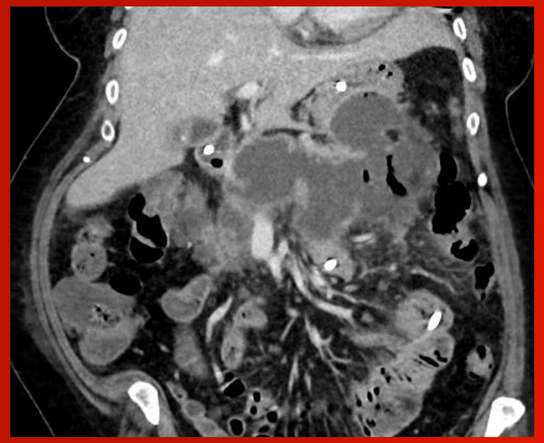
ACTIVE SCREENING STRATEGY FOR EXOCRINE PANCREATIC INSUFFICIENCY IN ACUTE NECROTIZING PANCREATITIS

Benjamin M Smith, MD; Gary C Vitale, MD; Robert C Martin, MD; Michael E Egger; MD, Prejesh Philips, MD

University of Louisville, Department of Surgical Oncology



Background

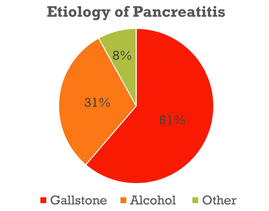
- Acute pancreatitis (AP) is an inflammatory process caused by multifactorial injury to the pancreas, most commonly from gallstones or alcohol abuse in the United States. (4)
- The Atlanta classification is the most widely accepted categorization system used currently
  - Interstitial edematous vs. necrotizing, with mild moderately severe, severe categories
- Bedside index for severity in Acute Pancreatitis (BISAP) is a validated index used to assess the clinical severity of pancreatitis
- AP outcomes have improved with minimally invasive techniques, although mortality approaches 30% in those with infected pancreatic necrosis (5,7)
- Exocrine pancreatic insufficiency (EPI) is the inability of the pancreas to produce, activate or deliver adequate pancreatic enzymes to aid in digestion
- Literature regarding EPI in the setting of AP is sparse
- There are two proposed mechanisms for the development of EPI in those with AP
  - Transient vs long term pancreatic cellular dysfunction from acute injury
  - Ductal disruption preventing pancreatic fluid flow from discontinuity or stricture
- Incidence of EPI related to AP is unknown (9)
- EPI treatment is inconsistent leading to nutritional deficiencies that are related to increased risk of mortality (8,10)
- Pancreatic enzyme replacement therapy (PERT) has aided in symptom management, nutritional status and overall patient well being (11)
- Research has shown increased incidence of EPI in those with moderately severe and severe pancreatitis
- There are no widely used screening tools for EPI in patients with AP(12)

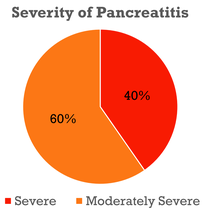
Methods

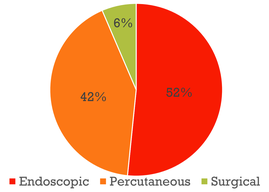
- Prospective cohort study 2019-2024: 62 patients with acute necrotizing pancreatitis undergoing drainage procedures due to local complications (walled off necrosis or acute necrotic collection)
- Excluded those with chronic pancreatitis, pancreatic cancer, and preexisting malabsorption syndromes
- Severity determined by Atlanta Classification; BSAP was calculated for each patient
- Patients were screened for EPI at time of resumption of non-clear liquid diet and at discharge
- An EMR template was utilized for screening
- PERT was initiated for a clinical or biochemical diagnosis of EPI
- Subjective global assessment form and serum albumin levels were recorded at discharge, 3, and 6 month follow up

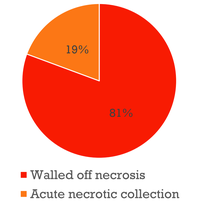
Results

N = 62







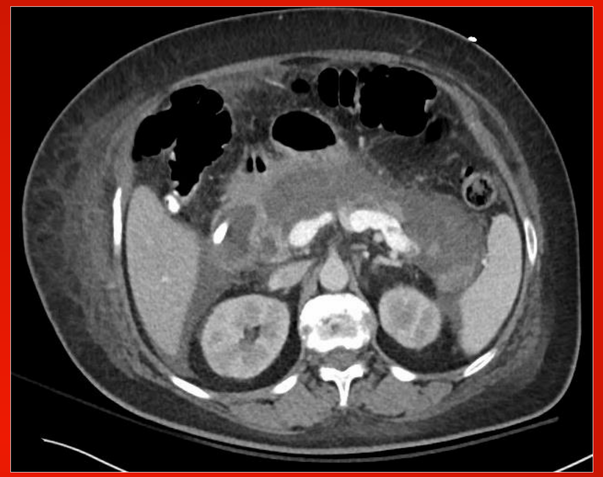


Results

EPI Prevalence: n=36 (58.1%)

Primary Drainage Procedure		Severity	
Endoscopic Drainage	32 (51.6%)	Moderately Severe	22 (61%)
Percutaneous Drainage	26 (41.9%)	Severe	14 (39%)
Surgical	4 (6.5%)	P- Value	0.788
Etiology		**Compliance**	**34 (81%)**
Gallstone	23 (54.8%)	Reason for noncompliance	
Alcohol	12 (28.6%)	Cost	5 (14%)
Other	3 (7.1%)	Intolerance	4 (11%)
P- Value	0.255		

Table 1: 36 out of 62 patients were found to have EPI. The primary drainage type of procedure did not impact the incidence of EPI (p=.153). The prevalence of EPI did not differ depending on the drainage procedure type, the etiology of the pancreatitis, the severity of the pancreatitis. Compliance was 81% and noncompliance was due to cost and intolerance issues.



Discussion

- We chose this relatively homogenous patient population with respect to the severity of their pancreatic parenchymal injury
- Screening at discharge increases compliance and allows us to intervene with enzyme replacement
- The median BISAP score was 4, indicating this cohort had very significant clinical pancreatitis
- Early screening during a patient's hospital stay may be too early and lead to erroneous findings given ongoing changes in these patient's clinical course
- Disease severity was not found change the incidence of developing EPI, although we only compared moderately severe to severe. This shows us that both patient populations are at high risk.
- We saw no evidence that the development of endocrine dysfunction was correlated with the development of EPI, indicating more complex pathophysiology at play.
- Endoscopic drainage procedures do appear to decrease the rate of EPI development in our patient population, potentially by allowing free communication of the pancreatic enzyme production into the gi tract.
- We found that PERT was well tolerated with an 80% compliance rate. This is surprising given the high cost of the medication and possibly corresponds to patient satisfaction with the medication effects.
- Around 40% of patients were able to stop therapy at 3 months and 90% at 6 months, indicating that EPI related to acute pancreatitis is transient. This is encouraging given the severity of the pancreatitis in our study group.
- This is a highly selective cohort of individuals. Even though we have a high-volume institution, it takes considerable time to develop a large enough cohort to observe small intergroup differences.
- This is a difficult patient population regarding reliable follow up; this may be a confounding factor
- Fecal elastase was not universally utilized as we relied more on a clinical diagnosis
- Future studies could have a control arm to identify quality of life differences

Conclusion

We found that early EPI in acute necrotizing pancreatitis is common but transient. We believe it often goes undiagnosed. An EMR based active screening strategy and intervention is important in optimization of these nutritionally fragile patients. This prospective cohort study is ongoing to help us identify the most appropriate role of EPI screening and treatment.

References:

1. Peery, A.F., et al., *Burden and Cost of Gastrointestinal, Liver, and Pancreatic Diseases in the United States: Update 2018.* Gastroenterology, 2019. **156**(1): p. 254-272 e11.
2. Maatman, T.K., et al., *The continuum of complications in survivors of necrotizing pancreatitis.* Surgery, 2020. **168**(6): p. 1032-1040.
3. Gapp, J., et al., *Trends and Outcomes of Hospitalizations Related to Acute Pancreatitis: Epidemiology From 2001 to 2014 in the United States.* Pancreas, 2019. **48**(4): p. 548-554.
4. Murtaugh, L.C. and M.D. Keefe, *Regeneration and repair of the exocrine pancreas.* Annu Rev Physiol, 2015. **77**: p. 229-49.
5. Banks, P.A., et al., *Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus.* Gut, 2013. **62**(1): p. 102-11.
6. Chua, T.Y., et al., *Necrotizing pancreatitis: Diagnose, treat, consult.* Cleve Clin J Med, 2017. **84**(8): p. 639-648.
7. Kokosis, G., A. Perez, and T.N. Pappas, *Surgical management of necrotizing pancreatitis: an overview.* World J Gastroenterol, 2014. **20**(43): p. 16106-12.
8. Singh, V.K., et al., *Less common etiologies of exocrine pancreatic insufficiency.* World J Gastroenterol, 2017. **23**(39): p. 7059-7076.
9. Peribtani, Y and C.E. Forsmark, *Update on the diagnosis and management of exocrine pancreatic insufficiency.* F1000Res, 2019. **8**.
10. Moore, J.V., et al., *Exocrine Pancreatic Insufficiency After Pancreatectomy for Malignancy: Systematic Review and Optimal Management Recommendations.* J Gastrointest Surg, 2021. **25**(9): p. 2317-2327.
11. Domínguez-Muñoz, J.E., *Pancreatic enzyme replacement therapy for pancreatic exocrine insufficiency: when is it indicated, what is the goal and how to do it?* Adv Med Sci, 2011. **56**(1): p. 1-5.
12. Hollemans, R.A., et al., *Pancreatic exocrine insufficiency following acute pancreatitis: Systematic review and study level meta-analysis.* Pancreatology, 2018. **18**(3): p. 253-262.