



Two Interesting Cases of Pancreatitis

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Disclosures:

- Boston Scientific Consultant



Patient A.R.

- July 2016: 65 y.o. man seen in office for recurrent acute pancreatitis
- Pancreatitis 6x (first in 2013 and intubated for 10 days during last admission in June)
- Sent to ER from my office
- Admitted and underwent Axios cyst gastrostomy
- Multiple necrosectomies
- Oct 2016 Stents removed and patient followed up annually

Patient A.R.

- March 2020 (7 years after 1st AP and 4 years after I first met him) complained of unintentional weight loss
- Imaging demonstrated a 30mm mixed solid/cystic



5 Pancreatic tail lesion

EUS FNB Results: Mucinous Neoplasm with at least High Grade Dysplasia

DIAGNOSIS(ES):

SPECIMEN ADEQUACY:
SATISFACTORY FOR EVALUATION

INTERPRETATION:
NEOPLASTIC: OTHER - Bethesda Category IV-B
Fine needle biopsy sections show:

Mucinous neoplasm with at least high grade dysplasia, see comment.

Comment: The biopsy sample show clusters of neoplastic cells demonstrating nuclear pleomorphism, including greater than 4 times variation in the size of nuclei and few single, signet ring type cells in background of abundant extracellular mucin. Also seen are abundant acute inflammation with granulation tissue, and necroinflammatory debris.

The overall findings are compatible with mucinous neoplasm with at least high grade dysplasia, cannot exclude invasion.

Clinical and radiologic correlation recommended.

Case reviewed at Intradepartmental Cytology Consensus Conference on 3/11/2020.

Patient A.R.

- Patient underwent a distal pancreatectomy
- Post resection course complicated by large fluid collection
- Another Axios cyst gastrostomy needed (!)
- Pathology revealed...

Invasive IPMN Associated Adenocarcinoma (Colloid Type) 6mm pT1b 0/33 LNs Negative

DIAGNOSIS(ES):

A. Stomach, partial gastrectomy:

Portion of stomach lined by oxyntic mucosa with mild chronic inflammation.

No adenocarcinoma is seen.

Immunostain for *Helicobacter pylori* will be reported as an addendum.

B. Tail of pancreas and spleen with portion of colon, pancreatectomy and splenectomy:

1. Invasive mucinous non-cystic adenocarcinoma (colloid carcinoma), moderately differentiated, 0.6 cm in greatest dimension (pT1b), arising from intraductal papillary mucinous neoplasm (IPMN) with high-grade dysplasia and ulceration. See microscopic description.

2. Margins (pancreatic and colonic) are negative for adenocarcinoma and high-grade dysplasia.

3. Thirty-three lymph nodes, negative for adenocarcinoma (0/33).

4. No lymphovascular or perineural invasion is seen.

5. Marked mixed inflammation with fat necrosis, scattered small abscesses, and multinucleated giant cells, with bridging inflammation through adhesion into wall of colon.

6. No adenocarcinoma is seen in attached portion of colon.

7. Unremarkable spleen and accessory spleen.

C. Colon, additional portion, colectomy:

Portion of unremarkable colon.

No adenocarcinoma is seen.

D. Omentum, resection:

Lobulated adipose tissue, consistent with omentum.

No adenocarcinoma is seen.

Patient A.R.

- Patient completed chemo and recently saw him in the office
- Now living in Florida (and driving a convertible)

Patient C.M.

- 47 year old transferred to Columbia with severe post ERCP necrotizing pancreatitis in 2012
- S/p CCY prior to ERCP and father died of pancreatic cancer
- Patient recovered without cyst gastrostomy
- Recurrent episodes of pancreatitis
- Underwent multiple subsequent ERCPs with PD stenting



Patient C.M.C

- In 2021 (9 years after first AP) patient complained of 15lb weight loss
- Cannot do MRI due to cochlear implants
- CT with BOP mass and upstream atrophy and dilated PD



EUS FNB:

Neoplastic with Tubular and Papillary Growth Pattern

DIAGNOSIS(ES):

SPECIMEN ADEQUACY:

SATISFACTORY FOR EVALUATION

INTERPRETATION:

NEOPLASTIC: OTHER - Bethesda Category IV-B

Smear, ThinPrep slide, and cell block section show:

Epithelial neoplasm with tubular and papillary growth pattern.

Note: The smears are very cellular and are composed of polygonal epithelial cells arranged in tubular and papillary growth pattern. The tumor cells show mild anisonucleosis and minimal pleomorphism. The cells contain central nuclei with prominent nucleoli and moderate amount of dense cytoplasm. Mitosis are extremely rare. Mucin characteristic of IPMN is not identified. Though, scant mucin is present. No tumor necrosis is identified.

A panel of immunostains show that tumor cells are positive for CK, CK7 and CDX-2. MUC profile shows that tumor cells are diffusely positive for MUC-6, focally positive for MUC-1 and negative for MUC-2, MUC-4 and MUC-5.

Tumor cells are negative for CK20, Hep-Par-1, inhibin and SATB2.

CD56, chromogranin and synaptophysin are focally positive. SMAD-4 is preserved. p53 is positive in rare cells. Acinic cell markers chymotrypsin and antichymotrypsin are negative.

Beta-catenin is normally expressed.

Overall, the morphologic features and immunoprofile are not specific. Differential diagnosis include intraductal tubular papillary neoplasm, however some of the features such as diffuse CDX2 expression are not typical of this entity.

Patient C.M.

- Patient referred for distal pancreatectomy
- Pathology revealed...

Invasive ITPN Associated Adenocarcinoma

2mm pT1a

0/32 LNs Negative

DIAGNOSIS(ES):

A. Pancreas, body and tail, partial pancreatectomy:

1. Microscopic foci of invasive pancreatic ductal adenocarcinoma, moderately differentiated, arising at the periphery of intraductal tubulopapillary neoplasm (ITPN), largest focus of invasion measures approximately 2 millimeters (pT1a). See microscopic description.
2. Thirty-two lymph nodes, negative for carcinoma (0/32).
3. See part C for final pancreatic margin status.
4. Perineural invasion is identified.
5. No lymphovascular invasion is identified.

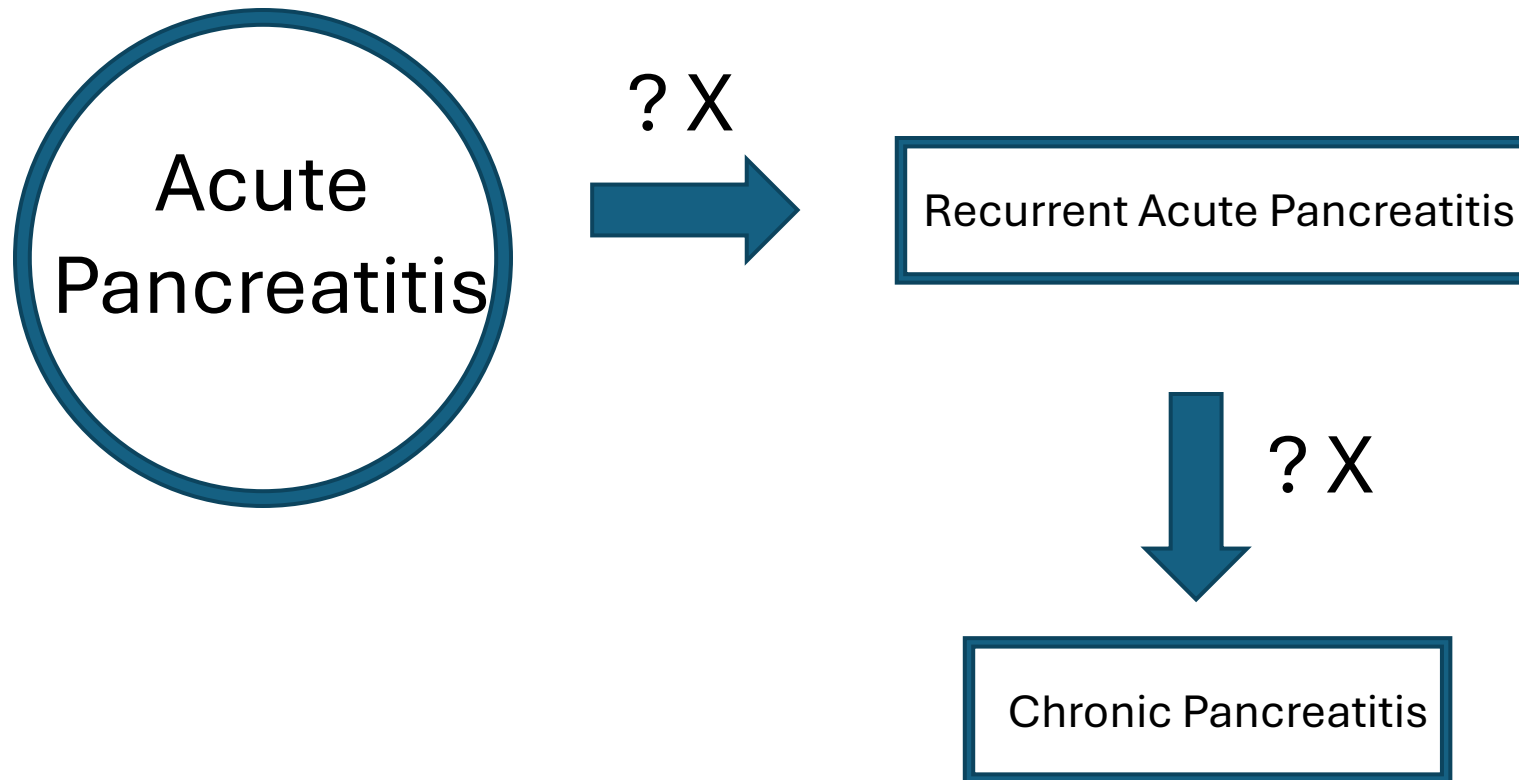
B. Spleen, splenectomy:

Fragments of unremarkable spleen, negative for carcinoma in representative sections.
One lymph node, negative for carcinoma (0/1).

C. Pancreas, partial pancreatectomy:

1. Intraductal tubulopapillary neoplasm (ITPN), no definite invasive carcinoma is seen.
2. New margin (smaller end) is negative for adenocarcinoma and ITPN.

Natural History of Acute Pancreatitis

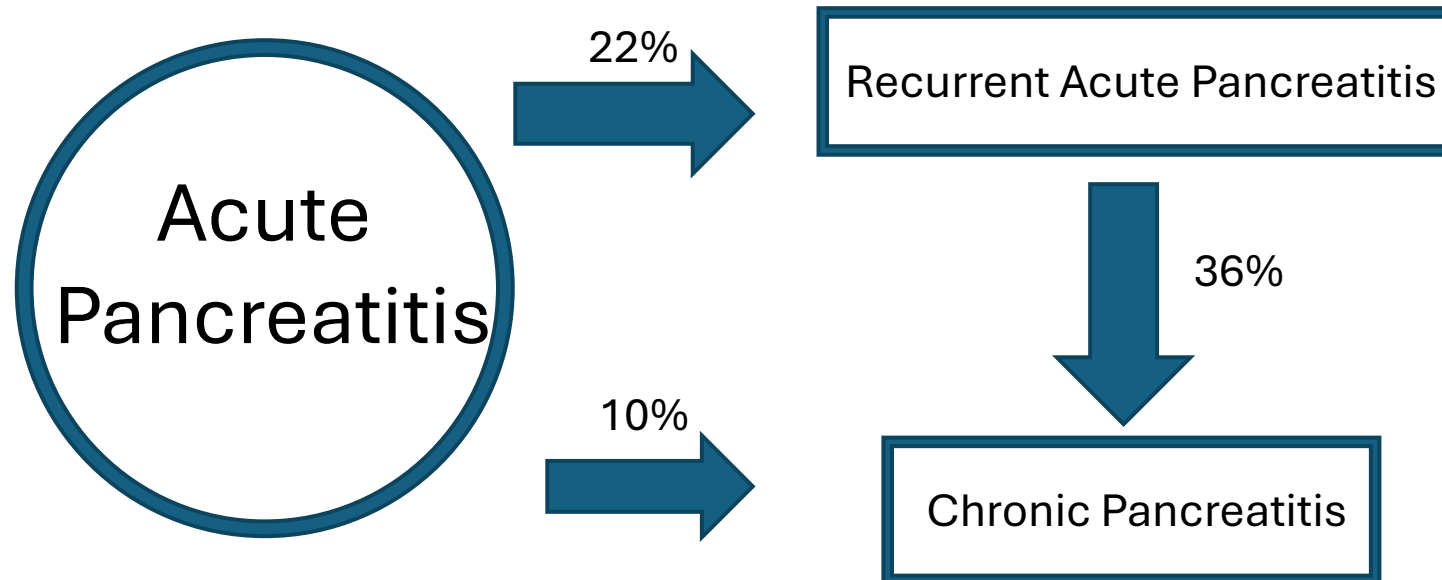


CLINICAL—PANCREAS

Frequency of Progression From Acute to Chronic Pancreatitis and Risk Factors: A Meta-analysis



- 14 Studies
- 8500 patients



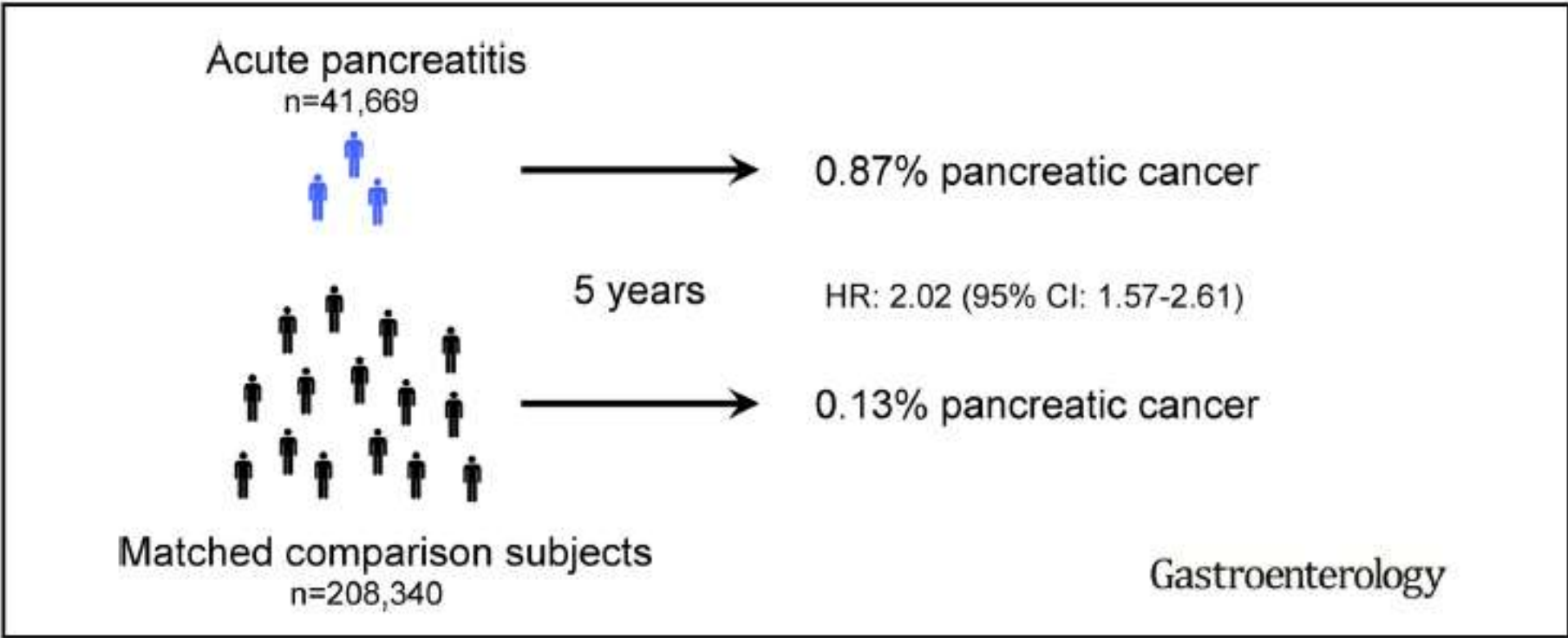
CLINICAL—PANCREAS

Acute Pancreatitis and Pancreatic Cancer Risk: A Nationwide Matched-Cohort Study in Denmark



Jakob Kirkegård,¹ Deirdre Cronin-Fenton,² Uffe Heide-Jørgensen,² and Frank Viborg Mortensen¹

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Acute Pancreatitis and Pancreatic Cancer Risk: A Nationwide Matched-Cohort Study in Denmark

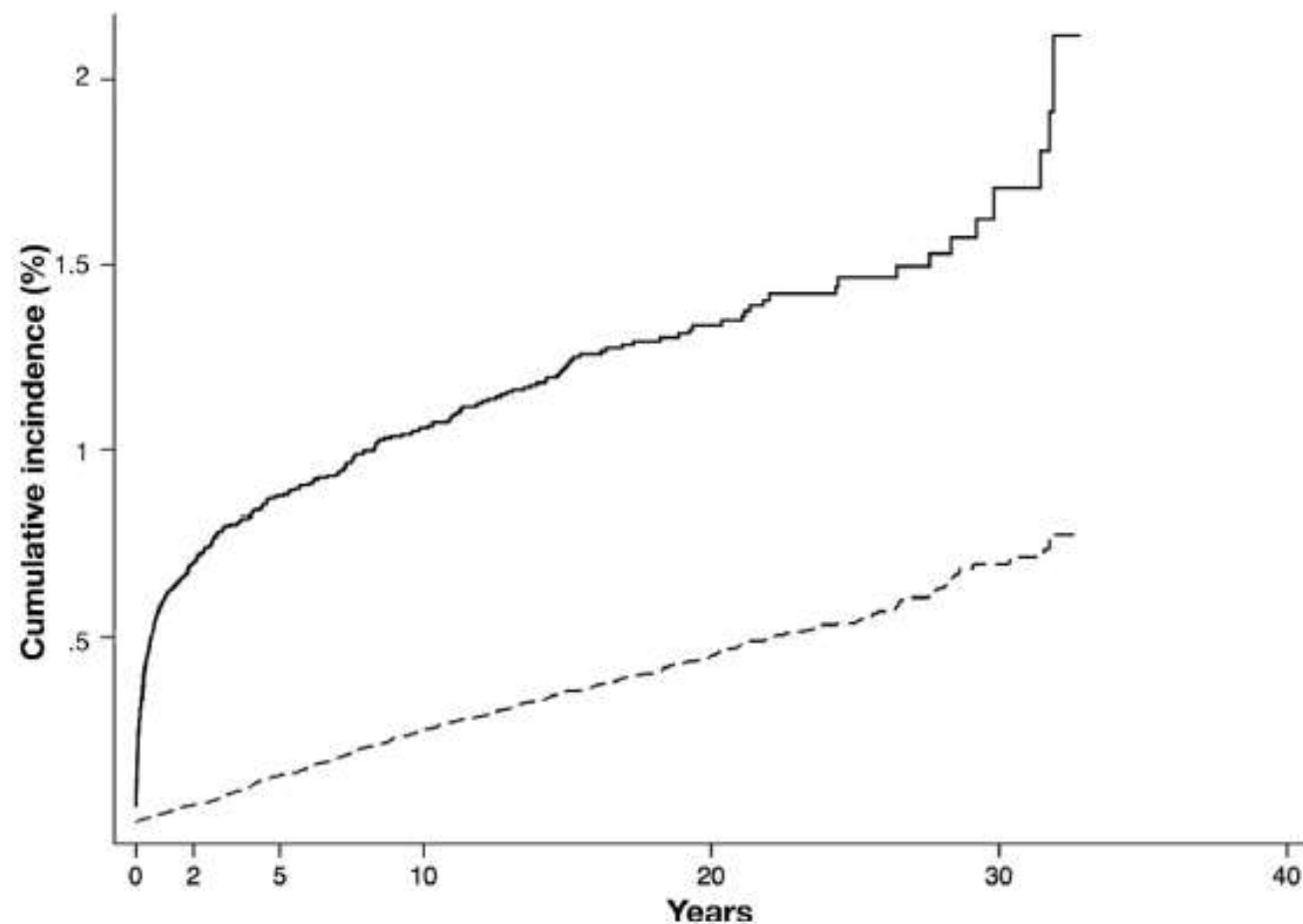
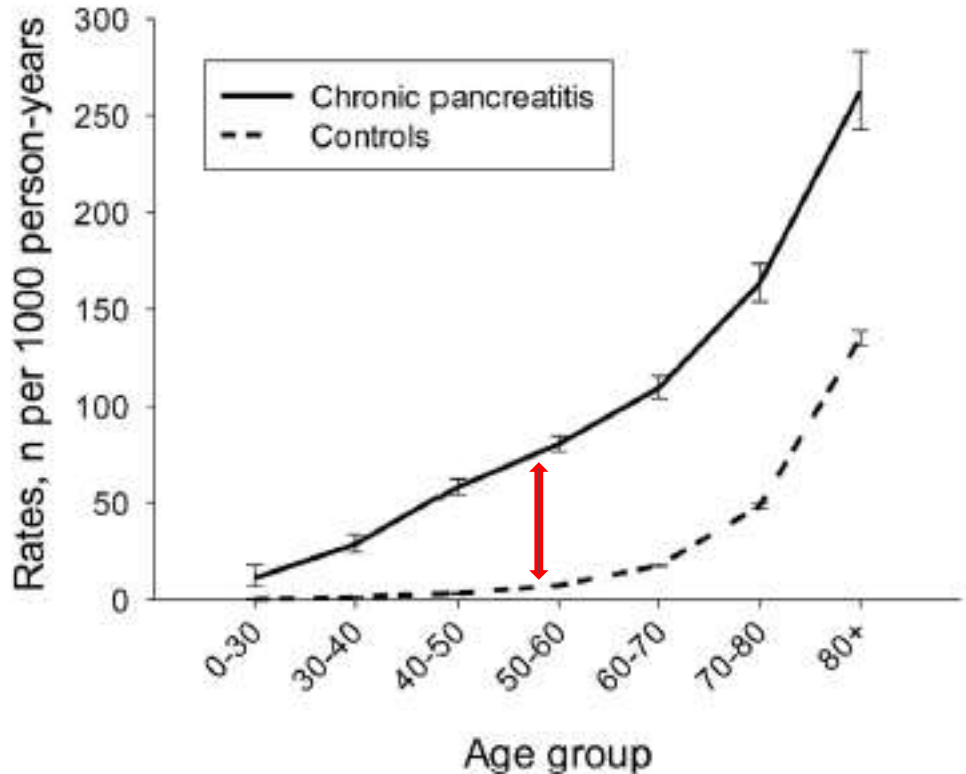


Figure 1. Cumulative incidence of pancreatic cancer in 41,669 patients diagnosed with acute pancreatitis and 208,340 comparison subjects. Full line: Patients with acute pancreatitis; dashed line: Matched comparison cohort.

CLINICAL—PANCREAS

Mortality, Cancer, and Comorbidities Associated With Chronic Pancreatitis: A Danish Nationwide Matched-Cohort Study

Ulrich Christian Bang,¹ Thomas Benfield,^{2,3} Lars Hyldstrup,^{1,3} Flemming Bendtsen,^{3,4} and Jens-Erik Beck Jensen^{1,3}



Supplementary Figure 1. Mortality rates in patients with CP and age- and sex-matched controls.

Mortality, Cancer, and Comorbidities Associated With Chronic Pancreatitis: A Danish Nationwide Matched-Cohort Study

Ulrich Christian Bang,¹ Thomas Benfield,^{2,3} Lars Hylstrup,^{1,3} Flemming Bendtsen,^{3,4} and Jens-Erik Beck Jensen^{1,3}

Table 2. Causes of Mortality Associated With CP Compared With Controls

	CP	Controls	HR ^a	95% CI
Number	11,972	119,720		
Person-years	71,814	917,436		
	% of total	% of total		
Death from all causes	46.4	13.0	5.0	4.8–5.2
Malignancies	10.2	3.3	1.4	1.3–1.5
Alimentary tract	10.6	0.4	26.1	23.1–29.4
Circulatory system	5.5	3.2	1.9	1.7–2.1
Respiratory system	2.8	1.0	3.3	2.8–3.8
Endocrine disorder	2.2	0.4	4.2	3.6–4.9
Psychiatric disorder	2.1	0.4	6.3	5.4–7.5
Infectious disease	0.6	0.1	4.4	3.2–6.0
Suicide	0.4	0.1	3.5	2.6–4.7
Accident	1.5	0.3	4.1	3.5–5.0
Missing diagnosis	7.8	2.7	N/A	N/A
Other diagnosis	2.1	1.1	1.3	1.1–1.4

HR, hazard ratio.

^aAdjusted for Charlson index and socioeconomic status.

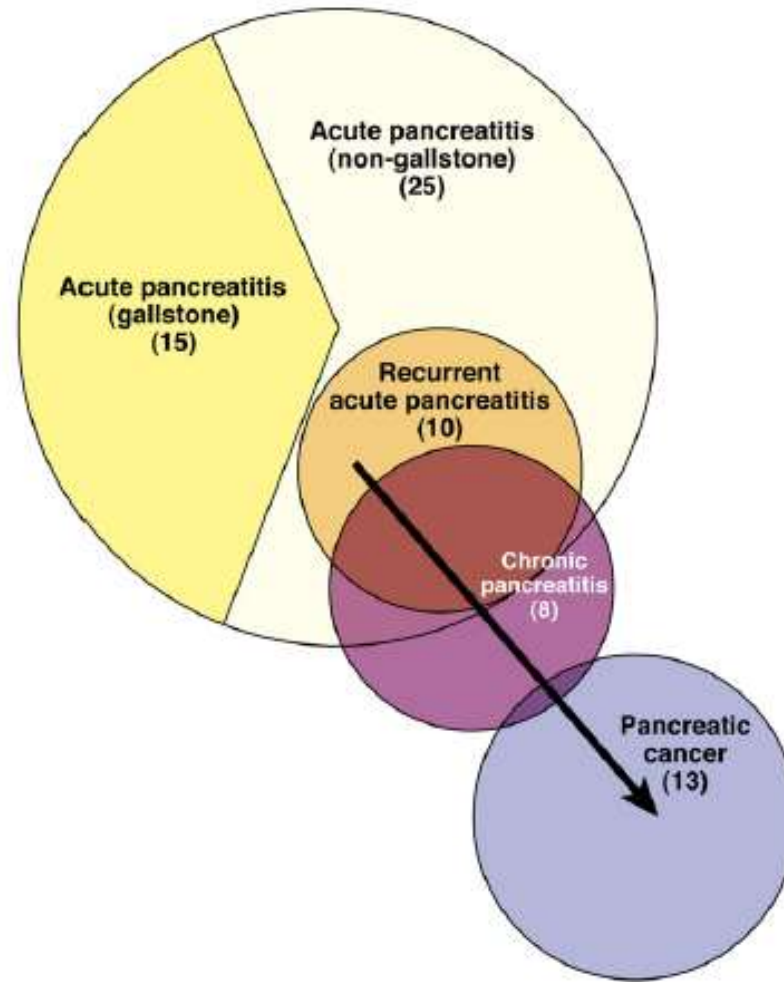


Figure 1. Incidence rates for pancreatitis and pancreatic cancer in the United States. *Numbers in parentheses* indicate approximate yearly incidence rates per 100,000 persons. The *arrow* indicates the relationship between benign and malignant disease. Recurrent AP develops predominantly in patients with non-gallstone-related pancreatitis, although it can develop in patients with gallstone-related pancreatitis when cholecystectomy has been delayed or refused.^{8,9,24}

Risk of Pancreatic Cancer After a Primary Episode of Acute Pancreatitis

- Dutch Pancreatitis Study Group
- Prospectively followed 731 with AP
- 7% progressed to chronic pancreatitis
- Rate Ratio of PDAC in CP 9x higher in CP patients
- Conclusion: Although a first AP episode may be related to PDAC, this risk is mainly in patient who progress to CP

Chronic Pancreatitis and Pancreatic Cancer Risk: A Systematic Review and Meta-analysis

- Meta-analysis of association of CP and PDAC
- 13 studies
- Effect estimates (EEs) stratified by length of follow up from CP to PDAC

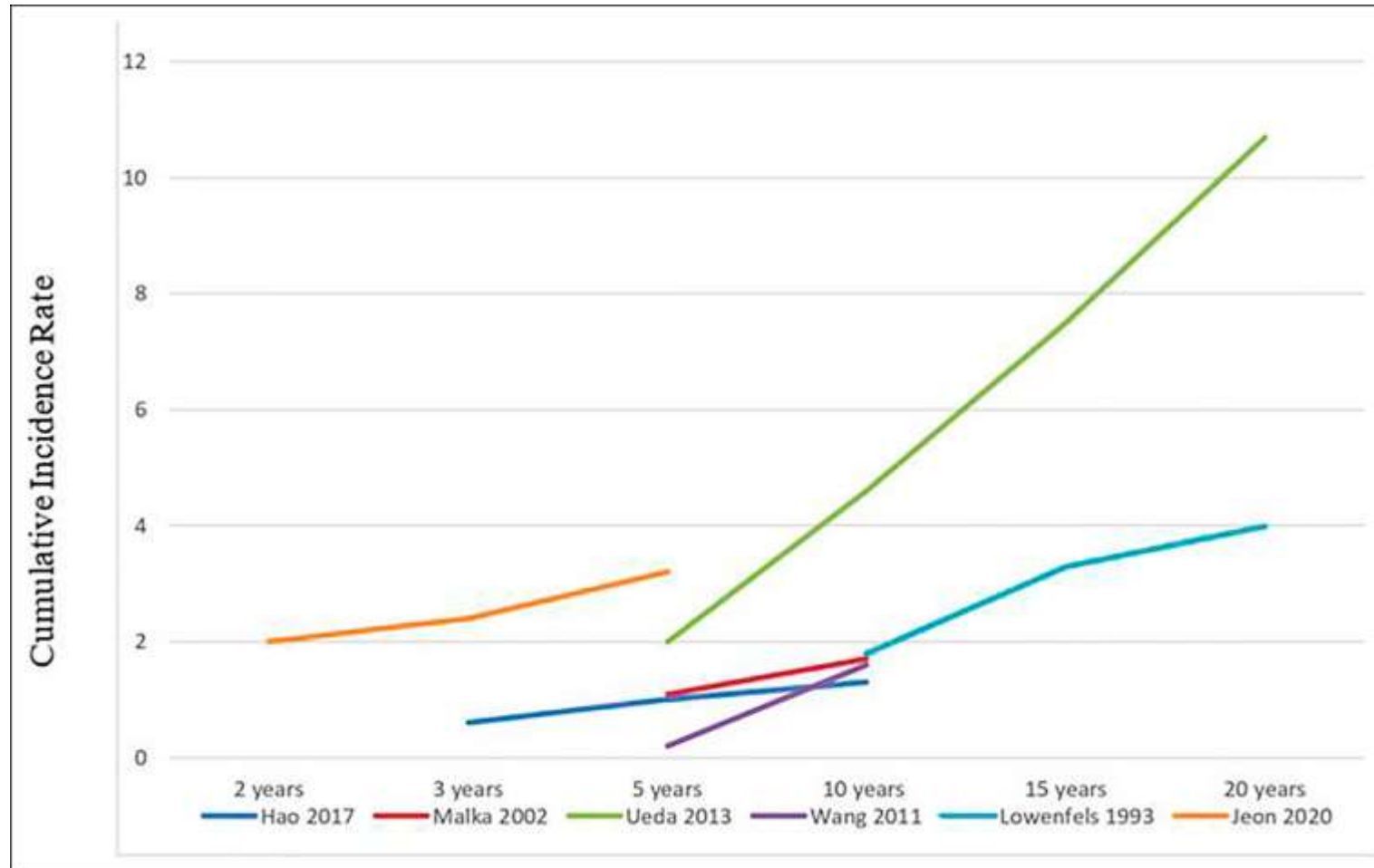
2 years	16.16	(95% CI: 12.59-20.73)
5 years	7.90	(95% CI: 4.26-14.66)
9 years	3.53	(95% CI: 1.69-7.38)

SUPPLEMENTARY MATERIAL is linked to the online version of the paper at <http://www.nature.com/ajg>

Am J Gastroenterol 2017; 112:1366–1372; doi:10.1038/ajg.2017.218; published online 1 August 2017

Chronic Pancreatitis Is a Risk Factor for Pancreatic Cancer, and Incidence Increases With Duration of Disease: A Systematic Review and Meta-analysis

Sonal Gandhi, MBBS¹, Jaime de la Fuente, MD², Mohammad Hassan Murad, MD³ and Shounak Majumder, MD²



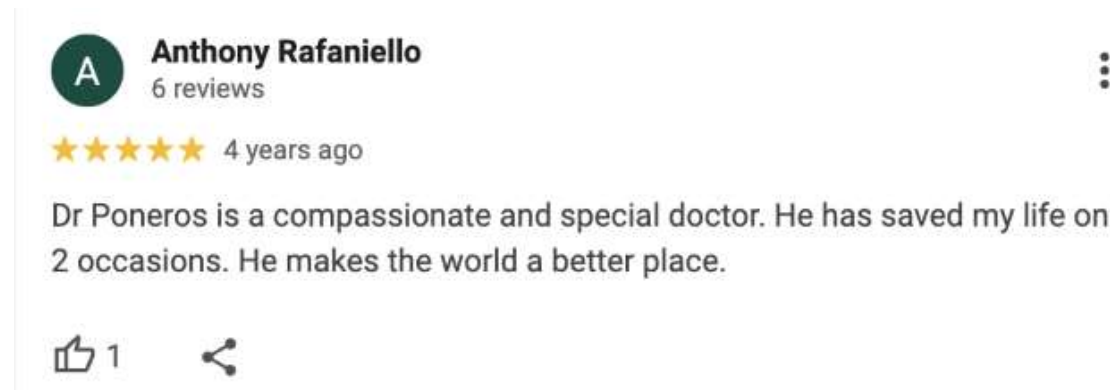
Two Interesting Cases of Pancreatitis

- By 2030, PDAC projected to be 2nd highest cause of US cancer death
- IPMN associated PDACs have better prognosis than non-IPMN associated PDAC
- Colloid IPMN carcinomas have more favourable prognosis than IPMN-PDAC
- ITPN even rarer than IPMN-PDACs (only recognized by WHO in 2010)
- ITPN is less aggressive than other PDAC (even IPMN associated)

Two Interesting Cases of Pancreatitis

“You don’t need to do extraordinary things to achieve extraordinary results”

-Warren Buffett



A screenshot of a review from a medical professional. The reviewer's name is Anthony Rafaniello, with a profile picture showing the letter 'A' and a note of 6 reviews. The review is dated 4 years ago and is a 5-star rating. The text of the review reads: "Dr Poneros is a compassionate and special doctor. He has saved my life on 2 occasions. He makes the world a better place." Below the text are icons for a thumbs up (with a '1') and a share icon.

Pancreatitis Program at CUMC



With the generous support from the Diller Von-Furstenberg Family Foundation, the newly established Pancreatitis Program at The Pancreas Center is dedicated to the treatment of acute and chronic pancreatitis. This multidisciplinary program offers advanced endoscopic care, surgical interventions including Total Pancreatectomy with Auto Islet Transplantation, pain management, genetic counseling, and nutrition services.

Our experienced medical team will streamline the care of patients living with this debilitating disease. The program strives to improve patients' quality of life through a personalized approach to these highly challenging cases. We understand that every patient's case is unique.

If you would like to refer a patient or learn more about the program please call 212-305-4795 or visit our website, www.pancreascenter.com

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