potentially Inappropriate Medications (PIMs) Across Four Geriatric Care Settings – Kathryn Choo Loy, MD

Podium 2 (9:00-9:15)
Enhancing Geriatric Outpatient Care: A Comprehensive Analysis of Geriatric Registered Nurse Phone Calls – Jason Shimoko

Podium 3 (9:15-9:30)
Risk Factors Associated with One-Year Mortality After Osteoporotic Hip Fracture in Hawaii: Higher Mortality Risk Among Native Hawaiians and Other Pacific Islanders – Luke Taylor, MS

Podium 4 (9:30-9:45)
Improving Chronic Kidney Disease Screening in Adults Living with Diabetes in the West O'ahu Community Hawai'i – Liza Mae Mamuad, MS

9:45– 10:15 am (30 minutes)
Poster Judging/Networking/Visit Exhibits

10:15 – 11:00 am (45 minutes)
Session #2 – *Obesity Management* - George Wirunsawanya, MD

11:00-12:00 Noon (60 minutes)
Session 3 – *Dr. Irwin J. Schatz MD, MACP Lectureship - Optimizing Physician Payment for a Well-Designed Single-Payer Healthcare System* - Stephen Kemble, MD

12:00-1:30 pm (90 minutes)
Lunch/Business Mtg/Networking/Visit Exhibits

1:30 – 2:30 pm (60 minutes)
Podium Presentations (4)

Podium 5 (1:30-1:45)
A case of Reversible Cerebral Vasoconstriction Syndrome (RCVS) on an Eclamptic Patient – Weiming Du, MD

Podium 6 (1:45-2:00)
Unmasking Graves' Disease: A Paralysis Case in the Emergency Department – Keith Nakamatsu, MS

Podium 7 (2:00-2:15)
Ockham's Razor and Bayes Theorem at work, a Case Series: Analyzing the sensitivity and specificities of Brugada, aVR Verecki, and Basel algorithms in evaluation of Wide Complex Regular Tachycardias amongst a Multiracial population – Clarke Morihara, MD

Podium 8 (2:15-2:30)
Findings from the First Systematic Survey of Surfer's Myelopathy Patients – Sarah Bellati, MS

2:30-3:00 pm (30 minutes)
Poster Judging/Networking/Visit Exhibits

3:00-3:45 pm (45 minutes)
Session #4 - *Advocacy Panel*
Stephen Kemble, MD/*ACP and Universal Healthcare* - Janet Onopa, MD, FACP /*Advocacy for Universal Healthcare* - Philip Verhoef, MD, FACP

3:45-4:30 pm (45 minutes)
Session #6 – *"Hāna Ho" - Update in Outpatient Medicine* – Robert Gluckman, MD, MACP

4:30-5:15 pm (45 minutes)
Break/Networking

5:15-5:45 pm (30 minutes)
Abstract Winners/Awards Presentation/Networking

5:45 pm – Doctor's Dilemma
Chief Medical Residents

Learning Objectives

At the conclusion of this activity, the participant will be able to know about:

- Advocacy Panel
- Internal Medicine Updates 2023
- Schatz Lecture
- Weight Management
- Doctor's Dilemma

CME Accreditation and MOC Points

The American College of Physicians is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The American College of Physicians designates this live activity for a maximum of **6.0** ***AMA PRA Category 1 Credit(s)*** ***TM***. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Successful completion of this CME activity, which includes participation in the evaluation component, enables the participant to earn up to **6.0** medical knowledge MOC points in the American Board of Internal Medicine's (ABIM) Maintenance of Certification (MOC) program. Participants will earn MOC points equivalent to the amount of CME credit claimed for the activity. It is the CME activity provider's responsibility to submit participant completion information to ACCME for the purpose of granting ABIM MOC credit.

Your Opinion Counts

At the conclusion of the meeting, please take a moment to complete the meeting survey form and verification of attendance form in your packet and return it to the registration desk. We value your opinion and use the surveys in planning future meetings.

Chapter Excellence Award

We are pleased to announce that our chapter is a recipient of the 2023 Chapter Excellence Award. The Chapter Excellence Award recognizes those chapters that excel in reaching the standards for managing a chapter, such as communicating to members, instituting Medical Students' and Resident/Fellow Members' activities and advancing and recruiting members.

Resident/Fellows' and Medical Students' Activities

Clinical vignettes, posters, and research papers prepared by Resident/Fellow Members and Medical Students will be presented at the meeting. Winners will receive a cash prize and be eligible for entrance into the national competition held during the ACP Internal Medicine National Meeting 2025.

Pathways to Fellowships

Attendance at chapter meetings can help all ACP members meet the qualifications for advancement to Fellowship. It is especially important for those applying under the pathway that calls for five years of activity as a member.

Governor

Samuel Evans, MD, FACP - Assistant Clinical Professor, Department of Medicine, University of Hawai'i and Hawai'i Pacific Health, Honolulu, HI
ACP Governor, Hawai'i Chapter
Email: samevansmd@gmail.com

Program Committee

Program Chair: Kuok-Chiang Lian, MD

Mary Ann Antonelli, MD, FACP
Javier Barranco-Trabi, MD
Joel Brown, MD, FACP
Lisa Camara, MD, FACP
Ethan Chun, MD
James Epure, MD, FACP
Alvin Furuike, MD, MACP
Robert Gluckman, MD, MACP
Joseph Go, MD
Donald Helman, MD, FACP
Helen Holmgren, MD
Jennifer Katada, MD, FACP
Stephen Kemble, MD
Philip Verhoef, MD, FACP
Helen Victor, BBA
Diana Kim, MD
Kuo Lian, MD, FACP
Todd Nagamine, MD
Ryon Nakasone, MD, FACP
Yoshito Nishimura, MD, PhD
Jenie Ogle, MD
Janet Onopa, MD, FACP
Abby Pandula, MD
Florian Sattlemachier, MD, FACP
David Spinks, MD
Philip Verhoef, MD, FACP
Sydney Tatsuno, MD, FACP
Arvin Tran, MD
William Wadzinski, MD, FACP
James Yess, MD, FACP

Faculty

Robert Gluckman, MD, MACP - Dr. Robert Gluckman has served in a variety of leadership positions with Providence. Prior to becoming chief medical officer for Providence Health Plans in December 2010, Dr. Gluckman served as chief medical officer for the teaching clinics at Providence Medical Group. He served on the faculty for Providence St. Vincent Internal Medicine Residency for 18 years, where he maintained an active internal medicine practice. Throughout his academic career he has focused on applying medical evidence to clinical practice, with an emphasis on increasing the value of care delivered to patients.

Dr. Gluckman graduated summa cum laude in 1978 from the University of Illinois and earned his medical degree in 1982 from the University of Chicago. He completed his residency at Michael Reese Hospital in Chicago and is board certified in internal medicine.

Dr. Gluckman is Treasurer Emeritus of the American College of Physicians and served on the Board of Regents for the American College of Physicians (ACP), the nation's largest physician specialty society. He brings a strong background in advocacy and public policy to his current role. He is past chair of the ACP's Medical Practice and Quality and ACP Services Political Action Committees. He received the ACP Oregon Chapter's Laureate Award in 2013 for his contributions to the internal medicine community. He is currently the American Health Insurance Plan's Liaison to the Center of Disease Control's Advisory Committee on Immunization Practices (ACIP), a member of the Board of the Oregon Medical Association and serves on the board for Stand for Children.

Iris Noh, MD – Dr. Iris Noh grew up in Hawaii. She attended the John A Burns School of Medicine at the University of Hawaii. She completed her Dermatology residency and Mohs fellowship at the University of Michigan, where she stayed on as faculty before moving back home. Dr. Noh is currently the Chief of Dermatology for the Hawaii Pacific Health Medical Group and practices Mohs micrographic surgery at the King street clinic at Straub Medical Center. During her free time, she enjoys spending time with her 2 kids.

Stephen Kemble, MD – Dr. Kemble attended medical school at University of Hawaii and Harvard, and he trained in both internal medicine and psychiatry. He retired from the private practice of psychiatry in 2017, but continued to work part-time until 2024 in Queen Emma Clinic, a hospital-based primary care clinic, including caring for mostly Medicaid patients and teaching psychiatric issues in general medical care to internal medicine residents.

He is an Assistant Clinical Professor of both Medicine and Psychiatry at the JABSOM. He is also a past president of both the Hawaii Psychiatric Medical Association and the Hawaii Medical Association. Dr. Kemble has a longstanding interest in health policy and health care reform, and he was appointed to the Hawaii Health Authority in 2011, charged with overall health planning for the State of Hawaii and with designing a universal health care system covering everyone in the State. He has been a member of Physicians for a National Health Program, a physician single-payer advocacy organization, since 1989, and he currently serves on the PNHP Board and chairs the PNHP Policy Committee.

Janet Onopa, MD, FACP – Dr. Janet K. Onopa is an internist in Honolulu, Hawai‘i. She received her medical degree from University of Hawaii John A. Burns School of Medicine and has been in practice for more than 20 years. Dr. Janet K. Onopa has expertise in treating Chronic diabetes, Sleep apnea, and Hypertension. She is an Assistant Clinical Professor of Medicine at the University of Hawai‘i John A. Burns School of Medicine.

Philip Verhoef, MD, FACP – Dr. Philip A. Verhoef is a med-peds intensivist, caring for patients in both the adult and pediatric critical care units at Kaiser Permanente Hawaii and Kapiolani Medical Center for Women and Children. In addition, he is an associate clinical professor of Medicine at the John A. Burns School of Medicine at the University of Hawaii and associate program director for the Kaiser Permanente Hawaii Internal Medicine Residency Program.

Dr. Verhoef graduated from the MSTP MD/PhD program at Case Western Reserve University with a PhD in pharmacology and completed residency training in internal medicine and pediatrics at UCLA and subspecialty MICU and PICU fellowship training at the University of Chicago.

As a junior faculty member at the University of Chicago, he was supported by several NIH awards to study the host response to infection using mouse models, patient samples and big data analysis of electronic health records. Since relocating to Hawaii in 2019, he has moved further into the big data space, combining analysis of immune responses with clinical data to further refine our understanding of subphenotypes in sepsis and COVID-19, as well as studying disparate health outcomes among patients in Hawaii.

Beyond his research, he is an active clinician and educator, with an interest in undergraduate and graduate competency-based clinical medical education. He loves spending time with his family, standup paddle surfing, and singing with the Oahu Choral Society.

Kamonkiat "George" Wirunsawanya, MD – Dr. Kamonkiat Wirunsawanya, aka Dr. George, currently practices as an endocrinologist at Straub and Pali Momi Medical Center. I am originally from Bangkok, Thailand and earned my medical degree from Rangsit University in Thailand. Afterwards, I came to the US to further specialize in internal medicine through a residency program at the University of Hawai'i Internal Medicine Residency Program, graduating in 2018 and completed my endocrinology fellowship at Boston University in 2020. My special interests are obesity management and the integration of technological advancements in diabetes treatment.

Doctor's Dilemma -

Javier Barranco-Trabi, MD - Chief Medical Resident, Tripler Army Medical Center, Honolulu, HI
Ethan Chun, MD – Chief Medical Resident, Kaiser Permanente, Honolulu, HI
Joseph Go, MD - Chief Medical Resident, UHIMRP, Honolulu, HI
Helen Holmgren, MD - Chief Medical Resident, Kaiser Permanente, Honolulu, HI
Todd Nagamine, MD – Chief Medical Resident, UHIMRP, Honolulu, HI
Yoshito Nishimura, MD - Chief Medical Resident, UHIMRP, Honolulu, HI
Jenie Ogle, MD – Chief Medical Resident, Tripler Army Medical Center, Honolulu, HI

New Fellows -

Shiuh-Feng Cheng, MD, FACP
Daven Chun, MD, FACP
Brandy Kaneshiro-Yeung, MD, FACP
Jennifer Katada, MD, FACP
Jacqueline O King-Jodi, MD, FACP
Linda Kuribayashi, MD, FACP
Sian Yik Lim, MD, FACP
Marisa E. Rivera, MD, FACP
Travis Watai, MD, FACP

2024 Chapter Awards

Distinguished Teacher/Mentor Award –	Brandy Kaneshiro-Yeung, MD, FACP
Diversity Award -	Ashley Morisako, MD
Hospitalist Award –	Sandra Loo, MD Helen Sim, MD
Resident of the Year Award -	Yoshito Nishimura, MD, PhD Arvin Tran, MD

New Members -

Abulhassan Ali
Kiara Arakawa-Taum
Christian Capirig
Slaton Case
Sean Choi
Horyun Choi
Juyoung Chong
Leslie B Chun, MD
Brianna Diaz
Weiming Du
Anna Eckart-Dodd, MD
Nathaniel Enriquez
Simone Evett
Rowena Feng
Elvelyn R Fernandez
Alyssa Finger
Bradley Fujiuchi
Malia Gacutan
Christine Anne T Galang
Alison Goo
Tony Head
Yusuke Hirao, MD
Aaron Hollenbaugh, DO
Joshua Hu
Ricky Huang
Ted V Jacoby
Jordan Jensen
Kirsten H Jin
Blake Kadomoto
Natalie Kamada
Meeta Kanwar
Brysa TW Kato
Koichi Keitoku
Janette M Keola
Jacqueline O King-Jodoi, MD FACP
Miki Kiyokawa, MD
John p Kristofich, MD
Philip M Lee
Barbara Lee, MD
Roy Levit, MD
Henry Lew, MD
Jordan R Li
Bao Xin Liang
Tyler Liu
Jhoanna D Mabutas, MD
Sharina Macapagal
Jamie Malone
Reed McCardell Malone
Trevor McCracken
Clarke Morihara
Ashley Morisako
Lauren Muraoka
Todd Nagamine
Keith Nakamatsu
Toru Nakata
Jadon Neuendorf
Yoshito Nishimura, MD PhD
Uzoagu Okonkwo
Christina Park
Charlotte Park
Frishan Paulo
Marisa E Rivera, MD FACP
Keith G Sablan
Stephen Sack
Parthav Shah
Kazushige Shiraishi, MD PhD
Ryan H Shontell
Aurelian Stewart
Bryce K Tanaka
Stephanie Tin
Kennedy Tobin
Darcy Tokunaga
Chalothorn Wannaphut, MD
Emily Weber
Sharon Wong
Marcus H Yamamoto
Sydney Yee
Thanaboon Yinadsawaphan

Membership 20, 30, 40, 50, 60, 74…..

74 Years –
Frederick L. Giles, MD, FACP
64 Years –
James W. Linman, MD, FACP
61 Years –
Robert Hockwald, MD, FACP
Leon Katz, MD, FACP
Alfred Morris, MD, FACP
50-59 Years -
James J Ball, MD FACP
Douglas B Bell, II, MD
Carl W Boyer, Jr, MD FACP
Joel D Brown, MD FACP
Winfred Y Chang, MD
Edward L Chesne, MD FACP
John S Falzarano, MD
Reginald C Ho, MD
George N Irwin, MD
William C James, MD FACP
Howard I Keller, MD FACP
John H Kim, MD FACP
Mark T Kuge, MD
James Lumeng, MD FACP
Virgil A Place, MD FACP
Harry Rubin, MD FACP
Dudley S Seto, MD
Arnold W Siemsen, MD FACP
Eugene G C Wong, MD FACP
40-49 Years –
Jonathan K Cho, MD
Marconi M Dioso, MD
Fortunato V Elizaga, MD FACP
Richard I Frankel, MD MPH FACP
Wilfred Y Fujimoto, MD FACP
Christine S Fukui, MD
Reuben C Guerrero, MD FACP
James E Hastings, MD FACP
Robert L Justice, MD
Roy O Kamada, MD FACP
William E Kaye, MD FACP
Stephen B Kemble, MD
Dukee Kim, MD
William K K Lau, MD FACP
Martin I Leftik, MD
Richard T Min, MD FACP
Roland C K Ng, MD FACP
Donald K Nikaitani, MD
Francis D Pien, MD FACP
Bruce J Purvis, MD
Werner G Schroffner, MD FACP
Edward N Shen, MD FACP
Gildo S Soriano, MD FACP
Howard Z Streicher, MD
George H Underwood, MD FACP
Chien-Fong Wu, MD
Tay-Ing Yang, MD
30-39 Years –
Gary W Ahn, MD
Gerard K Akaka, MD FACP
Kheng See Ang, MD
Julie Y Asari, MD
Steven S Azuma, MD FACP
Erlaine F Bello, MD FACP
Catherine A Bender, MD
Benjamin W Berg, MD FACP
Michael Bornemann, MD FACP
S Kalani Brady, MD MPH MACP
Charn-Sing Chan, MD
Freda E Chu, MD
John J Cogan, MD FACP
Andrew Wing Yan Dang, MD
Stephen H Denzer, MD FACP
Elaine M Doi, MD
James P Epure, MD FACP
Timothy E Fern, MD
David Fitz-Patrick, MD FACP
Nathan H Fujimoto, MD
Osamu Fukuyama, MD FACP
Alvin N Furuike, MD MACP
Leon P Garcia, III, MD
Kevin S Hara, MD FACP
Janice K Harada, MD
Ursula Heinz, MD
Birendra S Huja, MD
Joseph W Humphry, MD FACP
Yuri Imanishi, MD
Michael Ishioka, MD FACP
Arnold F Jacobson, MD
Laura L Jones, MD
Aaron S Kaichi, MD
Elliot J Kalauawa, MD
Richard T Kasuya, MD MSEd FACP
Keiichi Kobayashi, MD FACP
Shari L Kogan, MD FACP
Joseph K Koo, MD FACP
John p Kristofich, MD
Linda L Kuribayashi, MD FACP
Scott K Kuwada, MD FACP
David T Lee, MD
Randal J Liu, MD
Steven M Lum, MD FACP
Steven E MacBride, MD FACP
John B Marshall, MD FACP
John S Melish, MD FACP
Kenneth T Minami, MD
Dawn A Miura, MD
Edward J Morgan, III, MD FACP
Aaron H Morita, MD FACP
Ronald A Morton, MD
Mitchell S Motooka, MD FACP
Aaron K Nada, MD
Michael H Nagoshi, MD FACP
Bruce L Nelson, MD FACP
Stephen M Oishi, MD FACP
Janet K Onopa, MD FACP
Melvin P Palalay, MD
Edith L Pang, MD
Michael A Patmas, MD FACP
Kim-Thu C Pham, MD FACP
David M Saito, MD
Diane M Sakai, MD
Daniel M Saltman, MD FACP
Jorge C Samaniego, Jr, MD
William C Seal, MD FACP
Robin L Seto, MD FACP
Geoffrey S Sewell, MD FACP
William M Shapiro, MD FACP
Lorene K Siaw, MD
Edward A Silver, MD
Kenneth M Sumida, MD FACP
Mona N Suzuki, MD
Robin H Takata, MD
George L Talbot, Jr, MD
Laurie M Tam, MD FACP
Warren I Tamamoto, MD FACP

Benjamin A Tamura, MD
Naoto T Ueno, MD FACP
Adrienne Wing, MD FACP
James H Yamashita, MD
Russell D Yang, MD FACP
Guy N Yatsushiro, MD

20-29 Years –

Nadezna Lyn Penaranda Ang, MD
Mary Ann S Antonelli, MD FACP
Godofredo B Baclig, MD
Justin T Barratt, MD
Lisa B Barville, MD
Daniel H Belcher, MD
Jeffrey L Berenberg, MD MACP
Patricia A L Blanchette, MD FACP
Charles Block, MD
Dennis T Bolger, Jr, MD FACP
Bridget S Bongaard, MD FACP
Michael S Braun, MD
Terezia A Bush, MD
Stephen K Buto, MD FACP
Arlene A Cadelina, MD
Lisa A Camara, MD FACP
Dee-Ann L Carpenter, MD
Julie A Chang, MD FACP
Shiuh-Feng Cheng, MD FACP
Karen I Ching, MD
Chelsea K Ching-Endow, MD
Christina MB Chong, MD FACP
Daven K Chun, MD FACP
Bradley A Chun, MD
Carrie L Colmenares, MD
Jon P Cooney, MD
Irina R Crook, MD FACP
Gil A Cu, MD FACP
Arnulfo B Diaz, MD
Michael J Dimitrion, MD
Samuel J Evans, MD FACP
Daniel J Fischberg, MD
Roy L Foliente, MD
Richard A Girton, MD
Robert A Gluckman, MD MACP
Jodi Goh, MD
Eldi Han, MD
Eleanor R Hastings, MD FACP
Rick Y Hayashi, MD
Donald L Helman, MD FACP
Catherine T Ho, MD
John H Houk, MD FACP
Margaret Hu, MD
Reid K Ikeda, MD FACP
James H Ireland, MD
Dorothy M Iwanski, DO
Tad Jackson, MD FACP
C Mitchell Jenkins, MD FACP
Erma F Jose, MD
Joycelyn M Jurek, MD FACP
Leilani Kaanehe, MD
Robert S Kagawa, MD FACP
Brandy H Kaneshiro-Yeung, MD FACP
Mouhamed M Kannass, MD
Kevin K Kato, MD
Misao George Kawamura, MD
Kelly Y Kawaoka, MD FACP
John P Keenan, MD
Liliane Kheng, MD
Timothy D Kim, MD
Melanie Kim, MD
Paul H Kim, MD
Christine Kim, MD
Claudine K Kimura, MD
Tom-Oliver Klein, MD FACP
Joey Y Kohatsu, MD
Sreenandh Krishnagopalan, MD
Lance M Kurata, MD
Yvonne Yee-Wan Kwan, MD
Ronald C Kwon, MD FACP
Jason M Laird, MD
Thomas K Lau, MD
Sharon S Lawler, MD
Genevieve Ley, MD FACP
Gordon F H Liu, MD
Serena Lo, MD
William S Loui, MD FACP
James R Madison, DO FACP
Adelina V Matsui, MD
Eugene S Matsuyama, MD
Marjorie K Mau, MD MACP
Monica K Mau, MD
Lavonda Mee-Lee Nakamoto, MD
Francisco S Mercado, Jr, MD FACP
Florence Mimy, MD
Stephen K Miyasato, MD
David A Naai, MD FACP
Ryon K Nakasone, MD FACP
Craig Nakatsuka, MD FACP
R Craig Netzer, MD
David Ng, MD
Roger I Ogata, MD
David D Ono, MD
Roger L Palmer, MD
Ronald F Pangilinan, MD
Zelah G Pek, MD
Elizabeth G Quinn, MD FACP
Malia L Ramirez, MD
Glenn A Rediger, MD FACP
Erida Reichert, MD
LTC Jefferson R Roberts, MC USA
Nadine Tenn Salle, MD
Helen M Sim, MD
Bruce A G Soll, MD FACP
Charlie Y Sonido, MD
Beata W Summer-Brason, DO
Gerri D Sylvester, MD
David J Tamura, MD
Anne A Tanabe, MD
Sydney Y Tatsuno, MD FACP
Harvey B Ulano, MD PhD
Vuong M Uong, MD
Myrna S Uytingco, MD
Josephine T Waite, MD
Anthea Wang, MD
Aida B Wen, MD
Karen B White, MD FACP
Frank A Williams, MD FACP
Francis K L Won, MD
Lydia Wong, MD
Daniel W Wu, MD
Edward Y Yamada, MD
Teruo Yamauchi, MD
Jaelene Yates, MD PharmD
James Yess, MD FACP
Kelley B Yim, MD
Lester K Yim, MD
Lance A Yokochi, MD
Yuka Yonebayashi, MD
Clara P Yong, MD
Royden S Young, MD FACP

March Birthdays

Kheng See Ang, MD
Nobuhiro Ariyoshi, MD
James J Ball, MD FACP
Daniel H Belcher, MD
Sarah E Bellatti
Jeffrey L Berenberg, MD MACP
Eduardo B Biala, Jr
Michael S Braun, MD
Stephen K Buto, MD FACP
Dee-Ann L Carpenter, MD
Nicole Chong
Juyoung Chong
Ian K Chun
David W Coleman
Brianna Delamare
Ana Marie L Digao, MD FACP
Yue Fang
Daniel J Fischberg, MD
Roy L Foliente, MD
Victoria Fox-Behrle, MD
Alvin N Furuike, MD MACP
Eldi Han, MD
Reginald C Ho, MD
Veronica Icaza Galindo, MD
Florence Kan
Caleb Ko
Landon Kozai

Tiffany Kurozawa
Bao Xin Liang
Matthew C Linden
Stephanie Lum
John B Marshall, MD FACP
Adelina V Matsui, MD
Marjorie K Mau, MD MACP
John S Melish, MD FACP
Dawn A Miura, MD
Aiko Murakami
John Neighbors
Hanaa Al-Khansa B Nik Rushdi, MD
Stephen M Oishi, MD FACP
Tomifumi Onishi, MD
Kim-Thu C Pham, MD FACP
Aldrich Ricalde, MD
Marisa E Rivera, MD FACP
William M Shapiro, MD FACP
Katelyn Shirai
Eli Snyder
David J Spinks, MD
Alister Tang, MD
Sydney Y Tatsuno, MD FACP
Naoto T Ueno, MD FACP
Marko Vasic, MD
Kelley B Yim, MD
Miki Yokokawa, MD

PODIUM PRESENTATIONS

8:45 am Podium

Deprescribing of Potentially Inappropriate Medications (PIMs) Across Four Geriatric Care Settings

Kathryn Choo Loy, MD, Kapono Chang, DO; Jenny Davila, MD; Pedro Aguilar, MD; Kellie Kurasaki, MD; Kamal Masaki, MD; Aida Wen, MD; Cody Takenaka, MD; Gina Fujikami, MD; Jacob Moore, APRN; Sarah Racsa, MD
[1] University of Hawai'i John A Burns School of Medicine, Geriatric Fellowship Program, Honolulu, HI
[2] The Queen's University Medicine Group, Honolulu, HI

INTRODUCTION:

Polypharmacy, commonly defined as five or more medications, is seen with high prevalence among older adults. Appropriate deprescribing can help to reduce common problems associated with polypharmacy, including drug-drug interactions, adverse drug events and falls.

OBJECTIVE:

We conducted a quality improvement project to develop a deprescribing protocol for geriatric patients, and to assess if Beers criteria is a useful tool to help deprescribe PIMs.

METHODS:

Physicians and nurse practitioners working in four different service lines - inpatient, post-acute and long-term care, outpatient and home-based primary care (HBPC), each identified 3 patients with polypharmacy (5 or more routine meds) who had not seen a geriatrician within the past 12 months. Patients who were enrolled in hospice were excluded. Providers completed a pre-intervention data collection form which included total number of medications, names and drug categories, high-risk medications, dosing, frequency, and adverse drug reactions. Providers provided recommendations for dose reduction, tapering, or discontinuation. Post intervention data were collected at 1 month or at discharge for hospital patients, at 3 weeks or at discharge for SNF patients, and at 3 months or at death for clinic and HBPC patients. Pre and post intervention data were compared using paired t-tests.

RESULTS:

We analyzed preliminary data on 60 patients from 20 providers. Complete data collection will include N=112 patients. There was a significant reduction in number of medications per patient after intervention for routine meds (10.4 vs. 9.1, $p=0.0025$), PRN meds (2.2 vs. 1.6, $p=0.0004$), total meds (12.5 vs. 10.8, $p=0.0003$), and Beers criteria meds (2.3 vs. 1.5, $p<0.0001$). We saw deprescribing trends in medication classes of sedatives, muscle relaxants, anticoagulants, antihistamines/anticholinergics, opioids and antipsychotics.

CONCLUSIONS:

Using Beers criteria while reviewing the patient's medications reduced the amount of PIM medications prescribed. We saw trends in reduction of all high-risk medication classes. Of the Beers-criteria medications, opioids were the most commonly prescribed. Data from this QI project will inform educational interventions on deprescribing for primary care physicians.

9:00 am Podium

A Comprehensive Analysis of Geriatric Registered Nurse (RN) Phone Calls

Jason Shimoko[1], Megan Chock, MD[2,3], Serena Lo, MD[2,3]
[1] The University of Hawai‘i Mānoa, Honolulu, HI
[2] Kaiser Permanente Medical Group, Honolulu, HI
[3] University of Hawai‘i, John A. Burns School of Medicine, Honolulu, HI

Introduction: Geriatric medicine involves an interdisciplinary team to support the needs of older adults and their caregivers. There is ample literature about coordination of geriatric care in the hospital, but there are fewer examples of care in the outpatient setting. Our Geriatric Medicine department consists of three registered nurses (RNs) working with four geriatricians to conduct approximately 3,000 patient visits annually. Geriatric RNs call patients and caregivers to support physicians in follow-up and triage to improve coordination of care between geriatrician visits.

Objective: This study aims to describe the impact of geriatric RN phone calls by examining the reasons for and the time spent on each call-in order to understand what issues RNs manage and to quantify potential reduction in time burden for geriatricians.

Methods: From 9/6/2022 to 11/15/2022 (50 working days), documentation from all phone calls conducted by RNs in the Geriatric Medicine department were analyzed. The following information from each call was recorded: reason for the call, duration of the call, whether the call required geriatrician follow-up, and the date of the previous geriatrician visit. Calls that were not completed and calls that lacked documentation for the reason for and/or duration of the call were omitted from the final analysis. Data were entered into a secure Microsoft Access database.

Results: A total of 1348 calls were completed during the study period, with 944 calls included in the final analysis. On average, RNs spent 207 minutes daily on completed patient calls, approximately 173 total hours during the 50-day period. The most common reasons for RN calls were behavior management (22%), medication management (22%), caregiver support (21%), and triage (12%). Over three-quarters (75.6%) of RN calls did not require geriatrician follow-up. Calls that did require geriatrician follow-up were most often for triage (54%) and medication management (31%).

Conclusion: This study suggests that within the framework of our Geriatric Medicine department, geriatric RNs can effectively manage the majority of calls without action from a geriatrician. Our study found that the amount of time that RNs spent daily on completed calls was equivalent to almost half a workday for a full-time geriatrician, not including time spent on multiple or unsuccessful call attempts. Increasing resources for RN phone calls may be advantageous to improve quality and coordination of care by providing increased support for patients and caregivers, while alleviating time pressure on physicians.

9:15 am Podium

Risk Factors Associated with One-Year Mortality After Osteoporotic Hip Fracture in Hawaii: Higher Mortality Risk Among Native Hawaiians and Other Pacific Islanders

Luke Taylor, MS3[1], Masako Matsunaga, PhD[2],
Hyeong Jun Ahn, PhD[2], Sian Yik Lim, MD[3]
[1] University of Hawaii John A. Burns School of Medicine, Honolulu, HI
[2] University of Hawai‘i, John A. Burns School of Medicine,
Dept of Quantitative Health Sciences
[3] Hawaii Pacific Health, Straub Clinic, Honolulu, HI

Introduction:

Osteoporotic hip fractures represent a serious health concern in older adults and are associated with significant morbidity and mortality. Hawaii is home to an ethnically diverse population and a steadily increasing elderly population. However, studies investigating mortality after osteoporotic hip fracture in Hawaii are limited. This study aimed to estimate mortality rates and identify specific risk factors associated with one-year mortality after osteoporotic hip fracture in Hawaii.

Methods:

This study was a retrospective chart review of adults (â‰¥50 years old) hospitalized with an osteoporotic hip fracture at a large multicenter health care system in Hawaii from 2011-2019. Kaplan Meier curve and log-rank tests were performed to examine survival probability by sex, age group, race/ethnicity, primary insurance, body mass index (BMI), and American Society of Anesthesiologists (ASA) score. After accounting for potential confounders, adjusted hazard ratios (aHR) and 95% confidence intervals (CI) were obtained from Cox proportional hazards regression models.

Results:

We identified 1755 cases of osteoporotic hip fracture. The cumulative mortality rate one year after fracture was 14%. Older age (aHR 3.50; 95% CI 2.13-5.76 for 90+ years old vs 50-69 years old), higher ASA score (aHR 5.21; 95% CI 3.09-8.77 for ASA 4-5 vs ASA 1-2), and Native Hawaiian/Pacific Islander (NHPI) ethnicity (aHR 1.84; 95% CI 1.10-3.07 vs Caucasian) were independently associated with higher mortality risk. Female sex (aHR 0.64; 95% CI 0.49-0.84 vs male sex) and higher BMI (aHR 0.35; 95% CI 0.18-0.68 for obese vs underweight) were associated with lower mortality risk.

Conclusion:

Identifying populations at higher risk for mortality after hip fracture is necessary to provide appropriate interventions. In our study, men, older adults, and NHPIs were associated with significantly higher mortality. NHPIs are an especially vulnerable group and comprise a significant portion of Hawaii’s Hawai‘i’s population. Further research is needed to address the causes of higher mortality and interventions to reduce hip fractures and associated mortality in these populations.

9:30 am Podium

Improving Chronic Kidney Disease Screening in Adults Living with Diabetes in the West Oahu Community

Liza Mae Mamuad, MS2[1], Megan Yung, MS2[1],
Ross Takemoto, MS2[1], Jimmy Chen, MD[1]
[1] University of Hawai'i John A. Burns School of Medicine, Honolulu, HI

Background: Nearly 1 in 3 diabetic patients have chronic kidney disease (CKD). However, many of these patients are asymptomatic and undiagnosed until advanced stages of disease. Thus, routine screening, early detection, and treatment are crucial for reducing the morbidity and mortality of CKD. Kidney health evaluation fulfillment is defined as having both estimated glomerular filtration rate (eGFR) and urinary albumin-creatinine ratio (uACR) testing done within the measurement year. Currently, the national CKD screening rate is approximately 40% and even lower for marginalized groups. Similarly, we found the screening rate in the diabetic population at a primary care practice in West Oahu, which serves a large percentage of Native Hawaiians and Pacific Islanders, to be relatively low. The goal of this quality improvement (QI) project was to identify reasons for care gaps and implement interventions to improve the screening rate in this population.

Methods: Chart review of the electronic medical records of 315 diabetic patients at this primary care office was conducted. Dates and results of the most recent eGFR and uACR lab tests were recorded. Patients were then determined to be screened or unscreened. According to screening criteria, patients older than 75, receiving hospice/palliative care, or diagnosed with end-stage renal disease (ESRD) or stage 5 CKD were excluded. Based on the most common reasons for care gaps, three patient-centered interventions were implemented over 6 months. Two text message reminders about overdue lab tests were sent to unscreened patients, followed by two phone call reminders, and finally, two text message reminders to alternative phone numbers, if available. Chart reviews were conducted between each intervention to identify newly-screened patients.

Results: The baseline screening rate was found to be 52.4%. However, 57 patients did not meet inclusion criteria. Of the remaining patients, 58.1% fulfilled the criteria for CKD screening. The most common reason for care gaps among unscreened patients was due to incomplete uACR testing within the last 12 months (79.8%). Incomplete eGFR was the second most common (49%). Text message reminders, phone call reminders, and texts to alternative numbers increased screening rates by 10.8%, 5.5%, and 2.3%, respectively. At the end of 6 months, the screening rate increased by 29.4%, totaling 81.8% of patients screened for CKD.

Conclusion: Increased screening allows for earlier detection and treatment of CKD, thereby reducing its mortality and morbidity. Our findings suggest that suboptimal baseline CKD screening rates for diabetic patients can be increased through patient-centered interventions, with text messages being the most effective and efficient. Secondary analyses may be conducted to determine long-term outcomes, or if screening status is associated with other care measures or features of this population. While these initial results and interventions are limited to this setting, the results can be applied to other areas of primary care and expanded to include a wider scope of patients. Provider-centered interventions, either alone or in combination with patient-centered approaches, may also be a potential area of future research in increasing CKD screening rates.

1:30 pm Podium

A Case of Reversible Cerebral Vasoconstriction Syndrome (RCVS) in a Patient with Eclampsia

Weiming Du, MD[1], Chalothorn Wannaphut, MD[1], Yoshito Nishimura, MD, PhD[1], Christina Chong, MD[1,2], Travis Watai, MD1, FACP[1,2]
[1] University of Hawai'i Internal Medicine Residency Program
[2] The Queen's Medical Center

Introduction: Eclampsia generally resolves postpartum; however, seizures occurring beyond 48 hours post-delivery warrant investigation for alternate etiologies. RCVS, also named Call fleming syndrome is a very rare condition in which postpartum women develop reversible cerebral vasoconstriction after delivery usually within the first three weeks. Here we discuss a rare case of an 18-year-old female with eclampsia presenting with abnormal neurological exams and subsequently diagnosed with RCVS.

Case: An 18-year-old G1P0 Micronesian woman who is at 32 weeks pregnancy with a past medical history of bilateral hearing loss treated with bilateral cochlear implants who presented with altered mental status, bilateral lower extremities weakness and absent reflexes after an episode of eclampsia requiring emergent Primary Low Transverse Cesarean Section (PLTCS).

Patient was initially admitted for sepsis secondary to pyelonephritis with associated right hydronephrosis. During this hospitalization, patient had a tonic-clonic seizure which required PLTCS for non-reassuring fetal status (NRFHT). Patient was admitted to the intensive care unit (ICU) post operation and downgraded two days after but remained confused despite a normal neurological exam. She experienced another 3 minutes tonic-clonic seizure 48 hours after giving birth prompting neurology consultation. Prior to that, patient had no history of developmental delay or seizure. Patient was observed to have a flat affect and minimal verbal interaction. She was alert and oriented to name, place and year. She denied headache, fever, chest pain or shortness of breath. Vital signs were unremarkable except elevated blood pressure. Neurological exam was notable for gaze-evoked nystagmus in all four directions and diminished hearing, 3/5 in bilateral lower extremities, ataxia on finger-nose testing of bilateral upper extremities, bilateral hand pronator drift, and areflexia on patellar and Achilles tendon. Cerebrospinal Fluid Analysis revealed albuminocytologic dissociation with no viral or bacterial organisms identified. Brain MRI was normal, CTA Brain shows mild to moderate diffuse narrowing of the ACAs, MCAs, and PCAs. Diagnosis of RCVS was made based on CTA findings.

Patient was initiated with levetiracetam, verapamil and magnesium sulfate infusion. Despite initial neurological improvement, she became acutely unresponsive to noxious stimuli on day eleventh post-PLTCS. Repeat CTA revealed extensive worsening of cerebral vasoconstriction, prompting her transfer to the neuroscience ICU. Magnesium and levetiracetam were continued; verapamil was transitioned to nimodipine. Her condition gradually improved and she was discharged on nimodipine and levetiracetam.

Discussion: RCVS has been associated with a variety of conditions such as pregnancy, migraine, use of vasoconstrictive drugs, aneurysms and neurosurgical procedures. Presentations include thunderclap headache, seizures, aphasia, focal neuro deficits or visual deficits. CTA shows diffuse reduction in the caliber of the cerebral vessels and their branches. The treatments are usually calcium channel blockers with verapamil or nimodipine, as well as intravenous magnesium therapy given their vasodilation effects. Most patients show symptoms of resolution within days to weeks. Recurring episodes of RCVS are uncommon and the prognosis is generally good.

1:45 pm Podium

Unmasking Graves' Disease: A Paralysis Case in the Emergency Department

Keith Nakamatsu, MS[1], Miki Yokokawa, MD[2], Kuo-Chiang Lian, MD[3,4]
[1] A.T. Stills University School of Osteopathic Medicine
[2] University of Hawai'i Internal Medicine Residency Program
[3] University of Hawai'i Department of Medicine
[4] The Queen's Medical Center

Introduction: Thyrotoxic periodic paralysis (PP) is a sporadic form of hypokalemic PP that may occur in association with hyperthyroidism. Thyrotoxic PP is a rare, reversible condition often overlooked in the differential diagnosis of acute paralysis. We describe a case of thyrotoxic PP secondary to Graves' disease in conjunction with severe hypokalemia.

Case Description: A 32-year-old male with a past medical history of methamphetamine use disorder presented with acute quadriparesis. He had been in his usual state of health until he woke up from sleep when he found to have severe generalized weakness and was unable to move all limbs, leading to a fall. Upon arrival, he was alert and oriented, afebrile with blood pressure of 147/58 mm Hg and heart rate of 64 beats per minute. Neurological examination revealed symmetric proximal muscle weakness with a scale of 3 out of 5, diminished deep tendon reflexes and equivocal plantar reflex. There were no sensory abnormalities, and cranial nerves were intact. An extremely low potassium level (potassium <1.5 mEq/L) was identified on initial laboratory testing and low phosphorus (1.1 mg/dL) as well with otherwise normal chemistry profile. Electrocardiogram showed atrial fibrillation with prominent U waves along with ST depression. Further history revealed the patient had a high-carbohydrate meals one day prior to the presentation. There was no history of gastrointestinal or increased urinary losses of potassium, and no insulin use was noted. CT scans of the head excluded intracerebral and subarachnoid hemorrhages. The absence of cranial nerve involvement, sensory signs, muscle spasticity, or hyperreflexia ruled out brainstem stroke and spinal cord disease. Guillain-Barré syndrome was considered, given the progressive, symmetric muscle weakness, however there was no prodromal history of viral infection.

Aggressive electrolyte replacement was performed and after 100 mEq of potassium replacement, his potassium level was improved to 3.5 mEq/L. The patient had significant improvement in muscle weakness within a few hours with potassium repletion. Thyroid laboratory panel indicated overt hyperthyroidism (TSH <0.07 µIU/mL, free T4 4.8 ng/dL, free T3 17.6 pg/mL) with elevated TSH receptor antibody 3.95 IU/L (normal range <2.00 IU/L), revealing thyrotoxic PP secondary to hyperthyroidism caused by Graves' disease superimposed. Interestingly, the lack of classical findings including thyromegaly, nodular goiter, or exophthalmos made clinical suspicion of Graves' disease challenging in the initial workup. The patient started receiving methimazole along with prophylactic use of propranolol with close Endocrinology and Cardiology follow up planned.

Discussion: This case underscores the importance of considering thyrotoxic PP in the differential diagnosis of acute paralysis, especially in patients with significant electrolyte disturbances and atypical presentations. The necessity for a comprehensive evaluation is highlighted by the presence of cardiac manifestations like atrial fibrillation, which can accompany endocrine disorders such as Graves' disease, even in the absence of classical hyperthyroidism signs. Early detection and appropriate management of thyrotoxic PP are critical in preventing severe, potentially life-threatening complications.

2:00 pm Podium

Ockham's Razor and Bayes Theorem at work, a Case Series: Analyzing the sensitivity and specificities of Brugada, aVR Verecki, and Basel algorithm in the determination of Ventricular Tachycardia amongst a Multiracial population

Clarke Morihara, MD[1], Thanaboon Yinadsawaphan, MD[1], and Steven Azuma, MD[2]

[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI

[2] University of Hawai'i Department of Medicine, Honolulu, HI

Wide Complex Regular Tachycardias (WCT) defined as a regular rhythm, with rate greater than 100/min with QRS duration â‰¥ to 120 ms is a rare finding on 12 lead electrocardiograms (EKG's). Differential diagnosis includes ventricular tachycardia (VT), Supraventricular tachycardia (SVT) with aberrancy, antidromic bypass tract tachycardia, ventricular pacing and EKG artifact. Emergency treatment of wide complex tachycardia may include synchronized cardioversion irrespective of the etiology of the tachycardia. The differentiation between VT and SVT with aberrancy is clinically important to timely initiate acute and chronic management and guide further work up and treatment. There are historically many algorithms which are being applied but these have multiple, complex steps which can be difficult to recall in an emergency situation. Recently one algorithm (Basel) has been proposed which incorporates history of myocardial infarction, congestive heart failure with left ventricular ejection fraction of â‰¤35%, implanted cardioverter-defibrillator (ICD), or cardiac resynchronization therapy (CRT). This clinical information would be often available to the treating physician who can act on the result. The Basel algorithm has only 3 steps, can be performed in shorter time than the prior algorithms, can be performed by medical students with comparable result to experienced cardiologists, and has similar sensitivity and specificity.

Currently there are several established algorithms which provide an organized method to assist physicians in the differentiation of WCT and the diagnosis of VT or SVT. The Brugada1 Criteria (1991), aVR Vereckei2 algorithm (2008), and the Basel3 algorithm (2022). The original Brugada algorithm showed a sensitivity of 98.7% and specificity of 96.5%. The aVR Vereckei algorithm showed a sensitivity of 96.5% and specificity of 98.2%. The Basel algorithm showed a sensitivity of 92% and specificity of 89%.

Of 16,800 EKG's from September 2022 through December 2023, 16 patients (incidence 0.1%) from a multiracial population who presented between September 2022 to November 2023 at a community hospital had an EKG exhibiting WCT. This multiracial population consisted of individuals from Japanese (n=7), Native Hawaiian or Pacific Islander (n=3), Filipino (n=4) Chinese (n=1), or Caucasian ancestry (n=2). Additionally, this population was also assessed for the presence of structural heart disease (n = 8) which included either history of ICD, CRT placement, myocardial infarction, or history of transthoracic echocardiogram with an EF < 35%.

The three algorithms were separately applied to 16 EKG's by a cardiologist and 2 medical interns. The sensitivity and specificity of each algorithm to detect VT with was applied to a multiracial population. The sensitivity (Brugada 100%, aVR Vereckei 100%, Basel 100%) and specificity (Brugada 88.9%, aVR Vereckei 88.9%, Basel 79%) appeared to be similar to that of published data. We conclude the Basel algorithm is quicker, easier to apply, and more clinically relevant than the other algorithms. This may apply to hospitalists and first responders.

2:15 pm Podium

Findings from the First Systematic Survey of Surfer's Myelopathy Patients

Sarah Bellatti, MS[1], Joo Won Choi, MS[4], Jessica N. Wilson, PhD[1],
Ferdinand Hui, MD[2], Stacy C. Brown, MD[3],
[1] University of Hawai'i John A Burns School of Medicine, Honolulu, HI
[2] The Queen's Medical Center, Honolulu, HI
[3] The Queen's Medical Center – Neuroscience Institute, Honolulu, HI

Objective: To describe the presenting symptoms and circumstances of surfer's myelopathy (SM) based on systematically collected survey data in a prospective cohort study.

Background: SM is a rare, non-traumatic spinal cord injury affecting healthy, novice surfers. Existing literature consists only of case reports and case series that retrospectively summarize common findings.

Methods: We administered a survey electronically over the phone with eight patients treated for Surfer's Myelopathy in Honolulu, Hawaii between 2021 and 2023.

Results: Presenting Symptoms - Symptom onset after beginning to surf varied from 30-60 minutes (n = 4), within 30 minutes (n = 2), and 60-90 minutes (n = 2). 7/8 reported low back pain as their first symptom, while 1/8 reported leg weakness. All noted low back pain at onset of symptoms, ranging in intensity from 4 to 8 on a 10-point severity scale. 3/8 also reported pain radiation to their legs. When asked if the back pain began at an identifiable moment, 4/8 reported the pain progressed subtly overtime, 3/8 noted it when popping up to catch a wave, and 1/8 experienced it while paddling. Time between onset of the first symptom and the point when they could no longer walk varied from < 30 minutes (n = 3), 30-60 minutes (n = 3), and 2-4 hours (n = 1). One patient did not lose the ability to walk. Time between first symptom onset and the point they considered the peak severity of symptoms ranged from < 30 minutes (n = 3), 30-60 minutes (n = 2), 90-120 minutes (n = 2), and 60-90 minutes (n = 1).

Surfing history - All presented with symptoms during one of their first formal surf lessons. 0/8 patients had heard of SM prior to their diagnosis. 0/8 were informed of SM by their surf instructor. When asked how often their surf instructor advised them to sit and stretch their back, 5/8 reported never, 2/8 were told back pain was normal, and 1/8 was advised to sit and stretch after complaining of symptoms. Between waves, patients spent most of their time waiting in a prone position (n = 3), sitting up (n = 1), paddling (n = 1), or a combination of the prior (n = 3). The amount of consecutive time spent in the prone position was reported as > 1 hour (n =3), > 45 minutes (n = 3), and < 45 minutes (n = 2).

Prior athletic experience - Lifestyle activity level prior to injury included 2 professional/school athletes, 2 engaging in organized sports/regular fitness, and 4 with physical activities at least 3 days a week. Posture-based fitness activity experience varied, with 3/8 practicing frequently, 3/8 having tried, and 2/8 having never tried.

Prior back health history - 1/8 had prior chronic back pain. 1/8 had experienced prior traumatic back injury. 0/8 had prior back surgery.

Conclusions: Our systematic, survey-based data on the presenting history and circumstances of eight consecutive SM patients quantifies symptom variability and refines the illness script that informs the diagnosis of surfer's myelopathy.

POSTER PRESENTATIONS

Poster #1

Finding A Way with the Wayfinder Patient Navigation Program: Addressing Chronic Disease Disparities for COFA Migrant Populations in Honolulu

Sylvia Nguyen, MS[1], Kiara Arakawa-Taum, MS[1],
Christie Izutsu, MD, FACP[2], Bryan Brown, MD MHS[3,4]
[1] University of Hawai'i John A Burns School of Medicine, Honolulu, HI
[2] University of Hawai'i Department of Medicine, Honolulu, HI
[3] University of Hawai'i Office of Medical Education, Honolulu, HI
[4] The Queen's Medicine Center, Honolulu, HI

Historical events such as US thermonuclear weapon testing within their home lands left many Compacts of Free Association (COFA) citizens, including those of Chuukese, Marshallese, Pohnpeian, Yapese, and Kosraean descent with poor health and educational outcomes that led to their migration to Hawai'i.

A retrospective case-control study was conducted to quantify the severity of health disparities among COFA-affiliated patients receiving primary care in a hospital-embedded teaching clinic in Honolulu, HI by evaluating prevalence of diabetes, chronic kidney disease, end-stage renal disease, hypertension, stroke, heart disease, obesity, dyslipidemia, and asthma/COPD. In comparison to the age- and gender-matched control group, COFA patients have rates of diabetes two times greater (54.1%), chronic kidney disease 1.8 times greater (24.3%), and history of stroke two times greater (13.0%). Prevalence of uncontrolled diabetes in COFA patients was more than three times higher (16.1%), and end stage renal disease 3.7 times higher (9.6%) than that of the control group.

Within the same clinic, the Wayfinder Program was established to address the relative health burden by educating students on cultural differences and utilizing interpreter services to create connections with COFA patients. Medical students contacted patients using two telephone interpreter services, found that one agency had fewer wait times (average wait time 8 mins vs 24 mins), and implemented a clinic policy change for preference of interpreter companies. Key exemplary case studies were identified and reviewed, highlighting the benefit of the program for the migrant patient population.

Poster #2

Femoral Artery Occlusion as a Presentation of Mitral Valve Endocarditis

Brianna Delamare, DO[1], Robert McMurray, MD[2], David Peterson, DO[2], Jeremy Docekal, MD[2], Shaun Martinho, DO[2], Anthony Katras, MD2, Neil Stockmaster, MD[2], Derrick, Thiel, MD[2], Edwin Kamau, PhD[2], Viseth Ngauy, MD[2],

[1] Tripler Army Medical Center Internal Medicine Residency Program, Honolulu, HI

[2] Tripler Army Medical Center, Honolulu, HI

Introduction:

Staphylococcus epidermidis, a common bacterium found on the skin, is characterized as a gram-positive, coagulase-negative cocci with gamma-hemolysis. While frequently isolated in blood cultures, it is often considered a contaminant due to its low virulence. Although S. epidermidis may not produce toxins like other Staphylococcus species, it does have the ability to form biofilms on implanted medical devices, making it a well-known cause of prosthetic valve endocarditis (PVE). However, S. epidermidis is a rare cause of native valve endocarditis (NVE), with a reported incidence rate of 5% caused by coagulase negative staphylococci (CoNS), with S. epidermidis as the most commonly isolated species. We present a case of a young patient with a unique presentation of NVE due to S. epidermidis.

Case Report:

A 19-year-old U.S. Navy active-duty male presented with sudden onset unprovoked, right leg pain at rest, worse with activity while underway. He was seen 2 months earlier with right knee pain and prescribed non-steroidal anti-inflammatory medications for pain relief. He denied fevers but reported feeling uncomfortable for several weeks. Evaluation revealed leukocytosis and CT angiography in Guam revealed 20cm occlusion of the proximal right superficial femoral artery (SFA), requiring medevac to our facility. With a cool and painful right lower extremity and critical limb ischemia, urgent operative intervention was necessary. Open exploration demonstrated friable and inflamed femoral vessels. A common femoral arteriotomy was made in attempts to remove the thrombus, but was unsuccessful, due to the inflammatory disease. Surgical bypass was required to revascularize the right lower extremity. A common femoral artery (CFA) to SFA bypass with 8mm Gore polytetrafluoroethylene (PFTE) graft was performed, in addition to CFA to profunda femoris bypass with 6mm Gore PTFE. Echocardiogram revealed a large, pedunculated mass on the mitral valve with moderate to severe mitral regurgitation. Histopathology stains of the SFA specimen revealed numerous gram-positive cocci organisms, however, only S. epidermidis was isolated in 2/20 blood culture bottles. 16S rRNA PCR of the paraffin embedded SFA confirmed S. epidermidis. No involvement of other valves or end organs were identified on extensive evaluation. Antibiotic regimen included Vancomycin and Ceftriaxone for empiric coverage. Rifampin was added for prevention of biofilms with the newly placed grafts. He underwent mechanical valve placement and was transitioned to a prolonged course of Cefazolin and Rifampin based on the antibiotic susceptibility profile of the S. epidermidis.

Discussion:

This patient's epidermidis portal of injury is thought to be from skin abrasions sustained daily while working as an engine room mechanic on the ship. This case is unique as it demonstrates a subacute presentation of NVE in a young, healthy patient with substantial reserve and the low virulence of CoNS, which led to diagnosis late in the disease course with resultant severe valvular damage. Additionally, this case demonstrates a unique method of diagnosis with both histopathologic and PCR confirmation of the causative organism. Prolonged musculoskeletal symptoms accompanied by non-specific symptoms such as malaise, fatigue, or subjective fevers in young, healthy adults may be a harbinger of more life-threatening illness.

Poster #3

Hypotension, bradycardia and altered mental status: myxedema coma in a patient with undifferentiated shock

Bradley Fujiuchi, MD[1], Arvin Tan, MD1, Weiming Du, MD[1]
[1] University of Hawai‘i Internal Medicine Residency Program, Honolulu, HI

Myxedema coma is a rare, fatal complication of hypothyroidism with an estimated incidence of 0.22 per 1,000,000 per year. It can be precipitated by events such as severe hypothyroidism, sepsis, myocardial infarction, or surgery. Common presentations include altered mental status, hypotension, hypothermia, or hypoventilation. To date, about 300 cases have been reported.

This case is of an 82-year-old male with hypertension, hypothyroidism, and recent hospitalization for nonspecific interstitial pneumonitis and an ESBL UTI. The patient presented with hypotension to a systolic BP of 87 mmHg and was admitted for sepsis due to UTI and pneumonia. He was also bradycardic at 51 bpm and hypothermic at 33.5Â°C. Following admission, his blood pressure dropped to 90/38 mmHg and bradycardia persisted. He received fluid resuscitation without improvement. Hemoglobin was found to be 6.3 g/dL and he was transfused. Stress dose steroids were also started. Despite these interventions, the patient remained bradycardic and hypotensive, averaging a mean arterial pressure of 55 mmHg and HR of 45.

He was transferred to the ICU for undifferentiated shock and started on norepinephrine and dopamine. Temperature averaged 36Â°C even with the use of external warming devices. Throughout this, the patient was lethargic but arousable with intermittent confusion. TSH was severely elevated, >100 uIU/mL and T4 <0.4 ng/dL. His Levothyroxine was increased from 25 to 100 mcg/day with no improvements to his mentation. Endocrinology was consulted for suspected myxedema coma. His TSH and T4 improved with ongoing treatment, however the patient's mental status continued to deteriorate, ultimately requiring intubation for airway protection. Liothyronine was also started due to undetectable T3 levels despite treatment. Thyroiditis workup was negative and MRI ruled out stroke as the cause of his encephalopathy.

The patient was able to be weaned off vasopressors and passed spontaneous breathing trials, but with persistent encephalopathy, a goals of care discussion was held and the decision was made to transition to comfort oriented care. He was extubated, transferred to the floor, and passed within the following few days.

Myxedema coma is an uncommon presentation of hypothyroidism with significant morbidity and mortality. On admission, our patient was hypotensive, bradycardic, hypothermic and septic. These have been shown as significant predictors of mortality along with the need for mechanical ventilation, unresponsiveness to treatment, use of sedatives, lower GCS and high APACHE II scores, and SOFA scores > 6. Treatment includes supportive care and thyroid hormone replacement. No difference in survival has been shown in patients receiving oral vs. intravenous thyroxine, while treatment with a combination of levothyroxine and liothyronine has been shown to be successful. Patients treated with vasopressors with or without steroids have been shown to have increased in-hospital mortality. Unfortunately, despite prompt and aggressive intervention, mortality rates are high, described to be up to 60%. Our patient died after compassionate extubation to honor his advanced directives. Myxedema coma is a rare, emergent presentation of decompensated hypothyroidism that necessitates high clinical acuity, prompt recognition and early treatment.

Poster #4

Key Principles for Stroke Genetics Research Involving Indigenous People in Hawai‘i and the Pacific Islands

Christine Anne T. Galang, MS[1], Mālialani Kana’iaupuni[2], Selena Vanapruks, MS[1],
Alex Ueoka[3], Chaye N. Kauhola[2], Leah Dowsett, MD[4],
Keolu Fox, PhD[5], Kazuma Nakagawa, MD[2], Stacy C. Brown, MD[2]
[1] University of Hawai‘i John A Burns School of Medicine, Honolulu, HI
[2] The Queen’s Medical Center, Neuroscience Institute, Honolulu, HI
[3] Columbia University
[4] University of Hawai‘i John A. Burns School of Medicine, Dept of Pediatrics, Honolulu, HI
[5] University of California San Diego, Halıcıoğlu Data Science Institute, San Diego, CA

Objective: To outline key research principles that are prerequisite to advancing stroke genetics for Indigenous people in Hawai‘i and the Pacific Islands.

Background: Native Hawaiian and other Pacific Islander (NHOPI) people are disproportionately affected by stroke and comorbidities associated with cerebrovascular disease. Stroke risk is partially heritable, and there is growing research aimed at incorporating genetic information into clinical prevention and management strategies. Inclusion of NHOPI people in such research is paramount to reducing health inequities, however this must be undertaken with attention to Indigenous rights and cultural humility. The aim of this study was to review key principles concerning Indigenous people’s rights with respect to biomedical research.

Methods: We included all research relating to stroke disparities and stroke genetics research in Hawaii and Native Hawaiian and other Pacific Islander populations. We searched the following databases: MEDLINE via PubMed and Google Scholar (searched 2023). We also reviewed reference lists. Criteria for inclusion were peer-reviewed articles published in English that included Native Hawaiian and Pacific Islander populations, stroke genetics research, and ischemic stroke.

Results: We found 26 papers outlining stroke disparities in Indigenous people in Hawai‘i and the Pacific Islands and Indigenous peoples’ rights regarding stroke genetics. Traditional research institutions have historically failed to protect Indigenous peoples from group harm. Community-owned data governance structures and framework, like the Paoakalani Declaration, ensure research is conducted in a culturally sensitive and respectful manner. Education and outreach programs can be implemented to raise awareness about the importance and benefits of genetic and genomic research.

Conclusion: Considerations for research involving NHOPI Indigenous populations include free, prior, and informed consent (FPIC), indigenous data sovereignty, dissemination and application of results, and benefit sharing.

Poster #5

BAL-DFA negative PJP pneumonia

Yusuke Hirao, MD[1], Joseph Lee MD[1], Eyrica Sumida, MS[2],
Timothy Vossler, MD[3], Alena Velasco-Hughes, MD[3]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] University of Hawai'i John A Burns School of Medicine, Honolulu, HI
[3] The Queen's Medical Center, Honolulu, HI

Case Presentation: A 67-year-old male with Stage II Breast cancer vs metastatic carcinoma of unknown primary, polymyalgia, T2DM who presented to ED from the oncology office with dyspnea on exertion and hypoxia.

One week prior to admission, the patient started experiencing worse than usual fatigue where he just wanted to sleep all the time. He endorsed dyspnea upon exertion, particularly noticeable when ambulating rapidly. Notably absent symptoms included chest pain, shortness of breath at rest, and orthopnea. He denied fevers, chills, lightheadedness, abdominal discomfort, nausea, emesis, diarrhea, constipation, dysuria, hematochezia or hematuria.

Breast cancer was initially diagnosed eight months prior with a left axillary lymph node biopsy revealing invasive ductal carcinoma. He received three cycles of adjuvant adriamycin, cyclophosphamide, and pegfilgrastim, with the last administration occurring 12 days before admission. He was also taking prednisone 10mg for his polymyalgia.

On admission, vitals were significant for oxygen saturation of 82% which improved to 98% on 2L of oxygen. He was afebrile and his respiratory rate was normal. Physical exam was unremarkable including a normal lung exam. Labs were significant for leukocytosis at 17.78x10^3/ÂµL, with a neutrophil count of 15.03x10^3/ÂµL and a reduced lymphocyte count of 0.69x10^3/ÂµL. Other significant labs included a lactate dehydrogenase (LDH) level of 576 IU/L and Fungitell at 140 pg/mL. Computed tomography angiography of the chest excluded pulmonary embolism but revealed diffuse ground glass opacities. The patient was unable to produce sputum due to the absence of a cough. Following a bronchoscopy with bronchoalveolar lavage (BAL), he was started on empiric intravenous Bactrim at 20mg/kg of the trimethoprim component and Prednisone at 40mg twice daily. Direct fluorescent antibody (DFA) testing for pneumocystis jirovecii pneumonia (PJP) returned negative, but subsequent polymerase chain reaction (PCR) was positive. Patient gradually improved clinically over 9 days and was eventually discharged on 1.5L of oxygen, oral antibiotics and a prednisone taper.

Discussion: PJP is a prevalent cause of pneumonia in individuals who are immunosuppressed. Patients that are at higher risk include those with underlying malignancy, immunosuppressive treatment, and administration of corticosteroids. The gold standard for diagnosis of PJP from BAL fluid is cytology, followed by DFA. However, the lower burden of P. jiroveciiin non-HIV-immunocompromised patients remains a challenge for diagnosis. In this group, the sensitivity of conventional microbiological tests for sputum specimens is approximately 38 to 53%. Staining methods have largely been supplanted by high sensitivity PCR targeting P. jirovecii-specific genes. Recent meta-analyses have reported an aggregated sensitivity of 98%, 99%, and 97%, alongside a combined specificity of 91%, 90%, and 94%, predominantly in BAL samples.

In the presented case, PJP was postulated based on elevated levels of LDH/Fungitell and characteristic findings in imaging despite the lack of common presenting symptoms. It is pertinent to note that even in instances where DFA testing yields negative results, the execution of PCR testing remains a valuable diagnostic step, especially in cases of PJP among non-HIV patients.

Digital Poster #6

Missed Early Intervention in Uncontrolled Hypertension Leading to Early-Onset End-Stage Renal Disease and Heart Failure in a Young Female

Jonathan Hu, MS[1], Kevin Nebrejas, MD[2],
Yoshito Nishimura, MD, PhD, MPH[2], Jatinder Lachar, MD[3]
[1] University of Hawai'i John A Burns School of Medicine, Honolulu, HI
[2] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[3] The Queen's Medical Center, Honolulu, HI

Introduction: Uncontrolled hypertension is a common issue seen in outpatient practices. While most patients with hypertension have benign clinical courses, younger individuals with uncontrolled hypertension require greater attention as secondary causes such as endocrinopathies or renovascular diseases may be missed. Herein, we present a case of a young female with known untreated hypertension ultimately diagnosed with end-stage renal disease (ESRD) and heart failure due to Type 4 renocardiac syndrome.

Case Presentation: A 39-year-old Asian woman presented to the ED with a 3-week history of exertional dyspnea, orthopnea, nocturia, peripheral edema, and fatigue with notable deterioration over the past week. She does not regularly see a physician, although a detailed review of her outpatient records from three years ago revealed microscopic hematuria, subnephrotic range proteinuria (1025 mg/day), and uncontrolled hypertension with systolic pressures in the 180s. Her physical exam on admission demonstrated blood pressure of 206/130 mmHg, mild bibasilar inspiratory crackles, 2/6 holosystolic murmur best heard at the apex, and signs of fluid overload. Echocardiographic findings including LVEF of 35-40% with global hypokinesis and normal right ventricular function, coupled with elevated BNP, creatinine (6.8 mg/dL), and low hemoglobin (5.5 g/dL), led to a diagnosis of acute heart failure with reduced ejection fraction (HFrEF) with concomitant kidney injury of unknown chronicity and anemia. The patient was treated with intravenous diuretics, anti-hypertensives, and packed red blood cells and over her hospital course, achieved euvolemia and normotension without further need for transfusions. Her workup for newly-diagnosed HFrEF was largely unremarkable without ischemic findings and her anemia was attributed to kidney disease. However, her renal function continued to decline with creatinine increasing to 7.9 mg/dL. A renal ultrasound showed bilateral atrophic and hyperechoic kidneys, indicative of ESRD. Consultations with Nephrology and Cardiology were sought for further management. Tests for systemic infiltrative, autoimmune, and infectious etiologies were negative. Repeat 24-hour urine protein was similar to three years ago, demonstrating subnephrotic (1050 mg/day) proteinuria. A presumptive diagnosis of IgA nephropathy was proposed as the cause of her longstanding hypertension and ESRD. Patient declined renal biopsy for definitive diagnosis and was further managed with hemodialysis and conservative medical management.

Discussion: The case underscores the missed opportunity for early outpatient intervention in patients showing signs of proteinuria, microscopic hematuria, and uncontrolled hypertension. Routine urinalysis, often overlooked in hypertensive patients, should be considered for evaluating secondary hypertension in young, otherwise healthy individuals presenting with hypertensive urgency. Prompt recognition and management of possible underlying glomerulonephritis might have averted the progression to ESRD and development of renocardiac syndrome in this case. Additionally, earlier blood pressure control could have prevented the development of hypertensive cardiomyopathy and HFrEF. Clinicians should be vigilant in identifying hypertension coupled with urinalysis findings indicative of glomerulonephritis. Early recognition and intervention in such cases are essential to prevent adverse outcomes like those observed in this case.

Poster #7

Subdural Hemorrhage: A Complication of Lumbar Puncture

Joshua Hu, DO[1], Toshiaki Takahashi, MD[1],
Gavin Ha, MD[1], Kuo-Chiang Lian, MD2
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] The Queen's Medical Center, Honolulu, HI

Introduction: Lumbar punctures are a routine component of diagnostic procedures for central nervous system pathology, offering valuable insights despite the potential for complications such as headache, bleeding, infection, spinal hematoma, and, rarely, subdural hemorrhage. Previous studies have explored techniques and instrument types to minimize these complications. Here, we present a case of a young male undergoing a diagnostic lumbar puncture, complicated by subsequent post-dural puncture headache leading to subdural hemorrhage

Case Description: A 31-year-old male, with a history of alcohol and cocaine use, presented with upper respiratory symptoms, generalized headache, and leukocytosis. During his work-up no clear source of infection was identified so he underwent multiple diagnostic tests, including a lumbar puncture in the Emergency Department. This was performed under usual technique in the right lateral decubitus position, with opening pressure of 20mmHg and 10mL fluid removed for studies. The next day it was noted that his prior headache evolved with different characteristics, and was initially attributed to the lumbar puncture. However, despite treatment with common analgesic medications, the headache became refractory, prompting a head CT without contrast the next day. The imaging revealed a new acute 9mm right subdural hematoma. Further investigations, including MRI and angiogram/venogram, yielded no other apparent cause of bleeding. Over the subsequent days, the headache gradually subsided with supportive care, leading to the patient's discharge with outpatient follow-up.

Discussion: Annually, approximately 360,000 lumbar punctures are performed in U.S. Emergency Departments, with an estimated 37% incidence of post-dural puncture headache (PDPH). PDPH typically manifests within five days, remitting after two weeks or with autologous blood patch application. Described as a constant bilateral headache with a postural component, PDPH may present with accompanying symptoms such as nausea, cochlear, or ocular manifestations. The exact etiology remains unclear.

Subdural hematoma represents one potential complication of PDPH. When PDPH loses its postural nature, becomes severe, refractory, or is accompanied by new focal neurological deficits, subdural hematoma formation should be considered.

The incidence of post-dural puncture subdural hematoma is estimated at 1 in 220,000 cases. The proposed mechanism involves the removal of cerebrospinal fluid causing intracranial hypotension, leading to excessive traction and tearing of subdural bridging veins, culminating in hematoma formation. Beyond supportive care, treatment entails reversing anticoagulation and holding antiplatelet agents. Surgical management, is warranted when the hematoma exceeds 10mm or is accompanied by a midline shift greater than 5mm, irrespective of the Glasgow Coma Scale (GCS) score.

Conclusion: This case highlights the interval development of a subdural hematoma following a diagnostic lumbar puncture, necessitating swift advanced imaging to secure the diagnosis. The patient's familial history of migraines and social history of cocaine use added complexity to the clinical presentation. While the exact relationship between these factors and the heightened risk of subdural hematoma remains uncertain, it underscores the importance of considering individual risk profiles in such cases. As medical professionals, comprehensive understanding and awareness of potential complications, especially in patients with predisposing factors, are vital for effective intervention and management.

Poster #8

IgG4-Related Disease: A Polyclonal Disease Masking a Monoclonal Diagnosis

Samuel Hunt, DO[1], Dakota Rodgers, MD[1], Troy Denunzio, DO[2], James Nguyen, MD[2]
[1] Tripler Army Medical Center Internal Medicine Residency Program, Honolulu, HI
[2] Tripler Army Medical Center, Honolulu, HI

IgG4-related disease (IgG4-RD) is an auto-inflammatory disease that can affect virtually any organ system through a lymphoplasmacytic infiltration causing tumefactive fibrosis and organ dysfunction. IgG4-RD is a relatively new entity first discovered in association with autoimmune pancreatitis. It is now classified as a continuum that encompasses several previously named diseases, such as Riedel's thyroiditis or retroperitoneal fibrosis. Here we present a case of IgG4-RD that was initially concerning for chronic myelomonocytic leukemia (CMML) refractory to initial treatment and found to have IgG4-RD.

70 yo male with a past medical history of hypertension and seizure disorder who presents with myalgias, stiffness, fatigue, night sweats, and ESR over 100, originally diagnosed with polymalgia rheumatica. He was treated with 20mg daily prednisone with little improvement. Due to anemia and monocytosis, there was concern for malignancy and underwent bone marrow biopsy, immunofixation, and flow cytometry. This revealed normocellular bone marrow, an IgG kappa monoclonal gammopathy, and a small monotypic B-cell population. Imaging revealed metabolically active lymphadenopathy without osseous lesions. Lymph node biopsies revealed lymphocytic cell population without abnormalities. Treatment was started for presumptive CMML, there was marked increase in lymphadenopathy, increase in serum monoclonal protein, and dramatic worsening of renal function. Kidney biopsy revealed histopathologic signs for IgG4-RD. Prednisone was restarted at 40mg daily with subsequent improvement of anemia, renal function, monoclonal protein resolution, and symptoms.

This patient's initial presentation was concerning for CMML due to his b-symptoms, monocytosis, anemia, and monoclonal gammopathy, however, initial treatment with decitabine and cedazuridine was unsuccessful with interval worsening of kidney function and lymphadenopathy. In addition, there was normocellularity on bone marrow biopsy and only a small monotypic B-cell population on flow cytometry. Differential includes multi-centric Castleman's disease, early myeloproliferative disease, clonal hematopoiesis of indeterminate potential, clonal cytopenia of unknown significance, and IgG4-RD. Interleukin-6 was elevated but vascular endothelial growth factor levels were normal combined with monoclonal expansion as seen on flow cytometry and electrophoresis is atypical for Castleman's. Kidney biopsy confirmed stereotypical histopathologic characteristics for IgG4 related nephritis; this combined with high serum IgG4 levels, and enlarged kidneys with lymphadenopathy confirmed IgG4-RD. The patient was treated with a course of prednisone with improved glomerular filtration, improvement in anemia, disappearance of his monoclonal protein on electrophoresis, and a decrease in lymphadenopathy and symptoms. Monoclonal expansion is usually sufficient for diagnosing myeloproliferative disorder and excluding inflammatory diseases such as Castleman's or IgG4-RD as these diseases are polyclonal. There has been a case report showing an IgG4 producing myeloma, although it responded favorably to chemotherapy. It is likely that this case represents an early form of malignancy that may have triggered an inflammatory response, this malignancy was covered up by corticosteroid treatments. This case illustrates how multiple processes can obfuscate a definitive diagnosis.

Poster #9

A Palliative Care Approach to Skin Cancers: A Case of Advanced Squamous Cell Carcinoma

Ralina Karagenova, MS2[1], David J. Elpern, M.D. [2]
[1] University of Hawai'i John A. Burns School of Medicine, Honolulu, HI
[2] The Skin Clinic, Williamstown, MA

Introduction: Palliative care can improve symptoms, quality of life, and caregiver burden of patients with end-stage conditions1. Its use, however, remains unexplored in non-melanoma skin cancers and outpatient dermatology practices. We present an elderly woman with squamous cell carcinoma who benefited from a palliative care approach.

Case presentation: An 87-year-old woman with history of dementia and squamous cell carcinoma presented with a recurrence of an exophytic tumor of the scalp measuring on the mid-parietal scalp. The lesion was debulked, cultured, and a Xeroform dressing applied. Her daughter-in-law was instructed on how to change the dressings. Pathology showed a moderately differentiated squamous cell carcinoma extending to the base of the specimen. The patient later underwent outpatient Mohs micrographic surgery that showed carcinoma invading into the calvarium. Treatment options included intralesional 5 Fluorouracil (5FU) or topical 5FU and a short course of radiotherapy2. Chemotherapy with pembrolizumab may be a choice for healthier patients3, but was not suitable for this patient. In the end, the patient and her family decided they wanted to forego invasive or other treatments with the goal of supporting her quality of life at her home of many decades. Two months later the patient had a seizure and died. The tumor had eroded the calvarium and exposed her brain. She spent the last few months of her life comfortably at home being attended by her family and a visiting nurse.

Discussion: Squamous cell carcinoma can rarely present with extension to the skull and in this case, its localization, recurrence, and late diagnosis were poor prognostic factors4. There is a role for structured palliative care guidelines for life-limiting cutaneous conditions. Such patients will benefit from the interdisciplinary approach that palliative care offers.

References:
1. Thompson LL, Chen ST, Lawton A, Charrow A. Palliative care in dermatology: A clinical primer, review of the literature, and needs assessment. J Am Acad Dermatol. 2021;85(3):708-717. doi:10.1016/j.jaad.2020.08.029
2. Milena F et. al. A Short course Accelerated RadiatiON therapy (SHARON) dose-escalation trial in older adults head and neck non-melanoma skin cancer. Br. J Radiol. 2022 Jun 1;95(1134):20211347.
3. Burtness B, Harrington KJ, Greil R, et al. Pembrolizumab alone or with chemotherapy versus cetuximab with chemotherapy for recurrent or metastatic squamous cell carcinoma of the head and neck (KEYNOTE-048): a randomised, open-label, phase 3 study [published correction appears in Lancet. 2020 Jan 25;395(10220):272] [published correction appears in Lancet. 2020 Feb 22;395(10224):564] [published correction appears in Lancet. 2021 Jun 12;397(10291):2252]. Lancet. 2019;394(10212):1915-1928. doi:10.1016/S0140-6736(19)32591-7
4. Fidanzi C, Davini G, Dini V, et al. Palliative management of a recurrent destructive cutaneous squamous cell carcinoma of the scalp with brain exposure. Wounds. 2022;34(1):E7-E9. doi:10.25270/wnds/2022.e79

Poster #10

Chronicles of Insomnia: A Case of Non-24 Hour Circadian Sleep Rhythm Disorder in a Patient with Unilateral Blindness

Kristen A. Kircher, DO[1], Janet N. Myers, MD, FACP[2]
[1] Kaiser Permanente Hawaii Internal Medicine Residency Program, Honolulu, HI
[2] Kaiser Permanente Group, Honolulu, HI

Chronic insomnia is a common yet debilitating symptom that is often treated with cognitive behavioral therapy as a first-line therapy, however there are circumstances in which pharmacotherapy is useful. In patients with chronic insomnia refractory to usual treatment, it is important to investigate alternative causes. We present a case of a 27-year-old male who initially presented for evaluation of severe chronic insomnia, significantly impairing his everyday life and eventually leading to a diagnosis of major depressive disorder (MDD), now in remission, however insomnia remained persistent. Usual practices such as improvement in sleep hygiene, avoidance of blue light and limitation of caffeinated beverages were initiated without improvement. Upon further questioning, the patient reported he had a fitness sleep tracker, which seemed to show a predictable daily advancement in his sleep cycle. The patient additionally reported vision problems. He subsequently underwent a sleep study, which did not show evidence of sleep disordered breathing or other abnormalities. He was referred to ophthalmology for evaluation with noted history of congenital keratoconus, with significant visual impairment in his left eye (L eye 20/200, R eye 20/30). He underwent Actigraphy, and results were consistent with Non-24 hour Circadian Sleep Rhythm Disorder (N24SWD). N24WD is a sleep disorder usually noted in blind patients. Sleep disorders such as N24SWD are a result of the dysregulation of the circadian rhythm, which is controlled by the suprachiasmatic nuclei, which is regulated based on the daily light-dark cycle detected by the retina and synchronized to a 24-hour time period. In patients who suffer from visual impairments, the suprachiasmatic nucleus is unable to detect environmental cues and therefore cannot synchronize the patient's circadian rhythm to a normal 24-hour day. Our patient is unique, as his vision in his right eye is intact with 20/30 vision, therefore only blind in his left eye. The patient was prescribed melatonin initially with minimal improvement in symptoms. After initiation of Tasimelteon, a recently approved potent dual melatonin receptor agonist, the patient reported remarkable improvement in symptoms. This is the only medication that has been studied and proven to be effective in N24SWD and is the only approved treatment for N24SWD. This case highlights the importance of a thorough history and review of symptoms in patient's suffering from chronic insomnia. Clinicians should be aware of circadian rhythm sleep disorders that may masquerade as insomnia. Our case is a rare case of a patient that has unilateral visual impairment suffering from a N24SWD that demonstrated dramatic improvement with a melatonin receptor agonist.

Digital Poster #11

What's In a Name? Familial Atypical Multiple Mole Melanoma Syndrome: Patients at Risk For More Than Just Melanoma

Andrew J. Landau, MD[1], Detainee Morris, MD[1], Yula Geniza, MD[1],
Rowena Feng, MS[2], Pedro Manibusan, DO[3]
[1] Tripler Army Medical Center, Internal Medicine Residency Program, Honolulu, HI
[2] AT Still University, Mesa, AZ
[3] Tripler Army Medical Center, Honolulu, HI

Introduction:

Familial Atypical Multiple Mole Melanoma Syndrome (FAMMM) is an autosomal dominantly inherited syndrome associated with mutations in the CDKN2A gene on chromosome p9.21, which encodes multiple tumor suppressor genes, resulting in uncontrolled cell proliferation. People with FAMMM are at increased risk for melanoma and pancreatic cancer (mostly adenocarcinoma), and may be at increased risk for other malignancies including breast, renal, prostate, lung, esophageal, gastric and colorectal. Penetrance of pancreatic cancer in patients with CDKN2A mutations is estimated to be as high as 17% by age 75. It is important to recognize and understand the high risk for pancreatic and other gastrointestinal malignancies.

Case:

57-year-old female with history of GERD, hiatal hernia, esophagitis, gastritis, duodenitis, chronic iron deficiency anemia and known CDKN2A mutation without previous work up who presented to gastroenterology clinic for further evaluation and recommendations regarding her CDKN2A mutation. Family history is significant for FAMMM, pancreatic cancer in multiple first-degree relatives, melanoma, and colon cancer. She endorses some dysphagia and mild abdominal pain but no other new gastrointestinal symptoms. She had an EGD and colonoscopy 2 years ago; a colonic perineuroma was found and she was advised to repeat colonoscopy in 1-2 years. CT scan 3 months prior without new abnormalities and normal appearing pancreas. The patient underwent EGD with endoscopic ultrasound (EUS), and colonoscopy. EUS was unremarkable with normal appearing pancreas and no appreciable lymphadenopathy. There were no significant findings on EGD or colonoscopy. She will follow up annually for screening with imaging (CT or MRI) and/or EUS.

Discussion:

FAMMM is a rare hereditary syndrome that predisposes patients to melanoma, pancreatic and other malignancies. Pancreatic cancer risk is estimated to be as high as 17 % by 75 years of age. Furthermore, patients with a family history of pancreatic cancer and smokers are more likely to develop pancreatic cancer. While no guidelines exist to support pancreatic cancer screening, some experts recommend annual screening with CT, MRI/MRCP or EUS, with EUS being most sensitive for detecting pancreatic cancer. Increased frequency of screening colonoscopies may also be considered. It is important to understand the associated malignancies and have an active cancer screening plan when treating patients with FAMMM.

Poster #12

m&M: A Practical and Universal Equation to Calculate Ventilator Mechanical Power

Philip M. Lee, MS[1], Ehab Daoud, MD, FACP, FCCP[2]
[1] University of Hawai'i John A. Burns School of Medicine, Honolulu, HI
[2] Kuakini Health Center, Honolulu, HI

Mechanical power (MP) is an important surrogate for the energy delivered by a mechanical ventilator onto the lungs. It incorporates all the variables participating in ventilator-induced lung injury, including driving pressure, tidal volume, end expiratory positive pressure, and respiratory rate. The pitfall of mechanical power is its mathematical complexity. Although prior studies have attempted to create simplified equations, they lack clinical utility as calculations cannot be done by solely looking at ventilator settings, or they require manipulation of variables. There are also different formulas depending on the type of mode, such as volume versus pressure-controlled ventilation. This study offers a simplified, universal equation called the m&M equation which makes MP's clinical application more feasible. The equation utilizes 3 variables to calculate mechanical power, which can all be accessed bedside from the ventilator settings. The m&M formula is as follows: MP = 1.7 + (0.15 • mM), where mM = respiratory rate (bpm) • mean airway pressure (J/min) • tidal volume (mL).

Using the online SIVA simulator, four different respiratory mechanics scenarios in both the volume and pressure-controlled modes were created by adjusting different ventilator settings like resistance and compliance. More than 1,000 values were collected by adjusting different variables of mechanical power. Mechanical power was then calculated in 2 ways, either from the m&M equation or the current gold standard of geometrically deriving the area under the Pressure-Volume curve. A linear regression model and Pearson correlation were run to compare the relationship of these equations. There was a statistically significant linear relationship ($p > 0.001$) and strong correlation of determination ($R2 = 0.96$) between the m&M formula and the gold-standard method of calculating mechanical power.

The m&M equation is a reliable method of calculating mechanical power. The simplicity and universal nature of its calculation provides significant clinical utility. Physicians may consider using this equation in medical management of patients to minimize VILI.

Poster #13

An Uncommon Presentation of Cyclic Neutropenia in an Adult Active-Duty Male

Roy Levit, MD[1], Nicholas G Richwagen, MD[1,] Kelly Sun, MD[2], Ryan M Jones, MD[2]
[1] Tripler Army Medical Center Internal Medicine Residency, Honolulu, HI
[2] Tripler Army Medical Center Honolulu, HI

Introduction:

Cyclic neutropenia is a rare hematological disorder characterized by recurrent neutropenia, typically diagnosed in childhood. It occurs roughly in 1 of 1 million individuals. Presented is an unusual case of cyclic neutropenia in a 21-year-old active-duty male who was deployed to Japan. This case is particularly unique as the patient presented in adulthood, which is a rare occurrence.

Case presentation:

The patient presented with recurring fevers, hip pains, oropharyngeal ulcers, and lymphadenitis, with each episode lasting approximately one week. His vitals were stable and physical exam confirmed an oropharyngeal ulcer. Laboratory evaluations revealed severe Neutropenia (ANC 0.1) and a positive streptococcal infection during episodes, which required treatment with antibiotics. This infection happened soon after moving from his childhood home and being deployed to Japan for the first time.

The patient displayed a periodicity of neutropenia every 3 weeks raising suspicion of cyclic neutropenia. Genetic testing confirmed a heterozygous pathogenic mutation in the ELANE gene, confirming the diagnosis of the disease. Treatment with granulocyte colony-stimulating factor (G-CSF) resulted in appropriate increase in neutrophil counts and resolution of symptoms.

Conclusion:

This case highlights the rare occurrence of cyclic neutropenia in adulthood and the importance of considering the diagnosis in adult patients with recurrent infections, fever, and oral ulcers. While the etiology of cyclic neutropenia is not fully known, genetic factors, particularly mutations in the ELANE gene, play a crucial role in its pathogenesis. The patient tested positive for a heterozygous pathogenic mutation in the ELANE gene 416c>Tpro139Leu. This is a rare variant in the ELANE gene on chromosome 19. While being a dominant mutation leading to cyclic neutropenia, several carriers of the mutation have no signs or history of neutropenia. A de novo mutation is possible. However, it is also possible that the single nucleotide variant was inherited, and the phenotypic presentation of cyclic neutropenia arose later due to extrinsic and intrinsic factors affecting its expression.

Early recognition and treatment with G-CSF are essential for managing cyclic neutropenia, with individualized dosing regimens necessary and requiring close follow-up to maintain stable neutrophil counts.

Poster #14

A Case of Cyclic Thrombocytopenia, Navigating a Diagnostic Difficulty

Roy Levit, MD1; Karen J Shou, DO2; Jeffrey L Berenberg, MD, MACP2
[1] Tripler Army Medical Center Internal Medicine Residency Program, Honolulu, HI
[2] Tripler Army Medical Center, Honolulu, HI

Cyclic thrombocytopenia is a rare hematological disorder characterized by recurrent thrombocytopenia. Platelets may fluctuate over an interval of weeks and can fall to <5x10^3/µL, which is usually followed by a rapid increase to normal or elevated levels. The incidence is low, with less than 70 cases to date. Presented is a case of cyclic thrombocytopenia in a 27-year-old female. She was previously given the diagnosis of immune thrombocytopenic purpura (ITP) and received intravenous immunoglobulin (IVIG) and dexamethasone before moving to Hawaii. Her history is significant for seizure disorder secondary to cerebral palsy, requiring chronic treatment with multiple antiepileptic medications.

The patient initially presented with palpable purpura in her bilateral lower extremities, and her vitals were stable. Labs showed a platelet count of 2x10^3/µL. She was treated for ITP and given IVIG and dexamethasone. After treatment, her platelets increased to 62x10^3/µL. 4 weeks later, her platelet count fell to 51x10^3/µL. One week later, the platelets increased to 118x10^3/µL. Platelets were checked 4 weeks later and once again fell to 18x10^3/µL. At this time, she was given dexamethasone and rituximab and responded well initially; however, after 3 weeks, her platelets fell to 3x10^3/µL. Due to this, she was given prednisone 40 mg daily and eltrombopag. Her platelet count continued to fluctuate despite receiving treatment, rising to 685x10^3/µL before decreasing to 8x10^3/µL, which raised suspicion of cyclic thrombocytopenia. The evaluation showed an oscillation of her platelet count with a periodicity of 3-4 weeks. Oscillation was not observed with other cell lines. Eltrombopag is planned to be halted to observe and monitor the periodicity of thrombocytopenia over two months.

This case highlights the rare occurrence of cyclic thrombocytopenia and its susceptibility to be misdiagnosed as ITP. While the etiology of cyclic thrombocytopenia is not fully understood, impaired regulatory mechanisms, increased platelet destruction, or decreased bone marrow production could be at play. When platelets fall dangerously low, patients are at an increased risk of bleeding complications such as life-threatening gynecological bleeding and hemorrhagic stroke. Treatment involves a trial of cyclosporine or danazol. However, patients often do not respond to therapy. Cases have been reported of spontaneous resolution.
Cyclic thrombocytopenia should be considered in patients with a diagnosis of ITP who failed to respond to treatment and show periodic normal or high platelets. Suspending treatment or observation of cyclical platelet counts can unmask the diagnosis. Afterwards, treatment for ITP should be avoided to avoid harm.

Poster #15

Empyema due to Streptococcus intermedius: Uncommon consequence of Periodontal Diseases

Sharina C. Macapagal MD[1], Christian John Capirig MD[1],
Andrew Pham MD[1], Titus David MD[1], Yoshito Nishimura MD, PhD, MPH[1]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI

Introduction:

S. intermedius infection is uncommon in previously healthy people without apparent predisposing factors. Despite being an oral commensal bacterium, it can cause complicated infections that can present as liver, brain, and lung abscesses. Herein, we present a rare case of empyema due to S. intermedius in a healthy man.

Case Presentation:

An 82-year-old male with a history of hypertension and non-toxic multinodular goiter presented to the emergency room with a two-week history of dyspnea associated with intermittent fevers, chills, undocumented weight loss, dysphagia, and productive coughs. Despite a five-day course of Azithromycin, his symptoms persisted. No hemoptysis, night sweats, palpable lymphadenopathy, chest pain, or exposure to sick contacts were reported. He is a former tobacco smoker and denies illicit drug use. He traveled to Japan and Guam a month before his emergency room presentation.
Physical examination showed sinus tachycardia, tachypnea, and hypoxia without fever. While he had good dentition, there were dental caries. His left lung demonstrated decreased breath sounds with dullness to percussion and egophony.

Initial work-up revealed neutrophil-predominant leukocytosis of 32,400 (92.4% neutrophils and 3.8% lymphocytes). The basic metabolic panel was normal. Chest x-ray and computed tomography (CT) showed a large loculated left pleural effusion with the mediastinal shift to the right. A chest tube was inserted at the posterior left hemithorax, which initially drained purulent fluid with subsequent serosanguinous drainage. He was admitted with a presumptive diagnosis of left lung empyema, and he was started on intravenous ampicillin/sulbactam 3g every six hours. The pleural fluid was exudative, and the culture grew S. intermedius. He later required intrapleural fibrinolytic therapy, however, it was partially effective as repeat CT chest showed the presence of residual loculated effusions. As a potential nidus of recurrence, surgical decortication was performed. The source of the infection was considered to be a subclinical periodontal infection.

Discussion:

The case described the development of a large empyema due to S. intermedius in a relatively healthy patient. While S. Intermedius belongs to the S. anginosus group, which is a part of common oral floras, it is the most pathogenic among the group associated with significantly more extended hospital stay and morbidity compared to other species [1]. The most common risk factors for S. intermedius infections include dental manipulation and sinusitis [2]. While the exact cause of empyema in this patient is unclear, It might be attributed to a subclinical periodontal disease or silent aspiration of dental plaques [3]. In cases of severe S. intermedius infections, thorough oropharyngeal exams should be performed to identify an underlying cause.

Poster #16

Keeping up with the Jones? A case of adult, acute rheumatic fever with carditis

Brett W. Mathews, MD[1], Yuree J. Lin, MD[2], and Philip A. Verhoef, MD, PhD[1,2]
[1] Kaiser Permanente Hawaii Internal Medicine Residency Program, Honolulu, HI
[2] Hawaii Permanente Medical Group, Honolulu, HI

Introduction:

One of the nonsuppurative complications of group A Streptococcus (GAS) infection, acute rheumatic fever, is estimated to affect over 400,000 individuals worldwide annually. Prompt diagnosis and treatment is crucial as over 300,000 deaths worldwide are attributable to rheumatic heart disease each year. The majority of cases occur in resource-poor and low-middle income countries, impacting predominantly children aged 5 to 15 years of age. However, adults may present with primary acute rheumatic fever in resource-rich areas. Further, treatment recommendations may need to be sought from pediatric specialists, adding to the management challenge for providers who care for adult patients.

Case:

20-year-old female with history of obesity (BMI 34) and prediabetes presented to the ER with 6 days of subjective fever, substernal chest pain, shortness of breath, nausea, vomiting, and intermittent joint pains first starting in the feet than moving to her knees and shoulders. She had no prior episodes of chest pain or joint pain, and she denied neurologic symptoms, sore throat, cough, abdominal pain, diarrhea, or genitourinary symptoms. She had previously been seen at Urgent Care 3 days prior to presentation for myalgia and fatigue where she had normal vitals, physical exam, chest x-ray, and ECG and was discharged home. Initial ER vitals were T 99.3 HR 110 BP 106/53 RR 20 O2 98% on room air. Physical exam was notable for tenderness to palpation over the sternum, neck and right shoulder; no erythema or edema over the joint but reduced ROM with abduction; clear oropharynx; clear lungs; unremarkable cardiovascular exam apart from tachycardia; benign abdomen; and unremarkable neurologic exam. Labs were notable for leukocytosis 11.7, ESR 119, elevated D-dimer 3.9, elevated Troponin I 38, elevated BNP 357. ECG revealed sinus rhythm but with new first-degree AV block. CXR and CTA Chest unremarkable. TTE demonstrated preserved EF but mild-moderate mitral regurgitation. Rheumatologic work up including ANA, anti-CCP, RF, and complements were unremarkable. Blood cultures were negative. Strep PCR eventually returned positive, and additional questioning revealed that the patient was unhoused and sleeping in a car with her brother who recently had been admitted for GAS cellulitis. Upon consultation with Pediatric Cardiology and Pediatric Rheumatology, she was subsequently treated with naproxen, given a dose of IM benzathine penicillin, and discharged home with Pediatric Cardiology follow up.

Discussion:

The Revised Jones Criteria are used for diagnosing acute rheumatic fever; the 5 major manifestations include arthritis, carditis and valvulitis, CNS involvement (chorea), subcutaneous nodules, and erythema marginatum; the 4 minor manifestations include arthralgia, fever, elevated acute phase reactants (ESR, CRP), and prolonged PR interval on ECG. This patient met 2 major criteria (carditis, arthritis) and 3 minor criteria (fever, elevated ESR and CRP, and prolonged PR interval). Treatment of GAS pharyngitis is predominantly to prevent rheumatic fever and late sequelae.

Poster #17

A Case of Metastatic Intrahepatic Cholangiocarcinoma Without Risk Factors

Destinee Morris, MD[1], Andrew Landau, DO[1], Yula Geniza, MD[1],
John Lee, MD[2], Pedro Manibusan, DO[2]
[1] Tripler Army Medical Center Internal Medicine Residency Program, Honolulu, HI
[2] Tripler Army Medical Center, Honolulu, HI

Introduction: While intrahepatic cholangiocarcinoma is rare in high income countries such as the United States, its incidence has been increasing over the past four decades worldwide (1). Cholangiocarcinoma is an aggressive malignancy arriving from bile duct epithelial cells that can be subdivided into intrahepatic and extrahepatic lesions. Recognized risk factors for intrahepatic cholangiocarcinoma include primary hepatobiliary disease such as primary sclerosing cholangitis, cholelithiasis, cholecystitis, hepatolithiasis, chronic liver disease, genetic disorders, toxic exposures, infections, and metabolic syndrome. However, many patients do not have any of these risk factors (2). We describe a case of metastatic intrahepatic cholangiocarcinoma in a patient with no risk factors.

Case Presentation: A 43-year-old male presented to his primary care physician with two weeks of persistent right upper quadrant abdominal pain for 2 weeks. He described the pain as a sharp, stabbing pain that starts under his right rib cage and radiates to right upper back when he takes in a deep breath. He also endorsed intermittent nausea and decreased appetited. He denied changes to his bowel movements. He had never smoked or drank alcohol. He had no history of gallbladder or liver disease. Family history was negative for gastrointestinal disease or disorders. His exam was notable for tenderness to palpation in the right upper quadrant with mild guarding with palpation. His vital signs were unremarkable. His labs revealed elevated alkaline phosphatase (495), alanine transaminase (167), and aspartate transaminase (108) and negative hepatitis panel. Right upper quadrat ultrasound showed a right hepatic lobe mass with enlarged porta hepatitis lymph nodes and dilated common bile duct measuring 7mm. CT liver showed right hepatic lobe masses with adjacent lymphadenopathy and nodular liver contours consistent with cirrhosis. Interventional radiology performed liver biopsy and resulting pathology showed poorly differentiated adenocarcinoma. Gastroenterology performed esophagogastroduodenoscopy (EGD) with endoscopic ultrasound (EUS), colonoscopy, and lymph node fine needle aspiration (FNA). His EGD and colonoscopy were unremarkable and EUS showed enlarged porta hepatis lymph nodes. FNA of periportal lymph nodes showed no malignant cells on pathology. PET scan revealed multiple hypermetabolic lesions in the liver, mediastinum, lungs, and abdominal wall consistent with metastatic cancer. He was diagnosed with stage IV metastatic intrahepatic cholangiocarcinoma. He was seen by oncology and initiated on treatment with cisplatin, durvalumab, and gemcitabine for palliative chemotherapy.

Discussion
Our patient's next generation sequencing returned showing several sporadic genetic mutations that likely led to his development of cholangiocarcinoma to include CDKN2A, SMAD4, and TP53. This suggests cholangiocarcinoma may evolve based on sporadic genetic mutations alone without any other risk factors. As incidence is increasing, it is important that further research is pursued to determine pathogenetic pathways or molecular markers that could help in early detection, more specific treatment, and more favorable outcomes.

References
1. Saha SK, et al. Forty-Year Trends in Cholangiocarcinoma Incidence in the U.S.: Intrahepatic Disease on the Rise. Oncologist. 2016 May;21(5):594-9.
2. Palmer WC, Patel T. Are common factors involved in the pathogenesis of primary liver cancers? A meta-analysis of risk factors for intrahepatic cholangiocarcinoma. J Hepatol. 2012 Jul;57(1):69-76.

Poster #18

Pure Red Cell Aplasia- A Diagnostic Conundrum

Hema Narlapati DO[1], Ryan Jones MD[2], James Nguyen MD[2],
Karen Shou, DO[2], Jeffrey Berenberg MD MACP[2]
[1] Tripler Army Medical Center, Internal Medicine Training Program, Honolulu, HI
[2] Tripler Army Medical Center, Honolulu, HI

Introduction: Pure red cell aplasia (PRCA) is a rare disorder manifested as low reticulocyte count, normochromic normocytic anemia, and bone marrow absent of mature erythroid precursors in an otherwise normocellular bone marrow. Specifically, hallmark laboratory findings are isolated anemia with severe reticulocytopenia with an absolute reticulocyte count of under 10,000/microL, with definitive diagnosis proved with bone marrow biopsy revealing hypocellular erythroblast lineage (<1% makeup of total marrow), without abnormalities in the lymphocyteic, mekakargocyte, or myeloid lineages. Though common cases of acquired PRCA are from transient viral attack of red blood cell precursors like parvovirus B19, acquired pure red cell aplasia requires prompt identification commonly associated disorders to treat the underlying disease process and to recognize those who may benefit from immunosuppressive therapy. This case not only illustrates the diagnostic difficulty for pure red cell aplasia, but begs the question of whether PRCA should remain in diagnostic schemas to prevent later diagnoses which could portend a poor prognosis due to delay in treatment.

Case Presentation: A 79-year old male with a history of hypertension, hyperlipidemia and hyperthyroidism was admitted for acute anemia with a hemoglobin of 5.4 g/dL. With a history of chronic NSAID use of ibuprofen for knee pain, and reported melena, he underwent capsule endoscopy which revealed a clean based duodenal ulcer, and without and identified lower gastrointestinal bleed source identified on subsequent colonoscopy. He was started on an 8-week course of pantoprazole. Since initial presentation, his reticulocyte count was persistently low, initially at 0.3%. Iron panel in the acute phase noted an elevated ferritin. Despite guideline directed management for suspected upper gastrointestinal bleed, he continued to require red blood cell transfusions over the course of many weeks. Given the reticulocytopenia, and suspicion for pure red cell aplasia versus a myelodysplastic syndrome, the patient underwent a bone marrow biopsy, with findings consistent with pure red cell aplasia evident by absence of red blood cell precursors, minor increased reticulin, normal white blood cell morphology, and normal megakaryocytes. No chromosome or FISH abnormalities were noted from bone marrow biopsy. Parvovirus, Epstein Barr Virus, and cytomegalovirus were all negative. The patient was started on cyclosporine but had developed toxicity of rhabdomyolysis, hence treatment course was held for two weeks to allow for recovery. To this day, patient remains with improved hemoglobin levels ranging between 9.5-10.1 g/dL on cyclosporine without additional adverse effects.

Discussion: Given the rarity of pure red cell aplasia, earlier suspicions may be difficult, especially in the setting of more common etiologies to rule out. However, prompt diagnosis of PRCA would not only avoid unnecessary treatments, but allow for the clinician to identify whether a reversible secondary disease process can be managed to aid resolution of an acquired pure red cell aplasia. Additionally, early identification of pure red cell aplasia would prevent progression towards life threatening anemia and transfusion related complications from repeated blood transfusions, while allowing for timely treatment if immunosuppression remains an option for those without spontaneous remission or with explanation for an underlying etiology.

Poster #19

Catastrophic Antiphospholipid Syndrome: A Rare, Life-Threatening Complication in Systemic Lupus Erythematosus

Kevin Edward Nebrejas, MD[1], Manasawee Tanariyakul, MD[1], Kuo-Chiang Luo, MD[2]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] The Queen's University Medical Group, Honolulu, HI

A 33-year-old Micronesian woman presented as an inter-island transfer to our institution following a recent biopsy-proven diagnosis of class IV lupus nephritis (LN). She was initially treated with mycophenolate mofetil (MMF), hydroxychloroquine (HCQ) and prednisone, but developed hemoptysis with acute hypoxic respiratory failure requiring intubation and mechanical ventilation. CT scan of the chest demonstrated diffuse bilateral reticulonodular infiltrates and bronchoscopy revealed diffuse alveolar hemorrhage (DAH).

Laboratory evaluation revealed renal failure (SCr= 4.4mg/dL), hyperkalemia, metabolic acidosis, elevated anti-dsDNA, and low C3 and C4. Furthermore, the patient exhibited hemolytic anemia with blood smear consistent with microangiopathic hemolytic anemia (MAHA). ADAMTS13 level was normal. She was started on rituximab and pulse methylprednisolone. With improving respiratory status, the patient was later extubated. However, she subsequently developed neuropsychiatric symptoms with behavioral change and visual hallucination. MRI brain demonstrated diffuse punctate foci of acute to subacute infarction with narrowing of bilateral ACA, MCA, and left PCA.

Her hospital course was further complicated with widespread venous thrombosis (VTE) which initially presented as clotting of the guidewire during femoral non-tunneled dialysis catheter placement for continuous renal replacement therapy (CRRT). Imaging revealed VTE of upper and lower extremities of the left basilic and cephalic vein, right common femoral vein, left gastrocnemius, popliteal and femoral vein. Triple positive anti-phospholipid antibodies (anti-cardiolipin, beta-2-glycoprotein, and lupus anticoagulant) were consistent with findings for catastrophic antiphospholipid antibody syndrome (CAPS).

The patient initially received one dose of cyclophosphamide for LN and DAH, but was then switched to rituximab for a total of 4 doses due to persistent leukopenia. For CAPS, she was treated with heparin drip, prednisone, IVIg, and 6 cycles of plasmapheresis. Additionally, treatment included continuation of MMF. She gradually improved and eventually demonstrated evidence of good renal recovery with discontinuation of CRRT. Upon discharge, the patient transitioned to warfarin, MMF, prednisone, HCQ, and atovaquone.

The case highlights a rare presentation of CAPS in a patient newly diagnosed with Systemic Lupus Erythematosus (SLE) complicated with class IV LN. Her presentation fulfills the new 2023 ACR criteria for APS with the presence of at least 3 clinical domains and 2 laboratory domains. Clinically, the patient developed manifestations of VTE, intracerebral arterial thrombosis, suspected microvascular complication manifesting as pulmonary hemorrhage, livedo racemosa, and MAHA with thrombocytopenia. Laboratory criteria were met with triple aPL-positivity [1]. A minority of patients can demonstrate more accelerated life-threatening thrombosis and cataclysmic variant of APS termed CAPS using the Asherson criteria. CAPS has a more rapid onset with evidence of multi-organ involvement. Cornerstone to medical management is triple therapy consisting of anticoagulation, glucocorticoids, and therapeutic plasma exchange with IVIg. Due to the severity of presentation, the patient received additional immunosuppressive therapy. Prompt recognition and management of underlying CAPS is crucial given the mortality rate is as high as 30% [2,3].

Digital Poster #20

Confusion After Car Crash- An Unusual Presentation of Primary CNS Lymphoma

Jadon Neuendorf, MD[1], Sable Neuendorf, MS[2]
[1] University of Hawai‘i Internal Medicine Residency Program, Honolulu, HI
[2] California University of Science and Medicine

Introduction:
Primary central nervous system lymphoma (PCNSL), a non-Hodgkin lymphoma, is a rare condition with a rising incidence in the elderly population over the last two decades. The cause for the rising incidence is largely unknown, however aging of the global population and increased availability of and advances in diagnostic imaging are suspected to play a role. The most common clinical presentation of PCNSL involves focal neurologic deficits due to intracranial lesions. However, generalized neurocognitive changes may be present and falsely attributed to the aging process. Considering that PCNSL can involve different parts of the central nervous system, clinical presentation can be highly variable and vague systemic symptoms may be the presenting chief complaint.

Case Presentation:
We present a case of a 73 year-old male who presented to the emergency department status-post motor vehicle collision due to suspected confusion while driving. Elicited history included nine weeks of intermittent fevers with chills, night sweats, decreased appetite, weight loss, gait instability, and episodes of inattentiveness. The patient's family member had observed these inattentive episodes during meals but shared that the patient was arousable from them without difficulty. There was no reported seizure-like activity and the patient had no somnolence or weakness after these episodes. There were no reported vision changes or headaches. The patient reported he was up-to-date on his health screenings, his only recent travel was to California, and he had no unusual exposures.

On admission, the patient appeared appropriately nourished, although lethargic. He was noted to have a waxing and waning level of alertness, occasionally becoming inattentive and disoriented. He demonstrated a sluggish affect and slowed speech. Physical exam was notable for trauma to his right knee and a superficial laceration to his scalp, but was otherwise unremarkable. There was no cervical or axillary lymphadenopathy and no focal neurologic deficits. A noncontrast head CT was obtained due to the mechanism of injury which showed no intracranial bleed, however hypodensities were noted in the bilateral frontal lobes and left parieto-occipital lobes. The radiology impression indicated possible vasogenic edema. An MRI brain with and without contrast was obtained which showed multifocal enhancing lesions involving the anterior corpus callosum and left occipital lobe. These findings were concerning for a multifocal glioma or possible lymphoma and the radiology impression indicated the pattern was not typical of metastases, infarcts, or infection. EEG was negative. An infectious work-up including lumbar puncture with CSF testing was unremarkable and CSF cytology was negative for a clonal B or abnormal T-cell population. CT imaging of the chest, abdomen and pelvis was unremarkable and MRI of the spine did not demonstrate any lesions. Ultimately, a brain biopsy was performed and demonstrated Stage IE diffuse large B-cell lymphoma and a diagnosis of primary CNS lymphoma was made.

Discussion: Considering the variability in clinical presentation of PCNSL, clinicians should maintain high suspicion for this condition within a differential diagnosis for the elderly population in whom symptoms may be mistaken for age-related cognitive decline or other systemic illness.

Poster #21

A rare presentation of sarcoidosis as a large pulmonary mass

Sorawit Ongsupankul, MD[1], Witina Techasatian, MD[1], Bradley Fujiuchi, MD[1], Steven Namikim MD[1], Chalothorn Wannaphut, MD[1], Thanaboon Yinadsawaphan, MD[1], Sharina Macapagal, MD[1], Yusuke Hirao, MD[1]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI

Background: Sarcoidosis is an autoimmune disease with a diverse range of presentations. Common manifestations include constitutional symptoms, bilateral adenopathy, and reticulonodular opacity, while the presence of a solitary mass is rare. This disease is known for its ability to mimic malignancy, infection, and other granulomatous diseases. In this case, we discuss a patient with initially suspected lung malignancy based on imaging and preliminary pathological findings, highlighting the diagnostic challenges posed by sarcoidosis.

Case summary: We present a case involving a 52-year-old Caucasian woman with a 10-pack-year smoking history and chronic bronchitis who presented with shortness of breath persisting for two months. She was found to be hypoxic, and her chest X-ray showed infiltration in the right lower lobe. Initially treated for pneumonia, she showed no improvement in symptoms or radiological findings. A CT scan of the chest revealed focal, irregularly marginated, mass-like pulmonary consolidation in the right lower lobe, measuring up to 3.5 cm, accompanied by adjacent reticulonodular opacities and hilar lymphadenopathy. The patient was advised to continue with the budesonide/formoterol inhaler, along with albuterol and ipratropium bromide rescue inhalers. Six months later, a repeat CT scan displayed an enlarging mass-like consolidation in the right lower lobe, now measuring up to 6.3 cm, with stable mediastinal lymphadenopathy compared to the previous scan. Given the possibility of malignancy, an endobronchial ultrasound with transbronchial needle aspiration and transthoracic biopsy were performed. The initial preliminary results suggested small cell carcinoma of the lung, but the final analysis revealed non-caseating granulomas. The tests were also negative for acid-fast bacilli and fungal stains. She was diagnosed with stage 2 sarcoidosis and started on prednisone at 1 mg/kg. Her symptoms, including cough, sputum production, and dyspnea, subsequently resolved. Serial repeat CT scans at 6 and 17 weeks showed a progressive size reduction in the mass-like infiltration in her right lower lobe. The patient continued on a corticosteroid taper without a recurrence of her pulmonary symptoms.

Discussion: Atypical presentations of sarcoidosis are noted in 15-25% of cases, where patients may exhibit diffuse ground-glass opacities (GGO), honeycombing, multiple nodules, or necrotizing consolidation. Although mass-like infiltrations that mimic malignancy or infection are rare in sarcoidosis, there have been reports of nodular sarcoidosis with sizes reaching up to 7.5 cm.

It's important to note that local sarcoid reactions, characterized by non-caseating granulomas, can occur in or near tumors or regional draining lymph nodes in cases of various malignancies. The case discussed here exemplifies these diagnostic challenges: initial biopsies raised concerns for small cell lung cancer but were later confirmed to be non-caseating granulomas, highlighting the complexities in diagnosing certain cases of sarcoidosis.

Conclusion: The diagnosis of atypical manifestations of sarcoidosis presents significant challenges due to its resemblance to malignancy, infection, and other granulomatous lung diseases. When confronted with a lung mass, it is imperative to adopt a broad differential diagnosis. However, it is crucial to prioritize the exclusion of lung malignancy if suspicion is present.

Poster #22

A Case Report of Achromobacter xylosoxidans Cholangitis in a Patient with Hilar Cholangiocarcinoma

Nicholas Richwagen, MD[1], Joseph Rhee[2], Javier Barranco-Trabi, MD[1], Matthew Perkins, MD[3]; Edwin Kamau, PhD[4]

[1] Tripler Army Medical Center Internal Medicine Residency, Honolulu, HI
[2] Uniformed Services University of the Health Sciences, Bethesda, MD
[3] Tripler Army Medical Center, Honolulu, HI
[4] Tripler Army Medical Center, Department of Laboratory Science, Honolulu, HI

Introduction: Bacteria in the genus Achromobacter are increasingly linked to fatal infections, in particular devastating pneumonia in cystic fibrosis (CF) patients. Less common but still significant infections involve lung disease and bacteremia in non-CF hosts, with more rare manifestations documented across all organ systems. Risk of infection with these gram-negative, rod-shaped bacteria is highest among immunocompromised individuals and patients with indwelling lines, particularly those in the tropics. Globally, highly resistant Achromobacter infections are increasing in incidence. This report presents an unusual case of Achromobacter cholangitis, with only three prior documented cases.

Patient Case: A 73-year-old male with complaints of right upper quadrant abdominal pain, vomiting, and diarrhea was transferred from Guam to our hospital for evaluation of biliary obstruction. A percutaneous transhepatic cholangiography (PTC) was performed due to worsening of clinical condition after cholecystectomy for suspicion of choledocholithiasis or cholangitis. This resulted in stent and external drain placement for decompression. Laboratory results upon admission revealed leukocytosis and elevated liver function tests. He was placed on ciprofloxacin and metronidazole prior to transfer.

To evaluate the obstruction, Endoscopic Retrograde Cholangiopancreatography (ERCP) and MRI cholangiopancreatography (MRCP) suggested hilar cholangiocarcinoma (CCA) which was later confirmed by pathology. The patient developed fever and leukocytosis, despite a course of antibiotics, before a planned exploratory laparotomy which found the cholangiocarcinoma too extensive for surgical removal.

Following surgery, Achromobacter xylosoxidans was cultured in the patient's blood, which quickly progressed to sepsis. The hospital's infectious disease service was consulted. Antibiotics were changed to piperacillin-tazobactam and later cefiderocol, though bacteremia persisted despite initial clearance. Bile was identified as a bacterial reservoir. In the setting of persistent bacteremia and non-resectable CCA, the patient decided to pursue palliative management.

Discussion: Achromobacter is an uncommon human pathogen, mostly linked to CF pneumonia and less frequently with bacteremia in immunosuppressed patients. Hepatobiliary manifestations are rare, however a 2023 study found non-infectious Achromobacter associated with biliary flora in patients with cholangiocarcinoma (1). Apart from case reports, epidemiology, risk factors for infection, prognosis, and management recommendation are limited.

Longstanding biliary drainage lines, critical illness, malignancy, and residence in a tropical climate have been associated with Achromobacter infection. A. xylosoxidans is most often implicated in cases of Achromobacter bacteremia (97% of cases), with >50% of infections polymicrobial in nature, as evidenced by our patient with concomitant Enterococcus faecium and Staphylococcus aureus isolated from blood cultures.

The isolate from initial blood culture was resistant to trimethoprim-sulfamethoxazole, an antibiotic frequently documented as effective. He was placed on piperacillin-tazobactam based on initial results indicating susceptibility, though subsequent isolates developed intermediate resistance. Despite this, the patient demonstrated continued clinical improvement and was therefore kept on this therapeutic. Additionally, all isolates were imipenem-resistant, mirroring reports of increasing carbapenem resistance.

Cultures obtained from bile demonstrated less resistance than those from blood, suggesting poor biliary penetration. Bile likely acted as a reservoir for continuous bloodstream re-infection, making clearance impossible.

CITATIONS1. Poudel SK, Padmanabhan R, Dave H, et al. Microbiomic profiles of bile in patients with benign and malignant pancreaticobiliary disease. PloS one. 2023;18(4):e0283021

Poster #23

Minocycline-Induced Thrombocytopenia presenting with gross hematuria

Johnnie Saifa-Bonsu, MD[1], Karen Shou, DO[2], Jeffrey Berenberg, MD[2]
[1] Tripler Army Medical Center Internal Medicine Residency Program, Honolulu, HI
[2] Tripler Army Medical Center, Honolulu, HI

Introduction:

Minocycline is a commonly prescribed antibiotic with a well-established safety profile. However, cases of drug-induced thrombocytopenia related to minocycline use are rare. We present 29-year-old female who developed severe thrombocytopenia shortly after starting minocycline and spironolactone for acne.

Case Presentation:

The patient presented with a day's history of easy bruising after her child bumped into her eyeglasses leading to ecchymosis that was more than expected for the impact, as well as gross hematuria and right flank pain. Investigation on arrival resulted with a platelet of less than 2x10^9/L, from baseline of 354X10^9/L, WBC and Hemoglobin were in the normal range raising immediate concerns about the cause of her isolated thrombocytopenia. She had initiated minocycline and spironolactone therapy just one week prior to admission for the treatment of acne, which led to a clinical suspicion of drug-induced thrombocytopenia. Minocycline and spironolactone were promptly discontinued as there have been few reported cases of either drugs causing thrombocytopenia. Upon admission, and the patient was initiated on high-dose steroids and intravenous immunoglobulin (IVIG) therapy. Over the subsequent four days, her platelet count increased from 2 to 42 to 88 x 10^9/L, suggesting a favorable response to treatment. At this juncture, a diagnostic dilemma emerged, as clinicians needed to differentiate between ITP and minocycline-induced thrombocytopenia as other causes of thrombocytopenia such as HIV were ruled out. The decision was made to simultaneously administer treatment for both ITP and drug-induced thrombocytopenia. The patient responded well to this dual approach and was ultimately discharged from the hospital. To further confirm the diagnosis, laboratory testing revealed the presence of both IgG and IgM antibodies against minocycline in the patient's serum.
Discussion: The occurrence of unexplained thrombocytopenia is a frequently encountered clinical challenge, particularly in hospitalized patients who often receive various medications. Medications can induce thrombocytopenia through diverse mechanisms, including direct toxicity to the bone marrow or other organs. The use of cytotoxic drugs in cancer chemotherapy and other therapeutic interventions is recognized for causing dose-dependent reductions in platelet counts due to their impact on bone marrow function. While heparin-induced thrombocytopenia has been extensively studied, the available data on thrombocytopenia induced by other drugs, such as minocycline, are limited, primarily owing to its infrequent occurrence. Minocycline, a commonly prescribed antibiotic, has been associated with thrombocytopenia, with the most recent reported case dating back to 2003, where it manifested as purpura. This current case represents a unique instance of minocycline-induced thrombocytopenia, notably presenting with gross hematuria.

The rarity of minocycline-induced thrombocytopenia underscores the importance of vigilance and awareness among healthcare providers. Clinicians should consider this potential adverse effect, especially when evaluating patients with unexplained thrombocytopenia, even though it may be an infrequent occurrence. The manifestation of gross hematuria adds an unusual dimension to the presentation of minocycline-induced thrombocytopenia, emphasizing the diverse ways in which drug-induced hematologic complications can manifest. Importantly, further research and awareness are warranted to better understand the mechanisms of minocycline-induced thrombocytopenia and to refine treatment strategies for such cases.

Poster #24

FOXO3 Genotype Mitigates the Effect of Low Bioavailable Testosterone on Mortality: The Kuakini Honolulu Heart Program.

Ayumi Sakamoto, MS[1], Randi Chen, MS[2,3], Brian Morris, DSc, PhD[2], Aida Wen, MD[2], Timothy Donlon, PhD[3], Richard Allsopp, PhD[2], Brad Wilcox, MD[2,3], Kamal Masaki, MD[2,3]
1 University of Hawai'i John A Burns School of Medicine, Honolulu
2 University of Hawai'i John A Burns School of Medicine, Dept of Geriatrics, Honolulu, HI
3 Kuakini Medical Center, Honolulu, HI

Introduction:

Studies have found that age-related decline of testosterone is associated with increased risk of all-cause mortality. The gene FOXO3 regulates numerous homeostatic pathways, and the minor allele (G) of SNP rs2802292 has been associated with longer lifespan compared to major allele homozygote (TT). We studied whether bioavailable testosterone levels predict all-cause mortality in an older Asian male population, and are the first to examine its interaction with FOXO3 genotype.

Methods:

The Kuakini Honolulu Heart Program is a longitudinal cohort study of Japanese-American men in Hawaii since 1965. Serum testosterone was measured at Exam 4 (1991-93) when subjects were 71-93 years old. Bioavailable testosterone levels were calculated and quintiles were used for analysis. After excluding those missing data on hormone levels or FOXO3 genotype, our analytic sample included 3,138 men who were followed for mortality until December 31, 2022 (32 years). We also performed stratified analyses by FOXO3 genotype.

Results:

Age-adjusted mortality rates were significantly higher in lower quintiles of bioavailable testosterone (Q1=110.5, Q2=98.4, Q3=97.6, Q4=98.0, and Q5=93.5 per 1,000 person-years of follow-up, p for trend=0.0012). Multivariate Cox regression adjusted for baseline age, cardiovascular risk factors, prevalent chronic diseases (coronary heart disease, stroke, cancer, dementia) and FOXO3 genotype found an increased risk of total mortality in Q1 (HR=1.20, 95% CI=1.07-1.36, p=0.003), using Q5 as reference. There was a significant interaction with FOXO3 genotype (p=0.029). When stratified by FOXO3 genotype, we found a significant association between bioavailable testosterone and total mortality in the FOXO3 TT group (HR=1.34, 95% CI=1.14-1.59, p<0.001). However, in FOXO3 G allele carriers, there was no significant association between bioavailable testosterone and mortality. Mortality was not associated with bioavailable estradiol levels.

Conclusion:

Low bioavailable testosterone was associated with increased total mortality in older Japanese-American men with FOXO3 TT genotype, but not in those who were FOXO3 G allele carriers. The findings suggest a potentially protective role of the longevity-associated FOXO3 G allele by mitigating the adverse effects of low testosterone.

Poster #25

Bilateral renal vein thrombosis and chylous ascites in PLA2R-associated membranous nephropathy: a case report

Kazushige Shiraishi, MD[1], Chesta, MD[1],
Yoshito Nishimura, MD, PhD1, Christina M.B. Chong, MD[2, 3]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] University of Hawai'i, John A. Burns School of Medicine, Dept of Medicine, Honolulu, HI
[3] The Queen's Medical Center, Honolulu, HI

Chylous ascites is seldom seen with adult nephrotic syndrome, and its etiology and management in nephrotic syndrome are unclear, whereas renal vein thrombosis is a well-studied complication of nephrotic syndrome. Despite a single previous case report from an autopsy showing the coexistence of chylous ascites, nephrotic syndrome, and renal vein thrombosis, there have not been any cases showing these three phenomena in a single patient. We are presenting the first case of membranous nephropathy with bilateral renal vein thrombosis and chylous ascites that was successfully treated with anticoagulation and rituximab. Here, we report a rare case of PLA2R-associated membranous nephropathy complicated by bilateral renal vein thrombosis and chylous ascites successfully treated with anticoagulation and rituximab.

A 65-year-old African American male with a history of hypertension and peripheral arterial disease presented with acute onset abdominal pain and hematochezia, in addition to progressive lower extremity edema for one year. Four days prior to admission, the patient developed acute onset abdominal pain and hematochezia associated with abdominal distention on the day of admission. Except for blood pressure of 158/73 mmHg, his vital signs were within the normal limits. His abdomen was distended with periumbilical tenderness without rebound or guarding. He had bilateral pitting edema extending to the thighs. Rectal examination revealed bright red blood. Laboratory analysis showed hemoglobin 10.3 g/dL, creatinine 1.9 mg/dL, albumin 2.0 g/dL, cholesterol 222 mg/dL, and triglycerides 245 mg/dL. Twenty-four-hour urine chemistry revealed protein 6452 mg/24hr, creatinine 0.4 gm/24hr. The findings were consistent with nephrotic syndrome. Abdominal CT scan with contrast showed nonocclusive bilateral renal vein thrombosis with a moderate amount of ascites. Paracentesis revealed chylous ascites with cholesterol 10 mg/dL and triglycerides 131 mg/dL. The patient was started on an intravenous continuous heparin infusion for renal vein thrombosis and underwent endoscopy and colonoscopy for hematochezia, which did not reveal an active source of bleeding. Workup for nephrotic syndrome was positive for anti-PLA2R antibody. Rituximab successfully controlled the disease activity with improvement in renal function.

Chylous ascites is a rare complication of nephrotic syndrome. Its prevalence, characteristics, and management in the context of nephrotic syndrome are unclear due to the rarity. Some literature attributed chylous ascites in nephrotic syndrome to possible bowel edema. In our case, we hypothesize that the renal thrombosis increased renal lymphatic pressure, leading to leakage of lymphatic fluid into the peritoneal space, similar to chylothorax seen with central venous thrombosis. The case illustrates the importance of considering nephrotic syndrome as a cause of chylous ascites. The development of lymphatic imaging techniques to understand the fundamental mechanisms of chylous ascites in nephrotic syndrome is warranted to clarify the underlying pathophysiology.

Poster #26

A case of severe mineralocorticoid excess in abiraterone use for metastatic prostate cancer

Reese Suzuki, MD[1], Kellen Bean MD[1], Thomas Chen MD[2], Keola Beale MD[2]
[1] Kaiser Permanente Hawaii Internal Medicine Residency Program, Honolulu, HI
[2] Kaiser Permanente Medical Group, Honolulu, HI

Introduction: Abiraterone is an antiandrogen medication used for metastatic prostate cancer. Abiraterone selectively and irreversibly inhibits CYP17, an enzyme required for androgen biosynthesis which is expressed in testicular, adrenal, and prostatic tumor tissues; this also inhibits the formation of the testosterone precursors dehydroepiandrosterone (DHEA) and androstenedione. Due to CYP17 inhibition, mineralocorticoid excess and its clinical manifestations (i.e. hypokalemia, metabolic alkalosis, hypertension, fluid retention) are known side effects of abiraterone administration. We present a case in which a patient was hospitalized for mineralocorticoid excess symptoms from abiraterone use.

Case Description: A 78-year old male with PMH significant for hypertension, hyperlipidemia, type 2 diabetes mellitus, and metastatic prostate cancer, was admitted in 8/2022 for refractory hypokalemia and hypertensive urgency after the patient's third emergency department visit in 1 week. The patient was previously diagnosed with prostate adenocarcinoma one year prior via biopsy and was noted to have L1 and bilateral rib metastasis; shortly after diagnosis, he was started on bicalutamide and leuprolide (androgen deprivation therapy), and then 21 days later started abiraterone 1000 mg daily PO and prednisone 5 mg daily PO. One week prior to hospitalization, the patient presented to the ED with a potassium of 2.8 and generalized weakness; he was given 80 mEq total of potassium before discharge home with outpatient labs for follow up. He returned the next day to the ED with worsening weakness and a potassium of 2.7; he received a total of 90 mEq of potassium and 1L LR bolus. He was discharged with potassium 20 mEq BID PO. Three days later, the patient presented to the ED for the third time with hypertensive urgency (systolic blood pressure in the low 200s), potassium of 2.8 (despite taking his PO supplement as instructed), and an elevated bicarbonate level of 29. After discussion with nephrology, a urine potassium was obtained and showed increased excretion of potassium (urine potassium: urine creatine 35 mEq/g, normal <13 mEq/g, consistent with the diagnosis of mineralocorticoid excess from abiraterone use). After discussion with oncology, abiraterone was held and prednisone 5 mg daily PO was continued. The patient responded to potassium repletion over the next few days, was discharged on prednisone 5 mg BID PO, and instructed to hold abiraterone until oncology follow up.

Discussion: Abiraterone leads to a relative increase in mineralocorticoids and can lead to mineralocorticoid excess. To reduce the likelihood of this, abiraterone is often prescribed with a glucocorticoid. This case demonstrates that although abiraterone is prescribed with a glucocorticoid (specifically to suppress adrenal mineralocorticoid production by decreasing ACTH), there is still the possibility of developing mineralocorticoid excess with clinically significant findings. Patients on abiraterone therapy for metastatic prostate cancer should have their electrolytes and blood pressure checked frequently, and there should be a low threshold for holding abiraterone therapy if there are signs of mineralocorticoid excess.

References:
1. Yamamoto, Yutaka et al. "Serious Hypokalemia Associated with Abiraterone Acetate in Patients with Castration-Resistant Prostate Cancer." 2018 Sept 16.
2. Up to Date. "Abiraterone: Drug Information." Accessed 2023 Sept.

Poster # 27

Left-sided hepatic hydrothorax

Toshiaki Takahashi, MD[1], Reed McCardell Malone, MD[1], Luke Taylor, MD[1],
Yoshito Nishimura, MD, PhD, MPH[1], Christina M. Chong, MD, FACP[2]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] The Queen's Medical Group, Honolulu, HI

Introduction:
Hepatic hydrothorax is a complication seen in patients with liver disease. Left-sided hepatic hydrothorax is rare, accounting for only 13% of all cases, while 85% of patients develop it on the right side, which may suggest that anatomic factors dominate the localization of fluid collection in liver disease. Herein, we present a case of left hydrothorax secondary to decompensated alcoholic cirrhosis.

Case report:
The patient was a 32-year-old male with a history of alcoholic liver disease and non-bleeding esophageal varices who presented with progressive orthopnea and abdominal bloating for two weeks. He had a 27 kg weight gain in the past three months. Physical examination was notable for scleral icterus and decreased breath sounds on the left chest with dull sounds on percussion. The abdomen was distended with a positive fluid wave. The neurological exam revealed no focal deficits, except for right-sided asterixis. Laboratory results were remarkable for AST 98 IU/L, ALT 32 IU/L, alkaline phosphatase 223 IU/L, total bilirubin 9.2 mg/dL, total protein 7.2 mg/dL, albumin 2.9 mg/dL, LDH 426 IU/L, and PT-INR 1.8. Chest radiography demonstrated complete opacification of the left hemithorax with mediastinum shift to the right side. During the hospitalization, the patient underwent three thoracenteses with the removal of 4L of pleural fluid . While malignancy was an initial concerning differential diagnosis, the pleural fluid studies showed WBC 166/ul, protein < 1mg/dL, and LDH 61 IU/L, with no malignant cells seen on cytology. He was diagnosed with left hepatic hydrothorax secondary to decompensated alcoholic cirrhosis with portal hypertension, which was exacerbated by excessive sodium intake and increased amount of alcohol consumption. In addition to the therapeutic thoracenteses, he was treated with albumin and diuretics including spironolactone and furosemide, along with fresh frozen plasma and platelet transfusion with improvement in his symptoms.

Discussion:
In cases of cirrhosis with both pleural and abdominal fluid collection, the predominance of right over left-sided hydrothorax is not well understood. The hypoosmolar and portal hypertension physiology of liver disease easily explains abdominal fluid collection, supporting an equal prevalence of fluid collection in other compartments. If hydrothorax is caused by trans-diaphragmatic ascitic fluid flow or purely by low oncotic pressure, the incidence of hydrothorax would be expected to be equal on each side. Right predominance suggests that the liver itself, its relationship with the right diaphragm or the right chest, dominates the physiology of pleural fluid accumulation. Both the anatomy and physiology of liver disease make this patient's presentation with left hydrothorax a rare and complicated diagnosis.

Conclusion:
Our case highlights one of the rare complications of cirrhosis, emphasizing the need to recognize hepatic hydrothorax as a differential diagnosis for unilateral pleural effusion. Efforts to rule out other causes of unilateral pleural effusion such as infection and malignancy should be pursued as left-sided hepatic hydrothorax is a diagnosis of exclusion in patients with liver disease.

Poster #28

Androgenic Steroid Misuse in Military Personnel: A Case Study and Policy Implications.

Confesora Valdez A. MD[1], Robert Leimbach, MD[1]
[1] Tripler Army Medical Center, Honolulu, HI

Introduction: Androgens are potent drugs that require a prescription. Self -administration of androgens without a medical prescription is considered abuse for invalid, unproven, or off-label reasons. Moreover, the use of androgenic anabolic steroids (AASs) presents a multifaceted challenge within the military community, mainly because they are thought of as physical and performance enhancers, offering a revitalizing tone. Neglecting the potential health repercussions of anabolic steroid usage can result in significant adverse outcomes, encompassing hormonal disruptions, cardiovascular complications, and psychological issues, all of which have the potential to erode the operational readiness and long-term welfare of military personnel. AAS is widespread across different demographic groups. Anawalt's study in 2019 Estimated that 1-5% of the global population uses anabolic steroids. Larsen et al. studied active-duty military personnel and reported an incidence rate of 4.7. Several studies, including those by Eisenberg, Irving, and Miller, suggest that the use of AAS among teenage boys could be as high as 5.9%. Such misuse can result in many health complications, like gynecomastia, testicular atrophy, infertility, and cardiovascular problems, such as hypertension and a stroke. This case report aims to scrutinize the prevalence, ramifications, and attitudes associated with AAS use in the military.

Case: A male service member with a history of Obstructive Sleep Apnea, suffering from chronic fatigue and reduced libido for two years, visited the endocrinology clinic. He had erectile dysfunction, which did not respond to sildenafil but improved with tadalafil. After following a strict diet and exercise plan, he gained 20 pounds over a year, resulting in a BMI of 30.41. Laboratory tests showed the presence of stanozolol and FSH/LH at 0.1/0.9, hemoglobin at 18.7, hematocrit at 55.1, and total testosterone at 39. He admitted to using Deer Antler and "Bucked Up" supplements but denied androgen abuse. Despite advice to cease supplements, his lab results still showed abnormalities.

Discussion/conclusion: The increasing acceptance of AAS and other Performance-Enhancing Drugs (PEDs) within the military warrants an exhaustive policy reform. This case study highlights the challenges in diagnosing AAS misuse, exacerbated by underreporting due to perceived judgment. Patients frequently perceive healthcare providers as either stigmatizing or insufficiently informed about the subject. This perception is corroborated by research, revealing that a significant 56.1% of AAS users have never disclosed their use to healthcare providers. This lack of open communication obstructs effective treatment and counseling. Another complicating element is the role of commercial advertising, which often emphasizes benefits while downplaying risks. Our case report illustrates how this can lead to unregulated usage of such substances, often without medical oversight and an absence of transparent communication between healthcare providers and patients.

Poster #29

A Rare Case of Osteomyelitis Pubis with Abscess Formation

Cindy T. Vuong, MD[1], Raquel Tello, MD[2]
[1] Kaiser Permanente Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] Hawai'i Permanente Medical Group, Honolulu, HI

Introduction: Osteomyelitis pubis is a rare condition that can present with non-specific complaints such as pubic tenderness, antalgic gait, painful hip abduction, and fever. Diagnosis is often delayed. Timely recognition and further awareness of this condition is needed, especially within the diabetic population.

Case Description: A 54-year old female with type 2 diabetes experienced sudden chills, low back pain, nausea, vomiting, and diarrhea after her vacation. She was hospitalized for 2 days. Given an unclear etiology of her sepsis (suspected secondary to viral gastroenteritis) and a polymicrobial urinary tract infection, she was treated with 2 doses of IV ceftriaxone and 5 days of cefpodoxime. Preliminary blood cultures grew gram positive cocci, which was deemed a contaminant upon initial discussion with microbiology. After discharge, these blood cultures finalized with methicillin-sensitive staphylococcus aureus (1/2 sets) and streptococcus anginosus (2/2 sets).

The patient visited an urgent care clinic with new complaints of pelvic pain and pressure for 1.5 weeks following her discharge from the hospital. On labs - WBC 11.4, hemoglobin A1c 12.4.%. She was diagnosed with vaginitis (panel positive for candida), prescribed oral fluconazole, and sent home. A few days later, she was seen in the emergency room with worsening pelvic pressure and chills. Examination revealed tenderness over the pubic symphysis and discomfort upon hip abduction. Inflammatory markers included ESR >119 and CRP 8.3. Her CT Abdomen/Pelvis with contrast revealed mild stranding overlying the labia without evidence of underlying soft tissue gas. She was discharged home with naproxen but was later advised to return after the formal radiology read noted subtle erosive changes to the pubic symphysis and small pockets of soft tissue air concerning for osteomyelitis pubis.

Three weeks after the initial onset of symptoms, she was admitted. Blood cultures were negative. MRI of the pelvis revealed abnormal low T1 and increased T2 signal within the bilateral pubic symphysis with corresponding enhancement, faint fluid within the joint space with a thickened peripherally enhancing wall, and surrounding edema within the musculature. She underwent CT-guided joint aspiration, which yielded beige-colored purulent fluid with 3+ streptococcus anginosus on culture, presumed secondary to hematogenous seeding of the pubic symphysis from her partially treated gastroenteritis-induced bacteremia. She was treated with IV vancomycin and ceftriaxone for several days, then transitioned to IV cefazolin for 6 weeks. As an outpatient, although clinically improved, her CRP level remained elevated at 6.7. Repeat CT revealed further erosion of the pubic symphysis and development of a small abscess. Aspirate culture grew streptococcus anginosus and prevotella species. She completed an additional 1-month course of oral penicillin and metronidazole with normalization of her CRP level.

Conclusions: This case highlights the importance of maintaining an index of suspicion for rare diagnoses such as osteomyelitis pubis in the differential, particularly for diabetic patients. This population is more susceptible to infection and complications stemming from these infections. Early recognition and diagnosis would allow practitioners to initiate intravenous antibiotics to prevent future complications.

Poster #30

Infrequently encountered yet perilous: a rare case of the concurrent occurrence of thyroid storm arising from postpartum thyroiditis and lymphocytic hypophysitis

Chalothorn Wannaphut MD[1], Kamonkiat Wirunsawanya MD[2]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] University of Hawai'i, John A Burns School of Medicine, Dept of Medicine, Honolulu, HI

Introduction:
We present an extremely rare incidence of thyroid storm due to postpartum thyroiditis followed by the surprising diagnosis of lymphocytic hypophysitis, leading to panhypopituitarism. Two autoimmune conditions prominently manifest in connection with pregnancy: postpartum autoimmune thyroiditis and autoimmune hypophysitis. It is exceptionally uncommon for these two conditions concurrent severe form of postpartum thyroiditis and lymphocytic hypophysitis' to occur simultaneously. Due to its rarity, we believe it is worthwhile to share the clinical course and raise awareness of this unusual phenomenon.

Case presentation:
A 35-year-old woman, G1P1, presented with an ongoing depressed mood, inability to lactate, nausea, vomiting, and palpitations at two months postpartum. Upon arrival, she presented with hemodynamic instability and altered consciousness. Initial tests were positive for suppressed TSH (<0.01 mIU/L) and elevated free T4 (T4 5.2 mcg/dL). Her Burch-Wattofsky score was 45 based on the presence of fever, tachycardia, confusion, and GI symptoms consistent with thyroid storm. She was admitted to the ICU, and a treatment for thyroid storm was initiated, including methimazole, potassium iodide (SSKI), a beta-blocker, and stress dose hydrocortisone.

Further investigation for hyperthyroidism was conducted. Thyroglobulin antibodies, thyroid peroxidase (TPO) antibodies, and thyroid-stimulating immunoglobulin (TSI) all yielded negative results. Thyroid ultrasound indicated a normal thyroid gland with hypovascularity and no nodules. Given the absence of indicative signs of Graves' disease or toxic thyroid nodule as well as the rapid improvement in thyroid hormone levels, the decision was made to discontinue methimazole, suspecting postpartum thyroiditis. Subsequently, hydrocortisone and SSKI were stopped as clinical symptoms of thyroid storm improved. One day after discontinuing hydrocortisone, she exhibited a new onset of hypernatremia (Na 152 mmol/L) and borderline hypotension. Desmopressin and hydrocortisone were promptly initiated, addressing concerns of unmasked diabetes insipidus in the setting of adrenal insufficiency. Following oral desmopressin, her hypernatremia improved, and urine osmolality increased > 100%.

MRI of the pituitary gland with and without contrast revealed a normal-sized pituitary gland and homogeneously enhanced pituitary stalk with an absent posterior pituitary bright spot, suggesting lymphocytic hypophysitis. IgG-4 levels were normal. She was discharged in a favorable condition. During a close outpatient follow-up, further pituitary hormonal evaluation also revealed central hypothyroidism. At the present, she continues on a regimen of hydrocortisone, levothyroxine, and desmopressin for panhypopituitarism.

Discussion:
The postpartum period could lead to immune-related destruction of the thyroid and pituitary glands. Our case emerges as a pioneering instance, uniquely presenting with a thyroid storm and panhypopituitarism stemming from the concomitant occurrence of postpartum thyroiditis and lymphocytic hypophysitis. Highlighting the potential oversight of this occurrence underscores the importance of prompt diagnosis and intervention, which is crucial for avoiding delays in recognition and management.

Poster #31

Namast'ay In Bed - CSF leak presenting as chronic positional nausea

Chase Warashina, MD[1], Kevin E. Nebrejas, MD[1], Landon Kozai, MD[1], Traci Murakami, MD[2]
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] The Queen's Medical Group, Honolulu, HI

This case illustrates the burgeoning exploration of the neurogastric axis through the connection between cerebrospinal fluid (CSF) leak and unexplained chronic nausea, underscoring its relevance in the differential diagnosis within gastroenterology. We present a 32-year-old male with chronic nausea that was unresponsive to conventional medical management. Despite extensive workup, diagnostics were unrevealing. Following specialty [neurogastroenterology] center referral, MRI suggested perineural CSF collections. Empiric blood patch led to significant symptomatic improvement indicating a likely CSF leak despite atypical imaging findings. This case emphasizes the need for providers to consider CSF leaks as a cause of unexplained nausea, particularly when accompanied by other suggestive symptoms such as postural nausea.

Digital Poster #32

Idiopathic Intracranial Hypertension Masquerading as Giant Cell Arteritis

Marcus Yamamoto, MS[1], Ted Jabocy, MD[2], Christina Chong, MD, FACP[3]
[1] University of Hawai'i John A Burns School of Medicine, Honolulu, HI
[2] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[3] The Queen's Medicine Group, Honolulu, HI

Introduction:
Idiopathic intracranial hypertension (IIH) is a condition associated with elevated intracranial pressure (ICP) with normal CSF composition and imaging. IIH commonly presents with diffuse headache and visual changes with associated papilledema, often diagnosed in obese women ages 15-44.

Case Description:
A 72-year-old female with no significant past medical history presented to the emergency department with a headache and diplopia. The headache began nine days prior to presentation and is localized to the bilateral parieto-occipital region, but is worse on the right side. The pain is characterized as a 3/10 and is dull and constant in nature. Three days prior to presentation, the patient experienced diplopia that improved when closing one eye. On examination, her blood pressure was 170/79 mmHg with a pulse of 58 bpm and a temperature of 36.6 °C. She is 5 feet and 2 inches and weighs 84.5 kilograms, giving her a BMI 34.07 kg/m2. Physical examination revealed tenderness to palpation of the right temporal region. An ophthalmologist performed a dilated fundoscopic exam that was unremarkable. On neurological examination, a right eye abduction deficit was present and diplopia almost resolved with left gaze. There was no blurry vision, changes in color vision, ptosis, fatigable weakness, or jaw claudication. CRP and ESR were normal. Head and neck CT venogram, MRI brain with and without contrast, and MRI orbits were unremarkable. A lumbar puncture was performed, revealing an elevated opening pressure of 24 cm H2O and normal CSF composition. The patient's headache improved following the procedure. Though a diagnosis of IIH was most likely, giant cell arteritis (GCA) was also initially considered. The patient was started on acetazolamide 500 mg BID and prednisone 60 mg. A temporal artery biopsy was performed and was negative for arteritis and prednisone was subsequently stopped. With these findings excluding GCA and the clinical presentation of elevated ICP and diplopia in an overweight female, the patient was ultimately given a diagnosis of IIH.

Discussion:
This case illustrates the importance of identifying an isolated cranial nerve VI palsy in the diagnosis of IIH. Even in the absence of papilledema, the cranial nerve VI palsy pointed to the diagnosis of IIH, as this finding is seen in 30% of cases. This is because the abducens nerve is tethered within Dorello's canal and is susceptible to increased ICP. The patient's presentation was initially suggestive of GCA due to her presentation of headache associated with the temporal region tenderness. Although ESR and CRP were normal, these labs are found to be normal in 4% of GCA cases and biopsy should be done if one has a high suspicion.

Poster #33

Beyond Syncope: A Rare Case of Complete Heart Block due to Isolated Cardiac Sarcoidosis

Thanaboon Yinadsawaphan, MD[1], Ashwin Venkataraman, DO[3], Witina Techasatian, MD[1],
Clarke Morihara, MD[1], Steven Namiki, MD[1], Sorawit Ongsupankul, MD[1],
Kevin Benavente, DO[1], Jason Kuniyoshi, DO[1], Alamelu Ramamurthi, MD, FHRS[2],
Nath Limpruttidham, MD2, Abdulelah E Nuqali, MD2
[1] University of Hawai'i Internal Medicine Residency Program, Honolulu, HI
[2] The Queen's University Medical Group, Honolulu, HI
[3] Tripler Army Medical Center, Honolulu, HI

Introduction: Sarcoidosis is a rare systemic granulomatous disease of unknown etiology that can affect multiple organ systems, especially the lungs. Prevalence of sarcoidosis is unknown but cardiac sarcoidosis (CS) constitutes 25% of sarcoidosis cases. Isolated cardiac sarcoidosis (ICS) presents a diagnostic challenge due to the absence of extracardiac findings. This case report will discuss the case of ICS, discovered after a syncopal episode.

Case presentation: A 40-year-old male with an unremarkable medical history presented to a community hospital due to syncope. He initially felt dyspneic when he was walking and completely lost consciousness shortly after. His electrocardiogram demonstrated bradycardia with high-grade atrioventricular block (AVB). On arrival at an intensive care unit in a tertiary care center, the patient once again developed dizziness associated with an intermittent high-grade AVB requiring a temporary transvenous pacemaker. The high-grade AVB subsequently deteriorated to complete AVB without escaping rhythm within a few days, resulting in being completely pacemaker-dependent.

Routine laboratory tests were unremarkable. However, troponin, ESR, and CRP were slightly elevated. Further investigations were negative for c-ANCA, p-ANCA, anti-dsDNA, ANA, RPR, and Lyme disease. The echocardiogram showed LVEF of 50% without valvular abnormalities.

Positron emission tomography with 2-deoxy-2-[fluorine-18] fluoro-D-glucose integrated with computed tomography (18F-FDG PET/CT) revealed diffuse metabolic activity in the left ventricular lateral wall, suggestive of an inflammatory process, consistent with a diagnosis of ICS. There is no evidence of extracardiac sarcoidosis involvement, including the hilar and lung regions.

The patient was treated with 1 gram of intravenous methylprednisolone for 3 days, followed by oral prednisolone 40 mg per day. A dual chamber implantable cardioverter-defibrillator (ICD) was implanted successfully. The patient was discharged in good condition and was planned for an outpatient follow-up.

Discussion: Complete AVB is rare in generally healthy patients, accounting for less than 1 in 67000 healthy males. Possible reversible causes of AVB should be investigated before permanent pacemaker implantation. In this patient, 18F-FDG PET/CT supported a diagnosis of CS without extracardiac involvement. In this case, sarcoidosis affected mainly in a cardiac conduction system, resulting in the progression of intermittent high-grade AVB to complete AVB.

In addition, a potential myocardium scar caused by sarcoid granulomas could be a substrate for ventricular arrhythmia. Instead of a permanent pacemaker for a complete AVB with a low-normal LVEF in this patient, ICD implantation is strongly indicated for primary prevention of ventricular arrhythmia in this specific entity.

Corticosteroid is a drug of choice for treating most forms of sarcoidosis. Corticosteroid prevents deterioration of left ventricular function. However, AVB is recovered only in 40% of the patients after corticosteroid treatments but 0% in a non-treatment group. The effectiveness of corticosteroids in preventing ventricular arrhythmia in CS is unclear. The duration of treatment is uncertain. For follow-up, repeated 18F-FDG PET/CT, serum angiotensin-converting enzyme, and serum soluble interleukin-2 receptor might be used.

Conclusion: This case underlines the necessity of thorough investigations of complete AVB etiology. Diagnosis of CS warrants additional managements of complete AVB, including corticosteroids and an ICD.

Instructions for claiming CME credit and MOC Points

The survey/claim link for the 2024 ACP HI Annual Scientific Meeting is:
https://online2.snapsurveys.com/52zumh

Please note:

Survey/claim links are set to go live at noon on the last or only day of a meeting

The American College of Physicians (ACP) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The ACP designates this other activity (live component and enduring component) for a maximum ***6.0 AMA PRA Category 1 Credit(s)***™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Successful completion of this CME activity, which includes participation in the evaluation component, enables the participant to earn up to 6.0 medical knowledge MOC point in the American Board of Internal Medicine's (ABIM) Maintenance of Certification (MOC) program. It is the CME activity provider's responsibility to submit participant completion information to ACCME for the purpose of granting ABIM MOC credit.

Mahalo for joining us!

Mahalo to our Sponsors!

AstraZeneca plc is a multinational pharmaceutical and biopharmaceutical company headquartered in Cambridge, England. It has Research and development concentrated in three strategic centres; Cambridge, Gothenburg, Sweden and Gaithersburg, U.S. AstraZeneca has a portfolio of products for major disease areas including cancer, cardiovascular, gastrointestinal, infection, neuroscience, respiratory and inflammation. AstraZeneca AB, the Swedish arm of the company, is a subsidiary of Astra Zeneca plc.

https://www.astrazeneca.com/

Contact Specialist:

Michael Ocasek

AstraZeneca

Respiratory Specialty Team - Hawaii

(808) 255-8685 cell phone

Michael.ocasek@astrazeneca.com

AstraZeneca plc is a multinational pharmaceutical and biopharmaceutical company headquartered in Cambridge, England. It has Research and development concentrated in three strategic centres; Cambridge, Gothenburg, Sweden and Gaithersburg, U.S. AstraZeneca has a portfolio of products for major disease areas including cancer, cardiovascular, gastrointestinal, infection, neuroscience, respiratory and inflammation. AstraZeneca AB, the Swedish arm of the company, is a subsidiary of Astra Zeneca plc.

https://www.astrazeneca.com/

Contact Specialist:

Roy Simeon

AstraZeneca

Respiratory Specialty Team - Hawaii

Roy.Simeon@astrazeneca.com

GlaxoSmithKline

GSK is a science-led global healthcare company with a mission to help people do more, feel better, live longer. We research, manufacture and make available a broad range of medicines, vaccines and consumer healthcare products. Visit our exhibit for information about our products and resources.

http://www.gsk.com/

Account Specialist contact information:

Chris A. Furuya
GSK - Hawai‘i Representative
808-497-7571 Cell phone
Chris.a.furuya@gsk.com

A HEALTHIER career starts HERE.

Join the Hawai'i Pacific Health Medical Group, the employed provider group of Hawai'i Pacific Health formed to further our vision to be the leader in health care transformation and our mission to create a healthier Hawai'i. Comprised of more than 800 employed physicians, advanced practice registered nurses and physician assistants, HPHMG partners with Hawai'i Pacific Health's network of primary care providers and specialists to provide a distinctive and effective model of coordinated care for maintaining the health and well-being of our patients.

Together with our four medical centers – Kapi'olani, Pali Momi, Straub and Wilcox – and more than 70 convenient clinic locations statewide, our not-for-profit health care system is one of the largest in Hawai'i. HPHMG providers serve patients in both primary and specialty care on the islands of Hawai'i, Kaua'i, Lāna'i, Maui and O'ahu.

HawaiiPacificHealth.org/PhysicianCareers

Hawai'i Pacific Health is named Best Employer in Hawai'i by Forbes in 2023.

Forbes 2023
AMERICA'S BEST-IN-STATE EMPLOYERS
POWERED BY STATISTA

Scan the QR code to see our current physician job listings.

HAWAI'I PACIFIC HEALTH | KAPI'OLANI PALI MOMI STRAUB WILCOX
MEDICAL GROUP

HIPac FINANCIAL

Global Vision, Local Touch™

www.hipacfinancial.com

Jeffrey J. Kao
MBA, LUTCF®, CLTC®, FSCP™
Partner, Financial Adviser

808.529.6316
jeff@hipacfinancial.com

Kristi Tanigawa Chun
LUTCF®, FSCP™, CLTC®, ChFC®, CFP®
Partner, Financial Adviser

808.529.6311
kristi@hipacfinancial.com

Derek J. Tenn
Partner, Financial Adviser

808.529.6340
derek@hipacfinancial.com

IMIG

INTERNAL MEDICINE INTEREST GROUP

Our annual Subspecialty Mixer allows medical students to discover all internal medicine has to offer.

IMIG members volunteer at a blood pressure clinic.

Contact us!

uhimig@hawaii.edu

https://imig.uhmed.org

About Us

IMIG is a student-run interest group at the John A. Burns School of Medicine (JABSOM). We're dedicated to providing information about careers in internal medicine, fostering communication between students and physicians, and connecting with the community to promote health awareness.

Fall 2023 - Spring 2024 Events

- Subspecialty Mixer
- JABSOM IM Resident Panel
- ACP Pau Hana
- UHIMRP Primary Care Forum
- Farrington High School Capstone Project Mentorship
- Amyloidosis Bureau Patient Speaker
- Journal Club
- Check Your Pressure x IMIG: Blood Pressure Clinic
- Papakolea Health Fair (3/2/24)
- Lifestyle Medicine Speaker (3/13/24)

Opportunities for Physicians to Get Involved

- IM Clinical Specialties Experience
  - This program allows medical students to shadow physicians during their relevant preclinical units (e.g. shadow a cardiologist during MD2 [cardiac] unit)
  - Students typically join a specialist for one half-day (times and dates up to the physician's availability)
- Physician Mentorship Program
  - This mentorship program connects interested medical students with community physicians in IM and its subspecialties.
  - May entail shadowing, mentorship, and/or research opportunities
  - Expectations: have at least 2 one-on-one meetings per academic year. Time commitment is dependent on mentor's and mentee's preferences. Complete mid-year and end-year feedback surveys.

The #1 hospital in Hawai‘i is an ideal place to work, practice and grow.

The Queen’s Health System’s culture, diversity and benefits demonstrate the investment we are committed to making in our people—our most valued asset—and their career paths.

QUEEN’S UNIVERSITY MEDICAL GROUP

Scan the code to learn more!

Sanofi

Pharmaceutical Company

"We chase the miracles of science to improve people's lives".

linkin.bio/sanofi

Tim Cason
Medical Specialist –
Regeneron Healthcare Solutions
777 Old Saw Mill River Road
Tarrytown, NY 10591-6707
Cell: 949/342-0362
Email: timothy.cason@regeneron.com

Denise Grundy
Area Business Manager
Pulmonology Specialty Care Division
Sanofi
Cell Phone:310-968-6646/310-382-4102
Email: denise.grundy@sanofi.com

SANOFI
450 Water Street, Cambridge, MA 02141
Phone 1-617-252-7500




Maui Relief Support – Mahalo for going the extra mile!

Jennifer Agena, Pharmacist HPH
Jason Austria Staff Queen's
Wendy Burkholder Executive Queen's
George Burno, MD Queen's
David Cho, MD HPH
Amy Chong, Nursing Staff HPH
Carrie Colmenares, MD Kaiser
Shane Correira, Director HPH
Melisa Dias, Buyer HPH
Elaine Doi, MD Kaiser
Maegan Doi, MD Kaiser
Jeiko Durusan, FN HPH
Victoria Fox-Behrle, MD Kaiser
Raquel Guron, Buyer HPH
Amanda Haley, MD Queen's
Tiffanie Hipa Manager HPH
Marcus Iwane, MD Kaiser
Jennifer Katada, MD Kaiser
Timothy Kim, MD Kaiser
Yusuke Kobayashi, MD Queen's
Scott Kuwada, MD Queen's
Todd Kuwaye, MD Kaiser
Jordan McAngus Buyer HPH
Matthew Mitschele, MD Queen's
Nani Morgan, MD Queen's
Jana Moses Manager HPH
Jeremy Narca, Manager HPH
Ana Nguyen, MD Queen's
Maya Nordstom, APRN HPH
Hisami Oba MD Kaiser
Shanele Odan, Pharmacist, HPH
Abhinetri Pandula, MD Kaiser
Gypsy Paar, MD Queen's
Jonathan Paladino, MD HPH
Julia Park, MD Kaiser
Mike Pharaon, MD HPH
Elizabeth Quinn, MD Kaiser
Ashley Rodriguez, Supervisor HPH
Brandlee Sako, MD Queen's
Robert Schulz, MD HPJ
Blayne Tamada Manager HPH
Lisa Varville, MD Kaiser
Anthea Wang, MD Kaiser
Kim Webster Manage HPH

Index: