abnormal celiac serology and biopsy findings quantified by a Marsh score of 2 or greater. Serum hemoglobin and albumin were obtained and BMI was calculated for each patient. tTG IgA levels were expressed as the value/upper limit normal for the lab. Blood was obtained for genetic testing (genetic susceptibility score 1-8 using genetic markers HLA-DQ2/DQ8 haplotypes, Prometheus Labs) and determination of serum POP concentrations as recently described. The study was approved by the IRB of NYU School of Medicine. **Results:** Among the 30 CD patients (11 males, 19 females), 29 had elevated tTG IgA and the remaining child had IgA deficiency and elevated tTG IgG. 26/30 duodenal biopsies were classified as Marsh 3a or greater and 4 biopsies classified as Marsh 2. The children with Marsh 2 biopsies had elevated tTG IgA 2-10 times above the upper limit of normal, which normalized after adherence to a gluten-free diet. The genetic risk score had a significant positive correlation with the Marsh score while the serum albumin had a significant negative correlation (see table 1). For every 1 unit increase in risk score there was a 0.18 unit increase in Marsh score. None of the POP chemical values correlated with the Marsh score (table 2). **Conclusion:** Although environmental exposure combined with genetics appears to play a role in the development of celiac disease, our current results suggest that the degree of duodenal inflammation is related to genetic susceptibility. The negative correlation with serum albumin most probably just reflects the duodenal inflammation and possible albumin loss. Although previous studies have shown that tTG IgA > 100 is strongly associated with duodenal inflammation, we were not able to demonstrate a correlation between tTG IgA levels and degree of duodenal inflammation. Further studies are needed to understand the relationship between HLA-DQ2/DQ8 and duodenal response to gluten in children with CD.

Table 1 ? Patient characteristics and association with Marsh score

	Median (25th, 75th)	P	p-value
Age (yrs)	6.0 (3.0, 11.5)	-0.18	0.330
BMI	16.9 (15.4, 18.6)	-0.02	0.909
Genetic risk score	6.5 (4.0, 5.7)	0.39	0.034*
Marsh score	4.0 (3.0, 4.0)		
Albumin (g/dl)	4.3 (4.1, 4.6)	-0.40	0.036*
Hemoglobin (g/dl)	12.7 (11.3, 13.7)	-0.30	0.108
tTG IgA	12.0 (4.0, 35.0)	0.17	0.367

(*statistically significant at the p=0.05 level)

Table 2 ? POP chemical distribution and correlation with Marsh score

POP	Median (25th, 75th)	P	p-value
BDE47	0.02 (0.01, 0.03)	-0.03	0.885
BDE99	0.006 (0.002, 0.01)	-0.01	0.944
BDE100	0.007 (0.004, 0.01)	0.04	0.839
BDE153	0.02 (0.002, 0.04)	0.23	0.231
DDE	0.29 (0.20, 0.43)	-0.11	0.569
PFOS	2.02 (1.05, 2.90)	-0.27	0.143
PFOA	1.26 (0.88, 1.64)	-0.07	0.704
PFHxS	0.41 (0.27, 0.54)	-0.36	0.051
PFDA	0.06 (0.06, 0.18)	-0.17	0.378
PFNA	0.28 (0.16, 0.62)	-0.19	0.318

Tu1331

EMERGENCY PRESENTATIONS FOR GASTROSTOMY COMPLICATIONS IN ADULTS AND CHILDREN

Nina Chua, Jennifer F. McDonald, Megan Murray, Simon Craig, Edward M. Giles

Background: Gastrostomy insertion is associated with increased rates of emergency department (ED) visits and inpatient admissions. We aimed to identify patterns of emergency presentations of gastrostomy tube complications in adult and paediatric populations and examine the investigations and treatments provided. **Methods:** Retrospective chart review of patients with feeding tubes who presented to three Victorian EDs from 1 January to 31stDecember 2017. These patients had access to a single gastrostomy outreach service that was contactable by telephone 365 days per year. Cases were included if their ED presentation were partially or wholly due to a gastrostomy complication. **Results:** During the study period, 525 patients were under the gastrostomy service. 378 out of 225,800 ED presentations were by patients with a feeding tube, from 75 adults and 60 children. Visits relating to gastrostomy complications occurred in 23 adults and 20 children, with a total of 64 presentations. 23 presentations, 8 adult and 15 paediatric, were for a suspected infection, including presentations with leakage and erythema, and 7 of these were treated with antibiotics. 32 presentations, 18 adult and 14 paediatric, were for a mechanical problem including broken, dislodged or blocked gastrostomies, and of these 14 had a gastrostomy replacement. 9 presentations, 5 adults and 4 paediatric, were for a vomiting related issue and 6 presentations, 3 adults and 3 paediatric, for other reasons. **Conclusion:** In the setting of a hospital with a gastrotomy outreach service, ED attendances for gastrostomy patients are uncommon, and most visits are not due to complications from the device. Similar complications occur for children and adults, supporting the use of staff able to work in both patient populations.

Tu1415

MANAGEMENT OF ACUTE PANCREATITIS IN CHILDREN: A SINGLE-CENTER EXPERIENCE

Liron Birimberg Schwartz, Sara Rajiwate, Tanja Gonska

Over the past two decades the incidence of pediatric acute pancreatitis (AP) has increased. There has also been substantial progress in defining and characterizing AP in children. Furthermore, while recognizing the lack of evidence, guidelines for the fluid and nutritional management of children with AP were recently established (Abu-El-Haija et al, tifN, 2018). These are mainly borrowed from the experience and data generated with regard to adult patients presenting with AP. The current recommendations suggest aggressive fluid resuscitation in pediatric patients presenting with AP and early implementation of enteral nutrition. Nevertheless, while adult patients with AP are at high risk for developing multi-organ dysfunction and mortality, the disease course in children is usually mild. The Hospital for Sick Children is the largest referral center for pediatric diseases in Canada. Over the last sixteen years over 300 pediatric patients were admitted with the diagnosis of AP. This retrospective study sought to make use of this large single-center cohort to address the following questions: 1) Has the frequency of pediatric severe AP changed over time? 2) Does the clinically practiced management of pediatric AP align with the current guidelines? 3) Does more aggressive fluid management and/or early nutrition alter disease outcome? We have reviewed 333 medical charts and included 216 patients who met the strict diagnostic criteria of pediatric AP (Morinville, tifN, 2012) and for whom accurate data on fluid and nutritional management were available for our analysis. Disease severity was classified into mild, moderately-severe and severe AP according to the recently established criteria (Abu-El-Haija, tifN, 2017). Preliminary analysis showed that the incidence of severe AP has not changed significantly over the past years and stands at approximately 4.2% of the children presenting with AP. Furthermore, although 186 (86.9%) of the patients received intravenous fluid therapy, only 35 (18.8%) of them received the currently recommended rate of at least 1.5 maintenance. For the majority of patients (n=178, 82.4%), physicians continue to follow the NPO (nothing per os) approach in their initial management of AP. Differences in management now provide us with the opportunity to examine whether deviations from recommended guidelines for management of AP in children alters disease outcome.

Tu1416

PROLONGED HYPERAMYLASEMIA IN PATIENTS WITH ACUTE PANCREATITIS IS ASSOCIATED WITH RECURRENCE OF ACUTE PANCREATITIS

Kyu Won Lee, Tae Ho Kim, Sok Won Han, Jae Hyuck Chang

Objectives: Patients with acute pancreatitis often show serum amylase levels that remain or fluctuate above the upper normal limit for over a week. This study investigated the clinical characteristics of patients with prolonged hyperamylasemia and their prognoses, including recurrence. **Methods:** We retrospectively analyzed patients with first attacks of acute pancreatitis in a single center between March 2010 and December 2016. The patients were divided into two groups: patients in whom serum amylase levels were normalized (≤ 100 U/L) within a week (group 1) and patients who showed prolonged hyperamylasemia (> 100 U/L) over a week including fluctuation (group 2). We compared the clinical characteristics and prognoses between the two groups, and evaluated the factors related to recurrent pancreatitis, including the association with prolonged hyperamylasemia. **Results:** A total of 313 patients were enrolled after exclusion. The average follow-up period of these patients was 1,071 days (180?2,930 days). The serum amylase levels were normalized within a week in 205 patients (65.5%, group 1) and elevated over a week in 108 patients (34.5%, group 2). Group 2 was more related to alcohol, higher CT severity index, pancreatic necrosis, pseudocyst, and moderately severe pancreatitis than group 1 (p<0.05). Recurrent pancreatitis developed significantly more in group 2 (39.8%) than in group 1 (19.5%) (p<0.001). Serial daily average serum amylase levels according to recurrence of acute pancreatitis demonstrated that the serum amylase levels in patients with recurrent pancreatitis were significantly higher than those in patients without recurrent pancreatitis on days 5, 7, 8, and 9 (p = 0.020, 0.041, 0.023, and 0.009, respectively, Figure 1). The factors related to recurrent pancreatitis were amylase group, sex, alcohol, CT severity index, necrosis, and severity of pancreatitis (p<0.05). Multivariate analysis showed that recurrent pancreatitis was independently associated with amylase group (OR 2.123, p=0.007) and alcohol (OR 2.023, p=0.017) (Table 1). **Conclusions:** Prolonged hyperamylasemia over a week is associated with recurrence of acute pancreatitis. Therefore, closer observation and follow-up are needed in patients with prolonged hyperamylasemia.

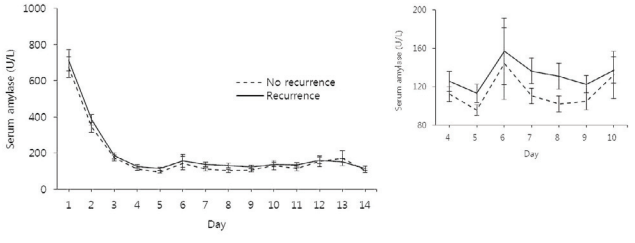


Fig. 1. Serial average serum amylase levels (±SD) according to recurrence of acute pancreatitis (* < 0.05; ** < 0.01).

Table 1. Multivariate analysis of factors associated with recurrent pancreatitis

Factors	Odds ratio (95% CI)	p value
Amylase group	2.123 (1.227?3.673)	0.007
Alcohol	2.023 (1.134?3.611)	0.017
Local complication	1.263 (0.246?6.489)	0.779
CT severity index (0?3 vs. 4?10)	1.432 (0.699?2.933)	0.326
Severity (mild vs. moderately severe/severe)	1.240 (0.246?6.246)	0.794

Tu1417

ACUTE PANCREATITIS IN PATIENTS WITH CYSTIC FIBROSIS

C. Roberto Simons-Linares, Mohannad Abou Saleh, Paul T. Kroner, Sunguk Jang, Amit Bhatt, John J. Vargo, Tyler Stevens, Prabhleen Chahal

Background & Aim: Acute pancreatitis (AP) in patients with cystic fibrosis (CF) is almost exclusively to pancreatic sufficient (PS) patients, with about 20% of PS patients developing AP. The severity of pancreatic ductal obstruction is related to the severity of the cystic fibrosis membrane receptor (CFTR) dysfunction. We aim to investigate the inpatient prevalence, outcomes of mortality, morbidity and cost of AP patients with CF. **Methods**: We performed a retrospective cohort study using the National Inpatient Sample (NIS) database from 2003 to 2013. The NIS was queried for patients with a discharge diagnosis of AP (ICD-9 577.0). The presence of CF was ascertained based on ICD9 codes 277.00-03 and 277.09. Outcomes included inpatient mortality, morbidity (SIRS, shock, ileus, AKI, ARDS), resource utilization (CT abdomen, MRCP, ERCP, parenteral nutrition), length of hospital stay (LOS), US$ inflation-adjusted Charges. **Results:** were adjusted for age, gender, race, Charlson comorbidity index (CCI), median income quartile, and hospital characteristics. Multivariable logistic regression models were constructed using SAS software version 9.4 **Results:** Over 2.8 million patients with acute pancreatitis were evaluated. 607 patients had CF (<1%). CF patients were most likely to be white (73% vs. 54%, p<0.001) and younger (mean age 33 vs. 53-year-old, p<0.001). No statistical difference in inpatient mortality (OR 0.002, P=0.76) and hospital charges (US$ - 1,657.52, p=0.31) was found. CF patients were less likely to have AKI (OR 0.54, P=0.061), ARDS (OR 0.23, p=0.039), gastrointestinal bleed (OR 0.30, p=0.097), upper endoscopy performed (OR 0.50, p=0.005), to receive blood transfusions (OR 0.39, p=0.062), cholecystectomy performed (OR 0.32, P<0.001), shorter length of stay, (-0.43 days, p=0.06) **Limitations**: Inpatient data only (missing post discharge outcomes), reliance on administrative data/billing codes for ascertaining outcomes and exposures. **Strength**: Biggest patient cohort to date. Adjusted for age, gender, race, Charlson comorbidity index (CCI), median income quartile, and hospital characteristics. **Conclusion**: Although patient with CF have a higher risk for developing AP than the general population, in this large inpatient study - CF patients hospitalized with AP did not have an increased mortality, morbidity or resource utilization. Interestingly, they were less likely to develop AKI, ARDS, GI bleed, and shorter hospital LOS. These findings could be explained by the significantly younger age of CF patients and/or it could be that CF patients do not have a significant amount of functional acinar tissue left to elucidate a severe AP attack.

Table 1a. Outcomes comparing CF vs. non-CF. Multivariate regression analysis (Adjusted).

Outcome	adjusted OR (95% CI)	P value
AKI	0.547 (0.291, 1.028)	**0.061**
Ileus	1.2 (0.716, 2.011)	0.489
Myocardial Infarction	0.003 (0, 1.698916e+20)	0.829
Shock	0.884 (0.22, 3.552)	0.862
ARDS and Mechanical Ventilation	0.231 (0.058, 0.926)	**0.039**
Bacterial Infection	0.002 (0, 2.693346e+27)	0.863
SIRS	0.384 (0.096, 1.54)	0.177
Parenteral nutrition	1.391 (0.936, 2.069)	0.103
CT abdomen	0.821 (0.47, 1.434)	0.488
MRCP	0.56 (0.179, 1.75)	0.319
ERCP	0.82 (0.388, 1.733)	0.604
EGD	0.507 (0.316, 0.813)	**0.005**
Transfusion	0.392 (0.146, 1.049)	**0.062**
GIB	0.308 (0.077, 1.238)	**0.097**
DIED	0.002 (0, 1.640994e+15)	0.761
Sepsis	1.007 (0.416, 2.435)	0.988
DVT	0.002 (0, 9.768183e+20)	0.82
PE	0.001 (0, 1.061499e+23)	0.829
PVT	0.432 (0.061, 3.086)	0.403
Cholecystitis	0.843 (0.315, 2.258)	0.734
CCY	0.32 (0.21, 0.487)	**<0.001**
PC	0.01 (0, 3.785894e+15)	0.823
PTHC	0.004(0, 3.05019e+18)	0.825

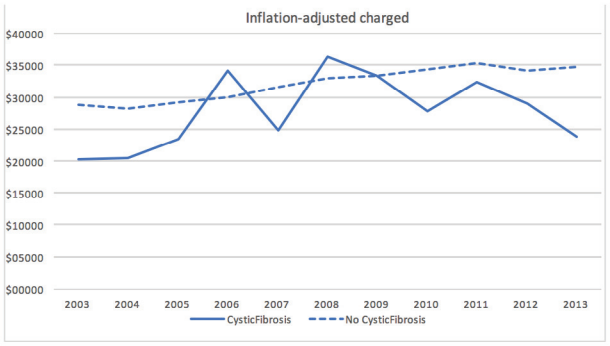
Table 1b. Association Between Cystic Fibrosis and Continuous Outcomes

	Cystic Fibrosis vs. Non- Cystic Fibrosis			
Factor	**Unadjusted estimate (95% CI)**	**Unadjusted p-value**	**Adjusted* estimate (95% CI)**	**Adjusted* p-value**
Length of stay (days)	**0.23 (-0.24, 0.70)**	**0.3357**	**0.43(-0.03, 0.90)**	**0.0685**
Inflation-adjusted Charges ($)	**-3908.59(-7241.58, -575.60)**	***0.0215***	**-1657.52(-4885.37, 1570.32)**	**0.3142**

*Adjusted for age, gender, race, median income quartile, CCI and hospital characteristics

Figure 1: Trend in Inflation-adjusted charges

Difference between groups p = 0.88



Tu1418

THE EPIDEMIOLOGY OF ACUTE PANCREATITIS IN ULCERATIVE COLITIS: A POPULATION-BASED STUDY FROM 2013-2018

George Khoudari, Mohannad Abou Saleh, Emad Mansoor, Mohamed M. Gad, Pooja Lal, C. Roberto Simons-Linares, Preetika Sinh, Prabhleen Chahal, Jean-Paul Achkar

Background and Aims: A few studies have suggested an increased prevalence of acute pancreatitis (AP) in ulcerative colitis (UC) with possible underlying etiologies of medication induced, autoimmune or idiopathic AP. These studies however were limited by small sample size. Using a large database, we sought to establish the epidemiology of AP in patients with UC and identify possible risk factors. **Methods:** We queried a commercial database (Explorys Inc, Cleveland, OH, USA), an aggregate of electronic health record data from 26 major integrated US healthcare systems. We identified a cohort of patients with a Systematized Nomenclature of Medicine?Clinical Terms (SNOMED-CT) diagnosis of UC from 2013-2018. We then investigated the prevalence rate of first ever diagnosis of AP after at least 30 days of being diagnosed with UC. We performed analyses to describe age-, race-, and gender-based distributions and to identify potential risk factors of AP in patients with UC. **Results:** Of the 38,289,490 individuals in the database, we identified 95,770 individuals with UC with an overall prevalence rate of 0.25% and 230,600 individuals with AP with prevalence rate of 0.6%. Of the individuals with UC, 1,570 developed first ever episode of AP after at least 30 days of UC diagnosis with prevalence rate of 1.6%. Patients with UC were more likely to develop AP when compared to individuals without UC [OR: 5.12, 95% CI: 4.94-5.30, p<0.0001]. They were also more likely to have a history of elevated IgG4 [OR 3.83; 95% CI: 2.65-5.52, p<0.0001], and an underlying diagnosis of idiopathic pancreatitis [OR 1.89; 95% CI: 1.57-2.28, p<0.0001]. Odd ratios of predictors of AP in UC patients who developed AP, when compared to UC patients who did not develop AP, are presented in table 1. The prevalence of AP in UC over a 5-year interval is presented in figure 1. **Conclusion:** This is the largest epidemiological study investigating the prevalence of AP in UC. We found the estimated prevalence of first ever occurrence of AP at least after 1 month of UC diagnosis to be 1.6% compared to 0.6% in the general population. Patients with UC were more likely to develop AP when compared to individuals without UC. Autoimmune pancreatitis, cholelithiasis/ choledocholithiasis and medication induced AP were the most common underlying etiology of AP in UC. The increased prevalence of idiopathic AP in UC however suggests a possible causal relationship.

Risk Factor		**OR (95% CI)**	**P-value**
Demographics			
	African American	2.05 (1.78-2.36)	<0.0001
	Male	1.05 (0.95-1.16)	0.34
	Age (18-65)	1.11 (0.10-1.23)	0.58
Pancreatitis Risk Factors			
	Autoimmune disease	2.05 (1.78-2.36)	<0.0001
	Hypertriglyceridemia	3.05 (2.50-3.72)	<0.0001
	cholelithiasis/choledocholithiasis	5.89 (5.24-6.61)	<0.0001
	Alcohol	1.26 (1.14-1.4)	<0.0001
	IgG4	3.83 (2.66-5.52)	<0.0001
Medications			
	NSAIDs	2.92 (2.44-3.49)	<0.0001
	Anti-TNF	1.38 (1.16-1.64)	0.0003
	Immunomodulators	1.52 (1.31-1.77)	<0.0001
	Methotrexate	1.81 (1.39-2.35)	<0.0001
Other			
	Smoking	2.27 (2.02-2.55)	<0.0001
	DM	2.44 (2.20-2.70)	<0.0001
	Obesity	2.10 (1.89-2.34)	<0.0001