



Connecticut
CHAPTER

25th Annual Meeting & Scientific Session

Wednesday • April 26, 2017

The Waterview • 215 Roosevelt Drive • Monroe CT 06468

Original Research & Case Vignette Recognition



Taking the DIE Out of DIET

Keynote Speaker:

Kim Allan Williams, MD, MACC, FAHA, MASNC, FESC

Professor of Medicine and Cardiology at Rush University Medical Center

CCACC • P.O. Box 1058 • Glastonbury, CT 06033

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Greetings from the CCACC

On April 26, 2017, over 100 healthcare providers and industry sponsors enjoyed the 25th Annual Meeting and Scientific Session of the Connecticut Chapter of the American College of Cardiology (CCACC). The afternoon poster session provided a superb array of clinical research and clinical vignette presentations by trainees in Cardiology and Internal Medicine from around the state. We invite you to peruse the abstracts of these presentations in the pages that follow.

The evening meeting included remarks from out-going Chapter President & Governor, Dr. Edward Tuohy, and incoming President & Governor, Dr. Kevin Kett. Dr. Kim Williams, an internationally renowned authority on diet and cardiovascular disease, then provided a stimulating review, peppered by his personal vegan experience, of the benefits of a plant-based diet on cardiovascular health. Based upon feedback of attendees, the day was enjoyed by all.

We thank the members of the Education Committee of the CCACC (page 3) for their diligent efforts in planning the meeting, the staff of The Waterview for excellent hosting, and the members of industry who provided valued support of the event (their names and corporations are listed on the back cover).

We hope you will join us for next year's Annual Meeting at The Waterview in Monroe, CT on Wednesday, April 11th, 2018 when our featured speaker will be Paul M. Ridker, MD, MPH, FACC, FAHA, Eugene Braunwald Professor of Medicine, Harvard Medical School, Brigham and Women's Hospital.

Cheryl Anderson

Cheryl Anderson, EdD, APRN
Co-Chair, Education Committee

Edward Tuohy

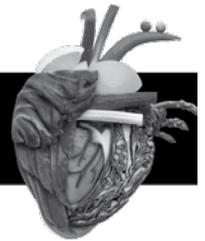
Edward Tuohy, MD, FACC
Past-President, CCACC

Craig McPherson

Craig A. McPherson, MD, FACC
Co-Chair, Education Committee

Kevin Kett

Kevin Kett, MD, FACC
President & Governor, CCACC



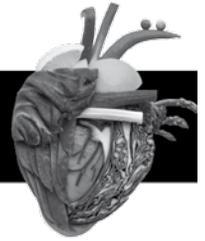
Poster Sessions & Industry Displays

Key Note Address:
“Taking the Die Out of Diet”
Kim A Williams, MD, FACC



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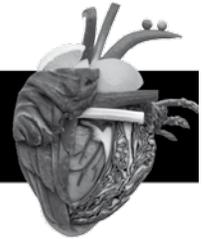
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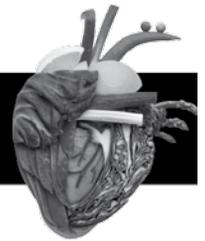
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KEYNOTE SPEAKER



Kim Allan Williams, MD, MACC, FAHA, MASNC, FESC

Dr. Kim Allan Williams is board certified in Internal Medicine, Cardiovascular Diseases, Nuclear Medicine, Nuclear Cardiology and Cardiovascular Computed Tomography.

Dr. Williams is the James B. Herrick Endowed Professor of Medicine and Cardiology at Rush University Medical Center. Dr. Williams has published numerous peer reviewed articles, monographs, book chapters, editorials, and review articles in the field of nuclear cardiology and minority health issues, with emphasis on education and innovations in perfusion imaging and quantitation of ventricular function. His research interests include selective adenosine receptor agonists, fluorinated perfusion PET imaging, health care disparities and payment policy, and appropriate use of cardiac imaging. He is a past president of the American College of Cardiology.







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Original Research Abstracts

ORIGINAL RESEARCH ABSTRACTS

VARIATION IN PRACTICE PATTERNS AND OUTCOMES ACROSS UNITED NETWORK FOR ORGAN SHARING (UNOS) ALLOCATION REGIONS

Dushyanth Srinivasan MD,¹ Benjamin Vaccaro MD,¹ Pooja Rao MBBS PhD,³ Rohit Ghosh B.Tech,³ Prashant Warier PhD,³ Tariq Ahmad MD, MPH,¹ and Nihar R. Desai MD, MPH^{1,2}

¹Section of Cardiovascular Medicine, Yale School of Medicine, ²Center for Outcomes Research and Evaluation, Yale New Haven Hospital, New Haven, CT, and ³Qure.ai, Mumbai, India

Original research



Dushyanth Srinivasan, MD
Yale School of Medicine

Background: The performance of heart transplantation in the United States is limited by organ availability and is managed by United Network for Organ Sharing (UNOS). Currently, efforts are underway to make organ allocation more equitable as demand continues to increase. However, little is known regarding the cotemporary patterns of care, wait time, and outcomes among patients undergoing heart transplant across UNOS regions. We sought to examine contemporary patterns of care, wait time, and outcomes among patients undergoing heart transplant across UNOS regions.

Methods: Adult patients undergoing first, single organ, heart transplantation between January 2006 and December 2014 were identified in the UNOS dataset. We compared sociodemographic and clinical profiles, wait times, use of mechanical circulatory support (MCS), status at the time of transplant, and 1-year survival across the 11 UNOS regions. Continuous variables were compared using ANOVA and categorical variables using

Results: There were 17,096 patients undergoing first, single-organ heart transplant. There were no significant differences in age, sex, renal function, and pulmonary vascular resistance across UNOS regions. However, there was 3-fold variation in median wait time (range 48-166 days, Region 5 and Region 1 respectively). Clinical severity as reflected by patient status at the time of transplantation varied widely with the proportion of patients undergoing transplant with Status 1A ranging from 36% (Region 8) to 79% (Region 1) ($P<0.001$). The percentage of patients who were hospitalized at the time of transplant varied from 41% (Region 6) to 98% (Region 1) and there was significant variation in the use of MCS (range 2.8%-13.0%, Region 3 and Region 11 respectively) and inotropes (range 8.2%-54.6%, Region 2 and Region 3, respectively; $P<0.001$ for all). One-year survival across regions varied from 88% to 93% ($P=0.026$).

Conclusion and Relevance: There are marked differences in patterns of care and wait time across UNOS regions despite similar clinical profiles of patients undergoing heart transplantation. Novel policy initiatives are required to address shortcomings in organ allocation.

ORIGINAL RESEARCH ABSTRACTS

Title: THE EFFECT OF PCSK9 INHIBITION ON STEROL ABSORPTION IN A COHORT OF REAL WORLD PATIENTS

Authors: Eric J Brandt¹ MD; Lane Benes, MD²; Linda Lee, MD³; Michael Davidson^{2,4} MD

Affiliations:

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Eric J. Brandt, MD
Yale School of Medicine

Original Research

Background: Proprotein convertase subtilisin/kexin type 9 (PCSK9) promotes degradation of low-density lipoprotein (LDL) receptors, inhibition of which leads to reduced serum LDL. PCSK9 is also present in the small intestine lining where its effect is not well understood, but is thought to increase intestinal triglyceride-rich apoB lipoprotein production, postprandial triglycerides, and chylomicrons, and decrease intestinal cholesterol excretion. As the effect of PCSK9 inhibition on sterol absorption has yet to be reported, we sought to investigate if PCSK9 inhibition in humans alters intestinal sterol absorption markers.

Methods: This was a retrospective cohort study of patients administered evolocumab [Repatha®] or alirocumab [Praluent®] between July 2015 and January 2017. Patients were excluded if they did not have sterol marker data (campesterol, sitosterol, and cholestanol) both prior to and following PCSK9 inhibitor initiation. When results fell below the detection limit, values were estimated as equivalent to the lower limit as to not bias results away from the null. Paired t-tests were used to compare mean serum sterol marker concentrations before and after PCSK9 inhibitor initiation. Analyses were repeated for those taking and not taking statins and those taking or not taking ezetimibe at both initiation and follow up and for each individual PCSK9 inhibitor.

Results: There were 62 possible subjects, 29 met inclusion criteria. Nine were prescribed evolocumab; 7 were prescribed alirocumab. Average follow up was 92.5 days. Mean campesterol (before 3.14 µg/mL, 95% CI 2.79 – 4.38), after 2.09 µg/mL, (95% CI 1.87 – 2.31 µg/mL), $p < 0.0001$), sitosterol (before 2.46 µg/mL (95% CI 2.23 – 2.70 µg/mL), after 1.62 µg/mL (95% CI 1.48 – 1.75 µg/mL), $p < 0.0001$), and cholestanol (before 3.25 µg/mL (95% CI 3.04 – 3.47 µg/mL), after 2.08 µg/mL (95% CI 1.96 – 2.21 µg/mL), $p < 0.0001$) all significantly decreased at follow up. Whether a patient was or was not on a statin, was or was not on ezetimibe, or selection of PCSK9 inhibitor did not significantly influence these findings.

Conclusion: PCSK9 inhibition with evolocumab or alirocumab was associated with decreased sterol absorption markers, regardless of statin or ezetimibe use.

ORIGINAL RESEARCH ABSTRACTS

THE LOWER THE BETTER- ELEVATED SYSTOLIC BLOOD PRESSURE IS ASSOCIATED WITH DIASTOLIC DYSFUNCTION

Suryateja Chaturvedula, MD, Elizabeth L. Wolfe, BS, Kai Chen, MD, PhD

University of Connecticut School of Medicine, Farmington, CT

Original research



Suryateja Chaturvedula, MD
University of Connecticut
School of Medicine

Background and Objective: In 2017, 34% of adults in the USA suffer from hypertension when prevalence is age-adjusted. Systolic blood pressure (SBP) is a well-established independent risk factor for the development of stroke, coronary events, heart failure and end stage renal disease. SPRINT trial concluded that intensive treatment of hypertension (SBP<120mm Hg), compared with the standard treatment (SBP<140 mmHg), significantly reduced the incidence of heart failure although the trial was not designed to further characterize the incidence of heart failure with either reduced ejection fraction (HFrEF) or preserved ejection fraction (HFpEF). It is plausible that an intensive BP control may protect against the development of HFpEF. We therefore sought to define the effect of BP control on diastolic dysfunction, a requisite component of HFpEF, as determined by echocardiography.

Methods: This is a retrospective cohort study by reviewing the medical record of ambulatory patients referred for echocardiography for diagnosis of dyspnea between 01/01/2015 - 09/30/2015. Patients with LVEF <50%, age < 50 years, or significant valvular diseases were excluded. The patients were classified as normotensive (SBP<130 mm Hg, n=78) or hypertensive (SBP>130 mm Hg, n=74). Demographic data, 3 serial blood pressure measurements, antihypertensive regimen including diuretic use were reviewed. Chamber quantification parameters, systolic function and diastolic parameters were assessed in all the patients. Differences between the two groups were assessed by student *t* test for continuous variables and chi square test for categorical variable.

Results: 152 patient were eligible, and the two groups were comparable with respect to age, gender, prevalence of diabetes. The mean SBP was significantly higher in the hypertensive group compared with that in the normotensive group (143 vs 119 mmHg, $p<0.01$). There was no significant difference between the groups in chamber size. However, evaluation of medial e' ($p=0.013$), lateral e' ($p=0.004$), medial E/e' ($p=0.006$), and lateral E/e' ($p=0.001$), and A wave ($p=0.01$), revealed statistically significant diastolic abnormalities in the hypertensive group compared to the normotensive group. There was no significant difference between groups in the number of antihypertensive drugs or diuretic therapy.

Conclusion: SBP control is an important determinant of diastolic function. Intensive BP therapy may be a crucial aspect of care in hypertensive and HFpEF patients. A larger trial with prospective design and clinical outcomes might be helpful in substantiating these findings.

ORIGINAL RESEARCH ABSTRACTS

IDENTIFYING AND IMPROVING APPROPRIATE UTILIZATION OF HIGHLY SENSITIVE CARDIAC TROPONINS AT A COMMUNITY HOSPITAL.

Rachna Kataria, MD¹, Lili Barsky, MD¹, Laia Jimena Vazquez Guillamet, MD¹, Kavitha Gopalaratnam, MD¹, Linsley Sikorsy, MD¹, David Hajdasz, PhD², Stuart W. Zarich, MD¹.



Rachna Kataria, MD
Bridgeport Hospital

¹ Yale New Haven Health System/ Bridgeport Hospital, CT, USA.

² Yale New Health System, CT, USA.

Original research.

Background: The diagnosis of acute coronary syndrome is a clinical one, and cardiac biomarkers are meant to assist in the diagnosis. With the availability of highly sensitive assays for measuring cardiac troponins, we are at risk of over diagnosing acute coronary syndrome. There are no guidelines at present for the role of positive cardiac troponin assays in non-cardiac diagnoses.

Objective: To develop, implement and assess the efficacy of a multifaceted intervention at reducing the number of inappropriate cardiac troponins (cTn) ordered among health care professionals at our hospital.

Method: A multidisciplinary clinical redesign team was formed. We then prepared and administered an anonymous online survey to all healthcare providers in our hospital, to assess troponin ordering attitudes in various clinical scenarios. Their responses were compared with those given by a panel of attending Cardiologists. A multifaceted intervention was then implemented to reduce the number of inappropriate troponin orders. We then performed a retrospective chart review in the 72 hours preceding and following our intervention; in order to determine the reduction in the number of inappropriately ordered troponin tests.

Results: Results of the survey revealed the degree of agreement between different health care professionals and our Cardiologists and brought to light the differences in troponin ordering attitudes. An automatic dashboard revealed that the average weekly cTn test volume during the 38-week period preceding the intervention was 442. In the 17-week period since our multifaceted intervention was implemented, the average weekly cTn test volume was 395. This 10.6% decrease in cTn test volume was found to be statistically significant as compared to the baseline period ($p=0.000$). A Pearson Correlation analysis found that the volume of cTn tests ordered was independent of ED volume. Retrospective chart review revealed a decrease in inappropriate troponin orders from 52% to 24%, with a relative reduction of 54%.

Conclusion: Calculating the pre test probability of acute coronary syndrome is quintessential in minimizing inappropriate utilization of cardiac troponins.

Evaluation of Left Atrial Appendage Following Closure with the LARIAT: A Case Series

Bani M. Azari MD, PhD¹, Brian Malm MD¹, Kelly Giordano BS¹, Paul Hermany MD¹, Eileen Bouman BA¹, Ben Lin MD, PhD¹, Joseph Akar MD¹ and Lissa Sugeng MD¹

1. Yale School of Medicine

Original Research



Bani M. Azari, MD
Yale School of Medicine

Background: Left atrial appendage (LAA) ligation is a strategy used for patients with atrial fibrillation and contraindications to anticoagulation for stroke prevention. The LARIAT suture delivery device is a pre-tied suture system designed to provide soft tissue closure. Although it is not FDA approved for LAA closure it is widely used off-label for the percutaneous LAA ligation. Initial trials of the LARIAT for LAA closure did not evaluate the presence or development of residual pouch. Additionally, follow-up evaluation of the ligated LAA was limited to patients with the presence of a leak at the time of LARIAT placement. This study evaluated the morphology of the ligated LAA following LARIAT placement.

Methods: 6 consecutive patients who underwent LAA ligation with the LARIAT were retrospectively studied. Medical charts and echocardiographic images were reviewed and evaluated for changes development or presence of a residual LAA pouch, leak or thrombus.

Results: Immediately following placement of the LARIAT transesophageal echocardiogram (TEE) demonstrated the presence of a residual pouch in 3 patients. Follow-up TEE of these patients demonstrated development of a thrombus in one, another continued to have a stable residual pouch and the third has not undergone surveillance TEE. For the 3 other patients that demonstrated complete LAA closure at the time of LARIAT placement, one developed a residual pouch, one developed a leak and the last one continued to have stable ligation of the LAA during surveillance TEE.

Conclusions: This case series illustrates that residual pouches can develop following LARIAT placement and surveillance TEE may be recommended in this patient population. The timeline for surveillance TEE following LARIAT placement is not clear at this point from these observations but may be considered as an addition to the protocol for future studies.

ORIGINAL RESEARCH ABSTRACTS

EFFECT OF ENHANCED PATIENT EDUCATION ON PATIENT SATISFACTION AND 30-DAY READMISSION RATES AFTER CARDIAC ARRHYTHMIA ABLATION

Kristin Bott DNP¹, Millicent Malcolm DNP¹, Juliette Shellman PhD¹, Suzanne Rose PhD²

¹ = University of Connecticut, ² = Stamford Hospital

Original Pilot Study

Patient satisfaction and 30-day hospital readmission rates are metrics used to assess quality of patient care. Dissatisfied patients pose a high risk of readmission (Boulding, Glickman, Manary, Schulman, Staelin, 2011) and during the vulnerable discharge period, 1 in 7 patients may experience an unavoidable readmission within 30-days post-procedure (Jencks, Williams, & Coleman, 2009). Successful patient education programs have been linked to improved patient satisfaction and subsequently reduced unnecessary readmissions (Murdock & Griffin, 2013; Hansen et al., 2011). Utilizing a framework of the Health Belief Model (Janz & Becker, 1984), an enhanced post cardiac arrhythmia ablation education intervention based on the Project Red framework for re-engineering the discharge process, was delivered to patients in one practice who underwent cardiac arrhythmia ablation procedure with the aim of increasing patient awareness of their disease, clarifying post procedural expectations with the aim of increasing patient satisfaction, and reducing 30-day readmission rate in this population. Results of this study support the implementation of enhanced patient education intervention during the vulnerable 24-72 hour post discharge period by showing a higher total patient satisfaction score in the group receiving the intervention (M=633, SD=78) compared to (M=508, SD= 137) in the control group with a statistically significant difference between the means of the groups (p=.005). This study also showed a lower rate of 30-day readmissions in the intervention group (7.1%) compared to (53.3%) readmission rate of the control group with a statistically significant association (p=.014) and large magnitude of effect. Results of this pilot study may be used for development and implementation of enhanced patient education programs aimed at increasing patient satisfaction and reducing 30-day readmission rates.



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Stamford Hospital

ORIGINAL RESEARCH ABSTRACTS

WHAT IS THE OPTIMAL METHOD TO MEASURE THE QT INTERVAL OVER TIME?

Tamta Chkhikvadze, MD¹; Craig A. McPherson, MD, FACC²

¹Internal Medicine Resident- PGY-2 Bridgeport Hospital/ Yale University Program

²Cardiology Program Director, Director Cardiac Electrophysiology Service, Clinical Professor of Medicine, Yale University School of Medicine, Bridgeport Hospital Yale New Haven Health



Tamta Chkhikvadze, MD
Bridgeport Hospital

Category of the Abstract: Original research

Background: Serial QT interval measurement in drug studies is subject to statistical errors.

Objective: To determine an optimal strategy for measuring change in serial QT measurements.

Methods: In a study of ziprasidone vs. placebo in young patients with pre-schizophrenia, 160 ECGs were recorded in 23 patients on placebo. The QT interval was measured by 4 strategies: 1) a board-certified cardiac electrophysiologist (BCEP) who was blinded to treatment allocation and read ECGs with the naked eye as patients progressed through the study (EP1); 2) a BCEP who used a jeweler's magnifying lamp to read ECGs that were blinded and randomized after study's end (EP2); 3) a 2nd year medical resident who read the ECGs as did EP2, but using naked eye; and 4) the QT measurements rendered by the ECG machine upon which ECGs were recorded. We analyzed the proportion of maximum QT intervals measured by each strategy in the 1st half of the study vs. the last half of the study period. The null hypothesis: maximum QT on placebo should be evenly distributed in the 2 halves of the study.

Results:

Measurement strategy	Maximum QT in 1 st half of study	Maximum QT in 2 nd half of study	p-value
EP1	7	17	0.009
PGY2	5	18	0.0003
EP2	9	14	0.24
Machine	9	14	0.24

Conclusion: A highly experienced observer reading in real-time as patients progress from "baseline" to "drug phase" may be prone to an expectation bias that leads to apparent QT prolongation (EP1). An inexperienced reader may be prone to random error (PGY2). An experienced reader using a magnifying lamp appears least prone to introducing error, but in a study of subjects with no heart disease, accepting the QT measurements of the ECG machine may be the optimal strategy.

EFFECTS OF SERIAL PHLEBOTOMY ON VASCULAR ENDOTHELIAL FUNCTION IN VOLUNTARY BLOOD DONORS; INSIGHTS FROM A PROSPECTIVE, RANDOMIZED, DOUBLE BLIND STUDY

Qurat-ul-ain Jelani, MD^{1,2}, Bisham Harchandani, MD², Ritchard G. Cable, MD⁴, Yu Guo, MS³, Judy Zhong, Ph.D.³, Timothy Hilbert, MD, Ph.D., JD⁴, Jonathan D. Newman, MD, MPH², Stuart D. Katz, MD, MS²

¹Bridgeport Hospital, Yale New Haven Health, ²New York University School of Medicine, Departments of Medicine and ³Population Health, New York University Langone Medical Center, New York, NY USA and ⁴American Red Cross, Farmington, CT USA



Qurat-ul-ain Jelani, MD
Bridgeport Hospital

Original Research.

Objective: Blood donation has been proposed to be associated with reduced risk of cardiovascular disease, but the effects of phlebotomy on vascular function in human subjects have not been well characterized. We conducted a prospective randomized double-blind study to determine the effects of serial phlebotomy on vascular endothelial function in the brachial artery.

Approach and Results: 84 iron (Fe)-replete, non-anemic subjects were randomly assigned to one of three study treatment groups: 1) four serial phlebotomy procedures each followed by intravenous infusion of placebo normal saline; 2) four serial phlebotomy procedures each followed by intravenous infusion of intravenous Fe to replete lost iron; and 3) four serial sham phlebotomy procedures each followed by infusion of placebo normal saline. Assigned phlebotomy procedures were conducted at 56-day intervals. We measured brachial artery reactivity (BAR, %) in response to oral methionine with high-resolution duplex ultrasound imaging before and one week after the fourth study phlebotomy. Before phlebotomy, oral methionine decreased BAR by -2.04% (95% CI -2.58, -1.50%), $p < 0.001$ with no significant difference between groups ($p = 0.42$). After phlebotomy, the response to oral methionine did not significantly change between groups ($p = 0.53$). Brachial artery nitroglycerin-mediated dilation did not change in response to phlebotomy.

Conclusions: Serial phlebotomy with or without intravenous iron supplementation did not alter the effects of oral methionine administration on BAR when compared with sham phlebotomy. These findings do not support a link between the effects of blood donation and vascular endothelial function.

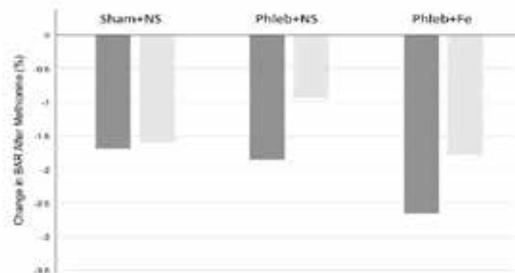


Figure 1. Mean brachial artery reactivity (BAR) response to oral methionine (%) before study phlebotomy (visit 2, dark gray bars) and one week after completion of study phlebotomy (visit 7, light gray bars) by randomized study phlebotomy groups. The post-phlebotomy decrease in BAR in response to oral methionine administration did not differ between randomized study phlebotomy groups (treatment by visit interaction $p = 0.53$).

HEART FAILURE SELF-MANAGEMENT USING A MOBILE WEB-BASED TELEMONITORING SYSTEM- IMPACT ON HOSPITAL READMISSIONS AND QUALITY OF LIFE. SELF-E HF

*OM Penciu MD PhD¹, I Tulai MD¹, A Butler MPH¹, AC Mihiu MD¹, R Bajwa MD¹, S
Osborne RN, BSN¹, K Doughty RN, BSN, CHFN¹, I. Galin MD¹*

¹Western Connecticut Health Network- Danbury Hospital, Danbury, CT, USA



Oana Penciu, MD, PhD
Danbury Hospital

Category: original research

Background: There is an increased focus on heart failure (HF) readmissions as the data is not only publicly reported, but a higher than average readmission rate can also lead to financial penalties. Reducing HF readmissions requires a multifaceted approach. One of these aspects is through tele-health. iGetBetter offers a potentially cost-effective, comprehensive patient care management program by patients taking regular at-home biometric measurements.

Objectives: This is a pilot study to assess the effectiveness of a system for disease self-management in reducing readmissions and improving quality of life in patients admitted to Danbury Hospital with a primary diagnosis of HF, as well as patients' satisfaction with the intervention and the perceived effect on HF related care over a period of 45 days.

Methods: This is a prospective, randomized study that includes patients admitted for a primary diagnosis of HF and discharged home with no services between January 2015 and February 2017. The patients were identified at the time of the index admission and were randomized to receive usual care versus participation in the iGetBetter program. Patients were instructed to check their biometrics daily using the provided equipment. Data was automatically recorded in a secure database. The investigator received alerts every time a patient had data outside the set thresholds. The patients' cardiologist was contacted to make recommendations regarding management. A Minnesota Living with Heart Failure Questionnaire was provided to all patients, at the time of enrolment and at the completion of the study. A questionnaire regarding participants' perceived effect of the intervention on the HF related care and one on the satisfaction with the intervention was provided to the iGetBetter patients at the end of the study.

Results: 26 patients were included (12 in the iGetBetter group, 14 in the control group). 3 patients were excluded from the analysis (2 in the iGetBetter group, 1 in the control group). The majority of patients had HF with reduced ejection fraction (16 of 23 patients in the study). There were 2 readmissions in the control group (both for HF) and one readmission in the iGetBetter group (for non HF). 11 out of 12 patients in the intervention group had more than one alert during follow up leading to medication changes and/or urgent outpatient follow up. All patients in the study reported significant improvement in their quality of life compared to baseline.

Conclusion: In this pilot study we show that iGetBetter is a practical way to follow a cohort of patients discharged with the diagnosis of HF and is a potential means to prevent readmissions. A larger scale trial with well-matched groups is indicated to further prove this idea.



Connecticut
CHAPTER

Case Vignette Abstracts

CASE VIGNETTE ABSTRACTS

RARE CAUSES OF HYPOXIA

Sadiya Thermidor, MD¹. Somera Chaudhry, MD¹.

¹ Stamford Hospital/Columbia University College of Physicians and Surgeons
Case Vignette



Sadiya Thermidor, MD
Stamford Hospital

A patent foramen ovale (PFO) occurs when the foramen ovale between the left and right atria fails to close after birth. While the prevalence of PFO's indicates that it is not an uncommon finding in our population – affecting 25 - 30% of individuals - they remain clinically silent in a majority of people. Neurological findings, such as CVA and migraines, are not uncommonly associated with PFOs; however, rare phenomenon such as platypnea-orthodeoxia syndrome, where dyspnea and desaturation occur in the upright position, co-existing anatomical variants, such as eustachian valve, and concurrent electrolyte abnormalities make an otherwise “benign” medical condition, become a surgical emergency.

An 82 year-old F with a past medical history of patent foramen ovale sent from her rheumatologist's office for progressively worsening shortness of breath and hypoxia. On admission, patient was notably hypoxic on room air with oxygen saturations in the low 80's and PaO₂ on ABG of 39. Patient was admitted with a presumption that worsening SOB and hypoxia was in the setting of a pulmonary versus a cardiac process. CTA showed no evidence of a pulmonary embolism, pneumonia, infiltrates, or interstitial changes. PFT's showed minimally reduced DLCO without evidence of restrictive or obstructive disease. Echocardiogram was subsequently done and showed a PFO with right-to-left intracardiac shunting with Valsalva that was not present on echocardiogram in 2011. With ongoing hypoxia and laboratory findings with evidence of severe hypophosphatemia, electrolyte abnormalities were considered as a possible culprit for hypoxia in the setting of respiratory muscle depression. Despite aggressive repletion and correction of hypophosphatemia she continued to have worsening hypoxia. Cardiac catheterization was done and showed no evidence of obstructive disease. Transesophageal echocardiogram confirmed a large PFO with right-to-left shunting and a eustachian valve in the right atrium. Given findings on investigative studies, patient was diagnosed with platypnea-orthodeoxia syndrome, which was exacerbated by a stretched PFO with a prominent eustachian valve further worsening hypoxia by directing blood flow towards the PFO. Patient was emergently transferred to Columbia University Medical Center for transcatheter closure of the PFO. Immediately following the procedure, patient's hypoxia improved with O₂ saturations increasing from 85% prior to closure to 98% following balloon closure. Since hospital discharge, patient has been weaned off of all supplemental oxygen, participated in a rehabilitation program, and is currently living independently.

Frequently, chronic medical conditions are over-looked in an acute setting. It is critical that physicians keep an open-minded approach and re-evaluate conditions that are otherwise thought to be stable. Although an enlarging PFO and the subsequent development of platypnea-orthodeoxia syndrome is uncommon, it is imperative to diagnose this rare cause of hypoxia in order to prevent delays in life-saving treatment.

CASE VIGNETTE ABSTRACTS

STEMI MANAGEMENT IN A PATIENT WITH ANEMIA AND OCCULT GI BLEEDING: WHAT IS THE BEST OPTION?

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Case Vignettes

Background: There are no clear guidelines to direct management of a STEMI patient with occult GI bleeding.

Case: A 68-year old man presented with 4 hours of chest pain and ECG showing anterolateral Q-waves and ST-segment elevation. He was rushed to the Cath Lab. Just as a 99% proximal stenosis of a large 1st diagonal artery was to be crossed admission CBC returned showing Hb=7.4 g/dL with low MCV of 73.3fL. A bare metal stent (BMS) was placed. Flow was restored. Dual anti-platelet therapy (DAPT) was initiated with aspirin (ASA) and Clopidogrel. Subsequent colonoscopy revealed an adenocarcinoma. Surgical resection was postponed for 6 weeks at which time Clopidogrel would be held and ASA continued. Was this the best strategy?

Decision-analysis: We performed a Markov-style decision analysis of 3 potential strategies: I: Plain Old Balloon Angioplasty (POBA) + ASA for 12 months (mos); II: BMS + DAPT for 1 mo, then just ASA for 11 mo; and III: Drug-eluting stent (DES) + DAPT for 6 mos, then ASA for 6 mos. We performed a literature review to estimate, for each strategy, the likelihood of “bad outcome,” defined as combination of coronary lesion MACE and severe bleeding. Results using clopidogrel are summarized in the table:

Strategy	6-month “Bad Outcome”			12-month “Bad Outcome”		
	MACE	Severe Bleed	Total	MACE	Severe Bleed	Total
I (POBA)	27.5%	0.3%	27.7%	34.8%	0.5%	35.3%
II (BMS)	12.0%	0.3%	12.3%	16.7%	0.57%	17.3%
III (DES)	8.2%	0.42%	8.6%	10.1%	0.84%	10.9%

Use of prasugrel resulted in total 12-month “Bad Outcome” of 17.3% for BMS and 11.2% for DES.

Use of ticagrelor resulted in total 12-month “Bad Outcome” of 19.3% for BMS and 19.1% for DES.

A sensitivity analysis in which bleeding risks were increased up to 10-fold did not alter the relative merits of the 3 strategies.

Conclusion: This case highlights the interplay of future bleeding risk and lesion outcome in the determining stent selection in STEMI patients who present with occult GI bleeding. Our analysis suggests that POBA is the inferior strategy and that outcome with DES is the superior strategy.



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CASE VIGNETTE ABSTRACTS

AN UNUSUAL CAUSE OF CONGESTIVE HEART FAILURE

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Third Place Winner

Iulia Tulai, MD
Danbury Hospital

Category: Case vignette

Introduction An overwhelming number of patients with heart failure with reduced ejection fraction (HFrEF) have coronary artery disease, valvular heart disease, toxic or viral cardiomyopathy (CMP) as underlying cause. We present the case of a patient with Systemic Lupus Erythematosus (SLE) and antiphospholipid syndrome (APS) who not only posed a challenging differential diagnosis, but had a surprising and rarely described etiology for her congestive heart failure (CHF).

Case presentation A 46-year-old female with a history of SLE, lupus associated aortic (AR) and mitral regurgitation (MR), APS and CMV viremia presented to an outside hospital with signs and symptoms of volume overload, presumably due to new onset nephrotic syndrome. A kidney biopsy was non-diagnostic and complicated by a perinephric hematoma, leading to discontinuation of her anticoagulation. Two weeks later, the patient presented to Danbury Hospital with worsening signs of congestion. Echocardiography revealed severe AR and MR, unchanged from the prior study. The left ventricle (LV) was now dilated, with global hypokinesis, and severely reduced ejection fraction of 30-35%.

Blood cultures were negative, and the TEE confirmed primary AR and MR, with no evidence of infectious endocarditis. The coronary angiogram showed normal coronary arteries. The patient had persistent CMV viremia, raising concern for viral myocarditis. Associated severe proteinuria and cardiac dysfunction pointed towards lupus nephritis and carditis. Furthermore, given her history of APS, thrombotic microangiopathy (TMA) with cardiac and renal involvement was also in the differential. Myocardial and renal biopsies were obtained; they both demonstrated findings consistent with TMA. The diagnosis of catastrophic APS (CAPS) was made and the patient was treated with anticoagulation, high dose steroids and plasmapheresis, followed by IVIG and Rituximab. Her CMP resolved and she underwent successful elective double valve replacement.

Conclusion TMA describes a pathological process of microvascular thrombosis, consumptive thrombocytopenia and microangiopathic hemolytic anemia, leading to end-organ ischemia. Most often this involves the kidneys and the brain. Cardiac involvement has also been described and has a significant impact on mortality. In a study of 220 patients with TMA, 9.5% of the subjects developed heart failure, which conferred a significant, two fold increase in mortality in comparison to the general population.

It is critical that physicians, and practitioners in general, are aware of the TMA predisposing conditions and recognize TMA in patients with otherwise unexplained heart failure, as individualized treatment is paramount. In our patient, we diagnosed TMA secondary to CAPS. This is the most severe form of APS with rapidly progressing multi-organ involvement. Specific CAPS treatment with anticoagulation, high dose steroids, plasmapheresis followed by IVIG and Rituximab saved our patient's life.

CASE VIGNETTE ABSTRACTS

LOPERAMIDE INDUCED VENTRICULAR TACHYCARDIA: AN ELECTRO-PHARMACOLOGICAL CASE STUDY

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Case vignette.



*Kwasi Dakwa, MD
Bridgeport Hospital*

Background: Loperamide is an over-the-counter anti-diarrheal agent. It activates μ -opioid receptors in the gastrointestinal tract thereby slowing peristalsis. At its recommended maximum dose of 16 mg a day, it is free from abuse potential owing to first pass metabolism and poor blood brain barrier penetration. High doses, however, may cause euphoric effects. This has led to its recent use as a cheap solution to reduce opioid withdrawal symptoms (a methadone alternative) and recreational abuse. In February 2016, the FDA reported that loperamide overdose had caused serious arrhythmias in 48 people. In vitro animal studies have demonstrated blockade of cardiac sodium (INa) and potassium (IKr) channels.

Objective: We report the course of a woman who took excessive doses of loperamide and whose clinical course demonstrated electro-pharmacological effects predicted by in vitro studies.

Case: A 24-year-old female daily loperamide user presented with recurrent syncope. Initial ECG showed monomorphic, wide, sine wave-like VT (QRS=310 ms); VT termination was followed by NSR with PR=240 ms and initial QRS=240 ms that narrowed to 180 ms within 20 seconds. A Brugada-like pattern was noted in V1-2. The QTc was >550 ms. The next day she experienced two episodes of torsades de pointes requiring defibrillation. ECG shortly after these episodes showed a QRS < 120 ms but QTc=560 to 610 ms. The QRS normalized 24 hours after admission; the QTC remained prolonged for 10 days.

Discussion: Klein et al and Kang et. al., using an in vitro model of human embryonic kidney cells, reported that loperamide blocked INa at very high concentrations [IC50=297 nmol] and IKr at lower concentrations [IC50=of 89 nmol]. Our patient's course provides in vivo support for the clinical relevance of these in vitro observations. At presentation, when serum levels would have been at their highest, ECG demonstrated rate-dependent QRS prolongation, very wide-complex monomorphic VT and Brugada-like effects, all features of INa blockade. After 24 hours, these effects dissipated (likely reflecting declining loperamide levels) and the ECG demonstrated QTc prolongation and torsades de pointes, features of IKr block. These IKr effects persisted for ~10 days, likely reflecting prolongation of loperamide clearance beyond its usual 12-hour half-life of elimination at high serum concentrations (in loperamide overdose, drug elimination half-life has been reported to be as long as 41 hours) and the lower serum concentrations needed to block IKr. Ultimately, all VT ceased, the ECG intervals normalized, and ICD implant was not a consideration. She was counseled to stop using loperamide!

CASE VIGNETTE ABSTRACTS

ACUTE CORONARY SYNDROME AND LONG QT- A CASE REPORT

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Resident Internal Medicine, Yale New Haven Health Bridgeport Hospital, CT

Case Vignettes



Mohammad Adeel, MD
Bridgeport Hospital

A 29 years old Caucasian female with no past medical history presented with acute inferior STEMI. Patient's social history was significant for 13 pack years of smoking. Patient received primary PCI with drug eluted stent to left circumflex artery and made good recovery. Post PCI EKG showed sinus rhythm, normal axis, resolution of ST segments and QTc of 589ms which was attributed to acute MI. Patient was discharge home after 5 days of uneventful hospital course, EF at discharge was 35-40% with inferior hypo kinesis.

A month after discharge, patient presented to hospital with generalized weakness, menorrhagia and PV bleeding. Patient was noted to have an Hb of 5.8 g/dl with low MCV. At that stage DAPT was held to carry out dilatation and curettage under GA for evaluation and treatment of bleeding. Procedure was complicated as patient developed cardiac arrest with pulseless VT and requiring CPR. After ROSC, loading dose of 300 mg IV amiodarone was given and patient was transferred to cath lab on continuous infusion. EKG obtained showed sinus tachycardia, no acute ischemic changes, and QTc of 671ms. This prolongation in QTc was attributed to amiodarone. Coronary angiogram performed showed unchanged coronary anatomy with patent stent. Amiodarone was discontinued.

With unclear cause of cardiac arrest, in next 24 hours patient had 4 further arrests with pulseless VT requiring multiple rounds of CPR. Patient eventually stabilized and underwent EP studies (EPS) to clarify the cause of malignant arrhythmias. EPS, however, failed to induce VT. Based on persistently prolonged QTc on serial EKGs, multiple VT events and EPS results, diagnosis of congenital long QT syndrome was made. This was later confirmed by genetic analysis showing mutation of gene *KCNQ1*. Patient received single lead ICD and was discharged home after uneventful 48 hours in hospital.

Within a day, Patient was brought back to hospital with VT storm. ICD interrogation showed 38 appropriate ICD shocks. Patient was stabilized and later ICD was upgraded to dual chamber ICD with override pacing. Patient remained asymptomatic after 1 year of follow up.

First, this case highlights the importance of keeping all possibilities in differentials when dealing with EKG changes like prolong QTc especially in acute setting. In this case different factors eluded the underlying diagnosis, including acute ischemia and use of QT prolonging medication i.e. amiodarone. Secondly the atrial pacing in an inherently unstable condition like LQTS is critical. Finally it is believed that recent ischemic insult coupled with adrenergic drive in setting of hemorrhage unmasked previously asymptomatic and unknown long QTc which to record is an unusual presentation of the condition.

CASE VIGNETTE ABSTRACTS

THE CHALLENGE OF COMBINED AORTIC AND MITRAL VALVE DISEASE

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Case Vignettes



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Clinical Presentation

A 28-year-old woman with history of endocarditis who underwent bioprosthetic aortic valve (AV) replacement and mitral valve (MV) repair 11 years prior due to severe aortic insufficiency now presents with dyspnea on exertion and chest pain. She had moderate AV stenosis noted on an echocardiogram 8 months before onset of symptoms, but had progressed to severe stenosis with a mean gradient of 58mmHg. On transesophageal echocardiogram (TEE), the AV had reduced opening with a peak velocity of 4.0 m/s, effective aortic orifice of 1.0 cm²; similar mean gradient and planimetry of 0.97cm² were obtained. The MV on initial TTE was thickened, had a mean gradient of 6 mmHg and on subsequent TEE, the MV repair appeared intact with trace mitral regurgitation, MV area was 1.5 cm² by pressure half time method, a right ventricular systolic pressure of 20-25mmHg and a dimensionless index of 1.7 at a heart rate of 70 bpm. Given concern of a redo sternotomy and presence of symptoms attributed to the aortic valve, further clarification of the MV was required.

Imaging Findings

On supine bicycle exercise stress echocardiogram the patient had a poor exercise capacity of 4 minutes and 21 seconds before developing symptom-limiting shortness of breath. Her heart rate and blood pressure had increased from 83 bpm and 98/67 mmHg at rest to 120 bpm and 124/60 mmHg with stress. Her MV gradient increased from 6 mmHg to 19 mmHg.

Role of Imaging in Patient Care

TEE elucidated the nature of the aortic bioprosthetic valve stenosis but the stress echocardiogram was critical in identification of significant mitral valve stenosis. The contribution of MV to dyspnea on exertion in setting of severe aortic stenosis would have been difficult to determine otherwise; therefore, planning for additional surgical intervention to the MV was included.

Summary and Discussion Points

Combined aortic and mitral valve disease is challenging especially when attempting to define which valve or both are responsible for symptoms. Regarding this patient's MV repair, her resting evaluation was somewhat abnormal but on bicycle stress revealed significantly different hemodynamics. This case highlights the importance of a functional evaluation of a prosthetic MV when evaluating for symptoms and especially when other cardiac surgery is planned.

CASE VIGNETTE ABSTRACTS

TO CLOSE OR NOT TO CLOSE? MANAGEMENT OF PATENT FORAM OVALE (PFO) IN THE TIMES OF UNKNOWN

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Yeunjung Kim, MD, MPH
Yale School of Medicine

Case Vignette

Background: While routine closure of patent foram ovale (PFO) is not recommended, the appropriate management of PFO remains unknown in patients with arterial thromboembolism that may be paradoxical.

Case: 43 year old Caucasian man with recurrent deep venous thrombosis (DVT) on rivaroxaban and reporting strict compliance, presented with severe right leg pain. CTA revealed multiple occlusive venous (right femoral and popliteal veins) and arterial thrombi (right superficial femoral and posterior-tibial artery, along with bilateral renal infarcts). The patient required urgent exploration and embolectomy, complicated by right lower extremity compartment syndrome, requiring fasciotomy and treatment with bivalirudin. Investigation of possible embolic sources revealed evidence of new apical left ventricular (LV) hypokinesis with thrombus, and a PFO on TTE. Subsequently, cMRI demonstrated delayed enhancement at the apical lateral wall (consistent with a transmural infarct) and associated LV thrombus, as well as an aneurysmal interatrial septum and prominent Chiari network. Coronary angiography demonstrated clean coronaries, and a hypercoagulability work up with genetic testing revealed heterozygous a factor V Leiden mutation. Given the risk of recurrent DVTs and paradoxical embolism, PFO closure was recommended along with traditional anticoagulation with Coumadin and daily aspirin.

Discussion: Diagnostic studies revealed multiple circumstances leading to arterial thromboembolism. Without evidence of coronary disease, isolated anterolateral transmural infarct on cMRI was suggestive of atypical variant Takotsubo cardiomyopathy (focal type) versus paradoxical embolus, both with associated LV thrombus. The cMRI obtained to characterize the LV thrombus and apical myocardium also revealed the presence of an interatrial septal aneurysm - not seen on TTE, providing evidence of added embolic risk. Given the findings and the identification of factor V Leiden on genetic testing, PFO closure was recommended and performed.

Conclusion: Multimodality imaging as well as genetic testing may be useful tools to determine future risk for thromboembolic events in setting of PFO and to guide further management.

CASE VIGNETTE ABSTRACTS

DIAGNOSIS ON THE TIP OF YOUR FINGERS

William Perucki, MD¹; Peter Schulman, MD¹

¹University of Connecticut School of Medicine

Case vignettes



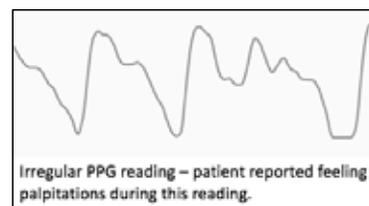
William Perucki, MD
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Introduction Palpitations are a common complaint that account for 16% of patient encounters with their primary care providers and prompt frequent referrals to a cardiologist. The differential diagnosis is broad and includes multiple types of arrhythmias, psychosomatic disorders, medications, stimulants, and recreational drug abuse. We present a case of a young patient with a prior history of SVT who presented years later with palpitations at rest.

Case: Our patient is a 32-year-old male with a past medical history of AV nodal reentry tachycardia (AVNRT) who presents years later for evaluation of recurrent palpitations. He first experienced palpitations at age 15 while training in a martial arts class. He was transported to a local ED where AVNRT was documented and terminated with a Valsalva maneuver. Evaluation at that time revealed no evidence of congenital or acquired heart disease; he was discharged on no cardiac medications. In the ensuing years, he experienced infrequent, brief episodes of palpitations until age 22; he was free of palpitations for 8 years. At the age of 30 he experienced 2 episodes of palpitations, each lasting a few minutes and terminating spontaneously. He was again free of symptoms for 2 more years, when he presented with 5 consecutive days of palpitations at rest without clear precipitation or alleviating factors. There was no associated syncope, presyncope, chest pain, orthopnea, paroxysmal nocturnal dyspnea, dyspnea at rest or with exertion. He drinks rarely though has never smoked tobacco or used illicit drugs. Physical exam and electrocardiogram were normal. Concerned about a recurrence of his previous AVNRT, the patient obtained a pulse reading during his episodes via an application on his mobile phone. Based on the tracing reviewed, the patient's palpitations were determined to be due to premature ventricular contractures.

Discussion: Traditional methods of detecting cardiac arrhythmias include electrocardiographic monitoring equipment that requires multiple electrodes to be worn by the patient. In our case, the patient used an "app" on his smart phone that utilizes photoplethysmography (PPG). PPG uses infrared light transmission through the fingertip to produce a wave form of peripheral blood volume changes that correlates with the patient's arterial pulse, and in our case, clearly demonstrated PVCs.

On arterial pulse tracings, PVCs appear as early beats often with a compensatory pause and result in a transient decrease and then an increase in arterial blood flow that is sometimes sensed by the patient. On the arterial pulse tracing, PVCs have a earlier and smaller than normal wave form than the normal sinus beats. Proposed algorithms for detecting premature ventricular contractions by PPG have been proposed by Sološenko et al, however these are not yet being commonly used.



By using data obtained from a mobile phone app, we were able to diagnose PVCs and avoid the cost and inconvenience of more extensive diagnostic testing.

CASE VIGNETTE ABSTRACTS

COMPLEX CONGENITAL HEART DISEASE, ATRIAL ARRHYTHMIA AND SINUS NODE DYSFUNCTION

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Case Vignette

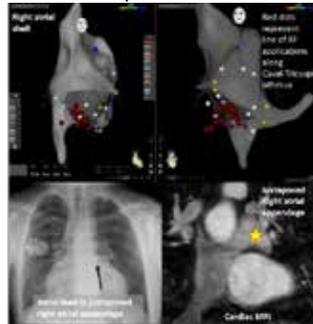
Introduction: Congenital heart disease (CHD) population has been on the rise. Long term issues in this population include arrhythmia, sinus node dysfunction and complex anatomy and vascular access limitations may pose challenges for electrophysiology interventions.

Case: A 14-year-old male with mesocardiac, D-transposition of the great arteries, ventricular septal defect (VSD) and juxtaposed right atrial appendage (RAA). He had been repaired with an arterial switch operation and he did have a residual restrictive VSD. The patient had underlying sinus dysfunction with sinus bradycardia, sinus pauses of up-to 6 seconds, junctional escape rhythms. The course was also complicated with episodes of atypical atrial flutter that required DC cardioversion and medical management (Flecainide). He underwent an electrophysiology study. Bilateral femoral venous occlusion, left subclavian occlusion limited vascular access for ablation. Radiofrequency (RF) ablation with line of RF applications along the caval tricuspid isthmus was created by utilizing right internal jugular access and guidance with CARTO™ 3D electroanatomic mapping. Subsequent to the ablation a single chamber pacemaker was implanted via the right axillary vein access, the lead tip was positioned in the juxtaposed RAA. Dual chamber pacemaker was avoided (residual VSD and intact AV condition). [Image]. Patient's medications were discontinued with no arrhythmia recurrence and improved exercise capacity. **Discussion:** Patients with complex congenital heart disease may require individualized approach to interventional arrhythmia management. Often limitations from complex anatomy, vascular access make interventional electrophysiology procedures a challenge.

Conclusion: Good understanding of anatomy in complex CHD patients and a good planning including the knowledge of vascular access limitations can lead to successful electrophysiology interventions in these patients.



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CASE VIGNETTE ABSTRACTS

5-FLUOROURACIL INDUCED TAKOTSUBO CARDIOMYOPATHY

Chandana Shekar MD, Supriya Tigadi MD

University of Connecticut Health Center

Case Vignette



Chandana Chekar, MD
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A 47 year old male with history of squamous cell carcinoma of the tongue, status post glossectomy, was admitted for chemotherapy for recurrent disease. He was on a standard chemotherapy regimen of cisplatin, docetaxel, and 5-Fluorouracil (5-FU). On the 3rd day of therapy with continuously infused 5-FU, he had an episode of syncope. Pertinent negatives included chest pain, shortness of breath and palpitations. He was hemodynamically stable and physical examination was within normal limits. Initial electrocardiogram (EKG) did not show any changes suggestive of ischemia or infarction, similar to baseline EKG. However, repeat EKG showed 1-2mm ST segment elevation in anterolateral leads. Emergent coronary angiography findings revealed no significant coronary artery disease. Echocardiogram was notable for a newly depressed left ventricular ejection fraction of 30–35 %, with moderate diffuse hypokinesis. Follow up echocardiograms over a week demonstrated an improvement in the EF to 45% and later to 50%. 5-FU therapy was discontinued. His presentation was attributed to Takotsubo Cardiomyopathy (TCM) with global affection of the left ventricle due to chemotherapy induced cardiotoxicity. He was treated with beta blockers and ACE-I. His EKG findings gradually resolved and he was discharged without symptoms.

Discussion: We describe a case of TCM after 5-FU exposure, supported by angiographic, electrocardiographic, and echocardiographic features. The antimetabolite chemotherapeutic agent, 5-FU interferes with DNA synthesis in cancer cells, leading to cell death. Cardiotoxicity due to this drug was first reported in 1975. The prevalence of 5-FU cardiotoxicity is reportedly 1-18% with a 0-13% mortality rate. The majority of cases present with anginal chest pain likely secondary to coronary vasospasm, which comprises 40–60 % of all initial cardiotoxic presentations; followed by myocarditis, cardiomyopathies and arrhythmias. TCM is also rarely reported as 5-FU cardiotoxicity. Myocardial microvascular spasm is one of the debated causal mechanisms underlying TCM. The toxic effect exerted by 5-FU and its oral prodrug capecitabine on the vascular endothelium involves the dysregulation of endothelial nitric oxide synthase and the induction of protein kinase C, leading to coronary spasms and endothelium-independent vasoconstriction. Alternative theories have implicated autoimmune phenomena and direct thrombogenic effects as well. Increased incidence of toxicity has been found with continuous infusion compared to bolus infusion. Discontinuation of 5-FU is required since relapse of symptoms occur in up to 90% of patients upon re-challenge. All patients on 5-FU infusion should be monitored for potential cardiotoxicity. As myocardial ischemia is the most frequent manifestation, early intervention with coronary angiography should be considered. Guideline directed medical therapy and immediate discontinuation of 5 FU is recommended once cardiotoxicity is detected. Potential genetic predisposition to cardiotoxicity from 5 FU should be investigated.

CASE VIGNETTE ABSTRACTS

MAY-THURNER SYNDROME: AN UNDERDIAGNOSED CAUSE OF DEEP VENOUS THROMBOSIS

Sara Villegas, PA-Student¹; Cindy Rossi, MHS, PA-C¹

1. Quinnipiac University

Case Vignette



Sara Villegas, PA-Student
Quinnipiac University

Introduction: May-Thurner Syndrome (MTS), also known as iliac vein compression syndrome, is caused by a common anatomical variant where the right common iliac artery (RCIA) overlies the left common iliac vein (LCIV) causing it to compress against the underlying vertebrae and obstruct venous flow. It was first described by R. May and J. Thurner in 1957. It is thought to be present in greater than 20% of the general population and to be responsible for 2-3% of all deep venous thrombosis (DVT). However, this is likely an underestimate of the true prevalence as it believed to be underdiagnosed. Patients with May-Thurner Syndrome most commonly present with signs and symptoms of extensive or recurring DVT formation in the left lower extremity without coagulopathies, risk factors, or other common causes. Currently, the standard treatment for MTS includes catheter directed thrombolysis, anticoagulation therapy, and stent placement to prevent recurrent DVTs and future pulmonary embolisms.

Case Description: A 44 year old male presented with pain, swelling and the inability to bear weight on the left lower extremity for two weeks. Symptoms began several days after a dog bite of the same leg. He denied any history of coagulopathies, contributing family history, or other DVT risk factors. The physical exam demonstrated edema and diminished dorsalis pedis and posterior tibialis pulses of the left lower extremity. The left calf was tender to palpation without erythema. The patient was afebrile and lung sounds were clear throughout. Doppler ultrasound confirmed extensive DVT formation in left common iliac, common femoral, popliteal and saphenous veins.

Outcome: The patient's treatment included ultrasound guided vascular access and initiation of tissue plasminogen activator (tPA) for thrombolysis for 24 hours in the intensive care unit. The following day, the patient returned to the operating room for mechanical thrombolytic therapy and visualization of the anatomy with intravascular ultrasound (IVUS). IVUS confirmed May-Thurner Syndrome as the diagnosis and the underlying cause of the venous compromise. After the diagnosis was confirmed, venous angioplasty and stenting of the LCIV were performed. Lastly, an inferior vena cava filter was placed and the patient was started on apixaban indefinitely for long-term anticoagulation therapy.

Conclusion: Currently, treatment of MTS focuses on thrombolysis of the DVT followed by correction of the underlying anatomical cause and anticoagulation therapy. Since MTS is thought to be present in at least 20% of the population and the underlying cause of many DVTs, it is imperative for clinicians to consider MTS in the differential diagnosis for patients presenting with extensive or recurring DVT formation. Accurate diagnosis of MTS is necessary so that the appropriate steps can be taken to treat the condition, avoid recurrence, and prevent life-threatening sequelae.



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