PancreasFest 2023

July 27-28, 2023
Pittsburgh, PA

FINAL PROGRAM

913.402.7102 | CAPER@lp-etc.com | www.caperpancreas.org

Thank you to the following companies for their support

Educational Grants:

AstraZeneca Pharmaceuticals
AbbVie Inc.
Boston Scientific Corporation
Cook Medical, LLC
Digestive Care, Inc.
Olympus Corporation of the Americas

Exhibitors:

AbbVie
Alcresta Therapeutics
Boston Scientific
ChiRhoClin, Inc.
Cincinnati Children's Hospital Medical Center
Cook Medical
Digestive Care, Inc.
Immunovia, Inc.
Ipsen
Fujifilm Healthcare Americas
National Pancreas Foundation
Nationwide Children's Hospital
Nestle Health Science I US Pharmaceuticals
Olympus
Regeneron Pharmaceuticals, Inc.
University of Pittsburgh
UPMC Cancer Pavilion
UPMC PancreaSeq
VIVUS

PancreasFest 2023 Organizing Committee

Co-Chairs:

Maisam Abu-El-Haija MD, MS
Randall Brand MD

Committee Members:

Kathryn Albers PhD
Melena Bellin MD
Darwin Conwell MD, MSc
Zobeida Cruz-Monserrate PhD
Steven Hughes MD
David Whitcomb MD, PhD
Dhiraj Yadav MD, MPH

Continuing Medical Education

In support of improving patient care, the University of Pittsburgh is jointly accredited by the Accreditation Council for Continuing Medical Education (ACCME), the Accreditation Council for Pharmacy Education (ACPE), and the American Nurses Credentialing Center (ANCC), to provide continuing education for the healthcare team.

Physician (CME)
The University of Pittsburgh designates this live activity for a maximum of 12.5 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Other health care professionals will receive a certificate of attendance confirming the number of contact hours commensurate with the extent of participation in this activity.

Faculty Disclosure:
All individuals in a position to control the content of this education activity have disclosed all financial relationships with any companies whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients.

All of the relevant financial relationships for the individuals listed below have been mitigated.

Venkata Akshintala	Origin Endoscopy Inc	Stockholder (privately held)
Venkata Akshintala	Abbvie	Grant/Research Support
Venkata Akshintala	Medtronic	Grant/Research Support
Venkata Akshintala	Boston Scientific	Grant/Research Support
Fiyinfolu Balogun	Bristol Myers Squibb Foundation	Grant/Research Support
Melena Bellin	Viacyte	Grant/Research Support
Melena Bellin	Vertex	Consultant
Melena Bellin	Dexcom	Grant/Research Support
Melena Bellin	Ariel Precision Medicine	Consultant
Randall Brand	Freenome	Grant/Research Support
Randall Brand	Immunovia	Grant/Research Support
Gregory Cote	Interpace Diagnostics	Consultant
Channing Der	Mirati Therapeutics	Consultant
Channing Der	Revolution Medicines	Consultant
Channing Der	SHY Therapeutics	Consultant
Channing Der	Reactive Biotherapeutics	Consultant
Channing Der	Deciphera Therapeutics	Consultant
Channing Der	Cellgen	Consultant
Phil Greer MS	Ariel Precision Medicine	Other: Employee
Aimee Lucas	Immunovia	Grant/Research Support
Aimee Lucas	ClearNote Health	Consultant
Ajith Siriwardena	ThermoFisher Scientific	Speakers' Bureau
Ajith Siriwardena	Bayer AG	Speakers' Bureau
Kristen Skvorak PhD	Codexis, Inc	Stockholder (publicly held)
Andrew Trout MD	Perspectum Inc.	Grant/Research Support
Aliye Uc	Abbvie Inc	Consultant
David Whitcomb	Ariel Precion Medicine	Consultant
David Whitcomb	Ariel Precion Medicine	Stockholder (privately held)
David Whitcomb	Nestle	Consultant
David Whitcomb	Organon	Consultant
David Whitcomb	AbbVie	Consultant

No other members of the planning committee, speakers, presenters, authors, content reviewers and/or anyone else in a position to control the content of this education activity have relevant financial relationships with any companies whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients.

AWARDS

CAPER 2023 Senior Mentor Award

In Recognition of Outstanding Achievements and Contributions to the Field of Pancreatology

Dhiraj Yadav MD, MPH
University of Pittsburgh Medical Center

CAPER Senior Mentor Award – Pasts Recipients

2013	Peter Banks MD
2014	Fred Gorelick MD
2015	David C Whitcomb MD, PhD
2016	Suresh Chari MD
2017	Vay Liang W. (Bill) Go MD
2018	Stephen Pandol MD
2019	Randall E. Brand MD
2021	Mark Lowe MD
2022	Darwin Conwell MD

The Ruth C. Brufsky Award for Excellence in Pancreatic Cancer


Ruth and Allen Brufsky

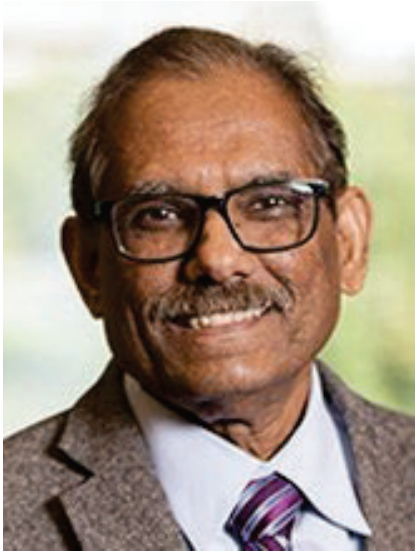
Ruth Brufsky was born in New York City in 1939. She was an only child. She met her husband Allen in Brooklyn when she was 13. They married in 1960.

She graduated from CCNY in 1960 and spent time as a school teacher in some of the most dangerous neighborhoods in New York City and Washington, DC before retiring to raise three children. She went back to law school in 1977, graduated on the law review at the University of Bridgeport Law School in 1980, and was an education lawyer in Southern Connecticut for 14 years until her death from pancreatic cancer in 1994. She was a dedicated attorney who represented most of the school boards in Southern Connecticut from Westport to New Haven. More importantly, most of her friends, family, and children remember her as one of the most generous and caring people they have ever known. She literally gave the shirt off of her back to her son Adam's wife Jill one day in a car in the early 1990s when Jill's shirt tore (a story we still recount almost 20 years later)—this was the kind of person she was. Everything in her life was to support her family and friends—she rarely if ever thought about herself.

Had she lived, she would have seen the success of the seeds she planted and nurtured. Her son Adam, a prominent breast cancer medical oncologist, is Professor of Medicine here at the University of Pittsburgh and an Associate Director of the Hillman Cancer Center. Her daughter, Naomi Brufsky Waltman, is a lawyer and a Corporate Vice President of CBS in New York in charge of all intellectual property litigation for the network. Her youngest son, Seth, is one of the founding partners of Ares Management, an alternative asset management firm based in Los Angeles. She would have seen eight grandchildren, two of whom are undergraduates at Stanford.

To honor her legacy, and to never forget the type of person she was and the life she led, Ruth's family has endowed the Ruth C. Brufsky Fund for Pancreatic Cancer Clinical Research. We are honored and delighted that this fund is being used not only to support clinical research in pancreatic cancer, but also to endow a yearly lecture during PancreasFest of a nationally prominent pancreatic cancer expert.

The Ruth C. Brufsky Award for Excellence in Pancreatic Cancer



Sudhir Srivastava PhD
National Cancer Institute

Ruth C. Brufsky Award for Excellence in Clinical Research on Pancreatic Cancer Recipients

2012	Ralph H. Hruban MD
2013	Anil K. Rustgi MD
2014	James L. Abbruzzese MD
2015	Peter J. O'Dwyer MD
2016	Terri Brentnall MD
2017	John Neoptolemos MD
2018	Eileen M. O'Reilly MD
2019	Brian M. Wolpin MD, MPH
2022	Diane Simone MD
2023	Sudhir Srivastava PhD

SCHEDULE-AT-A-GLANCE

Schedule-at-a-Glance

Thursday, July 27

7:15am – 8:45am	**Breakfast** *Kurtzman Room*
8:00am – 8:05am	**Welcome & Updates Since PancreasFest 2022** *Assembly Room*
8:05am - 8:35am	**Advances in Basic Science of Pancreatitis: Mechanistic Models** *Assembly Room*
8:35am - 9:35am	**Acute Pancreatitis Advancements** *Assembly Room*
9:35am – 10:35am	**Chronic Pancreatitis Advancements** *Assembly Room*
10:35am – 10:50am	**Morning Break** *Ballroom*
10:50am - 12:00pm	**Advancing Our Understanding of Common Conditions Which Pertain to Children and Adults** *Assembly Room*
12:00pm – 2:00pm	**Lunch** *Kurtzman Room*
1:15pm - 2:15pm	**Biomarkers, Genetics and Personalized Medicine in Pancreatitis** *Assembly Room*
2:15pm - 4:00pm	**CAPER Business Meeting, Awards & Lectures** *Assembly Room*
4:00pm	**Adjournment**
4:00pm - 5:00pm	**Poster Reception** *Kurtzman Room*
5:30pm - 7:30pm	**Attendee Reception** *Phipps Conservatory and Botanical Gardens*

Friday, July 28

7:15am - 8:45am	**Breakfast** *Kurtzman Room*
8:00am - 8:05am	**Welcome to PF23 Day 2** *Assembly Room*
8:05am - 10:05am	**Parallel Session | Pediatric Pancreatic Disease and Early Adulthood** *Breakout*
8:05am - 10:05am	**Parallel Session | Pancreatic Cancer** *Assembly Room*
10:05am - 10:20am	**Morning Break** *Ballroom*
10:20am - 11:15am	**The Ruth C. Brufsky Award for Excellence in Pancreatic Cancer** *Assembly Room*
11:15am - 12:15pm	**Advancing the Engagement of Underserved Populations in Pancreatic Diseases** *Assembly Room*
12:15pm - 1:30pm	**Lunch** *Kurtzman Room*
1:30pm - 3:30pm	**Interventions for Pancreatitis: New Approaches, Knowledge Gaps and Research Opportunities** *Assembly Room*
3:30pm	**Adjournment**

SCIENTIFIC PROGRAM

PancreasFest 2023 | Advances in Pancreatic Diseases

Wednesday, July 26, 2023

5:00pm – 6:00pm
CAPER Board Meeting
The Oaklander | The Henry Room

6:00pm - 7:30pm
Welcome Reception
The Oaklander | The Cathedral

Thursday, July 27, 2023

7:15am – 8:45am
Attendee Breakfast
Kurtzman Room

8:00am – 8:05am
Welcome & Updates Since PancreasFest 2022
Assembly Room
Maisam Abu-El-Haija MD, MS | Cincinnati Children's Hospital Medical Center
Randall E. Brand MD | University of Pittsburgh Medical Center

8:05am – 8:35am
Advances in Basic Science of Pancreatitis: Mechanistic Models
Assembly Room
Introduction: Randall E. Brand MD | University of Pittsburgh Medical Center
Miklos Sahin-Toth MD, PhD | David Geffen School of Medicine UCLA

8:35am - 9:35am
Acute Pancreatitis Advancements
Assembly Room
Moderators:
Georgios Papachristou MD, PhD | The Ohio State University Wexner Medical Center
Venkata Akshintala MBBS | Johns Hopkins University School of Medicine

Fluid Resuscitation
James Buxbaum MD | Keck School of Medicine of USC
Pancreas Club Best of the Best
A PROCALCITONIN-BASED ALGORITHM REDUCES ANTIBIOTIC USE IN PATIENTS WITH ACUTE PANCREATITIS: THE FINAL RESULTS OF THE PROCAP RANDOMISED CONTROLLED TRIAL
Ajith K. Siriwardena MD | University of Manchester
Do We Need a Cholecystectomy in the Index Admission of Acute Pancreatitis?
David C. Whitcomb MD, PhD | University of Pittsburgh School of Medicine
Panel Discussion

9:35am – 10:35am
Chronic Pancreatitis Advancements
Assembly Room
Moderators:
Phil Hart MD | The Ohio State University Wexner Medical Center
Juan Gurria MD | Cincinnati Children's Hospital Medical Center

Pain in CP
Vikesh Singh, MD, MSc | Johns Hopkins University School of Medicine
Pancreatic Drainage Procedures for Chronic Pancreatitis
William Nealon MD | Northwell Health
TPIAT for Chronic Pancreatitis
Gregory Wilson MD | University of Cincinnati Medical Center
Panel Discussion

10:35am – 10:50am
Break & Exhibits
Ballroom

10:50am – 12:00pm
Advancing Our Understanding of Common Conditions Which Pertain to Children and Adults
Assembly Room
Moderators:
Sohail Husain MD | Stanford University School of Medicine
Guru Trikudanathan MD | University of Minnesota

EUS Utilization From a Child to an Adult Pancreas
Roberto Gugig MD | Stanford University School of Medicine
Drug-Induced Pancreatitis Across the Life Span
Maisam Abu-El-Haija MD, MS | Cincinnati Children's Hospital Medical Center
Diabetes Pre and Post Surgery in Pancreatitis Patients
Melena Bellin MD | University of Minnesota
Panel Discussion

12:00pm – 1:15pm
Lunch & Exhibits
Kurtzman Room

1:15pm – 2:15pm
Advances in Biomarkers, Genetics and Personalized Medicine in Pancreatitis
Assembly Room
Moderator(s):
Elham Afghani MD, MPH | John Hopkins University School of Medicine
Greg Coté MD, MS | OHSU Healthcare

Imaging
Temel Tirkes MD | Indiana University School of Medicine
Circulating Protein Biomarkers of Pancreatitis (Acute and Chronic)
Zobeida Cruz-Monserrate PhD | The Ohio State University Wexner Medical Center
Panel Discussion

2:15pm – 4:00pm
CAPER Business Meeting, Awards, & Lectures
Assembly Room
Moderator: Maisam Abu-El-Haija MD, MS | Cincinnati Children's Hospital Medical Center

CAPER Business Meeting
Maisam Abu-El-Haija MD, MS | Cincinnati Children's Hospital Medical Center
CAPER Travel Scholarship Presentations
Melena Bellin MD | University of Minnesota
CAPER Senior Mentor Award
Introduction: Anna Evans Phillips MD, MS | University of Pittsburgh Medical Center
Attributes That Have Shaped My Career
Honoree: Dhiraj Yadav MD, MPH | University of Pittsburgh Medical Center
CAPER Young Investigator Presentations
Moderator: Mitch Ramsey MD | The Ohio State University
Acute Pancreatitis is Associated with Gut Dysbiosis in Children
Chinenye Dike, MD | University of Alabama at Birmingham
The Inflammatory Phenotype of Pancreatic Ductal Adenocarcinoma
Kelly Herremans MD | University of Florida College of Medicine
Leptin Expression and Cachexia in Pancreatic Ductal Adenocarcinoma
Andrea N. Riner MD MPH | University of Florida College of Medicine
Comparison of Current Assessment Tools for Children with Pancreatic Exocrine Insufficiency
Yuhua Zheng MD | Children's Hospital Los Angeles
Discussion

4:00pm
Adjournment

4:00pm - 5:00pm
Poster Reception *(not accredited, see poster listing for details)*
Kurtzman Room
Moderator: Kathryn Albers, PhD | University of Pittsburgh Medical Center

5:30pm – 7:30pm
Attendee Reception
Phipps Conservatory & Botanical Gardens

Friday, July 28, 2023

7:15am – 8:45am
Attendee Breakfast
Kurtzman Room

8:00am – 8:05am
Welcome to PF23 Day 2
Assembly Room
Maisam Abu-El-Haija MD, MS | Cincinnati Children's Hospital Medical Center
Randall E. Brand MD | University of Pittsburgh Medical Center

8:05am – 10:05am
Parallel Session – Pediatric Pancreatic Disease and Early Adulthood
Atrium
Moderator:
Andres Martinez MD | Vanderbilt University Medical Center

Clinical Trials Needed in Children
Aliye Uc MD | University of Iowa
Endoscopic Management in Pediatric Pancreatitis
Tom Lin MD | University of California San Diego
Which Imaging Do You Need for Your Patient with Pancreatic Disease?
Andrew Trout MD | Cincinnati Children's Hospital
Discussion

Case Presentations
Moderator: Madhura Phadke MD | Pittsburgh
Jay Freeman MD | Nationwide Hospital
Discussion
Megha S. Mehta MD | UT Southwestern
Discussion

8:05am – 10:05am
Parallel Session – Pancreatic Cancer
Breakout

Advances in Basic Science in Pancreatic Cancer
Moderator: Leopoldo Fernandez MD | Massey Cancer Center
Jami Salomon PhD | University of Pittsburgh Medical Center
Targeting KRAS for Pancreatic Cancer Treatment
Channing Der PhD | Lineberger Comprehensive Cancer Center
Anti-Tumorigenic Signaling
Kathleen DelGiorno PhD | Vanderbilt University Medical Center
Panel Discussion

Advances in Precision Approaches for Pancreatic Cancer: Are They Ready?
Moderators:
Jorge Machicado MD | University of Michigan
Daniel Delitto MD | Stanford University School of Medicine

Status for Defining at Risk Groups for PDAC- Beyond Hereditary Etiologies
Aimee Lucas MD, MS | Icahn School of Medicine at Mount Sinai
Predicting Response to Neoadjuvant Therapy
Alessandro Paniccia MD | University of Pittsburgh Medical Center
Pancreas Club Best of the Best | Uncinate Duct Dilatation Predicts Additional Risk for High-Grade Dysplasia or Invasive Carcinoma Among Fukuoka-Positive Intraductal Papillary Mucinous Neoplasms
Zhi Ven Fong MD | Massachusetts General Hospital
Panel Discussion

10:05am – 10:20am
Break & Exhibits
Ballroom

10:20am – 11:15am
The Ruth C. Brufsky Award for Excellence in Pancreatic Cancer
Assembly Room
Moderator: Randall E. Brand MD | University of Pittsburgh Medical Center

My Personal Reflections on Pancreatic Cancer Early Detection: Past, Present and Future
Sudhir Srivastava PhD, MPH | National Cancer Institute

11:15am – 12:15pm
Advancing the Engagement of Underserved Populations in Pancreatic Diseases
Assembly Room
Moderators:
Jose Trevino MD | VCU Health
Anwaar Saeed MD | University of Pittsburgh Medical Center

Improving Recruitment of Minority Populations in Pancreatitis Studies
Cemal Yazici MD | University of Illinois at Chicago
Improving Recruitment of Minority Populations in Pancreatic Cancer Trials
Fiyinfolu Balogun, MD, PhD | Memorial Sloan Kettering Cancer Center
Prevention of Pancreatic Diseases: NPF's Black/African American Initiative
Darwin Conwell MD, MSc | University of Kentucky
Panel Discussion

12:15pm – 1:30pm
Lunch & Exhibits
Kurtzman Room

1:30 pm – 3:30pm
Interventions for Pancreatitis: New Approaches, Knowledge Gaps and Research Opportunities
Assembly Room
Moderators: Dana Andersen MD | NIDDK / NIH & Jane Holt | The National Pancreas Foundation

Recap: 2023 NIDDK Pancreas Workshop Interventions for Pancreatitis: New Approaches, Knowledge Gaps and Research Opportunities
Steven J. Hughes MD | University of Florida
Outcomes of Interest for Clinical Trials
Dhiraj Yadav MD, MPH | University of Pittsburgh Medical Center
Lessons Learned From a Clinical Trial Design
Srikanth Iyer PhD | University of Alabama at Birmingham
Mission:Cure Initiative for PROs
Lola Rahib PhD | Mission: Cure
FDA Perspective in Survey Development
Chris St. Clair MD | FDA
Panel Discussion
Panelist | Erica Lyons MD | FDA

3:30pm
Adjournment

Poster Listing

1. NOVEL OPTOGENETIC MOUSE MODEL OF PANCREATITIS-RELATED PAIN
Olivia L. Babyok BS | University of Pittsburgh

2. A HISTORY OF ANY SURGERY IS A RISK FACTOR FOR CONSTANT ABDOMINAL PAIN IN PATIENTS WITH CHRONIC PANCREATITIS
Zachary Kassir MD | Johns Hopkins University School of Medicine Department of Gastroenterology

4. LONGITUDINAL CHANGES IN BODY COMPOSITION AND METABOLIC BIOMARKERS PRIOR TO DIAGNOSIS OF PANCREATIC DUCTAL ADENOCARCINOMA
Alexander Weston PhD | Mayo Clinic

5. REAL-TIME PROSPECTIVE DAXX/ATRX TESTING OF 186 CONSECUTIVE SURGICALLY RESECTED PANCREATIC NEUROENDOCRINE TUMORS REVEALS LOSS OF EXPRESSION CORRELATES WITH DISTANT METASTASIS
Phoenix Bell MD, MSc | UPMC

6. THE DETECTION OF ALT USING A NOVEL CISH ASSAY IS A POOR PROGNOSTIC BIOMARKER FOR PATIENTS WITH PANCREATIC NEUROENDOCRINE TUMORS
Simmi Patel MD | University of Pittsburgh Medical Center

7. DILATED MAIN PANCREATIC DUCT WITHOUT A VISIBLE PANCREATIC MASS: LONG-TERM FOLLOWUP AND PREDICTORS OF FUTURE PANCREATIC DUCTAL ADENOCARCINOMA
Arjun Chatterjee MD | Cleveland Clinic Foundation

8. ISOLATED INTRAMURAL GASTRIC METASTASIS OF PANCREATIC DUCTAL ADENOCARCINOMA (PDAC) DETECTED ON SURVEILLANCE ESOPHAGOGASTRODUODENOSCOPY (EGD) BEFORE ENDOSCOPIC ULTRASOUND (EUS) GUIDED BIOPSY
Arjun Chatterjee MD | Cleveland Clinic Foundation

9. SYNCHRONOUS PANCREATIC MASSES: A CASE SERIES
Arjun Chatterjee MD | Cleveland Clinic Foundation

11. A MODEL TO PREDICT SUBJECT RETENTION IN ACUTE PANCREATITIS STUDIES
Kathleen Tong | The Ohio State University Wexner Medical Center

12. INCIDENCE OF DIABETES FOLLOWING ACUTE PANCREATITIS: A MULTICENTER PROSPECTIVE COHORT STUDY
Joseph Bejjani MD | The Ohio State University Wexner Medical Center

13. THE INTERPLAY OF GENETIC AND ENVIRONMENTAL RISK FACTORS BUT NOT TRIGLYCERIDE LEVELS MEDIATE HYPERTRIGLYCERIDEMIC ACUTE PANCREATITIS: A CASE SERIES
Lara Cheesman | Johns Hopkins University

14. THE ASSOCIATION OF FATTY LIVER DISEASE AND UNDERLYING INFLAMMATION WITH ACUTE PANCREATITIS RISK
Sneh Patel | University of Illinois at Chicago

15. HYPERTRIGLYCERIDEMIA-INDUCED PANCREATITIS: A SINGLE CENTER EXPERIENCE
Monisha Shah MD | University of Texas Health Science Center at Houston

16. RACIAL DIFFERENCES IN POST ERCP PANCREATITIS
Vikash Kumar MD | Piedmont Athens Regional Medical Center

17. DISCORDANCE IN RADIOLOGY READINGS AMONG PATIENTS PRESENTING WITH SUSPECTED ACUTE PANCREATITIS
Anmol Singh MBBS | Johns Hopkins University

18. ALCOHOL INTAKE FREQUENCY REDUCES THE RISK OF GALLSTONES STRATIFIED FOR SEX, BMI AND POLYGENIC RISK SCORE: IMPLICATIONS FOR ACUTE PANCREATITIS
Phil Greer MS | Ariel Precision Medicine

19. SURVEY OF CLINICAL AND SUBCLINICAL EVEN RATES IN RAP
Phil Greer MS | Ariel Precision Medicine

20. SUB-NORMAL PANCREAS VOLUMES IN CHILDREN AND YOUNG ADULTS FOLLOWING ACUTE AND ACUTE RECURRENT PANCREATITIS
Andrew Trout MD | Cincinnati Children's Hospital Medical Center

21. DELETION OF NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN DECREASES PANCREATITIS SEVERITY AND BROWN ADIPOSE TISSUE WEIGHT IN A MURINE MODEL OF ACUTE PANCREATITIS
Zachary Hurst BS | The Ohio State University

22. NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN AND ITS HYDROPHOBIC LIGANDS ARE ALTERED IN THE BLOOD OF CHRONIC PANCREATITIS.
Kristyn Gumpper-Fedus PhD | The Ohio State University Wexner Medical Center

23. 10-YEAR OUTCOMES OF SMOKING CESSATION ATTEMPTS IN REDUCING LOW BONE DENSITY DISEASE IN CHRONIC PANCREATITIS
Arjun Chatterjee MD | Cleveland Clinic Foundation

24. ß-ADRENERGIC SWEAT TEST IN ACUTE RECURRENT AND CHRONIC PANCREATITIS WITH CFTR GENE MUTATIONS
Vybhav Gummadi MD | The Hospital for Sick Children

25. GERMLINE MULTIGENE PANEL TESTING IN ACUTE AND CHRONIC PANCREATITIS
Mitchell Ramsey MD | The Ohio State University

26. PANCREATIC STONE SIZE AND STONE DENSITY PREDICT THE NUMBER OF ESWL SESSIONS NEEDED FOR SUCCESSFUL FRAGMENTATION
Sneh Sonaiya MBBS | Johns Hopkins Medicine

27. CHARACTERIZING SYMPTOM BURDEN AND IMPACT OF CHRONIC PANCREATITIS IN AN ONLINE PATIENT COMMUNITY
Yasmin Hernandez- Barco MD | Massachusetts General Hospital

29. TRANSFER DELAY, LARGER NECROTIC COLLECTION AND NEED FOR ENTERAL NUTRITION ASSOCIATED WITH INCREASED UNPLANNED 30 DAY READMISSION IN NECROTIZING PANCREATITIS.
Gaurav Suryawanshi MD | University of Minnesota

30. NECROTIZING PANCREATITIS AMONG CHILDREN WITH OBESITY AND PANCREATIC STEATOSIS: A ONE YEAR REVIEW AT A HIGH-VOLUME CENTER
Muhammad Khan MD, MPH, FASGE, NASGPHAN-F | Nationwide Children's Hospital, The Ohio State University College of Medicine

31. INTRAOPERATIVE PYLORIC BOTULINUM TOXIN INJECTION FOR POST TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION GASTROPARESIS IMPROVEMENT IN CHILDREN
Juan Gurria MD MSc | Cincinnati Children's Hospital Medical Center

32. BRINGING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION TO A TERTIARY CHILDREN'S HOSPITAL: ASSIMILATION THROUGH SIMULATION
Christie Heinzman DNP, APRN | Nationwide Children's Hospital

33. A NOVEL TEAM MODEL FOR DIABETES CARE IN PEDIATRIC PATIENTS UNDERGOING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION
Jennifer Ladd MD, MSc | Nationwide Children's Hospital

34. ISLET CELL AUTOANTIBODY FORMATION AND ITS ASSOCIATION WITH BETA CELL FUNCTION FOLLOWING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION (TPIAT)
Colleen Lowe MSN, APRN-CNP, FNP-BC | Cincinnati Children's Hospital Medical Center

35. RATIO AND DOSE OF RC1: RC2 COLLAGENASES BASED ON PERCENT PARENCHYMAL MASS IMPROVES ISLET YIELD INTENDED FOR PEDIATRIC ISLET AUTO-TRANSPLANTATION
Chandrashekar B. Revanna PhD | Department of Pediatrics and Surgery, Center for Clinical and Translational Research, Nationwide Children's Hospital / Ohio State University

36. CHANGES IN PORTAL PRESSURE, BLOOD PRESSURE AND GLUCOSE LEVELS DURING ISLET INFUSION: CLOSE MONITORING FOR EVERY FIVE MINUTES DURING AUTO-TRANSPLANTATION
Krishna K. Samaga PhD | Department of Pediatrics and Surgery, Center for Clinical and Translational Research, Nationwide Children's Hospital / Ohio State University

37. ADHERENCE TO VACCINE RECOMMENDATIONS FOR PATIENTS POST SPLENECTOMY AFTER TOTAL PANCREATECTOMY ISLET AUTOTRANSPLANTATION
Angela Turner MSN, RN, CPN | Cincinnati Children's Hospital Medical Center

38. DUODENAL DUPLICATION CYST AS A RARE CAUSE OF PANCREATITIS IN A CHILD
Muhammed Colak MD | Nationwide Children's Hospital

39. IS NON-ETHANOL BASED ABLATION THERAPIES BETTER THAN ETHANOL ABLATION FOR PANCREATIC CYSTS?: A SYSTEMATIC REVIEW AND META ANALYSIS.
Yeshaswini Reddy MD | University of Illinois College of Medicine

40. CAN EUS-ABLATION REVOLUTIONIZE THE MANAGEMENT OF PANCREATIC CYSTS? A META-ANALYSIS OF SHORT-TERM OUTCOMES
Ahmed Salih MD | Indiana University School of Medicine

41. A DNA/RNA-BASED NGS PLATFORM TO IMPROVE THE CLASSIFICATION OF PANCREATIC CYSTS AND DETECTION OF PANCREATIC CANCER ARISING FROM PANCREATIC CYSTS
Aatur Singhi MD PhD | UPMC

42. TRENDS AND IMPACT OF EUS UTILIZATION FOR PANCREATIC CYSTS OVER TIME
Lillian Wang MD | Mayo Clinic Rochester

43. A NEW APPROACH TO EVALUATING THE MICROBIOME OF THE PANCREAS
Megan B. Ghai, MD, MPH, MA | University of Arizona College of Medicine Phoenix

44. CDX-7108: AN ENGINEERED GASTROINTESTINAL-STABLE LIPASE FOR THE ORAL TREATMENT OF EXOCRINE PANCREATIC INSUFFICIENCY
Kristen Skvorak PhD | Codexis

45. PANCREATIC TUBERCULOSIS IN A NEWLY DIAGNOSED HIV PATIENT: A RARE CASE REPORT
Mohammed Rifat Shaik MBBS | University of Maryland Medical Center Midtown Campus

46. EXOCRINE PANCREATIC INSUFFICIENCY TREATMENT IN PATIENTS WITH CHRONIC/RECURRENT ACUTE PANCREATITIS IMPROVES TREATMENT SATISFACTION AND DECREASES HEALTHCARE UTILIZATION: PACT-CP INTERIM FINDINGS
Jodie Barkin MD | University of Miami Miller School of Medicine

POSTERS

1. NOVEL OPTOGENETIC MOUSE MODEL OF PANCREATITIS-RELATED PAIN

OL Babyok, SA Fulton, JL Saloman
University of Pittsburgh

Background: Evaluation and management of chronic abdominal pain is upwards of a $30 billion burden annually in the United States alone. A significant portion of that cost is comprised of patients with suspected or diagnosed pancreatitis, an irreversible and progressive disorder that is characterized by inflammation and fibrosis of the pancreas as well as loss of exocrine and/or endocrine function. Patients report signs of central hyperexcitability such as an increase in pain to the abdomen as well as generalized hyperalgesia and allodynia. One of the main gaps in our knowledge is what factors are directly related to intrapancreatic pain sensation. It has been suggested that when acinar cells are damaged, they release neurotransmitters that may activate nociceptive fibers via their receptors. Available animal models such as cerulein, a cholecystokinin analog, were designed to elucidate the pathophysiology of pancreatitis development. While pain like behaviors are well documented in these models, it is near-impossible to tease out the underlying mechanisms. Due to the prevalence of hyperexcitability-related symptoms in patients and the gap of knowledge in the study of nociceptive-neuronal activation, we sought to further the development of a pancreatic pain model in which the nervous system could more precisely be manipulated to better understand neural regulation of the pancreas so that more targeted therapies can be developed in the future. This was attempted using the powerful technique of optogenetics coupled with transgenic mouse lines to create a mouse model of pancreatic inflammation that closely reflects patient symptoms. A pancreatitis-like state was assessed using a variety of behavioral, biochemical, and physiological measures both stand-alone and in comparison, to other animal models of pancreatitis. Using TRPV1-cre/ChR2 mice and the Neurolux μLED system, we found that ChR2 activation in TRPV1-cre neurons in the pancreas is sufficient to drive an increase in abdominal pain, edema of the pancreas, increase in pancreas-related enzymatic activity, and an upregulation of ATF3 in pancreas-innervating dorsal root ganglia. This data suggests our model is a viable model of pancreatitis that may allow for easier manipulation of the nervous system in order to study various components of pancreatic pain mechanisms.

2. A HISTORY OF ANY SURGERY IS A RISK FACTOR FOR CONSTANT ABDOMINAL PAIN IN PATIENTS WITH CHRONIC PANCREATITIS

Z Kassir, M Faghih, Z Yousefli, V Singh
Johns Hopkins University School of Medicine Department of Gastroenterology

Background: Chronic pancreatitis (CP) is a fibro-inflammatory disease that commonly manifests with abdominal pain. CP pain can be subdivided into two patterns: constant and intermittent. Constant pain has been shown to be associated with lower quality of life. We aimed to identify patient characteristics associated with constant pain in chronic pancreatitis.

Methods: All adult patients with CP referred to a multidisciplinary pancreatitis clinic between 2010-2022 were evaluated. CP was defined as the presence of calcification(s) on CT imaging and/or moderate to marked ductal changes on MRCP/ERCP as per M-ANNHEIM criteria. Exocrine insufficiency (FE-1 < 200 mcg/g) and/or typical histology were only found in patients with either of the aforementioned criteria. AP was defined as upper abdominal pain, serum amylase and/or lipase ≥ 3X upper limit normal and abdominal imaging showing changes and/or sequelae of AP (e.g. pseudocyst) if imaging was obtained after hospital discharge. We incorporated the presence, severity, and temporality of pain to classify patients into the following 3 categories: 1) No pain; 2) Intermittent mild to severe episodes of pain; 3) Constant mild to severe pain.

Results: 396 patients were included in the analysis. Of these, 75 (19%) had no pain, 155 (39%) had intermittent pain, and 166 (42%) had constant pain. The mean age was 50.1 (+/- 14.6); 45.7% female; 41.4% had alcohol etiology and 51.5% had idiopathic/genetic etiology. Female gender (OR=2.46 [1.26-4.82], p=0.008), younger patients (OR=0.95 [0.93-0.98], p< .001), depression (OR=2.9[1.17-7.1], p=0.022), and history of any surgery (defined as any abdominal, thoracic, pelvic, intracranial, musculoskeletal, or oropharyngeal procedure involving incision and tissue damage) (OR=2.02 [1.04-3.90], p=0.037) were associated with constant pain in the adjusted analysis. Patients with intermittent pain were more likely to have depression (OR=0.041[1.04-6.4], p=0.041), alcohol etiology (OR=3.73[1.2-11.94], p=0.027), and history of acute pancreatitis (OR=2.7[1.02-7.34], p=0.046). Both groups were less likely to have an idiopathic or genetic etiology (OR=0.22 [0.007-0.70), p=0.008). There were no significant associations between pain and BMI, smoking status, diabetes, and exocrine insufficiency.

Conclusion: Female gender, younger patients , higher rates of depression, a history of any surgery, and non-idiopathic/genetic etiology were significantly associated with constant pain in patients with CP.

Table 1 - Multinomial Regression Analysis; Factors Associated with Intermittent or Constant Pain (versus no pain).

Variable		Intermittent Pain AOR [95%CI]	P-value	Constant Pain AOR [95%CI]	P-value
Age		0.96[0.94-0.99]	<0.001*	0.95[0.93-0.98]	<0.001*
Gender	Male	Ref			
	Female	1.4[0.73-2.78]	0.30	2.46[1.26-4.82]	0.008*
BMI		0.97 [0.92-1.02]	0.22	0.98[0.92-1.03]	0.32
Insulin-dependent DM	Yes	0.97 [0.47-2.04]	0.94	1.3[0.63-2.70]	0.46
	No				
Depression	Yes	2.6 [1.04-6.4]	0.041*	2.9[1.17-7.1]	0.022*
	No				
Anxiety	Yes	1.7[0.73-3.9]	0.22	2.21[0.96-5.10]	0.63
	No				
Smoking status	Never				
	Active smoker	0.88[0.40-2.00]	0.77	1.56[0.68-3.55]	0.29
	Former smoker	1.19[0.53-2.7]	0.68	1.22[0.53-2.84]	0.64
Etiology					
Alcohol	Yes	3.73[1.2-11.94]	0.027*	2.21[0.69-7.13]	0.181
	No				
Idiopathic/Genetic	Yes	0.22[0.07-0.70]	0.008*	0.24[0.08-0.75]	0.014*
	No				
HX of Acute Pancreatitis	Yes	2.7[1.02-7.34]	0.046*	1.5[0.58-3.8]	0.41
	No				
EPI (FE<200 and/or Steatorrhea)	Yes	0.63[0.33-1.20]	0.16	0.56 [0.29-1.07]	0.08
	No				
Hx of Surgery	Yes	1.5[0.80-2.8]	0.21	2.02[1.04-3.90]	0.037*
	No				
Hx of other painful Conditions	Yes	1.4[0.56-3.7]	0.50	1.84[0.76-4.5]	0.18
	No				

4. LONGITUDINAL CHANGES IN BODY COMPOSITION AND METABOLIC BIOMARKERS PRIOR TO DIAGNOSIS OF PANCREATIC DUCTAL ADENOCARCINOMA

AD Weston, DCF Klatte, M Engels, A Ouni, A Bali, Y Ma, H Sledge, JE van Hooft, ME van Leerdam, B Ji, R Carter, C Bolan, MB Wallace, Y Bi
Mayo Clinic

Background: Shifts in body composition such as unexplained fat loss, muscle wasting, and changes in metabolic biomarkers such as increased blood glucose may be potential biomarkers for early-detection of pancreatic ductal adenocarcinoma (PDAC). However, shifts in body composition may be too subtle to detect from weight loss alone. We report longitudinal changes in body composition measured from abdominal CT as well as metabolic biomarkers preceding a diagnosis of PDAC.

Methods: We identified all patients (≥18 years) diagnosed with PDAC between 2000 and 2021. We collected data on demographics, comorbidities, and PDAC characteristics. We identified individuals with at least two prior abdominal CT exams as well as blood-based biomarkers within 3 years prior to date of diagnosis. From these CT exams, we applied a previously validated deep-learning algorithm to quantify visceral and adipose fat area, skeletal muscle area and density, and vertebral bone area and density from abdominal CT exams (Weston et al. Radiology 2019). Trends of body composition and metabolic biomarkers prior to a diagnosis of PDAC were estimated by linear mixed models.

Results: We identified 5,282 newly-diagnosed PDAC patients who had prior laboratory testing for metabolic biomarkers. Additionally, we identified 1,825 PDAC patients who had received abdominal CT imaging, of whom 581 (32%) had at least 2 pre-diagnostic CT exams eligible for analysis. The mean age at diagnosis was 68.4 (SD 11.3) years, 43.6% were female and the mean BMI was 27.0 (SD 5.5). The distribution of PDAC stage was as follows: 12.5% stage I, 32.6% stage II, 19.7% stage III, and 35.2% stage IV. Prior to a PDAC diagnosis, we observed significant decreases in subcutaneous and visceral fat volume, significant decrease in bone volume and near-significant decrease in muscle volume (P=0.067). We also observed changes in metabolic biomarkers including increased high-density lipoprotein, decreased low-density lipoprotein, increased HbA1c, and increased fasting blood glucose (Table 1). Concurrently, patients developed significant increases in white blood cells, hemoglobin, and albumin prior to a clinical PDAC diagnosis.

Conclusion: Significant alterations in several body composition and metabolic markers occurred in the years prior to development of PDAC.

Table 1. Trends of body composition and metabolic biomarkers within 3 years prior to a diagnosis of PDAC, estimated by linear mixed models. Area measurements were normalized by dividing by patient height, squared.

Body composition metrics	**N**	***β* (95%CI)**	***P***
Skeletal muscle area, indexed (cm^2 / m^2)	581	-0.24 (-0.50, 0.02)	0.067
Skeletal muscle density (HU)	581	-0.17 (-0.42, 0.07)	0.163
Visceral fat area, indexed (cm^2 / m^2)	581	-1.88 (-2.37, -1.40)	<.001
Subcutaneous fat area, indexed (cm^2 / m^2)	581	-2.59 (-3.18, -2.01)	<.001
Vertebral bone area, indexed (cm^2 / m^2)	581	-0.13 (-0.24, -0.02)	0.018
Vertebral bone density (HU)	581	2.21 (1.04, 3.38)	<0.001
Laboratory markers			
Fasting blood glucose (mg/dl)	991	4.84 (4.14, 5.55)	<0.001
HbA1c (%)	669	0.11 (0.08, 0.13)	<0.001
High-density lipoprotein (mg/dl)	859	0.87 (0.42, 1.33)	<0.001
Low-density lipoprotein (mg/dl)	762	-2.58 (-3.22, -1.94)	<0.001
Total cholesterol (mg/dl)	862	-2.56 (-3.13, -1.98)	<0.001
Triglycerides (mg/dl)	895	-1.37 (-2.39, -0.36)	0.008
White blood cells ($x10^9$/l)	3,594	0.33 (0.28, 0.38)	<0.001
Hemoglobin (g/dl)	3,605	-0.27 (-0.28, -0.26)	<0.001
Albumin (g/dl)	1,986	-0.09 (-0.09, -0.08)	<0.001

β=regression coefficient; CI=confidence interval. *β* values with 95% CIs are interpreted as the difference in means per 6 months closer to date of diagnosis.

5. REAL-TIME PROSPECTIVE DAXX/ATRX TESTING OF 186 CONSECUTIVE SURGICALLY RESECTED PANCREATIC NEUROENDOCRINE TUMORS REVEALS LOSS OF EXPRESSION CORRELATES WITH DISTANT METASTASIS

P Bell, K Smith, A Paniccia, K Lee, J Pingpank, M Hogg, H Zeh, M Nikiforov, A Singhi, A Zureikat
UPMC

Background: Pancreatic neuroendocrine tumors (PanNETs) are a heterogeneous group of neoplasms with increasing incidence and unpredictable behavior. Whole-exome sequencing studies of metastatic PanNETs found recurrent genomic alterations in DAXX and ATRX, which correlate with loss of protein expression. DAXX/ATRX loss in PanNETs is associated with adverse clinicopathologic features and poor disease-free survival (DFS); however, prior studies are retrospective. This study aim was to prospectively evaluate DAXX/ATRX status among surgically resected PanNETs and assess its prognostic significance.

Methods: Over a 5-year period, 186 consecutive patients underwent surgical management for a primary PanNET. This patient cohort included 175 non-functional PanNETs, 10 insulinomas, and 1 somatostatinoma. Additionally, 7 patients had multiple endocrine neoplasia type 1 and 1 patient had von Hippel Lindau syndrome. The status of DAXX/ATRX was immunohistochemically evaluated for each surgically resected PanNET. The results of DAXX/ATRX staining were correlated with clinicopathologic features and patient follow-up that ranged from 5 months to 5 years (median, 2.25 years).

Results: Loss of DAXX, ATRX, both proteins, or either protein within the prospective cohort was identified in 26 (14%), 21 (11%), 1 (1%), and 46 (25%) cases. DAXX/ATRX loss was associated with tumor size >2.0 cm (94% vs 56%, p< 0.001), higher WHO grade (G2/G3 vs G1, 41% vs 14%, p< 0.001), perineural invasion (72% vs 29%, p< 0.001), lymphovascular invasion (87% vs 42%, p< 0.001), synchronous distant metastasis (26% vs 9%, p=0.004), and metachronous distant metastases (35% vs 6%, p< 0.001). No statistical significance was observed between DAXX/ATRX status and functional status or predisposing syndrome. Among patients without synchronous distant metastases (n=162, 87%), the 3-year DFS for patients with DAXX/ATRX-negative PanNETs was 65% versus 95% for DAXX/ATRX-positive PanNETs. By multivariate analysis to include WHO grade, T-stage, and N-stage, loss of DAXX/ATRX was a negative, independent prognostic factor for DFS (p=0.023).

Conclusion: Consistent with retrospective studies, the detection of DAXX/ATRX loss in surgically resected PanNETs was associated with adverse clinicopathologic features and increased risk of developing distant metastatic disease. While long-term follow-up is being accrued for this cohort, the assessment of DAXX/ATRX should be considered in the prognostic evaluation of surgically resected PanNETs.

6. THE DETECTION OF ALT USING A NOVEL CISH ASSAY IS A POOR PROGNOSTIC BIOMARKER FOR PATIENTS WITH PANCREATIC NEUROENDOCRINE TUMORS

S Patel, K Smith, P Bell, CM Heaphy, AD Singhi
University of Pittsburgh Medical Center

Background: Alternative lengthening of telomeres (ALT) is a telomerase-independent telomere maintenance mechanism and defined by large ultra-bright, intranuclear foci of telomeric DNA using fluorescence in situ hybridization (FISH). The presence of ALT is an important prognostic biomarker for pancreatic neuroendocrine tumors (PanNETs) and correlates with early relapse-free survival (RFS). However, the routine adoption of ALT assessment within pathology laboratories has been limited due to the lack of widespread availability and technical challenges associated with telomere-specific FISH. To address these issues, chromogenic in situ hybridization (CISH) for telomeric DNA was developed and validated to assess for ALT in a large cohort of PanNETs.

Methods: A chromogenic probe consisting of repetitive telomeric sequences (CCCTAA) was designed for an automated stainer and optimized using 60 leiomyosarcomas with known ALT FISH status. Similar to telomere-specific FISH, ALT by CISH was defined by large chromogenic signals within the neoplastic nuclei as compared to the nuclei within the surrounding non-neoplastic stroma. Upon optimization, 360 sporadic, primary PanNET surgical specimens were evaluated by telomere-specific CISH, and correlated with patient age, gender, tumor size, WHO grade, lymphovascular and perineural invasion, T- and N-stage, status of ATRX/DAXX and ALT by FISH, synchronous and metachronous distant metastases, and RFS.

Results: The presence of ALT by CISH was identified in 112 (31%) PanNETs and had 100% correlation with ALT status by FISH. ALT CISH correlated with large mean tumor size, increased WHO grade, advanced T- and N-stage, loss of ATRX/DAXX protein expression, and the presence of both synchronous and metachronous distant metastases ($p < 0.01$). Among 271 patients without synchronous metastasis, the 5-year RFS rate for ALT CISH-positive patients was 39% as compared to 94% for ALT CISH-negative patients ($p < 0.01$). By multivariate analysis, ALT CISH was a negative prognostic factor for RFS, and independent of tumor size, WHO grade, lymphovascular and perineural invasion, N-stage ($p < 0.01$).

Conclusion: Using an automated stainer, a telomere-specific CISH assay has been successfully developed and can accurately detect ALT in surgically resected PanNETs. Further, ALT by telomere specific CISH was associated with multiple adverse clinicopathologic features and was a negative, independent prognostic factor for RFS among patients with PanNETs.

7. DILATED MAIN PANCREATIC DUCT WITHOUT A VISIBLE PANCREATIC MASS: LONG-TERM FOLLOWUP AND PREDICTORS OF FUTURE PANCREATIC DUCTAL ADENOCARCINOMA

A Chatterjee, V Lakhmani, HA Khasawneh, M Horibe, J De La Fuente, WR Bamlet, EE Carlson, AL Oberg, AM Delgado, KA Doering, N Takahashi, AH Goenka, GM Petersen, S Majumder
Cleveland Clinic Foundation

Background: Pancreatic ductal adenocarcinoma (PDAC) is a lethal disease frequently diagnosed at an advanced stage. Pancreatic imaging abnormalities that predate diagnosis of PDAC may serve as an early detection tool. Dilation of the main pancreatic duct (D-MPD) has been shown to occur up to a year before PDAC diagnosis.1 D-MPD in the absence of a visible pancreatic mass raises concern for occult neoplasm, the natural history of D-MPD remains poorly understood.

Methods: We identified adults (≥18-years-old) with D-MPD (MPD >3mm) on either abdomen/pelvis CT/MRI scans between 1/1/2012 and 12/31/2017. Patients with prior PDAC, pancreatic surgery, definite pancreas mass or other visible cause of D-MPD were excluded after expert radiologist review. Pertinent clinical, demographic, and imaging data were summarized. A stratified univariate Cox proportional hazards model was utilized to evaluate risk factors for PDAC development.

Results: 2307 patients (baseline CT:1615/ MRI:692) met our study criteria, 63.7% were female, median age was 71 (IQR:59.5-80.1), and median follow-up was 1466 days (IQR:1426-1509). The 1-year and 3-year event rates for PDAC were 2.70 and 2.99 per 100-patient years respectively. Out of sixty-three (2.7%) patients who developed PDAC within 3-years of the baseline scan, the majority (58/63) were within 1-year. Factors associated with a significantly increased 3-year risk of PDAC included male gender, BMI>25, diabetes mellitus, non-O blood type, elevated serum CA 19-9, focal narrowing of MPD, equivocal pancreatic mass, and suboptimal baseline scan quality.

Conclusion: D-MPD may predate the diagnosis PDAC and raises concern for neoplasm. However, only a small subset of D-MPD patients without an overt pancreatic mass developed PDAC on follow-up. The risk is highest in the first year with rare events after one year of follow-up. Risk factors identified in this study may enrich assessment and guide surveillance of patients with D-MPD.

Table 1. Univariate Stratified Survival Analysis assessing the risk of developing PDAC within 3 years from baseline CT/MRI in 2307 patients with D-MPD

Characteristics		Reference	Hazard Ratio	95% HR CI		p-value
Age (years)	≥50	< 50	7.11	0.98	51.19	0.0515
Body Mass Index (kg/m2)	**≥25**	**< 25**	**1.97**	**1.17**	**3.32**	**0.0105**
Maximum MPD diameter (mm)	**≥5-< 10**	**≥3-< 5**	**3.77**	**1.99**	**7.17**	**< .0001**
	≥10	**≥3-< 5**	**20.08**	**8.46**	**47.69**	**< .0001**
Serum CA19-9	**≥35**	**< 35**	**23.75**	**3.14**	**179.87**	**< .0001**
Gender	**Male**	**Female**	**1.94**	**1.18**	**3.18**	**0.0088**
ABO Blood type	**Type Non- O**	**Type O**	**2.24**	**1.16**	**4.31**	**0.0161**
Alcohol	Ever Use	Never Use	1.03	0.61	1.73	0.9137
Aspirin	Ever Use	Never Use	0.94	0.53	1.64	0.8177
Diabetes mellitus	**Yes**	**No**	**2.59**	**1.55**	**4.34**	**0.0003**
Chronic Pancreatitis	Yes	No	0.38	0.09	1.58	0.1847
Acute Pancreatitis	Yes	No	1.13	0.55	2.26	0.7609
Family History of PDAC	Yes	No	1.95	0.93	4.1	0.0772
Pancreas Cyst on imaging	Yes	No	1.44	0.86	2.43	0.1688
Equivocal Mass on imaging	**Yes**	**No**	**12.52**	**7.25**	**21.64**	**< .0001**
Image Quality	**Suboptimal but diagnostic**	**Optimal**	**2.24**	**1.31**	**3.84**	**0.0033**
Focal narrowing of MPD	**Present**	**Absent**	**6.23**	**3.79**	**10.23**	**<0.001**

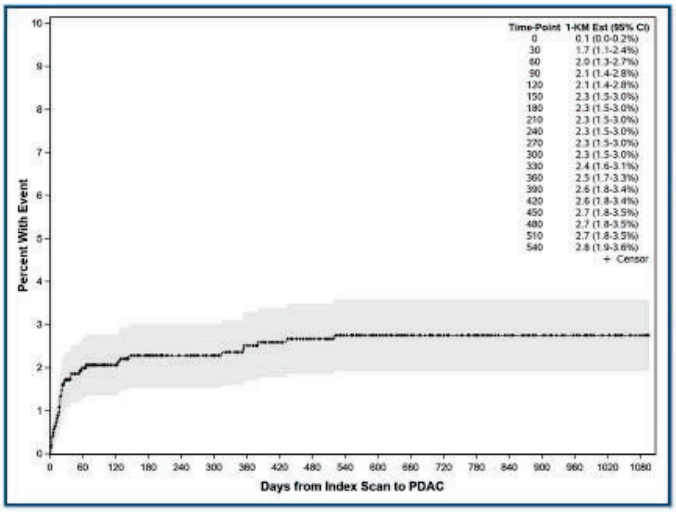


Figure 1: Kaplan-Meier curve estimate of PDAC event rates over the 3-year followup duration

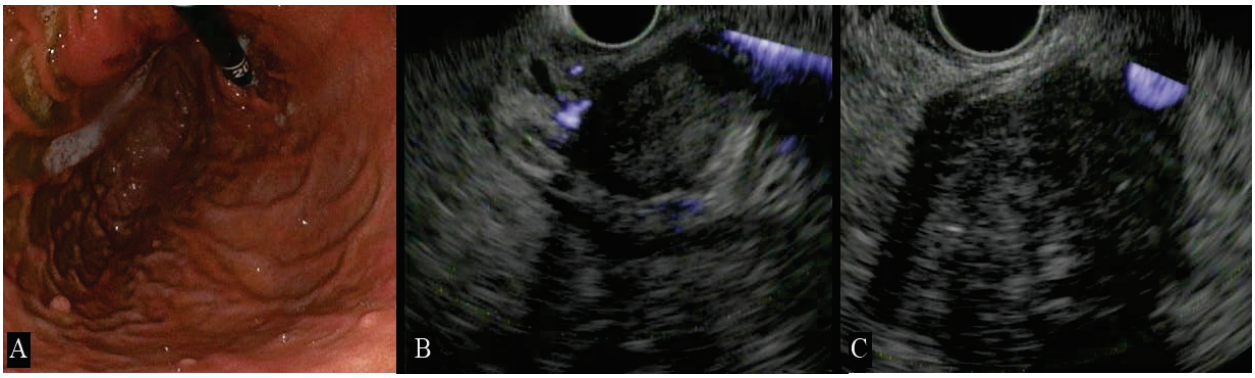
8. ISOLATED INTRAMURAL GASTRIC METASTASIS OF PANCREATIC DUCTAL ADENOCARCINOMA (PDAC) DETECTED ON SURVEILLANCE ESOPHAGOGASTRODUODENOSCOPY (EGD) BEFORE ENDOSCOPIC ULTRASOUND (EUS) GUIDED BIOPSY

A Chatterjee, A Ford, A Singh, P Chahal
Cleveland Clinic Foundation

Background: PDAC is an aggressive malignancy that requires prompt diagnosis and treatment to provide the patient with the best chance of long-term survival. Patients who have an imaging-confirmed solitary pancreatic mass generally undergo a EUS-guided fine needle biopsy (FNB) for histological confirmation of the diagnosis. We describe a rare case of solitary intramural gastric metastases discovered on surveillance EGD before EUS-guided biopsy.

Case: 63-year-old male with a past medical history of hypertension, chronic pancreatitis, and tobacco use presented with two months history of epigastric pain, and 5lbs weight loss. He denied jaundice, loss of appetite, pale-colored stools, or generalized itching. Initial CT-abdomen showed a 2.7cm x2.6cm mass in the pancreas body with associated proximal pancreatic duct dilatation and atrophy, suspicious of pancreatic neoplasm. Ca19.9 was 111, and CEA was 1.1. No evidence of metastasis was found on the CT chest/abdomen. EUS-guided FNB was scheduled. At the beginning of the procedure, a surveillance EGD showed a 2cm submucosal, noncircumferential mass in the gastric fundus (Fig1a). EUS was performed next, which confirmed a 2.1cm intramural mass in the gastric fundus originating from the muscularis propria (Fig1b), and a 3.9cm x 3.8cm mass in the pancreatic body with invasion into superior mesenteric vein, splenic vein, and splenoportal confluence with no noted arterial involvement (Fig1c). EUS-guided-FNB of both lesions was obtained using separate needles. Histopathology from both biopsies demonstrated invasive adenocarcinoma, histomorphologically similar and suggestive of a pancreatic primary. This changed the patient's staging from borderline resectable to metastatic, and he was referred for palliative chemotherapy.

Conclusion: Isolated gastric metastasis of PDAC is sporadic, and documented cases in the literature are from a prior diagnostic biopsy that created a seeding tract. Our case highlights two major points: 1) Though rare, isolated intramural gastric metastasis can occur and be missed on cross-sectional imaging. 2) Currently, no consensus or guideline mandates an EGD before EUS. This significant finding changed the staging, treatment, and prognosis and could have been missed with an oblique viewing EUS scope. At our center, we routinely perform EGD before EUS. Prospective studies are needed to confirm the utility of this practice.


A
B
C

9. SYNCHRONOUS PANCREATIC MASSES: A CASE SERIES

A Chatterjee, N Sharma, A Singh, M Franklin, R Garg, P Chahal
Cleveland Clinic Foundation

Background: Presence of synchronous multiple pancreatic masses is a rare finding, and any mass in the pancreas typically raises concern of undiagnosed pancreatic malignancy.

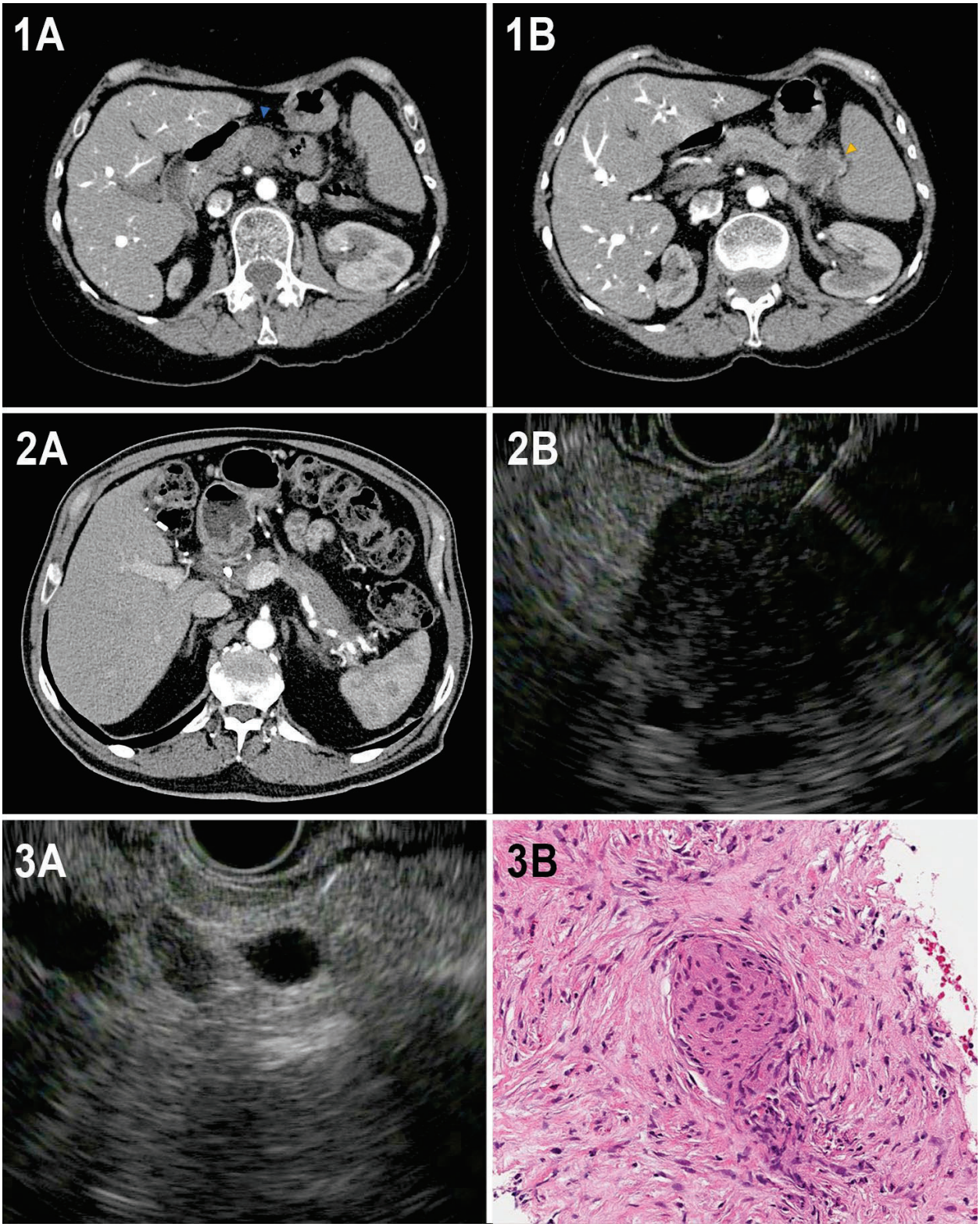
Aim: In this case series, patients presented with two or more synchronous solid masses as a result of pancreatic cancer (PC), autoimmune pancreatitis (AIP), and sarcoidosis.

Case One: 65-year-old female presented with abdominal pain and 20lbs unintentional weight loss over four months. CT scan revealed two suspicious solid masses in the body (Fig1a) and the tail of the pancreas (Fig1b). IgG4 level was normal, but CA19.9 was elevated at 75u/ mL. EUS with individual fine needle biopsies (FNB) of both masses confirmed infiltrative PC. Due to the significant cardiac history, the patient was deemed not a surgical candidate and was referred to oncology for chemoradiation/palliative therapy.

Case Two: 76-year-old male presented to the hospital with postprandial abdominal discomfort and unintentional weight loss. CT Abdomen demonstrated localized inflammation in the pancreatic tail.(Fig2a) EUS showed mass-like lesions in the pancreatic head and tail.(Fig2b) Immunohistochemistry was positive for IgG4-positive plasma cells. He was diagnosed with AIP and was started on steroids.

Case Three: 54-year-old male with complicated sarcoidosis (pulmonary/extrapulmonary involvement), presented with an abnormal PET scan showing focal increased uptake in the head/tail of the pancreas. His CT scan did not show any mass or duct dilation. EUS demonstrated ill-defined, infiltrative masses involving the pancreatic head and the tail.(Fig3a) FNB showed scattered non-necrotizing granulomas.(Fig3b) After excluding other causes of granulomatous diseases, he was diagnosed with pancreatic sarcoidosis.

Discussion: Only a few examples of synchronous pancreatic masses have been recorded in the medical literature. The clinical course for all patients differed greatly depending on the pathology. The plurality of solid masses and comparable imaging features of each with PC, the 4th highest cause of cancer-related deaths in the United States, is the highlight of this series. When encountering such individuals, a broad differential should be examined, as the clinical history of the illness varies. The whole pancreas should be investigated with multimodal imaging and EUS-guided acquisition histopathology to reach a precise diagnosis.


1A
1B
2A
2B
3A
3B

11. A MODEL TO PREDICT SUBJECT RETENTION IN ACUTE PANCREATITIS STUDIES

K Tong, PJ Lee, AE Phillips, M Nikahd, S Culp, P Paragomi, V Singh, E Afghani, I Lahooti, P Hart, S Han, L Lara, S Krishna, GI Papachristou
The Ohio State University Wexner Medical Center

Background: Recent studies of acute pancreatitis (AP) have focused on longitudinal outcomes with complete follow-up data being critical for their validity. Development of a model to predict long-term subject retention can guide resource assignment to maximize subject adherence.

Aim: To assess the drop-out rate of subjects enrolled in a prospective, longitudinal AP study and develop a model to predict subject retention.

Methods: This is a secondary analysis of the Post-AP Pancreatic Exocrine Insufficiency (PAPPEI) study, a study designed to assess the cumulative incidence of exocrine pancreatic insufficiency following AP. Eligible subjects were enrolled between 7/2017 and 9/2021. Follow-up questionnaires and blood/stool collection were completed at 3 and 12 months. A multivariable logistic regression model was built using forward stepwise AIC to predict the subject adherence at 3 months, defined as completion of questionnaires and stool sample submission. Candidate covariates considered for the model included baseline demographics, alcohol etiology, comorbidity burden, education, salary, lifestyle score, severity of AP, and submission of stool and blood samples at enrollment. Additionally, bivariate analysis for each candidate predictor vs. follow-up status at 3 months was performed using chi-square tests, Fisher's exact tests, and independent samples t-tests. Leave one out cross-validation (LOOCV) was used to assess internal validity.

Results: A total of 184 subjects completed the baseline questionnaires. Ninety of those completed the 3-month follow-up, representing a 3-month retention rate of 48%. High-school graduates or higher education degree, married status, older age, and submission of a baseline stool sample showed a significant association with adherence at 3 months. In the multivariable model, younger age and stool sample submission at baseline retained statistical significance. The final prediction model containing age, baseline stool sample, CCI, and salary produced the best predictive performance with an area under the curve (AUC) of 0.86 (sensitivity 0.85, specificity 0.72, accuracy 0.79). LOOCV results demonstrated comparable performance.

Conclusion: Only half of the enrolled patients completed the 3-month follow-up, highlighting the challenges with subject retention when conducting long-term observational AP studies. Herein, we propose a simple model that accurately predicts the odds of follow-up adherence and may guide resource assignment to maximize subject retention.

Table 1. Demographic and clinical characteristics by retention status* at 3-month follow-up (N=184)				
	3-Month Follow-up completed (n=90)	3-Month Follow-up lost (n=94)	Total (N=184)	p-value†
Age, mean (95% CI)	55.5 (52.6, 58.3)	47.2 (44.1, 50.4)	51.3 (49.1, 53.5)	**<0.001**
BMI, mean (95% CI)	31.8 (29.6, 33.9)	33.1 (30.8, 35.4)	32.4 (30.9, 34.0)	0.40
Male, N (%)	40 (44.4%)	49 (52.1%)	89 (48.4%)	0.30
Race, N (%)				
White	79 (87.8%)	74 (78.7%)	153 (83.2%)	0.28
Black	8 (8.9%)	14 (14.9%)	22 (12%)	
Other	3 (3.3%)	6 (6.4%)	9 (4.9%)	
Etiology, N (%)				
Alcoholic	9 (10.0%)	17 (18.1%)	26 (14.1%)	0.10
Non-Alcoholic	81 (90.0%)	75 (79.8%)	156 (84.8%)	
History of AP, N (%)	31 (34.4%)	41 (43.6%)	72 (39.1%)	0.20
Revised Atlanta Classification				
Mild	56 (62.2%)	60 (63.8%)	116 (63.0%)	0.63
Moderately severe	25 (27.8%)	20 (21.3%)	45 (24.5%)	
Severe	6 (6.7%)	10 (10.6%)	16 (8.7%)	
CCI, mean (95% CI)	0.9 (0.6, 1.2)	1.1 (0.8, 1.4)	1.0 (0.8, 1.2)	0.47
Healthy Lifestyle Score‡, mean(95% CI)	3.7 (3.5, 4.0)	3.8 (3.5, 4.0)	3.7 (3.6, 3.9)	0.90
Education, N (%)				
< High school graduate	1 (1.1%)	8 (8.5%)	9 (4.9%)	**0.001**
High school graduate/GED	16 (17.8%)	33 (35.1%)	49 (26.6%)	
Post-high school training	70 (77.8%)	52 (55.3%)	122 (66.3%)	
Marital Status, N (%)				
Widowed/divorced/ separated/single	31 (34.4%)	48 (51.1%)	79 (42.9%)	**0.0033**
Married	58 (64.4%)	43 (45.7%)	101 (54.9%)	
Subject annual income, N (%)				
<$50,000	27 (30.0%)	47 (50.0%)	74 (40.2%)	0.051
$50,000-$100,000	26 (28.9%)	20 (21.3%)	46 (25.0%)	
>$100,000	18 (20.0%)	12 (12.8%)	30 (16.3%)	
Baseline blood submitted N (%)	69 (76.7%)	80 (85.1%)	149 (81.0%)	0.14
Baseline stool submitted, N (%)	79 (87.8%)	38 (40.4%)	117 (63.6%)	**<0.001**

*Patients were considered "retained" at 3-month follow-up if they completed both the follow-up questionnaire and stool sample at 3-month follow-up; ‡Patients were scored on a scale from 0-7; each patient received 1 point if they consumed ≤ 2 alcoholic beverages per day for men or ≤ 1 for women, never smoked, had a normal BMI (18-24.99), were active or moderately active, consumed ≥124 oz of fluids/day for men or ≥92 oz of fluids/day for women, did not often consume or never consumed red meat, did not often consume or never consumed chocolate or candy, and had more than 3 servings of fruits/vegetables per day.

†p-value from chi-square or fisher exact (when expected counts ≤ 5) test for categorical predictors and two-sample t-test for continuous predictors

12. INCIDENCE OF DIABETES FOLLOWING ACUTE PANCREATITIS: A MULTICENTER PROSPECTIVE COHORT STUDY

J Bejjani, A Evans Phillips, S Culp, K Duncan, PJ Lee, J Machicado, P Paragomi, J Chennat, V Singh, E Afghani, A Lahooti, S Han, L Lara, M Ramsey, S Krishna, GI Papachristou, PA Hart
The Ohio State University Wexner Medical Center

Background: Diabetes mellitus (DM) following an episode of acute pancreatitis (AP) is an increasingly described complication. However, the pathogenesis remains poorly understood and prospective studies assessing the cumulative incidence are lacking. The aim of this study is to prospectively assess the short-term incidence and risk factors of diabetes following an episode of AP.

Methods: Adults (≥18 years) hospitalized with AP were consecutively invited to participate in this multicenter, prospective cohort study from 06/2017-10/2021. Patients with pancreatic cancer, chronic pancreatitis, exocrine pancreatic insufficiency, or other malabsorption disorders at baseline were excluded. For this study, we restricted the analysis to participants who completed the 12-months questionnaire data. Diabetes status was evaluated using a combination of physician diagnosis/self-report and/or protocol-directed glycated hemoglobin (HbA1c) values. Baseline characteristics were compared between those who did versus did not develop DM.

Results: 98 participants completed the 12-months follow-up questionnaire. Approximately one-third (30/98, 30.6%) had pre-existing DM at enrollment and were not included in additional analyses. In the remaining at-risk participants, the cumulative incidence of DM (based on either physician diagnosis or protocol-directed testing) was 4.4% (3/68) at 3 months and 10.3% (7/68) at 12 months. There were no differences in demographic or pancreatitis-related characteristics between those who did versus did not develop DM (Table). The proportion of participants with new hyperglycemia (i.e., either pre-DM or DM) was 5.6% at 3 months and 11.1% at 12 months. In a subgroup analysis limited to at risk participants with HbA1c values at baseline, a paired HbA1c value was available for 14 and 16 participants at 3 and 12 months, respectively. In this subgroup, the rate of hyperglycemia was 21% (n=3) at 3 months and 13% (n=2) at 12 months.

Conclusions: The prospectively ascertained cumulative incidence of new onset DM within 1 year following AP is approximately 10%. There were no observed clinical risk factors, but the analysis is limited by the statistically small sample size. Self-reporting of DM underestimates the true disease burden, so additional, larger prospective studies are needed to investigate the incidence, risk factors, and mechanisms of DM following AP.

Table. Comparison of baseline characteristics by diabetes status 12 months following an acute pancreatitis episode.

	No New DM (n=61)	**New DM (n=7)**	**p-value**
Age, Median (IQR)	54 (22.2)	59 (35.1)	.512
Female sex	34 (55.7)	5 (71.4)	.690
White race	58 (95.1)	6 (85.7)	.359
Hispanic/Latino ethnicity	1 (1.6)	0 (0)	1.000
BMI, Median (IQR)	29.6 (9.9)	29.6 (5.9)	.801
BMI, obese category (vs. other)	28 (45.9)	3 (42.9)	1.000
Current Smoker	8 (13.3)	1 (14.3)	1.000
Current Alcohol	30 (49.2)	3 (42.9)	1.000
Prior episode of AP	17 (27.9)	3 (42.9)	.411
Etiology:			
Biliary (vs. other)	25 (41.0)	2 (28.6)	.694
Alcoholic (vs. other)	7 (11.5)	1 (14.3)	1.000
Mild clinical severity (vs. other)	41 (68.3)	3 (42.9)	.221
Pre-existing pre-DM	12 (19.7)	2 (28.6)	.627
Family History of DM	19 (31.1)	4 (57.1)	.216

13. THE INTERPLAY OF GENETIC AND ENVIRONMENTAL RISK FACTORS BUT NOT TRIGLYCERIDE LEVELS MEDIATE HYPERTRIGLYCERIDEMIC ACUTE PANCREATITIS: A CASE SERIES

L Cheesman, M Faghih, Z Kassir, V Akshintala, E Afghani, S Martin, D Whitcomb, VK Singh
Johns Hopkins University

Background: Hypertriglyceridemia (HTG) is reported to be the third most common cause of acute pancreatitis (AP) in the United States. However, it is not known why only 5-10% of patients with TG levels ≥1,000 mg/dL develop AP. Both HTG and AP are mediated by genetic and environmental risk factors but the overlap and association are poorly understood.

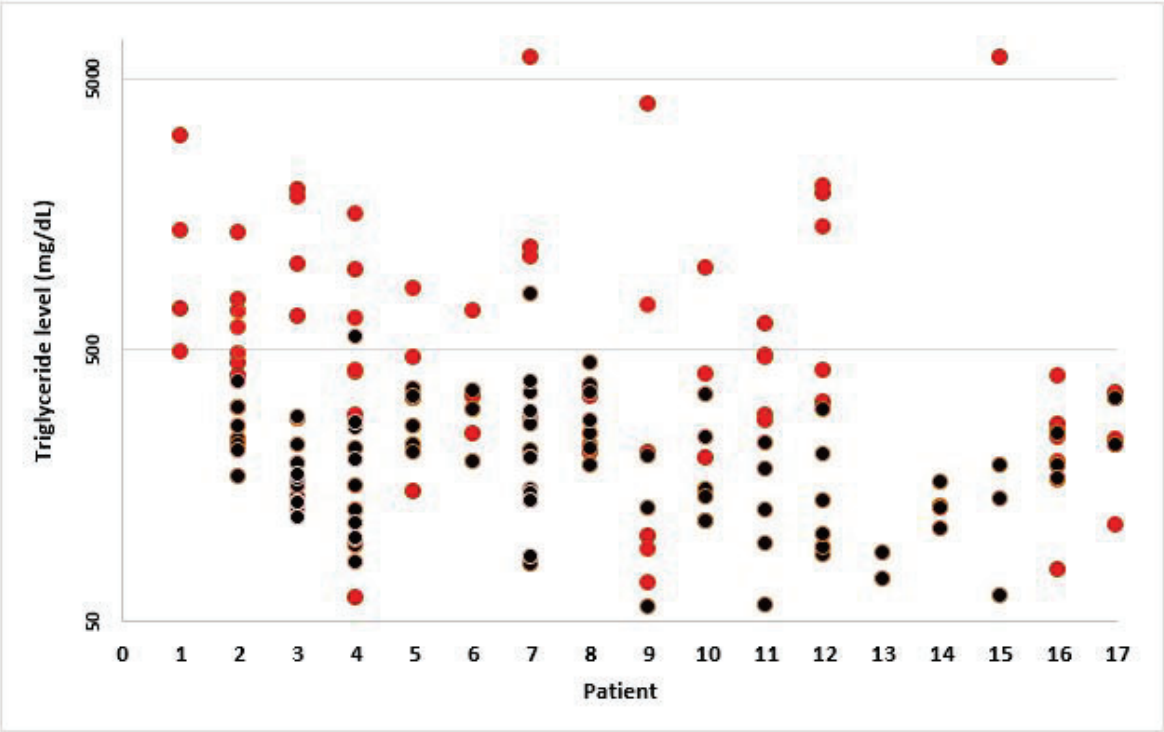
Aim: To evaluate the prevalence of pancreatitis and lipid metabolism genetic variants as well as environmental risk factors in patients with presumptive HTG-recurrent acute pancreatitis (RAP)

Methods: We evaluated consecutive subjects with presumptive HTG-RAP with or without underlying chronic pancreatitis from an outpatient pancreatitis clinic between 2018 to 2022. TG levels were recorded during all AP episodes requiring hospitalization and as well as during outpatient clinic follow-up. All subjects underwent pancreatitis genetic testing (PRSS, CFTR, SPINK1, CASR, CTRC, CEL, UBR1, SDBS, CPA1, GGT1, two regions of interest near CLDN2, and a region of interest in SLC26A9) as well as a targeted sequencing testing panel evaluating lipid metabolism variants (APOB, LPL, PPARG, FABP4, APOC2, APOA5). Environmental risk factors were recorded for each patient, including diabetes, exogenous hormone use (e.g. tamoxifen and oral contraceptives), twin gestation, and obesity (BMI ≥ 30.0).

Results: Over a 6-year follow-up, 17 subjects had 70 AP episodes, with only 24.2% (17 episodes) linked to high TG levels (≥1,000 mg/dL). In addition, all identified subjects had ≥1 environmental risk factor including diabetes (52%), exogenous hormone use (17%), and obesity (52%) which may affect hypertriglyceridemia-induced pancreatitis. All identified subjects (100%) had likely risk variants for pancreatitis in CFTR (17/100%), UBR (2/11.7%), SLC26A9 (12/70.5%), CASR (7/41%), CLDN2 (6/35%), CEL (1/5%) including ten (58%) subjects who had pathogenic variants for pancreatitis [CFTR (6/60%), PRSS1 (2/20%), SPINK1 (3/30%), CTRC (2/20%)]. In addition, fifteen (88%) subjects had lipid metabolism variants associated with increased serum triglyceride levels.

Conclusion: Patients with HTG-RAP are enriched for risk variants in genes for pancreatitis and lipid metabolism as well as environmental factors. TG levels alone did not correlate with risk of RAP. Future studies should include patients with HTG and no history of AP who have undergone pancreatitis genetic testing.

Figure1. Vertical triglyceride scatter plot level among 17 study subjects. Red dots represent triglyceride level(s) during hospitalization for acute pancreatitis and the black dots represent triglyceride level(s) between acute pancreatitis episodes.



14. THE ASSOCIATION OF FATTY LIVER DISEASE AND UNDERLYING INFLAMMATION WITH ACUTE PANCREATITIS RISK

S Patel, P Sarraf, B Boulay, H Kim, Y Dai, K Danielson, U Bagci, T Tirkes, E Mutlu, B Layden, C Yazici
University of Illinois at Chicago

Background: Fatty liver disease (FLD) is an increasingly common condition, attributable in part to a Western diet, sedentary lifestyle, and increased alcohol consumption. FLD has been associated with AP, but the relationship between the two diseases is not well understood. We hypothesize that patients with FLD have an increased risk for AP and this is in part due to an increased baseline inflammatory state in FLD.

Methods: In this case-control study, subjects were enrolled during hospitalization. AP was diagnosed by the presence of at least two of the following: i) abdominal pain consistent with AP, ii) serum amylase or lipase ≥ 3 times upper limit of normal, and iii) features of AP on cross sectional imaging. Controls were chosen among patients hospitalized for non-pancreatic disease related care. Baseline clinical and sociodemographic data were collected. In this cohort, FLD was diagnosed by CT, US and/or MRI imaging. CRP level was quantified using ELISA.

Results: One hundred subjects were enrolled. Fifty-two AP and fifteen control subjects had imaging data available for analysis. There were no significant differences in baseline characteristics including age (p = 0.529), sex (p = 0.844), BMI (p = 0.998), waist circumference (p = 0.179), pre-existing diabetes status (p =1.0) or current alcohol use (p=0.326) between the two groups. Control and AP groups differed by race (86.7% versus 38.5% African-American, p = 0.003) and ethnicity (6.7% versus 42.3% Hispanic, p = 0.024). A significantly higher percentage of AP patients had FLD (71.2%) compared to control patients (33.3%, p = 0.018), and AP subjects with FLD had higher CRP levels (35.6 mg/L) compared to AP subjects without FLD (20.8 mg/L, p=0.014) (Table 1).

Conclusion: Our results demonstrate that the prevalence of FLD is significantly higher in patients with AP compared to controls, and that AP patients with FLD have significantly higher CRP levels compared to AP patients without FLD. These results indicate that the presence of FLD is associated with an increased inflammatory state in patients with AP. Further studies are needed to explain the association between FLD and acute pancreatitis.

Table 1. Differences in key clinical and sociodemographic characteristics of acute pancreatitis and control patients			
Variable	**Control (n =15)**	**Acute Pancreatitis (n= 52)**	**P-value**
Age, years (SD)	48.8 (13.3)	46.2 (14.4)	0.529
Female, n (%)	8 (53.3)	24 (46.2)	0.844
Race: African American, n (%)	13 (86.7)	20 (38.5)	**0.003**
Ethnicity: Hispanic, n (%)	1 (6.7)	22 (42.3)	**0.024**
Body Mass Index (SD)	30.3 (5.76)	30.3 (7.77)	0.998
Waist size, cm (SD)	74.9 (26.0)	84.9 (24.7)	0.179
Pre-existing diabetes, n (%)	5 (33.3)	9 (32.1)	1
Current alcohol use, n (%)	8 (53.3)	37 (71.2)	0.326
Fatty Liver Disease, n (%)	5 (33.3)	37 (71.2)	**0.018**
CRP, mean (SD)	9.18 (15.0)	31.6 (18.2)	**<0.001**
Acute Pancreatitis Severity, n (%)			
Mild		42 (80.8)	
Moderately severe or severe		10 (19.2)	
Acute Pancreatitis etiology, n (%)			
Gallstones		13 (25.0)	
Alcohol		22 (42.3)	
Idiopathic		6 (11.5)	
Hypertriglyceridemia		5 (9.6)	
Other		6 (11.5)	

15. HYPERTRIGLYCERIDEMIA-INDUCED PANCREATITIS: A SINGLE CENTER EXPERIENCE

M Shah, C Abrenica, S Nair, JM Rhoads, N Thosani
University of Texas Health Science Center at Houston

Background: Severe hypertriglyceridemia, defined as triglyceride levels >1000 mg/dL, is estimated to cause ~6% of cases of acute pancreatitis (AP) in children. The aim of this study is to determine the incidence, clinical course, management strategies and prognosis of hypertriglyceridemia-induced acute pancreatitis (HTG-AP) in children and compare the findings to AP due to other etiologies.

Methods: Single center, IRB approved, retrospective chart review of pediatric patients admitted to Children's Memorial Hermann Hospital between January 2010- June 2022 with ICD 10 codes of 577.0 Acute pancreatitis, 577.1 Chronic pancreatitis, and K85 Acute pancreatitis.

Results: A total of 55 patients were admitted with a diagnosis of acute pancreatitis. We identified 10 patients with HTG-AP, contributing to 18% of total admissions for acute pancreatitis. M:F ratio was 1:1, all but two were Hispanic. Most patients were between 11-17 years of age. 7 of the 10 patients had a concomitant diagnosis of diabetes mellitus (5 with type 2 DM, 2 with type 1 DM). 3 patients had a genetic cause of hypertriglyceridemia (two with Berardinelli-Seip congenital lipodystrophy, one with familial chylomicronemia). All patients with HTG-AP were started on insulin drip on admission which helped in decreasing triglyceride levels. One patient with a genetic cause of hypertriglyceridemia required plasmapheresis. Enteral feeds were delayed beyond 48 hrs due to pain and rising triglyceride levels in all but two patients. 4 patients had complications including pseudocyst formation and development of necrotizing and recurrent pancreatitis. One patient required 13 admissions due to development of necrotizing pancreatitis with pseudocyst requiring necrosectomies, cystogastrostomy and external drain placement. Patients with HTG-AP had longer hospital stays as compared to AP due to other etiologies.

Conclusion: The incidence of HTG-AP may be higher than previously estimated. Underlying DM and Hispanic ethnicity may be potential risk factors. Early initiation of insulin should be strongly considered in patients with HTG-AP along with nil per os and intravenous hydration. Underlying etiology of hypertriglyceridemia may influence the response to insulin, and genetic variants may require use of plasmapheresis. HTG-AP tends to have a more prolonged course with delayed initiation of feeds, longer hospital stays and possible risk of complications.

Table 1. Hypertriglyceridemia-induced acute pancreatitis (HTG-AP) compared to acute pancreatitis due to other etiologies

Diagnosis	No. of cases	Ethnicity	Length of hospitalization (mean avg in days)	Prolonged initiation of enteral feeds
Hypertriglyceridemia-induced Pancreatitis	10	80% Hispanic, 20% African American	14.3 days	85%
Pancreatitis due to other etiologies	45	37% Hispanic, 44% Caucasian 18% other Ethnicities	6.5 days	9%

16. RACIAL DIFFERENCES IN POST ERCP PANCREATITIS

R Nana Sede Mbakop, A Forlemu, P Bandaru, V Gayam, V Kumar, P Garlapati, V Ronda, M Reddy, D Amakye
Piedmont Athens Regional Medical Center

Background: Post ERCP Pancreatitis (PEP) is reported at rates from 2 to 10%. Race has been shown to impact the risk of PEP in a prior study using a National Inpatient sample (NIS) data with ICD 9 codes. However, ICD 10 codes have been shown to be more specific in identifying health conditions and quality of patient care. The aim of this study was to investigate racial disparities in patients with PEP using the NIS nationwide database with ICD 10 codes.

Methods: We used the NIS for the years 2016-2017. Patients 18 years or older, who underwent inpatient ERCP were identified using ICD10 codes. PEP was defined as having a subsequent diagnosis of acute pancreatitis. ERCP was classified by intervention (diagnostic, hepatobiliary, pancreatic, and both). Demographic information on age, gender, race, and insurance status was collected. Unadjusted odd ratios with PEP as dependent variable were obtained using Chi square. Multivariate logistic analysis was used to identify independent predictors of PEP. P-value was set at < 0.05.

Results: A total of 10716 patients with median age of 63 years, and 52% females were identified. Overall, the PEP rate was 8.5%. Hispanics had a higher rate of PEP (10.2%) compared to Whites, Asians and Blacks (8.5%, 7.7%, and 6.8% respectively, p< 0.001). PEP rates were higher in patients who had pancreatic interventions compared to hepatobiliary interventions (14.2 vs 9.6%, p< 0.001, respectively). Blacks were more likely to have pancreatic interventions compared to Whites, Hispanics, Asians, and Native Americans (29% vs 24.5%, 24.8%, 21.9%, 17.1% respectively, p< 0.01). Also Blacks and Hispanics were more likely to be obese (15.1% and 15.5% vs 14%, 6.5%, p< 0.01) and be on Medicaid or self-pay compared to other races (p< 0.01). Multivariate analysis showed that after controlling for the ERCP intervention, Blacks and Hispanics continued to have higher odds of PEP (Table 1).

Conclusion: Blacks and Hispanics appear to have the highest rates of PEP and worse outcomes compared to other races. They are also more likely to have insurance disparities and be obese, which may have played a role in the higher rates of PEP and poorer outcomes.

Table 1: Multivariate analysis with outcome of PEP

	Adjusted odds (CI)
Age, years	0.98, 0.85 – 0.99
Sex	
Female	
Male	0.61, 0.53 – 0.70
Race	
White	
Black	1.40, 1.11 – 1.77
Hispanic	2.44, 1.21 – 4.90
Asian	1.22, 0.80 – 1.88
Native American	0.81, 0.63 – 1.04
Insurance	
Medicare	
Medicaid	0.77, 0.55 – 1.09
Private insurance	1.08, 0.87 – 1.35
Self-pay	0.45, 0.21 – 1.02
Admission day	
Weekday	
Weekend	1.14, 0.99 – 1.30
Obesity	1.17, 1.14 – 1.78
ERCP intervention	
No intervention	
Hepatobiliary	0.91, 0.79 – 1.04
Pancreatic	1.79, 1.55 – 2.06
Both	1.92, 1.87 – 2.10

17. DISCORDANCE IN RADIOLOGY READINGS AMONG PATIENTS PRESENTING WITH SUSPECTED ACUTE PANCREATITIS

A Singh, F Bhullar, A Kamal, M Faghih, N Bush, MA Khashab, E Afghani, VK Singh, A Zaheer, V Akshintala
Johns Hopkins University

Background: Acute pancreatitis (AP) is among the most common causes of inpatient hospitalization in the USA. Characteristic imaging features are an important part of the diagnostic criteria for AP. The subtle nature of the imaging findings of AP with subjective assessment, especially in mild AP can lead to discordance among the reading radiologists in some cases, which can have an impact on the clinical care of these patients.

Methods: All patients with lipase >3-fold the ULN were identified using a real-time notification system in the EMR. Patients' medical records were screened for demographics, clinical information, and the type of diagnostic imaging. Those who experienced abdominal pain and underwent abdominal imaging (CE-CT/MRI) were included in the study. 2 radiologists who were aware of the elevated lipase levels but were blinded to all other clinical information performed repeat examinations of the imaging studies for all the patients and the results were compared to the initial report of the imaging. Fleiss's kappa test was used to check the agreement between the two readings. A p-value of < 0.05 was regarded as statistically significant.

Results: Lipase elevation notifications were received for 575 unique adult patients of whom 234 had abdominal pain and underwent diagnostic abdominal imaging (CE-CT/MRI). Among these patients, 122 (52.1%) were diagnosed with AP on initial imaging review. On repeat examination of the imaging studies 141 (60.2%) patients were found to have AP, with a discordance in 45 (19.2%) reports. 28.5% (32/112) of the originally negative reports were reported positive, while 10.6% (13/122) of originally positive reports were considered negative. Fleiss' kappa showed moderate agreement between the radiologist's judgments, $\kappa = 0.609$ (95% CI, 0.605 – 0.613), $p < 0.001$.

Conclusion: The discordance rate for reading diagnostic imaging of AP was 19%, which is within range of what has been observed for other diseases. Thus imaging results should be interpreted by taking into account the complete clinical picture to avoid any significant changes in clinical management.

18. ALCOHOL INTAKE FREQUENCY REDUCES THE RISK OF GALLSTONES STRATIFIED FOR SEX, BMI AND POLYGENIC RISK SCORE: IMPLICATIONS FOR ACUTE PANCREATITIS

PJ Greer, S Adams, DC Whitcomb
Ariel Precision Medicine

Background: Acute pancreatitis (AP) is one of the most common gastrointestinal diseases resulting in hospitalization. Gallstone disease (GSD) is the most common etiology for AP with risk associated with female sex, obesity and genetics. Alcohol is the second most common etiology of AP with risk associated with men drinking >4 drinks per day with possible protection from AP with moderate drinking. We tested the hypothesis that moderate alcohol intake reduces the risk of GSD as a mechanism for potential protective effects from AP.

Methods: We evaluated the UK Biobank (UKBB) cohort of 449,999 subjects with complete data. We computed the Polygenic Risk Score (PRS) for GSD as published by Tanigawa Y et al. (2022) using the PGS Catalog utilities. Alcohol intake frequency was recategorized as never drinking to ~1/month, ~1/week, and >=3days/week. GSD presence or absence was determined using ICD10-codes, ICD9-codes, death certificates, and self-reported interview data. Associations were compared using Chi squared test. Multiple factors were evaluated using logistic regression.

Results: In the UKBB, GSD was noted in 7.8% of subjects. Men were less likely to have GSD than women (OR: 0.582, p=1.25e-58), risk of GSD progressively increasing with BMI above 25 with BMI 25-30 62% and >30 266% higher, and increase in risk with PRS from lowest to highest quintiles (OR 2.890, p=0.0e+00). The PRS effect was greater in women than men, and greater with higher BMI. When compared to non-drinkers+~1/m, the risk of GSD was reduced with drinking ~1day/week (OR: 0.772, p=1.94e-74) and reduced further when drinking >=3days/week (OR: 0.602, p=1.140e-305). GSD subjects showed similar PRS across all alcohol intake frequency categories (p=0.316).

Conclusion: We replicated the finding of Lim et al, where the risk of GSD is higher in women than men with progressive risk from higher BMI and higher PRS. The risk of GSD is reduced by 22.8% with ~1day/week of alcohol, and 39.8% with >=3days/week of alcohol. The benefit of moderate alcohol consumption in reducing AP may be through reduced risk of biliary pancreatitis. However, after an initial AP attack the effects of any alcohol appears to increase risk of RAP and chronic pancreatitis.

19. SURVEY OF CLINICAL AND SUBCLINICAL EVEN RATES IN RAP

P Greer, J Gibson, J Swoger, T Moore, D Whitcomb
Ariel Precision Medicine

Background: Acute pancreatitis (AP) is one of the most common gastrointestinal diseases resulting in hospitalization. Recurrent acute pancreatitis (RAP) occurs on 10-20% of all AP subjects, but the frequency and severity of RAP attacks is difficult to estimate from hospitalizations alone since many patients learn to manage episodes at home. Prior estimates range from 0.2 to 3.5 AP events per year.[Ramsey, Akshintala, de Jong] Few have attempted to collect data on subclinical events that do not result in hospitalization.

Methods: We created an anonymous, 2 minute, on-line survey to collect information on clinical and subclinical events for RAP subjects who were referred to Ariel Precision Medicine for genetic testing. The survey included seven questions: Have you been diagnosed with AP?, Number of lifetime AP hospitalizations?, Number of AP hospitalization in the past two years?, Number of days of missed school/work/social engagements due to pancreatic abdominal pain in the past two years? Number of events of pancreatic abdominal pain with no attempt to receive medical care in the past year? Have you been diagnosed with a pain syndrome? The survey was emailed out with a reminder 1 week later. The survey results were collected, and all statistical tests were run in R (R-project.org)

Results: A total of 198 people responded to the survey with a median time to completion of 98 seconds. After filtering for RAP, incomplete answers, constant moderate-to-severe pain lasting over 3 months (chronic pain syndrome confounding estimates of RAP attacks) and impossible answers (AP in last 2 years greater than lifetime), 98 subjects with usable data remained. The rate of AP hospitalization was 1.44 events per year, ER visits without hospitalization were lower at 1.37/year. The number of missed work/school/social engagements were 2.46/year and the number of severe pain attacks without seeking medical care was 3.95/year.

Conclusion: Counting attacks of RAP by the number of hospitalizations appears to grossly underestimate the burden of RAP on patients. Future studies should include measures to capture RAP events managed at home.

20. SUB-NORMAL PANCREAS VOLUMES IN CHILDREN AND YOUNG ADULTS FOLLOWING ACUTE AND ACUTE RECURRENT PANCREATITIS

B Fortson, N Mahalingam, T Thompson, M Abu-El-Haija, A Trout
Cincinnati Children's Hospital Medical Center

Background: Pancreatic atrophy is a subjective but important finding of chronic pancreatitis (CP). The purposes of this study were to: (1) quantify pancreatic volume by magnetic resonance (MR) and computed tomography (CT) in children and young adults with either (a) a single prior episode of acute pancreatitis (AP) or (b) acute recurrent pancreatitis (ARP), and (2) compare these volumes with established normative volumes.

Methods: Participants were selected from a registry of patients with clinically confirmed AP and ARP. Age, height, and weight were abstracted from the medical record. The closest CT or MRI examination, obtained more than 30 days after the date of AP or ARP, without findings of active AP, was selected for segmentation. The pancreas was manually segmented in 3D Slicer by a pediatric radiology fellow with segmentations refined by a board-certified pediatric radiologist. Volumes were compared to previously published percentile values defined by age and body-surface area (BSA).

Results: 15 patients were in the AP group (mean age 14.3±3.3 y) and 20 patients were in the ARP group (mean age 12.9±5.3 y), 10 of whom were also in the AP group. Mean parenchymal volumes were 56±24 mL (range: 16-101 mL) and 49±33 mL (range: 5-111 mL) in the AP and APR groups, respectively.

Among the 15 examinations from patients with AP, 6 (40%) total patients had pancreatic volumes < 50th percentile for BSA, 5 (33%) of whom had volumes < 25th percentile. Two (13%) patients had volumes < 5th percentile for age, one (7%) of whom had volume < 5th percentile for both BSA and age.

Among the 20 examinations from patients with ARP, 10 (50%) total patients had pancreatic volumes < 25th percentile for BSA, 8 (40%) of whom had volumes < 5th percentile. Six (30%) total patients had volumes < 5th percentile for both BSA and age.

Conclusion: Pancreas parenchymal volumes are sub-normal (< 5th percentile) in a small percentage of pediatric and young adult patients after a single attack of AP and are sub-normal in a substantial percentage of patients with ARP. Pancreatic parenchymal volume deserves additional investigation as an objective marker of progressive pancreatitis in children.

21. DELETION OF NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN DECREASES PANCREATITIS SEVERITY AND BROWN ADIPOSE TISSUE WEIGHT IN A MURINE MODEL OF ACUTE PANCREATITIS

Z Hurst, K Gumper-Fedus, S Knoblaugh, K Chasser, V Pita-Grisanti, E Velez-Bonet, Z Cruz-Monserrate
The Ohio State University

Background: Acute pancreatitis (AP) is a common inflammatory disorder of the pancreas with an incidence of up to 35 per 100,000 individuals in the United States. Approximately 25% of patients with AP develop severe AP (SAP), which can result in life-threatening complications including sepsis and multiple organ dysfunction syndrome. Neutrophil gelatinase-associated lipocalin (LCN2/NGAL) is a secreted adipokine that plays a complex role in the pathophysiology of pancreatic diseases previously shown to contribute to the development of pancreatic fibrosis and regulation of inflammation in pancreatic cancer and is upregulated in AP within 24 hours of an acute attack. Additionally, LCN2 has been shown to play a role in the activation of brown adipose tissue (BAT) via a non-adrenergic mechanism. In this study we sought to determine the effects of systemic and pancreas-specific deletion of LCN2 expression in AP.

Methods: AP was induced using six hourly intraperitoneal injections of 50µg/kg body weight caerulein, an analog of cholecystokinin which results in pancreatic overstimulation, for two consecutive days in male control wild-type mice (C57Bl/6J, n=5) and mice with systemic (LCN2-/-, n=4) or pancreas-specific (LCN2flox/flox/PDX-CRE, n=4) deletion of LCN2 expression. To assess AP severity, we measured serum amylase and lipase and performed histology on the pancreas. The mouse pancreases were evaluated for acinar cell loss, mononuclear inflammation, and stromal fibrosis using a pancreatitis index scoring system.

Results: LCN2flox/flox/PDX-CRE mice demonstrate a significantly lower pancreatitis index score (p=0.0383) compared to the control mice, indicating a less severe AP. However, LCN2-/- mice demonstrated only a trend toward lower pancreatitis index scoring compared to control mice. We also observed a decrease in the trend of serum amylase in both LCN2-/- and LCN2flox/flox/PDX-CRE mice compared to control mice. Interestingly we observed a significant decrease in the BAT weight of LCN2-/-, but not LCN2flox/flox/PDX-CRE, mice compared to the control mice.

Conclusion: Our results suggest that loss of LCN2 expression reduced the severity of AP in a murine model warranting its further investigation as a potential therapeutic target in AP. Moreover, the reduction in BAT suggests that LCN2 expression could contribute to BAT adipogenesis and/or the regulation of adipose tissue browning.

22. NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN AND ITS HYDROPHOBIC LIGANDS ARE ALTERED IN THE BLOOD OF CHRONIC PANCREATITIS.

K Gumpper-Fedus, KM Chasser, O Crowe, V Pita-Grisanti, N Badi, R Cole, MA Belury, S Culp, A Hinton, PA Hart, SG Krishna, LF Lara, ML Ramsey, L Li, W Fisher, S Pandol, EL Fogel, SS Vege, W Mark, C Forsmark, M Bellin, J Serrano, D Yadav, DL Conwell, Z Cruz- Monserrate
The Ohio State University Wexner Medical Center

Background and Aims: Pancreatitis is a fibroinflammatory disorder lacking therapies or biomarkers that distinguish acute and chronic disease. Neutrophil gelatinase-associated lipocalin (NGAL) is a proinflammatory adipokine elevated in the blood during inflammatory diseases and can be secreted by several cell types to bind hydrophobic ligands like the fatty acid (FA) linoleic acid. Here, we aimed to examine changes in the expression of NGAL and its hydrophobic ligands in the blood of chronic pancreatitis (CP) subjects and assess whether NGAL could be a biomarker for CP.

Methods: NGAL was measured in biospecimens (plasma and urine) from a discovery cohort of samples provided by the multicenter PROspective Evaluation of Chronic Pancreatitis for EpidEmiologic and Translational StuDies (PROCEED) study in acute and recurring acute pancreatitis (AP/RAP), CP and a control group. Plasma FA abundance was assessed using gas chromatography.

Results: NGAL was significantly elevated in the plasma of subjects with CP compared to control ($p < 0.0001$, AUC = 0.777) and subjects with AP/RAP ($p < 0.0001$, AUC = 0.754), but there was no difference in urine NGAL levels. Propensity score matching further showed a significant elevation in plasma NGAL in CP compared to controls ($p < 0.05$) or AP/RAP ($p < 0.01$). NGAL was elevated in subjects with CP and diabetes compared to CP cases without diabetes alone ($p < 0.0001$). Linoleic acid, a strong binding partner of NGAL, was less abundant in the blood of CP compared to AP ($p < 0.05$). FAs downstream of linoleic acid metabolism like dihomo-γ-linoleic acid and adrenic acid were more abundant in the blood of CP compared to AP/RAP and controls.

Conclusion: Plasma NGAL may be a potential biomarker of CP, especially if combined with other clinical characteristics; however, this will need to be validated in a separate cohort. Furthermore, the elevated NGAL levels in CP may be contributing to metabolic dysfunction by binding and trafficking linoleic acid into cells for increased conversion to its metabolites, suggesting a possible mechanism for the pathophysiology of CP.

23. 10-YEAR OUTCOMES OF SMOKING CESSATION ATTEMPTS IN REDUCING LOW BONE DENSITY DISEASE IN CHRONIC PANCREATITIS

A Chatterjee, V Chittajallu, J Perez, P Chahal
Cleveland Clinic Foundation

Background: Chronic pancreatitis (CP) patients are at increased risk of low bone density, and tobacco smoking is an independent risk factor for developing low bone density in CP patients. We aimed to determine if smoking cessation would lower the risk of low bone density in CP patients.

Methods: We conducted a retrospective cohort study utilizing TriNetX, a global federated health research network of electronic medical records. Utilizing ICD-10 codes, we identified CP patients with tobacco smoking history and divided them into two cohorts. Smoking cessation was defined as a prescription for varenicline/bupropion or a smoking cessation counseling visit. Propensity score matching (PSM) was performed for demographics and medications (pancreatic enzyme replacement, calcium, and vitamin D). We assessed 10-year outcomes for osteoporosis, osteopenia, fractures, malnutrition, calcium deficiency, vitamin-D deficiency, and mortality.

Results: We identified 9,142 patients with smoking cessation attempts (Cohort 1) and 52,679 patients with no smoking cessation attempts (Cohort 2). After PSM, both cohorts had 9,085 patients. Cohort 1 was more likely to be prescribed lipase, pancrelipase, vitamin-D, and calcium supplements when compared to Cohort 2. There was lower 10-year-mortality (OR:0.85, 95% CI:0.79-0.92, p< 0.001) and malnutrition (OR:0.77, 95% CI:0.71-0.82, p< 0.001) in Cohort 1 compared with Cohort 2. A sub-analysis, excluding patients on lipase, pancrelipase, vitamin-D, and calcium supplementation, still noted lower 10-year-mortality (OR:0.71, 95% CI:0.58-0.86, p=0.001) and malnutrition (OR:0.44, 95% CI:0.34-0.55, p< 0.001) in patients who received smoking cessation therapy. No differences were noted in the 10-year development of osteoporosis, osteopenia, or fractures, regardless of smoking cessation attempts.

Conclusion: CP patients are ideally managed in a multidisciplinary fashion and necessitate close follow-up with their primary care physicians (PCP), gastroenterologists, and pain management specialists.

Table 1. Demographics before and after propensity score matching

Characteristic	Before propensity match		After propensity match	
	Smoking Cessation (N=9,142)	No Smoking Cessation (N=57,679)	Smoking Cessation (N=9,085)	No Smoking Cessation (N=9,085)
Age — yr (mean ± SD)	53.2 +/- 12.0	53.4 +/- 14.5	53.2 +/- 12.0	53.3 +/- 12.0
Female — no. (%)	4537 (49.9)	24236 (42.4)	4537 (49.9)	4520 (49.8)
Caucasians — no. (%)	6444 (70.9)	37409 (65.5)	6444 (70.9)	6461 (71.1)
American Indian or Alaska Native — no. (%)	48 (0.5)	306 (0.5)	48 (0.5)	38 (0.4)
Black or African American — no. (%)	1952 (21.5)	12953 (22.7)	1952 (21.5)	1963 (21.6)
Hispanic or Latino — no. (%)	276 (3.0)	2404 (4.2)	276 (3.0)	259 (2.9)
Asian — no. (%)	38 (0.4)	437 (0.8)	38 (0.4)	37 (0.4)
Alcohol related disorders — no. (%)	2486 (27.4)	9236 (16.2)	2486 (27.4)	2472 (27.2)
Lipase — no. (%)	1495 (16.5)	3769 (6.6)	1495 (16.5)	608 (6.7)
Vitamin D — no. (%)	649 (7.1)	1884 (3.3)	1237 (13.6)	800 (8.8)
Calcium — no. (%)	2574 (28.0)	10475 (18.3)	2547 (28.0)	1893 (20.8)

Table 2. Ten-year outcomes after propensity score matching

Outcome	Smoking Cessation (N = 9,085)	No smoking cessation (N=9,085)	Odds ratio	95% Confidence Interval
Osteoporosis	859 (9.5%)	635 (7.0%)	1.39	1.24-1.54
Calcium deficiency	10 (<0.1%)	17 (<0.1%)	0.59	0.27-1.28
Vidamin D deficiency	1978 (21.8%)	1195 (13.2%)	1.84	1.7-1.9
Malnutrition	1746 (19.2%)	2151 (23.7%)	0.77	0.71-0.82
Mortality	1447 (15.9%)	1659 (18.3%)	0.85	0.79-0.92

24. ß-ADRENERGIC SWEAT TEST IN ACUTE RECURRENT AND CHRONIC PANCREATITIS WITH CFTR GENE MUTATIONS

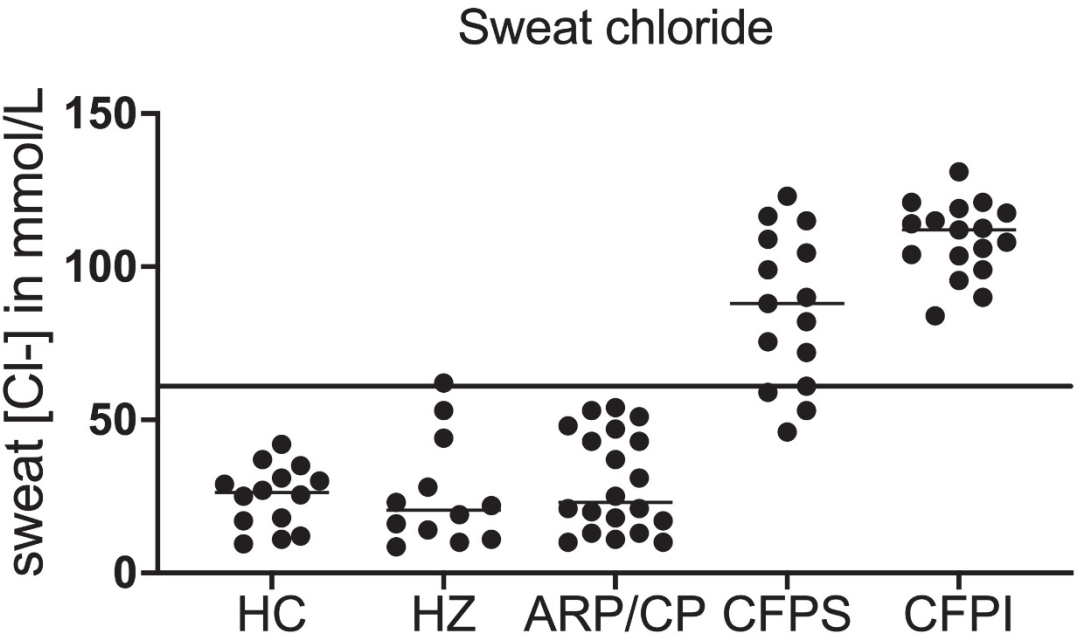
V Gummadi, J Avolio, T Gonska
The Hospital for Sick Children

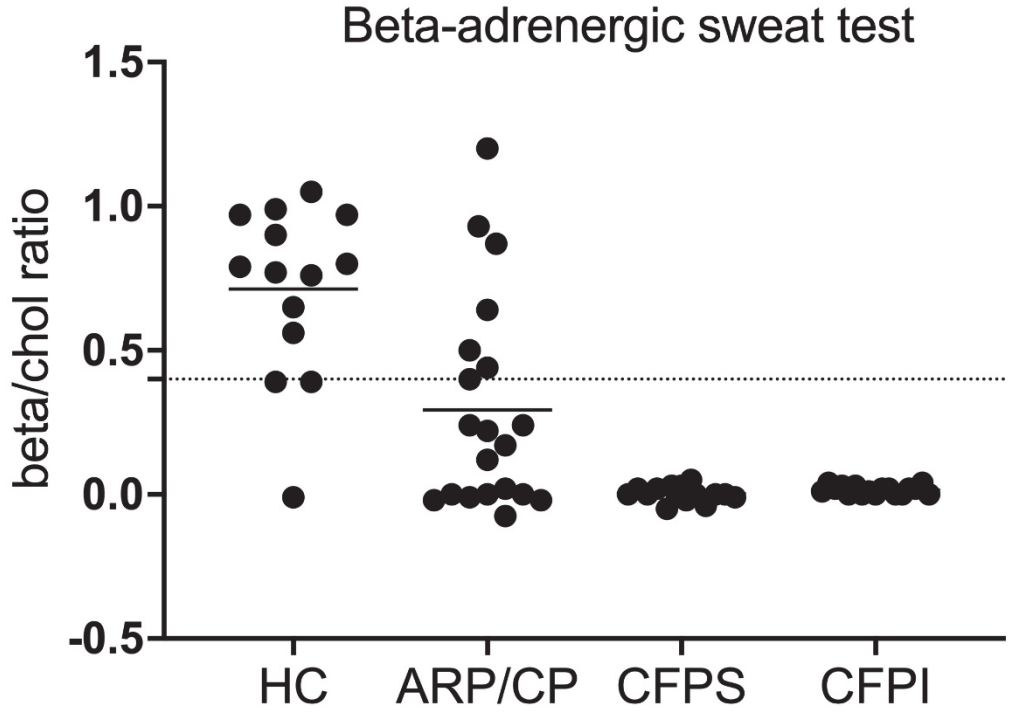
Background: CFTR gene mutations are significant contributors to the risk of acute recurrent pancreatitis/chronic pancreatitis (ARP/CP) in individuals with and without cystic fibrosis (CF). With the availability of CFTR modulator therapy, there is growing clinical relevance to their use in non-CF individuals like those with ARP/CP who harbor CFTR gene mutations. But, given that there >2000 CFTR gene mutations with varying degrees of CFTR dysfunction and disease-causing effects proven only in some mutations, it is difficult to determine as to who would benefit from the CFTR modulator therapy. We describe the use of ÃŸ-adrenergic sweat test to address this clinical question.

Methods: 20 individuals (median age 18.5; range 7-43yrs) with ARP/CP, who had a CFTR genetic analysis result were prospectively recruited from 2013-2017 to undergo the sweat test. ß-adrenergic sweat secretion test (ratio of ß-adrenergic and cholinergic sweat rates-ß/chol ratio) and the classic sweat chloride test were performed on each subject. For comparison, we also included individuals covering the spectrum of CFTR dysfunction-CFPI (CF pancreatic insufficient, n=17), CFPS (CF pancreatic sufficient, n=15), and healthy controls (HC, n=14).

Results: Mean sweat chloride levels and ß/chol ratios in the ARP/CP group were 29.3±16 mmol/L and 0.29±0.36 respectively. On comparison with other groups, the mean ß/chol ratios are significantly different ($p < 0.05$) between the groups; with CF patients having absent ß-adrenergic sweat secretion whereas the sweat chloride levels overlapped between the groups. None of the individuals in the ARP/CP group had a sweat chloride level >60mmol/L and 13 (65%) had a ß/chol ratio of < 0.40. There is no correlation between the sweat chloride and ß-adrenergic sweat secretion tests ($r=0.02$, $p=0.93$) in the ARP/CP group.

Conclusion: ß-adrenergic sweat test distinguishes between subjects with variable CFTR function. It can be helpful to determine the contribution of CFTR dysfunction to the pancreatic disease and thus identifying individuals with ARP/CP who might benefit from CFTR modulator therapy, if it were to be used.


Sweat chloride
sweat [Cl-] in mmol/L
150
100
50
0
HC
HZ
ARP/CP
CFPS
CFPI


Beta-adrenergic sweat test
beta/chol ratio
1.5
1.0
0.5
0.0
-0.5
HC
ARP/CP
CFPS
CFPI

25. GERMLINE MULTIGENE PANEL TESTING IN ACUTE AND CHRONIC PANCREATITIS

ML Ramsey, B Heald, Y Gokun, J Baker, JR Groce, S Han, PA Hart, SG Krishna, LF Lara, PJ Lee, GI Papachristou, R Pearlman, S Poll, ME Roberts, PP Stanich
The Ohio State University

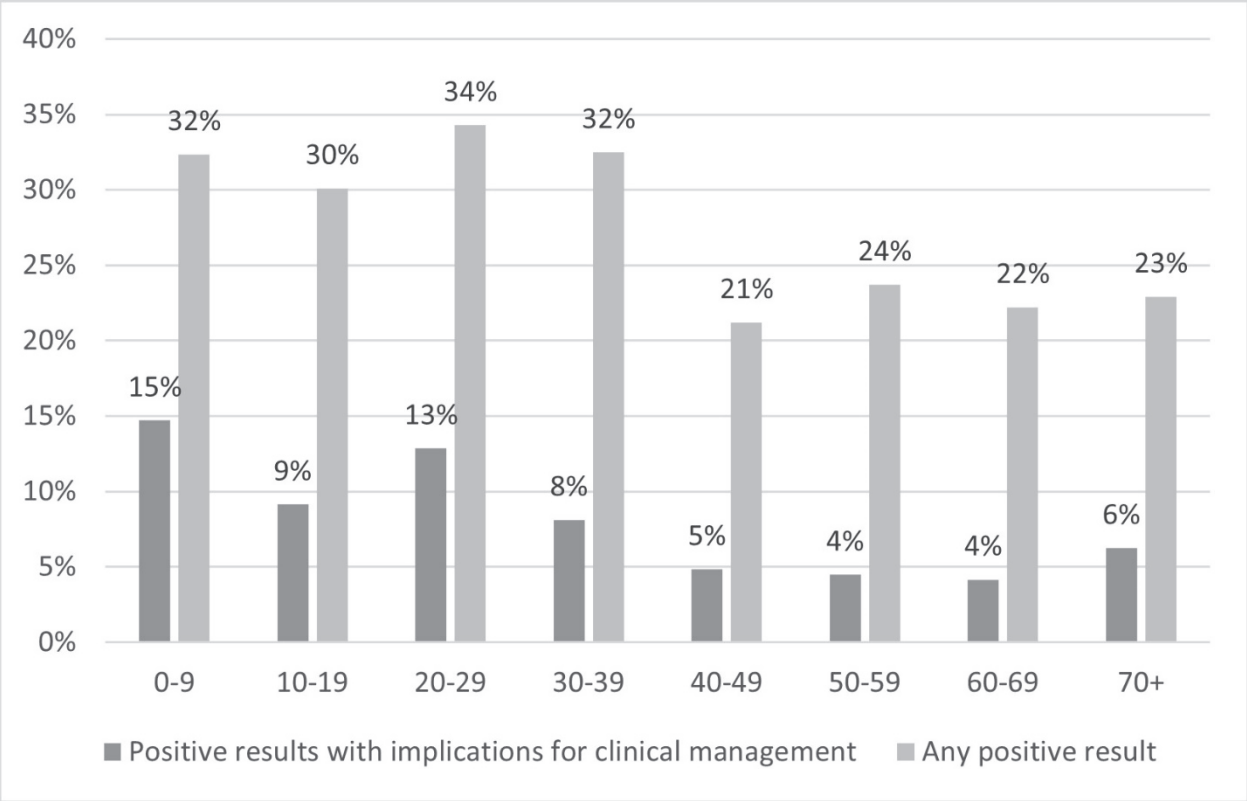
Background: Germline genetic testing is recommended for younger patients with idiopathic pancreatitis, but there is no consensus recommendation for those over age 35. We aimed to analyze genetic testing results across a variety of ages.

Methods: Individuals (ages 0-90yr) who underwent germline multigene testing for pancreatitis susceptibility genes (CASR, CFTR, CPA1, CTRC, PRSS1, SPINK1) through a large commercial laboratory between 2017 and 2022 were selected. Multivariable logistic regression models were performed to identify factors associated with a positive result (defined as at least one pathogenic, likely pathogenic, and/or increased risk variant in a pancreatitis-related gene). Clinically significant results were defined as positive results in PRSS1, biallelic CFTR or SPINK1, or polygenic (i.e., CFTR and CASR).

Results: Overall, 2,468 subjects with primary indication of pancreatic cancer (n=128), acute pancreatitis (AP; n=401), chronic pancreatitis (CP; n=631), or other indications (n=1,308) completed panel testing including pancreatitis genes. Among patients with pancreatic cancer, 10.2% had monoallelic variants in CFTR, but none had biallelic variants in CFTR. Among subjects with AP or CP (n=1,032), the most common variants were monoallelic CFTR (18.8%), monoallelic SPINK1 (6.9%), PRSS1 (4.5%), polygenic (2.0%), CTRC (1.6%), and biallelic CFTR (1.3%). The frequency of pathogenic variants varied according to age at the time of testing (Figure 1).

Among subjects with AP or CP, a positive result was more common at < 35 vs ≥35yr (32.1% vs 24.5%, p=0.007). Similarly, a clinically significant result was more common at < 35 vs ≥35yr (10.8% vs 5.4%, p=0.001). After adjusting for age, sex, race/ethnicity, primary indication for testing, and family history of pancreatitis, a positive family history of pancreatitis was associated with increased odds ratio (OR) of 8.59 (95% confidence interval (CI) 2.92-25.25) for a clinically significant panel result while each 5-year increase in age at test completion had lower odds (OR 0.89, 95% CI 0.83-0.95).

Conclusion: The yield of germline genetic testing is highest in younger individuals with a positive family history of pancreatitis, which supports current recommendations for testing. However, a clinically significant result was found in approximately 5% of older adults suggesting that germline testing could be considered for patients of all ages.


40%
35%
30%
25%
20%
15%
10%
5%
0%
32%
30%
34%
32%
21%
24%
22%
23%
15%
13%
9%
8%
5%
4%
4%
6%
0-9
10-19
20-29
30-39
40-49
50-59
60-69
70+
Positive results with implications for clinical management
Any positive result

26. PANCREATIC STONE SIZE AND STONE DENSITY PREDICT THE NUMBER OF ESWL SESSIONS NEEDED FOR SUCCESSFUL FRAGMENTATION

S Sonaiya, N Bush, P Chandragiri, N Gaurav, I Lahooti, A Singh, L Patel, M Khashab, P Lee, R Talukdar, V Singh, S Lakhtakia, S Han, M Tandan, V Akshintala
Johns Hopkins Medicine

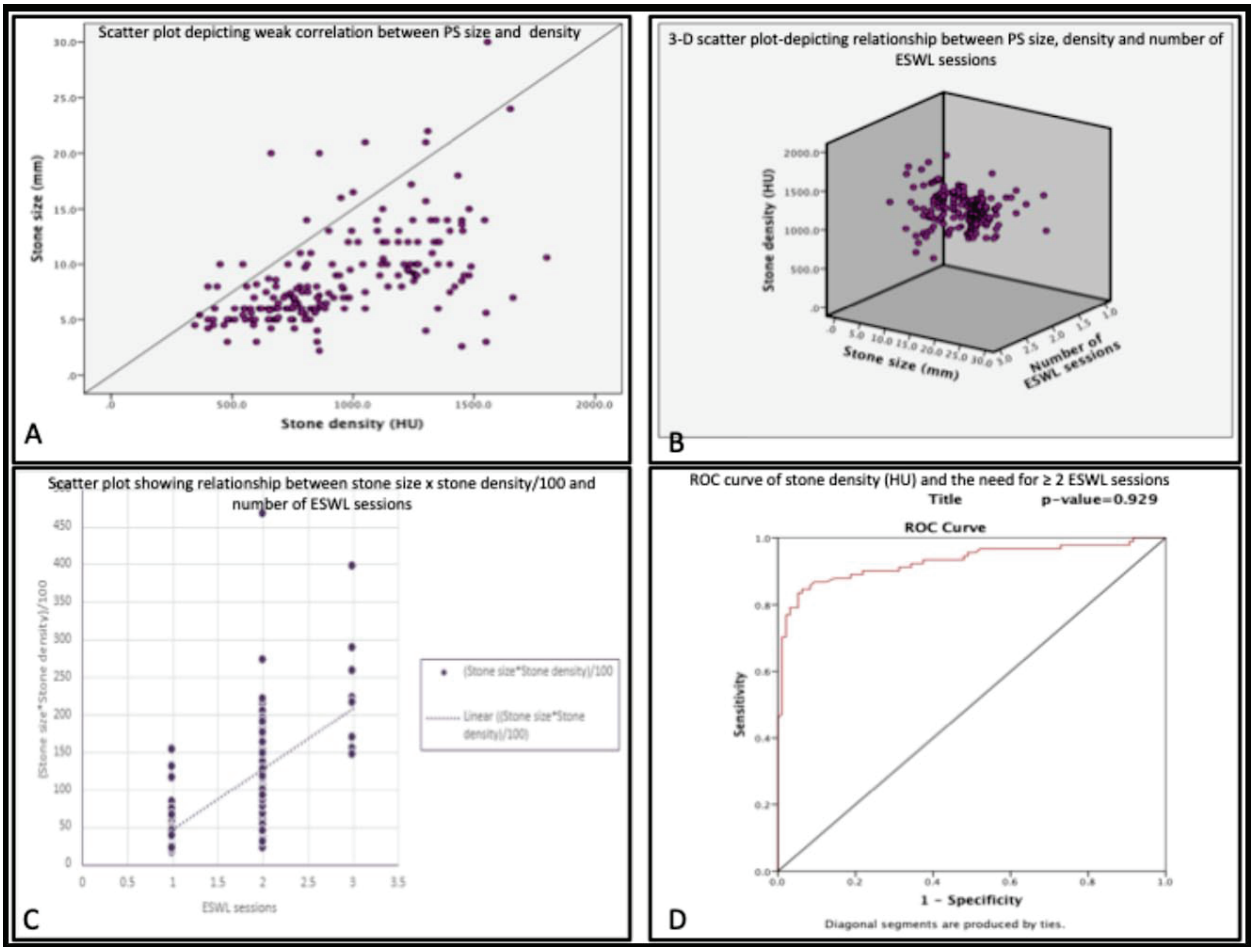
Background: Extracorporeal shock wave lithotripsy (ESWL) is the first-line treatment for pancreatic stones (PS) in chronic pancreatitis (CP). The role of computer tomography (CT) scan-based stone density in determining the efficacy and the number of ESWL sessions in pancreatic stones has been poorly understood.

Methods: We retrospectively evaluated CP patients with PS who underwent ESWL therapy for stone fragmentation at 3 academic centers in India and the US. Successful fragmentation was defined as the breakdown of the stone to a size < 3mm. In this study, we aim to create a model for predicting the number of ESWL sessions needed based on stone size (mm) and CT stone density (HU) for CP patients with PS.

Results: 187 subjects (Mean age 36.9±12.3 years, 61.5% male) underwent ESWL. The mean PS size was 8.7±4.2 mm (2.2-30 mm) and the mean density measured by CT attenuation value was 940.3±328 HU (346-1800 HU). 96 (51.3%) needed a single session, 83 (44.4%) needed 2 sessions, and the remaining 8 (4.3%) needed 3 ESWL sessions for successful fragmentation. The clinical success rate measured by the difference in pain scores was 59.5±29.4% (Table 1). We found a positive direct correlation between the mean PS density and the number of ESWL sessions (r=0.746, CI 0.675-0.804, p-value < 0.01).

We created a model based on PS stone size(mm), stone density(HU), and the Pearson-coefficient r to create a prediction model for the number of ESWL sessions needed. The model was found to have a significant relationship with the number of ESWL sessions (R2 0.47, r=0.685 95% CI 67.98 to 92.73, p-value < 0.01). The PS density metric had an AUROC of 0.929 in predicting the number of ESWL sessions (p-value < 0.01) and a density threshold of 895 HU could predict the need for multiple ESWL sessions with an accuracy of 89.1% (Figure 1).

Conclusion: PS size and CT Stone density are strong predictors of the technical efficacy of ESWL in terms of determining the number of sessions required for successful fragmentation. This information would help the provider in discussing the procedure plan with the patient and planning the logistics.


Scatter plot depicting weak correlation between PS size and density
Stone size (mm)
Stone density (HU)
A
3-D scatter plot-depicting relationship between PS size, density and number of ESWL sessions
Stone density (HU)
Stone size (mm)
Number of ESWL sessions
B
Scatter plot showing relationship between stone size x stone density/100 and number of ESWL sessions
(Stone size*Stone density)/100
ESWL sessions
(Stone size*Stone density)/100
Linear ((Stone size*Stone density)/100)
C
ROC curve of stone density (HU) and the need for ≥ 2 ESWL sessions
Title
p-value=0.929
ROC Curve
Sensitivity
1 – Specificity
Diagonal segments are produced by ties.
D

27. CHARACTERIZING SYMPTOM BURDEN AND IMPACT OF CHRONIC PANCREATITIS IN AN ONLINE PATIENT COMMUNITY

Y Hernandez-Barco, J Twal, J Pack, V Powell
Massachusetts General Hospital

Background: Due to a deficiency in functional digestive enzymes, patients with chronic pancreatitis (CP) may experience exocrine pancreatic insufficiency (EPI), maldigestion/malnutrition, and reduced quality of life. A 2-pronged approach using a US-based registry for patients with EPI taking pancreatic enzyme replacement therapy (PERT) and an online patient community with structured/unstructured data collection activities is being implemented to capture data/experiences to better appreciate the patient's lived experience, needs, and burden of illness. The study goal was to understand CP with EPI from the patient's perspective based upon online community discussion.

Methods: The online community, administered and moderated by Mission: Cure, includes patients, caregivers, and advocates. Data from 3 quick polls and 1 community survey conducted in 2022 were collected/analyzed.

Results: In quick poll 1, 17 of 32 (53%) respondents (patients with CP) reported having to restrict/change their diet "all the time" to avoid CP symptoms and 1 reported never doing so. Most (18/34; 53%) respondents (patients with CP) in quick poll 2 stated that diet had a major impact on CP-related abdominal pain; no respondents considered diet to have no impact. Stomach pain (n=10), flatulence (n=9), and bloating (n=9) were the most common symptoms of EPI considered bothersome by the 14 respondents (patients with EPI due to CP) of quick poll 3. The community survey was conducted in patients with CP (N=44); 4 (9%) had been diagnosed with EPI and recurrent acute pancreatitis. Results showed that 28 of 44 respondents experienced bothersome symptoms at least once per day and considered stomach pain (n=26), bloating (n=6), and steatorrhea (n=5) to be the most bothersome CP symptoms. Most respondents stated their most bothersome CP symptoms had major impacts on daily life, including effects on emotional well-being (77%), social life (73%), intimacy (66%), physical activities (60%), and work life (59%).

Conclusion: This analysis identified symptoms that patients consider most bothersome, the impact of these symptoms on daily life, and how diet can affect symptom burden. Ongoing analysis of community discussions and comparisons to US-based registry data with patients taking PERT will help develop more robust understanding of patient experiences and burden of CP.

29. TRANSFER DELAY, LARGER NECROTIC COLLECTION AND NEED FOR ENTERAL NUTRITION ASSOCIATED WITH INCREASED UNPLANNED 30 DAY READMISSION IN NECROTIZING PANCREATITIS

G Suryawanshi, A Terwillger, W Xiao, DE Jonason, S Amateau, N Azeem, S Mallery, B Kuzmak, M Freeman, G Trikudanathan
University of Minnesota

Background: Hospital readmission rate is a key metric of patient care quality and represents a substantial burden to both the individual patient and the health care system. Necrotizing pancreatitis (NP) with its protracted course characterized by organ failure, need for intervention and ICU stay, malnutrition, and physical deconditioning, is associated with increased readmission rate. The aim of this study was to estimate the incidence and cause for 30 day unplanned readmission following index hospitalization for NP and to identify independent risk factors for readmission.

Methods: Adult NP patients managed at our center between 2009-2022 were identified from a prospective database and categorized into 2 groups based on readmission status. Patients with no follow up, who died during index admission or within 30 days of discharge were excluded. Baseline demographic, disease severity, discharge, intervention, and radiologic characteristics were collected. Multivariable analysis was completed to identify independent predictors of 30 day readmission; a p-value < 0.05 was considered significant.

Results: Among 505 patients [males – 345 (68%), median age 50 years] included, 201 (40%) had at least one unique readmission within 30 days of index discharge. There were 258 unique readmissions, with common etiologies for readmission being abdominal pain (37%), sepsis (25%), gastric outlet obstruction (4%), and feeding tube dysfunction (4%). On readmission, 127 patients (65%) required percutaneous/endoscopic/surgical intervention, 88 (46%) underwent necrosectomy, and 15 (8%) required ICU stay. Readmitted patients were at higher risk for mortality at 6 months (25% vs. 6%, P=0.04). Significant independent predictors of 30 day readmission on multivariable analysis were length of stay (LOS) ≥ 14 days at outside hospital (OSH) prior to transfer [AOR 2.16 (1.12 – 4.18), P= 0.02], need for enteral nutrition on discharge [OR 1.87 (1.08 – 3.24), P= 0.03], and size of necrotic collection ≥ 6 cm [OR 1.99 (1.04 – 3.80), P= 0.04].

Conclusion: Readmission following NP is common (40%) and is associated with greater mortality at 6 months. Expedited transfer to tertiary center for timely care and intervention, assiduous follow up of other high risk patients (large collection and those who need enteral nutrition) could avoid readmissions and optimize outcomes.

	Readmitted within 30 days N=201	Non-readmitted N=304	Odds Ratio (OR)	P-value
Demographics, *n (%)*				
Gender, male	137 (68.2)	208 (68.4)	1.01	*0.95*
Age, median (IQR)	50 (35-62.5)	50 (38-63)	0.61	*0.22*
Race, white	176 (87.6)	266 (87.5)	1.01	*0.98*
Etiology of pancreatitis				
Alcohol	67 (33.3)	122 (29.3)	0.75	*0.12*
Biliary	68 (33.8)	89 (29.3)	1.24	*0.28*
Hypertriglyceridemia	12 (6.0)	11 (3.6)	1.69	*0.21*
Idiopathic	23 (11.4)	52 (17.1)	0.63	*0.08*
Medication	6 (3.0)	6 (2.0)	1.53	*0.47*
Post-ERCP	13 (6.5)	10 (3.3)	2.03	*0.09*
Other	11 (5.5)	12 (4.0)	1.41	*0.42*
Obesity (BMI ≥ 30)	90 (44.8)	111 (36.5)	1.41	*0.06*
ASA grading ≥ 3	119 (59.2)	201 (66.1)	0.74	*0.11*
Charlson Comorbidity ≥ 4	31 (15.4)	51 (16.8)	0.90	*0.68*
BISAP, mean	1.52	1.59	0.76	*0.51*
Characteristics of disease severity, *n (%)*				
Transferred from OSH	117 (58.2)	161 (53.0)	1.24	*0.25*
Length of stay ≥ 14 days at OSH	31 (26.7)	27 (16.4)	1.86	***0.04***
Total length of stay, median in days (IQR)	11 (6-23)	10 (5-22)	1.09	*0.90*
SIRS on admission	139 (69.2)	223 (73.4)	0.31	*0.81*
Persistent SIRS	96 (47.8)	144 (47.5)	1.01	*0.95*
Need for ICU admission	57 (28.4)	83 (27.3)	1.05	*0.80*
Presence of				
Cardiovascular failure	37 (18.4)	42 (13.8)	1.41	*0.16*
Renal failure	15 (7.5)	17 (5.6)	1.36	*0.40*
Respiratory failure	62 (30.9)	91 (30.0)	1.04	*0.83*
Multiorgan failure	32 (15.9)	39 (12.8)	1.29	*0.36*
Persistent organ failure	53 (26.4)	80 (26.3)	1.00	*0.99*
Infected necrosis	100 (49.8)	130 (42.8)	0.75	*0.12*
Interventions during hospitalization, *n (%)*				
Endoscopic transluminal drainage (ETD)	83 (41.3)	132 (43.4)	0.92	*0.64*
Percutaneous drainage	49 (24.4)	64 (21.1)	1.21	*0.38*
Open Necrosectomy	10 (5.0)	16 (5.3)	0.94	*0.89*
Endoscopic Necrosectomy	59 (29.4)	89 (29.3)	1.00	*0.99*
LAMs placed during ETD	55/83 (66.3)	90/132 (68.7)	0.89	*0.71*
Discharge characteristics, *n (%)*				
Need for enteral nutrition	72 (35.8)	70 (23.0)	1.87	***<0.01***
Need for TPN	2 (1.0)	7 (2.3)	0.43	*0.30*
Tolerating PO intake	127 (63.2)	230 (75.7)	0.55	***<0.01***
Need for narcotics at time of discharge	125 (62.2)	194 (63.8)	0.93	*0.71*
Discharge destination, home	154 (76.6)	243 (80.0)	0.82	*0.37*
Imaging characteristics, *n (%)*				
Size of necrotic collection ≥ 6 cm *	143/189 (75.7)	169/267 (63.3)	1.80	***<0.01***
Extent of pancreatic necrosis, >30% *	87/172 (50.6)	136/252 (54.0)	0.87	*0.49*
Location of necrotic collection *				
Pancreatic	2/188 (1.1)	5/270 (2.2)	0.47	*0.35*
Peri-pancreatic	21/188 (11.2)	26/270 (9.6)	1.18	*0.59*
Both	164/188 (87.2)	238/270 (88.2)	0.92	*0.77*
Presence of				
Disconnected pancreatic duct	64 (31.8)	101 (33.2)	0.94	*0.75*
Portal vein thrombosis	10 (5.0)	21 (6.9)	0.71	*0.38*
Superior mesenteric vein thrombosis	8 (4.0)	11 (3.6)	1.10	*0.83*
Splenic vein thrombosis	21 (10.5)	35 (11.5)	0.90	*0.71*
Pseudoaneurysm	2 (1.0)	3 (1.0)	1.00	*1.00*
Ascites	136 (67.7)	185 (60.9)	1.37	*0.10*

30. NECROTIZING PANCREATITIS AMONG CHILDREN WITH OBESITY AND PANCREATIC STEATOSIS: A ONE YEAR REVIEW AT A HIGH-VOLUME CENTER

MA Khan, A Flood, C Gariepy, JM Grisham, C Heinzman, J Nathan, AJ Freeman
Nationwide Children's Hospital, The Ohio State University College of Medicine

Background: Necrotizing acute pancreatitis (NAP) is thought to occur in < 1% of children with acute pancreatitis (AP). We note an increase in its presentation, and thus aim to describe its presentation, course, complications and therapy.

Methods: We performed a retrospective study of children with NAP during the last 12 months at Nationwide Children's Hospital, identifying those with NAP based on INSPPIRE definition for AP and computed tomography (CT) imaging. Patient demographics, etiology, clinical course, complications, therapeutics, and outcomes were reviewed.

Results: Four patients (mean age 16.9 years; range 13.2-20.2 years) had NAP out of approximately 50 patients (8%) with AP during the same time frame. Mean BMI was 37 kg/m2 (range 30.5-43.7). Gallstones pancreatitis was the suspected etiology for 1 patient, Post-ERCP pancreatitis for 1 patient, and the other 2 were considered to be idiopathic. All patients underwent endoscopic ultrasound (EUS); all 4 patients were determined by EUS to have hyperechoic pancreatic parenchyma typical of pancreatic steatosis. Length of stay (LOS) ranged 25-33 days (mean 29 days), including re-hospitalization in 75%. Admission to the intensive care unit was necessary for 50% of the patients with NAP due to sepsis, fluid overload, and/or respiratory failure. Three patients developed walled-off necrosis, and required EUS-guided cystgastrostomy with lumen apposing metal stent placement (LAMS 20 mm lumen diameter) to facilitate drainage and/or subsequent necrosectomy; one patient also required percutaneous cystic drain as a portion of the collection was not initially amenable to endoscopic drainage. There were no surgical interventions or deaths. None had persistent pancreatic exocrine or endocrine insufficiency after recovery.

Conclusion: Among our cohort of patients with necrotizing pancreatitis, all were obese, had EUS-determined pancreatic steatosis, and most developed peri-pancreatic fluid collections requiring EUS-guided cystgastrostomy with LAMS placement. Further studies evaluating obesity and pancreatic steatosis are warranted in NAP and AP.

31. INTRAOPERATIVE PYLORIC BOTULINUM TOXIN INJECTION FOR POST TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION GASTROPARESIS IMPROVEMENT IN CHILDREN

J Gurria, C Lowe, T Jenkins, M Ogg, A Bondoc, G Tiao, D Vitale, M Abu-El-Haija
Cincinnati Children's Hospital Medical Center

Background: Acute Recurrent Pancreatitis (ARP) and Chronic Pancreatitis (CP) in children may cause severe recurrent abdominal pain, nausea, vomiting, significant malnutrition, opioid dependency, and impairment in mental health and quality of life. After medical and interventional therapy has been exhausted, surgical management plays a vital role. Total Pancreatectomy with Islet Autotransplantation (TPIAT) is a surgical option. Pancreatitis causes gastroparesis that could be potentiated by surgery. Botulinum toxin (BT) injections of the pylorus have been used in children for gastroparesis during endoscopic and surgical procedures to prevent its morbidity.

Methods: Single institution retrospective cohort study of 10 pre-BT and 10 post-BT injection patients who underwent TPIAT from 2021-2022 to evaluate the outcomes of intraoperative pyloric BT injections including: time to achieve full oral nutrition, weight difference between preop admission to 3 months post op, post op days with emesis, post op days when gastrostomy tube (G) required venting to drainage due to nausea/vomiting, and length of stay (LOS). We hypothesized that pyloric BT performed during TPIAT surgery will decrease the incidence of gastroparesis symptoms post-operatively.

Results: Both groups predominantly had a CP diagnosis (8 of 10), were similar by sex (BT, 40% male vs 50% in no BT). Bivariate assessment found that BT patients were older, had fewer days to achieve full nutrition by mouth, had fewer days when G tube had to be opened to allow drainage due to symptoms, had fewer emesis days, and had similar LOS and 3-month weight change compared to no BT patients (TABLE). After adjustment, BT patients had fewer G tube days ($p< 0.05$) and lower 3-month weight loss ($p=0.02$), but group differences with full nutrition and emesis days were not significant.

Conclusion: Intraoperative pyloric BT injection improves postoperative gastroparesis symptoms of patients undergoing TPIAT. This procedure is only performed at a few pediatric institutions and to our knowledge, this is the first pediatric study aimed to improve the significant morbidity caused by gastroparesis from this surgical approach on an already high-risk population with gastroparesis due to ARP or CP. Prospective data will be collected as part of the follow up to this pilot study.

Patient Characteristics & Outcomes by Botox Status.

	Botox=Yes	Botox=No	p-value
N	10	10	
Age at TPIAT (years)	15 (13, 17)	8.5 (4, 14)	0.019
Male	40% (4)	50% (5)	1.00
Diagnosis=CP	80% (8)	80% (8)	1.00
Genetics			
PRSS1	30% (3)	50% (5)	0.65
CFTR	60% (6)	60% (6)	1.00
SPINK1	20% (2)	0% (0)	0.47
CTRC	0% (0)	10% (0)	1.00
Other	20% (2)	0% (0)	0.47
Weight at admission (kg)	59.8 (52.5, 77.9)	37.8 (20.1, 61.3)	0.11
BMI percentile at admission	72 (68, 92)	88 (56, 97)	0.82
Weight at 3 months (kg)	59.3 (49.5, 70.7)	34.3 (20, 59.1)	0.05
Weight change at 3 months (kg)	-2.6 (-4.4, 0.2)	-2.6 (-4.3, -0.5)	0.68
Length of Stay (days)	16 (15, 19)	16 (15, 18)	0.93
Days to Full PO	24 (16, 29)	41.5 (28, 50)	0.027
GT Days thru POD 14	7 (4, 11)	11.5 (9, 13)	0.008
Emesis Days thru POD 14	0.5 (0, 3)	6 (2, 9)	0.024

* Values reported as median (Q1, Q3), or % (n).
Group comparisons made using Fisher's exact tests or Exact Wilcoxon tests.

32. BRINGING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION TO A TERTIARY CHILDREN'S HOSPITAL: ASSIMILATION THROUGH SIMULATION

C Heinzman, L Vohsing, C Camacho, T Maa, CE Gariepy, AJ Freeman, JD Nathan
Nationwide Children's Hospital

Background: Total pancreatectomy with islet autotransplantation (TPIAT) is a complex surgical procedure requiring intensive post-operative care and is offered at few pediatric hospitals nationwide to children with debilitating pancreatitis. Prior to introduction of a TPIAT program at our tertiary children's hospital, new workflows involving multiple care teams needed to be developed and tested to reduce preventable errors with potential for patient harm. Simulation provides opportunities to model these complex processes and learn in a safe and controlled environment.

Methods: The simulation, medical and surgical teams performed TPIAT-specific scenarios across various phases of care. The primary goal was to review patient management including the newly developed order sets and new equipment as well as corresponding policies and procedures. The secondary goal was to educate staff on these new processes. Onsite simulations using actual medical equipment typically available in the clinical environments were conducted on various hospital units including the primary admitting unit, operating room, pediatric intensive care, and stepdown units. Simulations were conducted with day and night shift staff, the surgeon, advanced nurse practitioner, nursing educator, nursing informatics, pharmacy, clinical leadership, endocrinology, and simulation team.

Results: Interprofessional simulations involving 65 participants occurred in July 2022. Multiple latent safety threats involving team communication, equipment, personnel, medications, and the electronic health record were identified. Standardized operating procedures were created for use of new continuous glucose monitoring technology. Safety concerns, risk assessment and mitigation plans were iteratively developed and tested in simulations.

Conclusion: When properly employed, simulation-based training allows opportunities to learn new skills and provide real-time feedback. In this patient population, our team used simulation-based learning to assimilate care teams to a complex patient population that suffers from debilitating pain from pancreatitis and are scheduled to undergo TPIAT. By involving stakeholders and frontline staff in simulation-based healthcare systems testing, we were able to successfully address concerns, answer questions, boost staff confidence and safely perform the institution's first TPIAT.

33. A NOVEL TEAM MODEL FOR DIABETES CARE IN PEDIATRIC PATIENTS UNDERGOING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION

JM Ladd, K Friesner-Gephart, K Gandhi, MJ Okafor, CA Heinzman, SK Rasmussen, CE Gariepy, AJ Freeman, JD Nathan
Nationwide Children's Hospital

Background: Total pancreatectomy with islet autotransplantation (TPIAT) is a complex surgical option offered to patients with chronic pain and reduced quality of life due to acute recurrent or chronic pancreatitis which is not responsive to other therapies. From an endocrine perspective, all patients have insulin-dependent post-pancreatectomy diabetes immediately following TPIAT. Although isolated islets are reinfused after pancreatectomy, usually into the portal vein, these islets are susceptible to apoptosis, and long term exogenous insulin independence is variable. As hyperglycemia affects islet survival, tight glycemic control in the immediate post-operative period and the months that follow is critical. Diabetes technology, including insulin pumps and continuous glucose monitors (CGMs), can greatly assist in glycemic control but are complex technology requiring intensive education and supervision.

Methods/Results: The Diabetes/Insulin Inpatient Service Consult (DIISC) team is an innovative branch of the Endocrinology division in our large, tertiary care pediatric academic center. Pre-operatively, after the initial physician assessment for pediatric TPIAT candidates, the DIISC advanced practice practitioners and diabetes educator take a lead role in providing new-onset diabetes education including hands-on technology skills over several weeks. During the latter half of the hospitalization following TPIAT, the DIISC team provides 24/7 in-house support for these patients and collaborates with multiple disciplines to provide comprehensive and optimal diabetes care. The DIISC team also guides 48 hours of care during which time families primarily manage pumps and CGMs in preparation for home-going. After hospital discharge, in addition to regular physician clinic visits, the DIISC team proactively follows up with families to make frequent insulin regimen adjustments to maintain euglycemia and to address questions related to post-TPIAT diabetes care.

Conclusion: The DIISC team is a unique, innovative component of the pediatric TPIAT team at our institution and is integral in maintaining tight glycemic control. Future studies are planned to assess glycemic outcomes and long term insulin independence in our center under this novel diabetes care model. Additionally, future research will evaluate comfort level with diabetes technology and diabetes-related quality of life in pediatric TPIAT patients at our center.

34. ISLET CELL AUTOANTIBODY FORMATION AND ITS ASSOCIATION WITH BETA CELL FUNCTION FOLLOWING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION (TPIAT)

A Lavik, C Lowe, S Tellez, L Hornung, M Abu-El-Haija, D Elder
Cincinnati Children's Hospital Medical Center

Background: The function of transplanted beta cells following TPIAT range from sufficiently intact function that allows for insulin independence to negligible function requiring high dose daily insulin (>0.5 units/kg/day). Markers of pancreatic islet cell autoimmunity are routinely used in children to predict risk of type 1 diabetes, but studies evaluating these markers in children following TPIAT are lacking. Similarly, the association between islet cell autoimmunity and beta cell function in children following TPIAT is unknown.

Methods: Children who completed TPIAT at our center between 2015 and 2021 were eligible for inclusion. Individuals were screened for islet cell autoantibodies (Glutamic Acid Decarboxylase (GAA), Islet Antigen-2 (IA-2), Insulin (IAA), or Zinc Transporter 8 (ZnT8)) prior to and every six months following TPIAT. From a total of 23 patients, we identified 7 patients who were positive for GAA and/or IA-2 within 18 months of TPIAT and 16 patients who remained negative for all four islet autoantibodies. Given that patients are placed on exogenous insulin of varying degrees post-TPIAT we didn't count the presence of IAA as positive. The GAA/IA-2 antibody-positive patients' BMI percentile was significantly higher than the negative group (see Table; 78.6 vs 49 (p=0.04)). We compared metrics of stimulated beta cell function by mixed meal test (MMT) between groups.

Results: We found GAA/IA-2 antibody-positive patients produced significantly more C-peptide and endogenous insulin in response to MMT than islet antibody-negative patients (see Table; Peak C-peptide 3.2 vs 1.6 (p=0.01); AUC C-peptide 144 vs 62 (p=0.02); C-peptide Secretion Index 0.04 vs 0.02 (p=0.02); Insulinogenic Index 0.9 vs 0.4 (p=0.003)). This difference in C-peptide and endogenous insulin persisted regardless of whether patients were receiving exogenous insulin therapy. After controlling for BMI percentile (p=0.31), insulinogenic index was still significantly higher in the GAA/IA-2 positive group (p=0.01). There was no difference in glucose metrics between groups.

Conclusion: In summary, MMT performed 12 months post-TPIAT, GAA/IA2 positive patients released significantly more endogenous insulin even after controlling for BMI percentile in order to achieve similar glucose control to those negative for islet autoantibodies. This observation suggests islet cell autoantibodies may contribute to beta cell dysfunction following TPIAT.

MMT within 18 months: Islet positive vs negative groups

	Positive GAA/IA2 N=7	All negative* N=16	P-value
TPIAT age (years)	11.1 (9.6-15.1)	7.1 (4.4-12.9)	0.15
Sex (male)	3 (43%)	10 (63%)	0.65
BMI percentile	78.6 (67.9-91.6)	49.0 (16.4-67.3)	**0.04**
Off insulin **On insulin** **TDD/kg**	5 (71%) 2 (29%) 0.42 (0.04-0.80) *n=2*	8 (50%) 8 (50%) 0.17 (0.05-0.35) *n=8*	0.41
HbA1c (%)	5.7 (5.5-6.0)	5.6 (5.5-5.9)	0.57
Fasting C-peptide (ng/mL)	1.2 (0.7-1.5)	0.5 (0.4-0.9)	0.07
Peak C-peptide (ng/mL)	3.2 (2.4-3.6)	1.6 (1.3-2.2)	**0.01**
Fasting glucose (mg/dL)	94 (89-105)	92 (87-99)	0.42
AUC glucose	2220 (1260-2925)	2078 (1185-2985) *n=14*	0.82
AUC C-peptide	144 (98-183)	62 (42-84) *n=15*	**0.02**
Insulinogenic Index	0.9 (0.6-1.2) *n=6*	0.4 (0.3-0.4) *n=14*	**0.003**
C-peptide Secretion Index	0.04 (0.04-0.06)	0.02 (0.02-0.03) *n=14*	**0.02**
Disposition Index: C-peptide	0.04 (0.02-0.05)	0.04 (0.02-0.06) *n=14*	0.68
Proinsulin **<1.6** **=1.6**	 2 (29%) 5 (71%)	 10 (62.5%) 6 (37.5%)	0.19

Data is presented as median (25^{th}-75^{th} percentile).

Insulinogenic Index = Δ Insulin $_{30\text{-}0\ min}$ / Δ Glucose $_{30\text{-}0\ min}$

C-peptide Secretion Index = Δ C-peptide $_{30\text{-}0\ min}$ / Δ Glucose $_{30\text{-}0\ min}$

Disposition Index: C-peptide = Δ C-peptide $_{30\text{-}0\ min}$ / Δ Glucose $_{30\text{-}0\ min}$ x 1/fasting C-peptide

* All negative – never positive (for any of the 4 islets) at any point less than 18 months post-TPIAT

35. RATIO AND DOSE OF RC1: RC2 COLLAGENASES BASED ON PERCENT PARENCHYMAL MASS IMPROVES ISLET YIELD INTENDED FOR PEDIATRIC ISLET AUTO-TRANSPLANTATION

KK Samaga, AA Kodipad, S Narayanan, JD Nathan, AN Balamurugan
Department of Pediatrics and Surgery, Center for Clinical and Translational Research
Nationwide Children's Hospital / Ohio State University

Background: Digesting chronic pancreatitis pancreases (CPP) during islet isolation is challenging The dose and composition of the collagenase enzyme blends (CEB) used in the islet isolation process is a critical. In this study, recombinant Clostridium histolyticum class I (rC1) and class II (rC2) collagenase on human pancreas digestion efficacy in combination with P. polymyxa protease (BP-protease) enzyme blend (rCEB) was assessed.

Methods: No animal components were used in the generation of the raw material or purification of rC1, rC2, or BP-Protease. In our initial studies, we tested rCEB in 22 human research pancreases utilizing split lobe and full lobe models. In the current study, we introduced rCEB for clinical islet auto-transplantation (n=5) we also tested this enzyme blend in two validation islet isolations (n=2) before introduced in clinical. The unit of collagenase used was >19 Wunsch units/gram pancreas (20- 40 unit) and >14.000 protease unit/gram pancreas (15,000-53,000 unit). Dosing of rCEB was done based on the assessment of percent parenchymal tissue mass in each chronic pancreatitis pancreas. rCEB enzyme blend was injected into the pancreatic duct and digestion was done in Ricordi chamber. Pancreas digestion profiles (islet number, IEQ, islet/acinar morphology, tissue pellet volume, undigested pancreas) were carefully monitored and compared to isolations performed with traditional enzyme formulations.

Results: The rCEB efficacy and ratios were identified followed by islet functions were carefully monitored before introduced to clinical islet auto-transplantation. Assessing percent parenchymal mass by visual and histology methods guided us to select the correct enzyme dosing. Thedigestion efficacy of rCEB in all five isolations were >90%, the undigested portion of pancreases were < 10%. The pancreasdigestion profiles were normal, and the final islet viability was >70% in all preparations. The final islet per gram pancreaswas comparable to our previous islet isolation performed with traditional collagenase enzyme blends.

Conclusion: Dosing rCEB based on percent parenchymal mass successfully digested the entire fibrotic pancreas and released all islets. Purified P. polymyxa protease in combination with rC1rC2 collagenases can be effectively applied to digest fibrotic chronic pancreatitis pancreases for maximizing islet yield intended for clinical islet auto-transplantation.

36. CHANGES IN PORTAL PRESSURE, BLOOD PRESSURE AND GLUCOSE LEVELS DURING ISLET INFUSION: CLOSE MONITORING FOR EVERY FIVE MINUTES DURING AUTO-TRANSPLANTATION

AA Kodipad, CB Revanna, BN Appakalai, JD Nathan
Department of Pediatrics and Surgery, Center for Clinical and Translational Research
Nationwide Children's Hospital / Ohio State University

Background: Chronic Pancreatitis (CP) is a life debilitating illness in children impairing quality of life and it is associated with incapacitating pain, repeated hospitalizations, and an elevated risk of narcotic dependence. Pediatric patients with severe and intractable chronic pancreatitis, total pancreatectomy (TP) and islet auto-transplant (IAT) may be undertaken. Introducing islets through the portal vein make it susceptible to elevation of portal pressures, portal thrombosis, or bleeding. Close monitoring of portal vein pressure (PVA), CVP, blood pressure for every five-minute during islet infusion by using a computerized manometer placed inline through a three-way stopcock and simultaneous monitoring of blood glucose, and ACT may prevent portal vein thrombosis. This is the first study reporting portal pressure monitoring every five minutes during islet infusion in pediatric patients.

Methods: Pediatric patients underwent total pancreatectomy followed by islet auto-transplantation (n=25). Human islets were isolated following young donor islet isolation protocol for CP pancreas. Unpurified islets were infused into portal vein and PVA, CVP, blood pressure, blood glucose, and ACT were monitored for every five minutes throughout the islet infusion in operating room.

Results: Blood glucose (BG) levels were well controlled (below 180mg/dL) during islet infusion due to close monitoring of BG every five minutes. Drop in BG levels due to dumping insulin from infused islets was not observed. The average pancreas weight was 46 grams. Patients received a mean islet dose of 6368 IEQ per kg body weight, corresponding to a tissue volume of 9.4ml. Elevation in portal pressure was observed during infusion of >0.25ml tissue/kg body weight of recipient. Every five-minute monitoring allowed us to carefully assess portal vein pressure (PVA), CVP, blood pressure, blood glucose and ACT levels and control their levels.

Conclusion: This is the first report, monitored several key parameters for every five-minute during islet cell infusion and improved our understanding of blood glucose, blood pressure and portal pressure changes. This method of slow infusion and frequent monitoring helped to prevent adverse reaction.

37. ADHERENCE TO VACCINE RECOMMENDATIONS FOR PATIENTS POST SPLENECTOMY AFTER TOTAL PANCREATECTOMY ISLET AUTOTRANSPLANTATION

A Turner, M Ogg, C Lowe, C Kohlrieser, M Abu-El-Haija
Cincinnati Children's Hospital Medical Center

Background: The post splenectomy patient is at increased risk of rapidly progressing sepsis due to bacteria and encapsulated organisms. Overwhelming Post Splenectomy Infection (OPSI) is associated with a high mortality rate up to 50 %. Fever in a patient without a spleen is considered a medical emergency. The spleen pays an important role in the immune system response. Prevention of OPSI include education, vaccination, and prophylaxis therapy. The vaccination guidelines for patient at high risk due to asplenia can be cumbersome and questioned by healthcare providers which further confuses patients and families. Overall vaccination adherence of the asplenic patient is estimated to be at 15%. Prevention requires active engagement of both patients and the health care team to ensure adherence to vaccine recommendations.

Methods: The purpose of this project is to implement a process and strategies that increase vaccination rate of patients with acquired asplenia after a Total Pancreatectomy Islet Autotransplantation. This project utilized the improvement process Plan Do Study Act (PDSA) cycles to plan, test, and evaluate strategies. The project utilized existing practice standards for vaccination of high-risk populations and the Electronic Medical Record (EMR) to generate letters to obtain updated vaccine records, perform existing pre-visit planning and collaborate with primary care providers. Vaccination records were obtained in advance of specialty clinic visits and pre-visit planning was completed. Vaccination records were reviewed by infectious disease team and vaccinations were given if indicated. Vaccinations that were due between interval office visits were communicated with the family and primary care provider. Data was collected before and after improvements via chart review.

Results: By using the strategies to increase vaccination of the asplenic patient, there was an increase of vaccines given during the clinic visit from 16% to 75%. Vaccination records obtained prior to the visit was increased from 42% to 76%.

Conclusion: The project simplified vaccination adherence tracking by utilizing the EMR to efficiently implement strategies to collaborate with the health care team. Further work needs to be done to facilitate long term vaccination adherence by empowering families and patients through guidance and education.

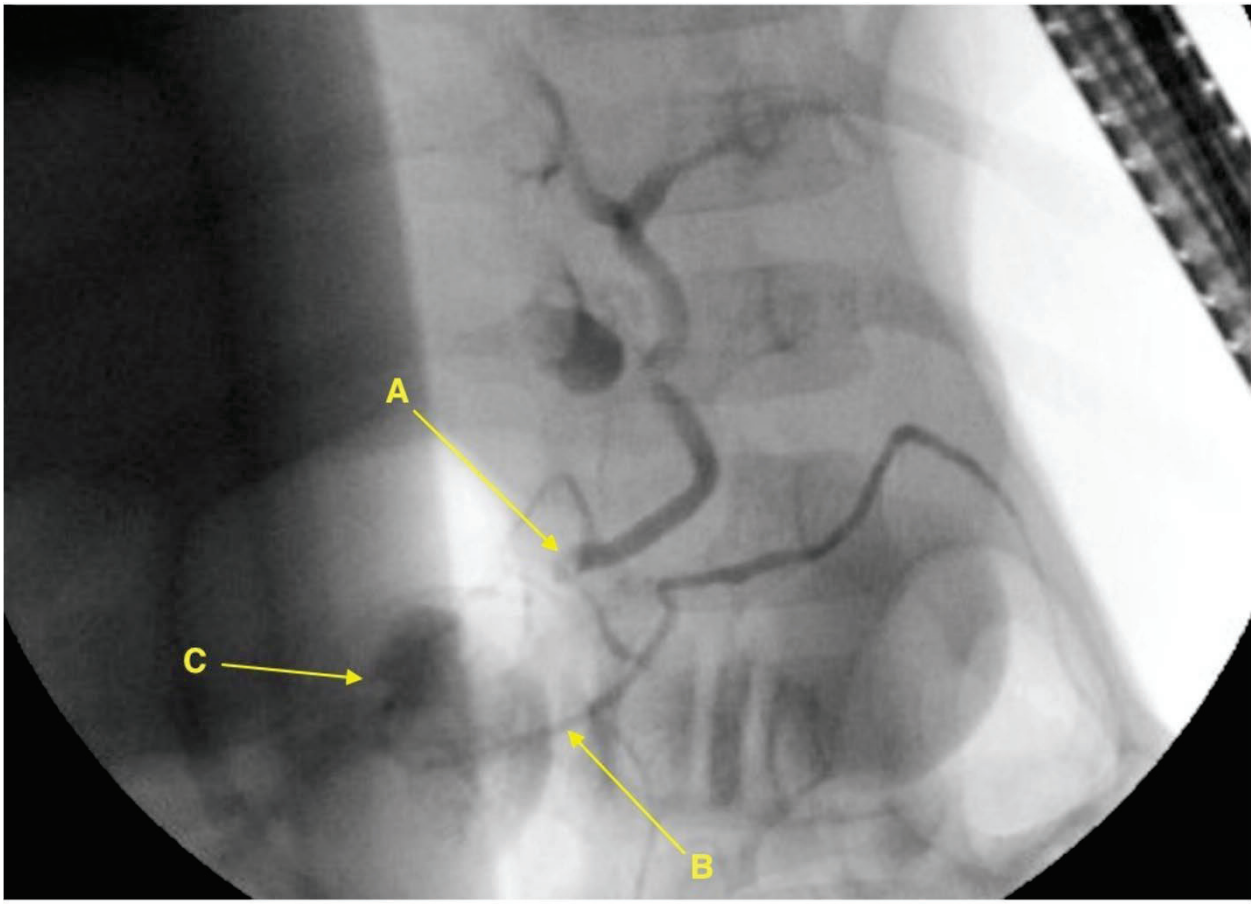
38. DUODENAL DUPLICATION CYST AS A RARE CAUSE OF PANCREATITIS IN A CHILD

MA Colak, L Pruitt, C Heinzman, SK Rasmussen, CE Gariepy, DS Vitale, A Trout, JD Nathan
Nationwide Children's Hospital

Background: Duodenal duplication cyst (DDC) is a rare congenital malformation of the gastrointestinal tract with estimated incidence of < 1/100,000 live births. DCC typically presents with abdominal pain, nausea/vomiting, or pancreatitis. We report a case of DDC discovered in a 2-year-old boy causing acute pancreatitis.

Case: A 1-year-old boy with history of preterm birth (26 weeks) complicated by IUGR/oligohydramnios presented with abdominal pain and poor weight gain. Upper gastrointestinal series demonstrated an intraduodenal soft tissue impression in the second portion of the duodenum (D2). Esophagogastroduodenoscopy showed a well-defined cystic lesion protruding into the duodenal lumen. Diagnostic laparoscopy and intraoperative endoscopy failed to identify the mass/cyst. Abdominal pain and feeding issues resolved without need for enteral feeding. At two years old, he presented with abdominal pain, and CT revealed a 1.6 cm well-circumscribed cystic intraduodenal lesion. MRCP demonstrated an intraluminal cystic lesion in D2 with features suggestive of type III choledochal cyst. He was referred to hepatopancreatobiliary surgery but failed to attend clinic. He developed recurrent epigastric pain with lipase elevated at 1741 U/L (ref range < 202 U/L). Further questioning revealed complaints of similar abdominal pain 6 months prior. Endoscopic ultrasound (EUS) and ERCP were performed to define mass etiology and anatomic relationships. EUS showed a 1.8 cm cystic lesion in D2 distal to the ampulla with a double wall sign, typical of DDC. ERCP (Figure) showed an aberrant pancreatic duct branch inserting into the cyst with otherwise normal biliary and pancreatic ducts. He underwent transduodenal partial cystectomy as definitive management with copious debris evacuated from within the cyst. Portions of the cyst wall were left in situ due to proximity to the aberrant pancreatic duct. Microscopic examination of the cyst revealed duodenal tissue, confirming a DDC. During 8-month follow-up, he continues to grow well on all oral diet without any subsequent episodes of pancreatitis.

Conclusion: DDC can be a cause of pancreatitis, particularly when the cyst is in proximity to drainage of the pancreatic duct. Insertion of an aberrant pancreatic duct into a DDC is a unique finding, in which obstruction of drainage by intracystic debris can result in pancreatitis.



Arrow A: Ampulla
Arrow B: Aberrant pancreatic duct
Arrow C: Cyst filled with contrast

39. IS NON-ETHANOL BASED ABLATION THERAPIES BETTER THAN ETHANOL ABLATION FOR PANCREATIC CYSTS?: A SYSTEMATIC REVIEW AND META ANALYSIS.

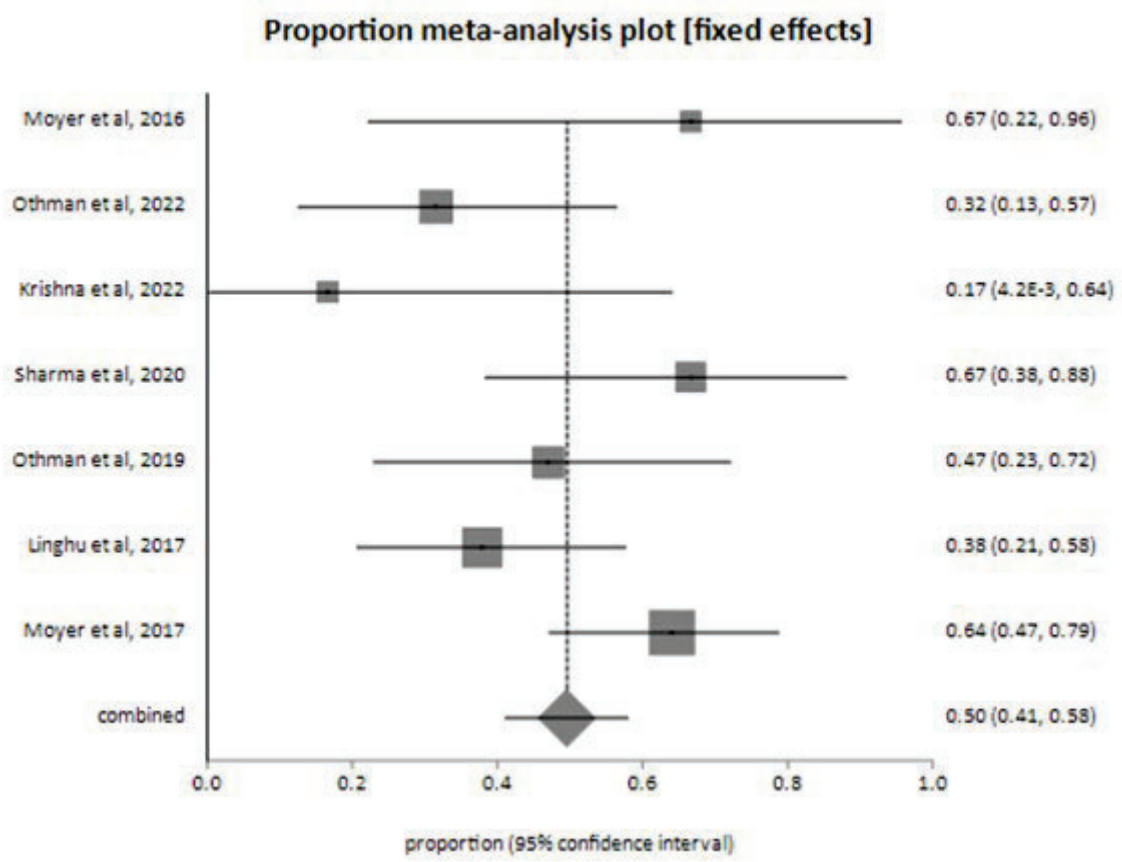
Y Reddy, N Singh, L Chawla, S Puli, S Dhillon
University of Illinois College of Medicine

Background: Endoscopic Ultrasound (EUS) guided Ethanol Ablation of the pancreatic cyst has been used as an alternative for surgery in recent years. In this meta-analysis we compare the outcomes of pancreatic cysts ablation with ethanol based ablation therapy versus non ethanol based ablation therapies.

Methods: Data was collected and extracted from Medline, Pubmed, and Ovid journals. Statistical analysis was done using Fixed and random effects models to calculate the pooled proportions.

Results: Data was extracted from 19 studies (n=609) which looked at EUS guided ablation of pancreatic cysts. Of the 19 studies, 8 (n=390) studies used EUS guided ethanol ablation. There were 4 (n=88) studies that used ethanol with a paclitaxel combination for ablation, and 7 studies (n=131) used non ethanol based ablations alone. The pooled proportion of patients with complete cyst resolution in the ethanol group was 61.11% (95% CI = 56.25 to 65.86), ethanol with paclitaxel group was 54.34% (95% CI = 44.03 to 64.46), and non ethanol group was 49.59 % (95% CI = 41.19 to 58.01). Procedure related complications included pancreatitis that was noted in a pooled proportion of 8.08% (95% CI = 5.51 to 11.11) in the ethanol group that was relatively higher compared to 5.82% (95% CI = 1.95 to 11.56) in the ethanol with paclitaxel group, and 3.91% (95% CI = 1.31 to 7.83) in the non ethanol group. Publication bias calculated using Harbord-Egger bias indicator gave a value of 2.3 (p = 0.09). The Begg-Mazumdar indicator gave Kendall's tau b value of 0.28 (p = 0.39).

Conclusion: EUS guided pancreatic cyst ablation is an alternative therapy for non surgical candidates. This study showed that complete cyst resolution was comparable in patients with ethanol and non ethanol ablation. Procedural adverse events were very minimal in all the treatment groups suggesting that pancreatic cyst ablation is safe.


Proportion meta-analysis plot [fixed effects]
Moyer et al, 2016
Othman et al, 2022
Krishna et al, 2022
Sharma et al, 2020
Othman et al, 2019
Linghu et al, 2017
Moyer et al, 2017
combined
0.67 (0.22, 0.96)
0.32 (0.13, 0.57)
0.17 (4.2E-3, 0.64)
0.67 (0.38, 0.88)
0.47 (0.23, 0.72)
0.38 (0.21, 0.58)
0.64 (0.47, 0.79)
0.50 (0.41, 0.58)
0.0
0.2
0.4
0.6
0.8
1.0
proportion (95% confidence interval)

40. CAN EUS-ABLATION REVOLUTIONIZE THE MANAGEMENT OF PANCREATIC CYSTS? A META-ANALYSIS OF SHORT-TERM OUTCOMES

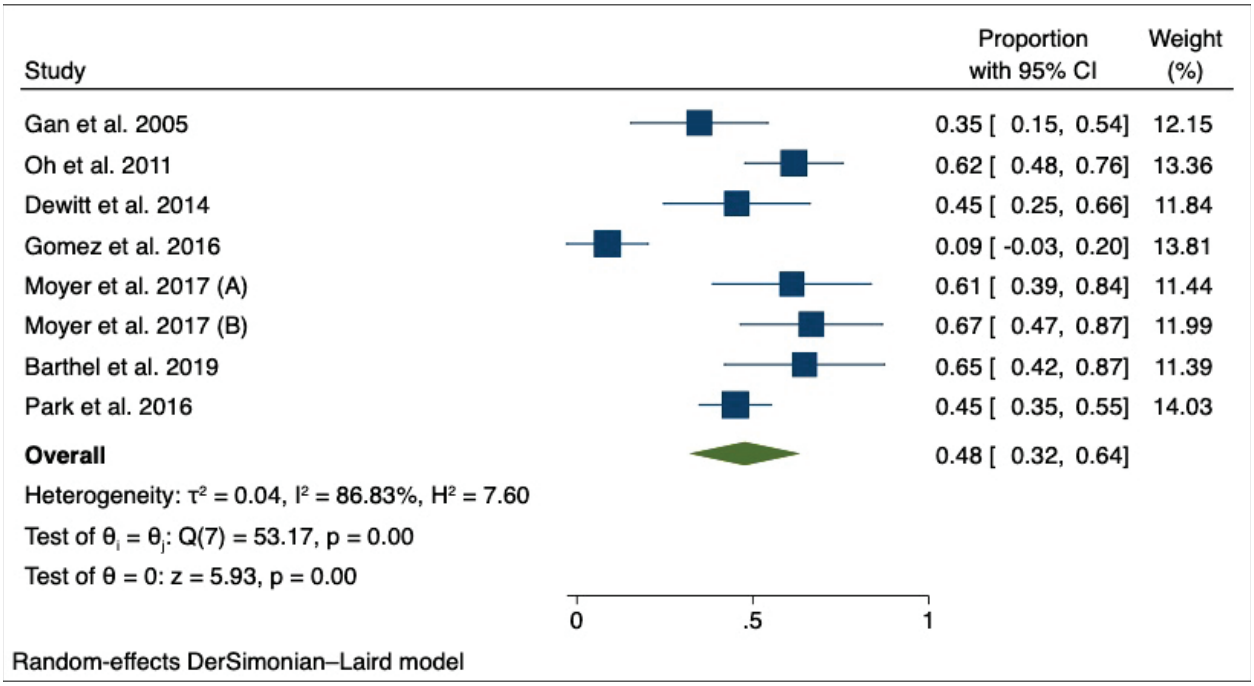
A Salih, J Emran, S Menakuru, M Salih, D Harris, D Echols, B Ji, Y Bi
Indiana University School of Medicine

Background: Management of pancreatic cysts remains challenging. Stratification of cyst malignancy potential needs improvement. Whilst surgical resection of the pancreas carries significant morbidity and mortality, cyst ablation is a promising option for cyst management. We conducted a systematic review and meta-analysis to study the efficacy and safety of endoscopy ultrasound (EUS) guided pancreatic cyst ablation.

Methods: A systematic literature search was conducted on clinical trials of EUS pancreatic cyst ablation and reported following the recommendation of Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) guidelines. The outcomes were the cysts' resolution on imaging at 12 months and the presence of complications. Trials that studied neuroendocrine cysts or followed the patients for less than 12 months were excluded. The inverse-variance method was used to calculate the weighted pooled proportion under the random effect model.

Results: The initial search identified 6,274 reference studies, from which 182 articles were selected and reviewed. Seven studies met our inclusion criteria with a total of 262 patients. Among them, 175 (67%) were females. Pancreatic mucinous cysts reflected 44% of all cysts (116), serous cystadenomas were 8% (22), while 46% (121) were indeterminates. The mean diameter of the cysts was 26.89mm and ranged from (1.55-68mm). A total of 104 cysts were located in the head of the pancreas, 84 in the body, and 51 in the tail. EUS was used to ablate cysts in all patients. Alcohol was used in 52% (137) of cases, Paclitaxel and alcohol in 26% (69), Paclitaxel and Gemcitabine in 6.8% (18), and Paclitaxel and Gemcitabine and alcohol in 8% (21). About 6.4% (17) of cysts received radiofrequency ablation. The pooled proportion for complete cyst resolution on imaging after 12 months of EUS ablation was 48% [95% CI 32, 64, I2=86.83%]. shows the individual study proportions and the pooled estimate. Partial cyst resolution was 27% [95% CI 9, 45, I2=93.88%], and cyst persistence was 19% [95% CI 11, 26, I2=55.34%].

Conclusion: Our meta-analysis supports EUS-PCA as a viable option for managing pancreatic cysts, with acceptable complication rates. Further randomized controlled trials comparing EUS-PCA to conventional surgery are needed.


Study
Proportion with 95% CI
Weight (%)
Gan et al. 2005
0.35 [0.15, 0.54]
12.15
Oh et al. 2011
0.62 [0.48, 0.76]
13.36
Dewitt et al. 2014
0.45 [0.25, 0.66]
11.84
Gomez et al. 2016
0.09 [-0.03, 0.20]
13.81
Moyer et al. 2017 (A)
0.61 [0.39, 0.84]
11.44
Moyer et al. 2017 (B)
0.67 [0.47, 0.87]
11.99
Barthel et al. 2019
0.65 [0.42, 0.87]
11.39
Park et al. 2016
0.45 [0.35, 0.55]
14.03
Overall
0.48 [0.32, 0.64]
Heterogeneity: τ² = 0.04, I² = 86.83%, H² = 7.60
Test of θi = θj: Q(7) = 53.17, p = 0.00
Test of θ = 0: z = 5.93, p = 0.00
0
.5
1
Random-effects DerSimonian–Laird model

41. A DNA/RNA-BASED NGS PLATFORM TO IMPROVE THE CLASSIFICATION OF PANCREATIC CYSTS AND DETECTION OF PANCREATIC CANCER ARISING FROM PANCREATIC CYSTS

M Nikiforova, A Wald, D Spagnolo, M Melan, M Grupillo, Y Lai, R Brand, A O'Broin-Lennon, K McGrath, W Park, P Pfau, P Polanco, N Kubiliun, J DeWitt, J Easler, A Dam, S Mok, M Wallace, V Kumbhari, B Boone, W Marsh, S Thakkar, K Fairley, E Afghani, Y Bhat
UPMC

Background: Despite a multidisciplinary approach, the classification of pancreatic cyst type, such as a cystic precursor neoplasm (IPMNs, MCNs, and IOPNs), and detection of high-grade dysplasia and early adenocarcinoma (advanced neoplasia) can be challenging. Next-generation sequencing of preoperative pancreatic cyst fluid improves the clinical evaluation of pancreatic cysts, but the recent identification of novel genomic alterations, such as gene fusions, necessitates the creation of a comprehensive panel and the development of a genomic classifier to integrate the molecular results.

Methods: A 74-gene DNA/RNA-targeted NGS panel (PancreaSeq Genomic Classifier) was created to evaluate 5 classes of genomic alterations to include gene fusions and gene expression. Further, CEA mRNA (CEACAM5) was integrated into the assay using RT-qPCR. Separate multi-institutional cohorts for training (n=108) and validation (n=77) were tested and justified based on statistical power analysis, and diagnostic performance was compared to clinical, imaging, cytopathologic, and guideline data.

Results: Upon creation of a genomic classifier system, PancreaSeq GC yielded a 95% sensitivity and 100% specificity for either an IPMN, MCN, or IOPN, and the sensitivity and specificity for advanced neoplasia were 82% and 100%, respectively. In comparison, high-risk stigmata/worrisome features, such as associated clinical symptoms, large cyst size (>3.0 cm), main pancreatic duct dilatation, the presence of a mural nodule, increasing cyst size, and malignant cytopathology, had lower sensitivities (41-59%) and lower specificities (56-96%) for advanced neoplasia. Applying the AGA and the IAP/Fukuoka guidelines to the entire cohort yielded sensitivities and specificities of 64% and 66%, and 77% and 40%, respectively. However, incorporating PancreaSeq GC into the AGA and the IAP/Fukuoka guidelines increased the sensitivity to 91% and 95%, respectively, while maintaining the inherent specificity of each guideline. Finally, the inclusion of gene expression analysis, such as chromogranin A (CHGA), for pancreatic neuroendocrine tumors (PanNETs) had 100% sensitivity and 99% specificity for pancreatic neuroendocrine tumors (PanNETs), while cytopathology yielded an 83% sensitivity and 100% specificity.

Conclusion: Combined DNA/RNA NGS was not only accurate in predicting pancreatic cyst type (e.g., IPMNs and PanNETs) and advanced neoplasia, but also improved the sensitivity of current pancreatic cyst guidelines.

42. TRENDS AND IMPACT OF EUS UTILIZATION FOR PANCREATIC CYSTS OVER TIME

L Wang, F Gleeson, M Levy, S Majumder, P Vatsavayi, S Umar, S Velaga, E Rajan, B Abu Dayyeh, A Storm, W Harmsen, S Vege, V Chandrasekhara
Mayo Clinic Rochester

Background: Neoplastic pancreatic cysts (PC) are increasingly noted on cross-sectional imaging. Multiple management guidelines have been published to improve risk stratification and resource utilization. This study aims to evaluate trends in endoscopic ultrasound (EUS) utilization and agreement between pre-EUS cross-sectional imaging and EUS for specific PC features.

Methods: This single center retrospective cohort study included consecutive adults undergoing EUS for PC detected with CT/MR between 2013-2015 (Cohort 1) and 2018-2020 (Cohort 2). Patients without CT/MR and those undergoing EUS for non-PC indications were excluded. Clinical, imaging, cytologic and outcomes data were collected. PC were defined as high-risk (having any Fukuoka high risk (HR) or worrisome feature (WR)), low risk (cyst size 10-29mm, without any HR or WR), and very low risk (cyst size < 10mm, without any HR or WR). Chi-square, Fisher's exact test, and Wilcoxon rank-sum test were used to compare categorical and continuous variables. P< 0.05 was significant.

Results: Of 203 included patients, 95 (46.8%) were in Cohort 1. Pre-EUS CT was preferred in Cohort 1 (62.1% vs 37.9%, P=0.046), versus MRCP/MRI in Cohort 2 (51.9% vs 48.1%, P=0.046). More patients in Cohort 1 underwent EUS for non-high-risk PC on pre-EUS imaging (68.4% vs 40.7%, P< 0.001), and fewer had a high-risk PC detected on EUS (22.1% vs 43.5%, P=0.006) (Table 1). Agreement between CT/MR and EUS for absence or presence of any WR was 79.3%, and 97% for any HR (Table 2). EUS refutation of a high-risk PC (P=0.20) or upgrade of a non-high-risk PC (P=1.0) was similar over time. FNA impacted diagnosis and management in 10 patients (15.9%), without difference over time (P=0.49). Median surveillance duration was 1124 days (IQR 352, 1522 days), with pancreatic adenocarcinoma diagnosed in 3.4% of patients and pancreatic neuroendocrine tumor in 3.9%, similar between cohorts (P=0.89 and P=0.79). Surgical referral was higher in Cohort 2, with trend towards significance (26.9% vs 18.8%, P=0.06).

Conclusion: In this series, utilization of EUS for low-risk PC decreased over time. A trend towards increased surgical referral for EUS-confirmed HR or WR was seen. These data suggest recent guidelines have improved EUS resource utilization for PC.

Table 1: Comparison of Baseline Characteristics and Pancreas Cyst Features in Patients undergoing EUS Over Time

			COHORT 1 (2013-2015) *(N = 95)*	COHORT 2 (2018-2020) *(N = 108)*	P-value
Age, median (IQR), years			67 (IQR 58, 73)	67 (57, 74)	0.94
Male gender			55 (57.9%)	49 (45.4%)	0.08
Genetic Mutation and Syndromes	Genetic testing not performed		87 (91.6%)	99 (91.7%)	0.64
	Genetic Testing Performed	No high-risk mutation identified on genetic testing	3 (37.5%)	5 (55.6%)	
		High-risk mutation identified on genetic testing	5 (62.5%)	4 (44.4%)	
Serum CA 19-9, median (IQR), (U/mL)			21 (IQR 8, 39)	11 (IQR 6, 35)	0.29
Pancreas Cyst Risk* based on pre-EUS cross-sectional imaging	Very Low Risk		12 (12.6%)	5 (4.6%)	**0.0003**
	Low Risk		53 (55.8%)	39 (36.1%)	
	High-Risk		30 (31.6%)	64 (59.3%)	
Pancreas Cyst Risk* based on EUS	Very Low risk		14 (14.7%)	12 (11.1%)	**0.006**
	Low Risk		60 (63.2%)	49 (45.4%)	
	High-Risk		21 (22.1%)	47 (43.5%)	
FNA performed for fluid analysis and cytology during EUS			28 (29.5%)	35 (32.4%)	0.65
Outcomes	Surgical Referral		15 (15.8%)	29 (26.9%)	0.06
	Malignancy Diagnosed during Follow-up	PDAC	3 (3.3%)	4 (3.7%)	0.89
		PNET	4 (4.4%)	4 (3.7%)	0.79

EUS, *endoscopic ultrasound*; IQR, *interquartile range*; GI, *Gastroenterology*; FNA, *fine needle aspiration*; PDAC, *pancreatic ductal adenocarcinoma*; PNET, *pancreatic neuroendocrine tumor*

Definition of pancreatic cyst risk:* ***Very Low Risk* *(cyst <10mm, and not having any high risk or worrisome features as defined by the 2017 revised Fukuoka Guidelines);* ***Low Risk*** *(cyst 10-29mm, and not having any high risk or worrisome features as defined by the 2017 revised Fukuoka Guidelines;* ***High-Risk*** *(cyst having any high risk or worrisome feature as defined by the 2017 revised Fukuoka guidelines)*

Table 2: Pancreatic Cyst Features: Agreement between pre-EUS cross-sectional imaging and EUS

Pancreas Cyst Feature		Total Number (%) of patients with a specific pancreas feature on pre-EUS imaging	Total Number (%) of patients with a specific pancreas feature on EUS	Number of patients and overall % Agreement between pre-EUS cross-sectional imaging and EUS for presence and/or absence of specific cyst feature	Number of patients and overall % Disagreement between pre-EUS cross-sectional imaging and EUS for a presence and/or absence of a specific cyst feature
Worrisome Features	Cyst size ≥ 3cm	40 (19.7%)	34 (16.7%)	178 (87.7%)	25 (12.3%)
	MPD dilation 5-9mm	16 (7.9%)	18 (8.9%)	191 (94.1%)	12 (5.9%)
	Abrupt MPD caliber change with upstream pancreatic atrophy	7 (3.4%)	3 (1.5%)	197 (97.0%)	6 (3.0%)
	Cyst growth ≥5mm/2 years	30 (14.8%)	23 (11.3%)	194 (95.6%)	9 (4.4%)
	Enhancing mural nodule < 5mm	3 (1.5%)	1 (0.5%)	201 (99.0%)	2 (1.0%)
	Thickened cyst wall	6 (3.0%)	8 (3.9%)	197 (97.0%)	6 (3.0%)
	Any Worrisome Feature	90 (44.3%)	57 (28.1%)	161 (79.3%)	42 (20.7%)
High Risk Features	MPD dilation ≥ 10mm	6 (3.0%)	5 (2.5%)	202 (99.5%)	1 (0.5%)
	Enhancing mural nodule ≥ 5mm	0 (0.0%)	6 (3.0%)	197 (97.0%)	6 (3.0%)
	Any High-Risk Feature	6 (3.0%)	8 (3.9%)	197 (97.0%)	6 (3.0%)

Worrisome and high-risk pancreas cyst features were defined as per the revised 2017 Fukuoka guideline. Clinical pancreatitis attributed to cyst, obstructive jaundice, and enhancing cyst wall were excluded.

EUS, *endoscopic ultrasound*; cm, *centimeters*; MPD, *main pancreatic duct*; mm, *millimeters*

43. A NEW APPROACH TO EVALUATING THE MICROBIOME OF THE PANCREAS

MB Ghai, J Yang, J Aponte-Pieras, M Bayoumi, S Marsh, MP Celaya, F Zenhausern, W Wassef
University of Arizona College of Medicine Phoenix

Background: Chronic pancreatitis (CP) is an inflammatory-mediated condition with irreversible morphological changes leading to pancreatic endocrine and exocrine dysfunction. Disruptions in the microbial balance can be associated with many inflammatory-mediated pathologies including CP. The analysis of the microbiome in pancreatic disease has largely relied on using fecal or salivary samples. Few studies have directly analyzed duodenal fluid aspirate of pancreatic exocrine secretions. In this pilot study, we aimed to prove feasibility of using duodenal fluid aspirate and molecular profiling to characterize the pancreatic microbiome.

Methods: Included were adults who had already been referred for an endoscopic ultrasound (EUS) with secretin testing for work up of suspected CP. Those excluded were exposed to antibiotics within 30 days of EUS, undergoing chemotherapy, or had a history of Roux-en-y gastric bypass. Secretin was administered pre-procedurally. Using EUS, duodenal fluid was aspirated at time t= 0, 15-, 30-, 45-, and 60-minutes post-secretin administration. Microbial analysis was performed on isolated DNA with the IS-pro Microbiota kit and software. The microbial analyses were correlated to the secretin study results to identify participants with CP.

Results: A total of 28 participants were enrolled, 53.6% were found to have CP. Descriptive statistics are presented in Table 1. Of note, the average age of those with CP was higher than those without CP (without CP= 48.9 years, CP= 53.7 years). A history of smoking was reported by 46.2% of those without CP and 60% of those with CP. Microbial DNA isolated and sequenced from the duodenal aspirate of two participants demonstrated that the participant with CP had greater microbial diversity as compared to the participant without CP.

Conclusion: In this pilot study, we demonstrated that our method of collecting and preparing pancreatic exocrine secretions successfully allowed for the identification of bacterial species. We were also able to characterize differences in microbial diversity between those with and without CP. The combination of minimally invasive endoscopy to directly collect pancreatic secretions with a non-selective molecular culture assay may provide a fingerprint of the pancreatic microbiome that allows for a faster and more accurate CP diagnostic.

Table 1: Descriptive Statistics

		Without Chronic Pancreatitis	Chronic Pancreatitis
Enrolled participants, n(%)		13 (46.4%)	15 (53.6%)
Patient Characteristics			
Sex, n	Male	8	10
	Female	5	5
Mean Age, years		48.9	53.7
Race, n	Non-Hispanic White	9	9
	Black	1	4
	Hispanic White	1	1
	Asian or Pacific Islander	1	0
	Native American	0	0
	Other	1	0
	Unknown	0	1
Insurance Status, n	Medicare	5	6
	Medicaid	3	3
	Private	5	5
	Unknown	0	1
Current alcohol use, n	Yes	4	3
	No	7	11
	Unknown	2	1
History of smoking (>100 cigarettes), n	Yes	6	9
	No	7	5
	Unknown	0	1

44. CDX-7108: AN ENGINEERED GASTROINTESTINAL-STABLE LIPASE FOR THE ORAL TREATMENT OF EXOCRINE PANCREATIC INSUFFICIENCY

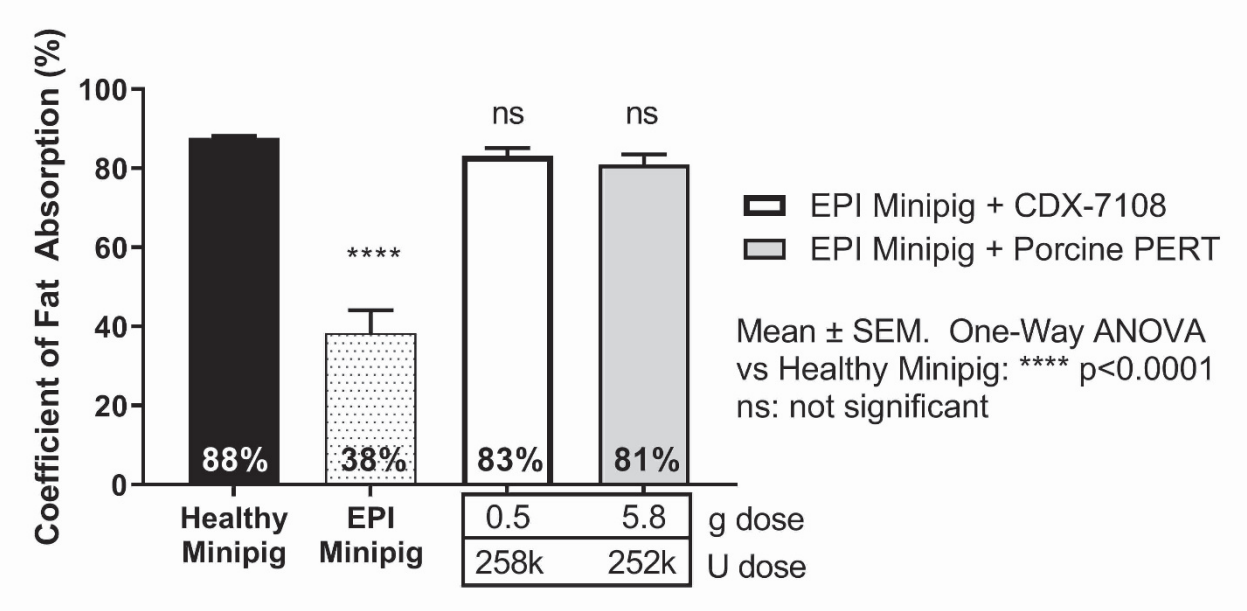
C Chng, R Garcia, WC Hallows, K McCluskie, N Kruse, A Lao, J Hang, B Sato, GW Huisman, Y Wang, AP Silverman
Codexis

Background: Exocrine pancreatic insufficiency (EPI) results when pancreatic secretions do not maintain normal digestive function, which can lead to malabsorption, malnutrition, vitamin deficiencies, and other symptoms significantly impacting health and quality of life. Oral porcine-derived pancreatic enzyme replacement therapy (PERT) is standard of care (SOC) for EPI. Though safe, the efficacy of porcine-derived PERTs are limited by lipase stability and activity, necessitating enteric coating and a high pill burden in many patients, sometimes ≥5 pills taken before, during, and after every meal. The pill burden leads to reduced compliance in many patients and persistence of morbidities.

Methods: To address patient need for a more effective PERT, we engineered a highly active and gastrointestinal-stable lipase enzyme that effectively metabolizes dietary fats within the gastrointestinal (GI) tract, without the need for enteric coating. Starting with a broad specificity bacterial lipase, we screened >13,000 variants over seven rounds of directed evolution to identify CDX-7108, a lipase with high stability in GI conditions including low pH, pepsin, and a bile acid while retaining broad activity in the intestine. To test the efficacy of CDX-7108, we created an minipig model of EPI through surgical ligation of the pancreatic duct.

Results: CDX-7108 showed remarkable stability to simulated gastric conditions, as well as high activity to a variety of natural lipid sources in simulated intestinal conditions. When compared in the EPI minipig model at an equivalent USP unit dose to porcine PERT, CDX-7108 achieved similar levels of correction of percent coefficient of fat absorption (%CFA) at a ~10x lower dose by mass. In subsequent studies evaluating lower dose units, CDX-7108 demonstrated significant %CFA improvements compared to EPI minipig control at doses as low as 56 mg (33.7 kU)/minipig/day ($p < 0.0001$).

Conclusion: These preclinical results highlight the ability of CDX-7108 to improve fat absorption in an EPI animal model with substantially reduced dosing requirements as compared to porcine pancreatin. CDX-7108 is presently being evaluated in the clinic (NCT05082051).


Coefficient of Fat Absorption (%)
100
80
60
40
20
0
ns
ns
****
88%
38%
83%
81%
Healthy Minipig
EPI Minipig
0.5
5.8
g dose
258k
252k
U dose
EPI Minipig + CDX-7108
EPI Minipig + Porcine PERT
Mean ± SEM. One-Way ANOVA
vs Healthy Minipig: **** p<0.0001
ns: not significant

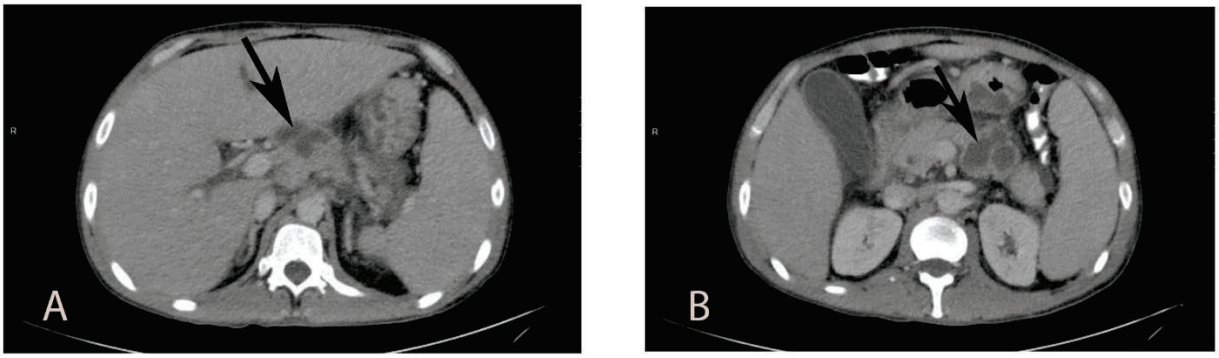
45. PANCREATIC TUBERCULOSIS IN A NEWLY DIAGNOSED HIV PATIENT: A RARE CASE REPORT

MR Shaik, NA Shaik, E Yunasan
University of Maryland Medical Center Midtown Campus

Background: Although the incidence of tuberculosis associated with HIV infection has risen in recent years, pancreatic tuberculosis has been infrequently reported. We present a case of pancreatic tuberculosis in a Hispanic male in the setting of a newly diagnosed HIV infection with acquired immunodeficiency syndrome (AIDS).

Case Presentation: A 38-year-old man from Honduras with no significant medical history presented with odynophagia, abdominal pain, and lower gastrointestinal bleeding of one week duration. He had also experienced significant weight loss and failure to thrive over the past three months. On physical examination, he appeared frail with oral thrush. Diagnostic tests showed that he was HIV-1 positive with a CD4 count of 11/mm3. A CT scan of his abdomen revealed multiple pancreatic collections. He was started on Rifampin-Isoniazid-Pyrazinamide- Ethambutol (RIPE) therapy, antiretroviral therapy, and fluconazole. AFB cultures of sputum were positive for mycobacterium tuberculosis complex. The patient started experiencing new onset fevers during the next week, and a repeat CTA revealed an increase in the size of the pancreatic collections. Despite broad antibiotic coverage, the patient did not improve, so an endoscopic ultrasound (EUS) was performed, which revealed three cystic lesions in the pancreatic head, genu, and tail. Fine needle aspiration of the cyst fluid was performed, which tested positive for AFB bacilli. With supportive management and RIPE therapy, the patient improved clinically over the next few days. He was scheduled for a follow-up with repeat abdominal imaging on discharge to monitor the cyst resolution.

Discussion: Pancreatic tuberculosis is rare and it is believed that the pancreas is naturally protected from tuberculosis infection due to the presence of pancreatic enzymes that inhibit bacterial growth. It can cause a range of symptoms, including abdominal pain, nausea, vomiting, weight loss, and fever. Pancreatic tuberculosis should be in the differential when evaluating patients with nonspecific abdominal pain and risk factors such as being from an endemic area, or being HIV positive. Diagnosis is typically made through EUS or CT-guided fine-needle aspiration biopsy. With anti-tuberculosis treatment, the prognosis is good, but if left untreated, it can be fatal.



A and B are CT abdomen images demonstrating fluid collections in the pancreatic head and body respectively

46. EXOCRINE PANCREATIC INSUFFICIENCY TREATMENT IN PATIENTS WITH CHRONIC/RECURRENT ACUTE PANCREATITIS IMPROVES TREATMENT SATISFACTION AND DECREASES HEALTHCARE UTILIZATION: PACT-CP INTERIM FINDINGS

JA Barkin, Y Hernandez-Barco, S Al-Kaade, R Pannala, J Twal, J Pack, T Delk, VJ Powell, D Whitcomb
University of Miami Miller School of Medicine

Background: The needs of patients (pts) with exocrine pancreatic insufficiency (EPI) due to chronic pancreatitis (CP) are poorly understood. To further characterize the challenges for pts with CP and EPI receiving pancreatic enzyme replacement therapy (PERT), a prospective, pt-driven registry was established to collect data from pts and their providers. We present interim findings from the PAtient-CenTric Chronic Pancreatitis (PACT-CP) Registry.

Methods: This longitudinal, noninterventional study (NCT05762445) in the US, follows pts up to 5 years. Eligible adults are receiving PERT with suspected/confirmed EPI and diagnosed with CP/recurrent acute pancreatitis (RAP) at enrollment. To understand impact of clinical practices/EPI clinical course, multivariate modeling will be used to analyze effects of medical history, comorbidities, and treatments on EPI history/progression, quality of life, and healthcare resource utilization (HCRU).

Results: As of March 15, 2023, 91 pts are enrolled. Median age is 60.0 years (Table). Alcohol (susceptibility/progression), diabetes mellitus, and tobacco smoking are the most common CP etiologies. Pts (n=76) reported a median of 6 pills of PERT taken daily at Visit 1; median number was generally similar at subsequent visits. Most pts responding about PERT impact on symptoms at Visit 1 through the 18-month (mo) visit noted PERT greatly/somewhat improved symptoms (Visit 1, 78%; 3-mo, 71%; 6-mo, 71%; 9-mo, 77%; 12-mo, 73%; 15-mo, 50%; 18-mo, 100%). At Visit 1, 15 pts had recently been hospitalized because of abdominal pain; numbers declined at subsequent visits. Nineteen pts needed emergency room visits over the past 3 mo at Visit 1; numbers declined at subsequent visits. Full-time employees took a median of 2.5 days off over the past 3 mo at Visit 1 (n=28); days off at subsequent visits were ≤2.0. Mean global treatment satisfaction scores fluctuated and generally improved from Visit 1 to subsequent visits.

Conclusion: Many pts with CP/EPI receiving PERT have unmet needs in their CP/EPI management with impacts on work productivity and HCRU. Treatment of EPI with PERT may mitigate overall HCRU in pts with EPI due to CP/RAP. Future analyses will continue to clarify areas of unmet needs and provide insights with PERT use and other aspects of disease management.

Table. Patient Demographics and Disease Characteristics at Baseline

Characteristic	Total N=91
Patient demographics[a]	
Age (years), median (range)	60 (27–83)
Sex, n (%)	
Male	42 (53)
Female	37 (47)
Race, n (%)	
White	63 (80)
Black or African American	7 (9)
Asian	2 (3)
American Indian or Alaska Native	2 (3)
Other	8 (10)
Ethnicity, n (%)	
Hispanic or Latino	12 (15)
Not Hispanic or Latino	67 (85)
Chronic pancreatic etiology[b,c,d], n (%)	
Toxic-metabolic	
Alcohol (susceptibility/progression)	33 (37)
Tobacco smoking	19 (21)
Hyperlipidemia (fasting >300 mg/dL, nonfasting >500 mg/dL)	9 (10)
Medications	5 (6)
Toxins, other	3 (3)
Toxins, other, not otherwise specified	3 (3)
Hypercalcemia (total calcium levels >12.0 mg/dL or 3 mmol/L)	2 (2)
Toxins, chronic kidney disease (stage 5, end-stage renal disease)	2 (2)
Not applicable	43 (48)
Metabolic, other	
Diabetes mellitus	28 (31)
Not applicable	62 (70)
Idiopathic	
Late onset (>35 years of age)	42 (47)
Early onset (<35 years of age)	7 (8)
Not applicable	41 (46)
Evidence of suspected/confirmed EPI diagnosis[b,d], n (%)	
Weight loss	39 (44)
Clinical steatorrhea	31 (35)
Abnormal fecal elastase-1 test	27 (30)
Vitamin deficiency	18 (20)
Pancreatic function testing	6 (7)
Other	27 (30)
Not applicable	13 (15)
Clinical manifestations of EPI[b,d], n (%)	
Gastrointestinal symptoms	
Diarrhea/loose stool	65 (73)
Abdominal pain	60 (67)
Bloating	46 (52)

Steatorrhea	32 (36)
Flatulence	29 (33)
Not applicable	5 (6)
Nutritional	
Unintentional weight loss/difficulty gaining weight	38 (43)
Vitamin D deficiency	24 (27)
Other vitamin/mineral deficiencies	13 (15)
Malnutrition	12 (13)
Other	1 (1)
Not applicable	40 (45)

[a]Data were available for 79 patients.

[b]Data were available for 89 patients.

[c]Selected chronic pancreatic etiology; other categories not shown include genetic, autoimmune, recurrent and severe acute pancreatitis, obstructive, and other.

[d]Multiple options may be selected for individual participants.

Abbreviation: EPI, exocrine pancreatic insufficiency.

SAVE.THE.DATE

PANCREASFEST 2024

July 25-26, 2024
Cincinnati, OH