生病的故事討論會

acute pancreatitis→Chronic p.→Pancreatic cancer

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2025.03.28

- Acute pancreatitis,
- repeated
- Chronic pancreatitis
- Calcification

Etiology

Pancreatic cancer

疾病有長程的故事,從查明原因(Past).到現階段的治療(Present illness)—以後還會怎麼樣(Future) 這些都是生病的故事要探討的

- Disease =problems
- Past : Roots
- Past : Risk factors
- PI—>RR.SOAP
- Management and response
- Outcome—near future
- Future

生病的故事討論會

臨床推理.

Problems 解析

思考問題如何發生

有無危險性

確定診斷及治療方針

注意治療反應

推定治療效果及預後

後續應該注意的事項

減少合併症

減少再發

減少再入院

降低死亡率

注意討論會之結論

完成疾病劇本

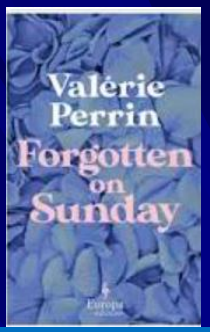
疾病的三段故事

- Acute pancreatitis

Chronic pancreatitis

- Pancreatic cancer
- Pancreatic cancer
- Pancreatic cancer

兩段胰臟炎的故事,走進生命的故事研討會



- 醫學上的兩段故事,卻是綿延相關的事件,每一段都值得深入考量.兩段之間有密切的關聯.因果相連.問題是為什麼發病(roots), 喝酒引起的問題還是家族因素.**每一段都要暢所欲言. 仔細推敲.**故事即將開演.布幕緩緩升起.

- 也許每個人都有兩段人生一段能暢所欲言,另一段則絕口不提,讓沉默遮掩難言之隱
- 瓦萊莉.貝涵
- (瓦萊莉．貝涵 Valerie Perrin
- 1967- 法國新一代小說明星,)
- 48歲出第一本小說創作《星期天被遺忘的人》，2015年在法國藉由口碑效應，持續醱酵，2018年法國書店員票選為「年度最愛」，讀者留言深情擁戴，九千位讀者4.5顆星感動好評。

布幕緩緩升起，生病的故事要開演了

一定是生病了去看病
一小部分是健檢發現的

門診/急診--→住院

重點

- 1.病史摘要—注意問題重點-Problem list →臨床推理的開始,
- →最可能的 診斷
- 2. 尋找**診斷依據**—Lab. Images and others.
- Dx. Evidence—characteristic symptoms, signs, lab.
- And images.
- 3. 從一開始想到以後可能的變化以及後果.—從小到大,一生
- 的影響. 避免癌症/重要合併症
- **疾病原因(Roots)**—可否避免,
- 有發病之**危險因素(risk factors)**特別是是一些生活習慣.
- **減少再發是衛教重點**-→Chronic pancreatitis
- 4. 治療及生活改變 Goal of therapy
- 5. 新觀念 Advance in etiology, Dx. And Rx..

有兩段病史，終身困擾

-喝酒造成的問題

- 從小時候—就有喝酒的經驗
- 2016，第一次出現急性胰臟炎的明顯病史，
- 住院確診,(有依據)
- 2016-2024.05 間斷出現急性胰臟炎，
- 糖尿病低血糖症+胰臟鈣化
- 2024.05.慢性胰臟炎+ 胰臟外分泌缺乏症EPI
- 再入院補充胰臟酵素後改善
- 2024.07-2044:如何發展.且聽下回分解

他有兩段病史: 2016病史概要-1

- 病人是某化妝品公司的產品經理, 男性,35歲, 已婚,育有一男一女.**平時愛喝酒**,下班后常與好朋友喝酒聊天.9點以後才回家
- 2016,03.03. 他的大學好朋友從美國回國, 他約4名老同學陪他一起吃晚餐,(6:30pm) 也**喝了不少酒**. **9 pm開始覺得上腹部不舒服**, 有些噁心(當時還在居酒屋餐廳).9:50 pm 回到家後疼痛加劇. 並出現嘔吐現象. 不過吐得不多.病人以前喝酒也常有**胃痛**現象.不以為意.找2包強胃散.就坐在沙發上休息看電視影片 .

2016病史概要-2

- **疼痛斷斷續續, 沒有減輕**.他喝了1杯熱牛奶上床休息.一個小時後,(11pm)疼痛越來越厲害.吐了兩口, 只好坐起來.他發現吐的時候, 胃痛加劇. 但是**向前彎曲的時候**卻感覺好一些.躺平不能減輕疼痛.
- 疼痛越來越厲害,也有畏冷現象11:55 pm 由太太陪同到醫院急診.**2016.03.04 清晨0:15 抵達急診.**

急診醫師問什麼1.(問清楚病人的問題)

- 1.何時開始疼痛: 9 pm, 2016.03.03
- 2.疼痛的位置:上腹部
- 3.會不會痛到背後: (+),轉到右肩部(-)
- 4. 是悶痛還是絞痛—開始是悶悶的不舒服.疼痛一直增加.11pm以後**腹痛如絞**.吃藥沒有好轉,吐時疼痛加劇, **向前彎曲覺得舒服一點**.
- 5.**從前有過這樣的情形嗎--有**,喝了酒以後有點胃痛,以為是喝酒引起的胃炎,吃吃胃藥就好了.沒看醫師.

急診醫師問什麼2

- 6.有噁心嘔吐嘛?—(+), 吐不多, 吐了兩次,最近的一次是來急診之前,吐不多,只吐一小口,黃黃的水,大約是是11點40分
- 7.胃口受影響嘛?—不太想吃
- 8.有發燒嘛: 沒有, 但11:50 pm 出門時有一點畏寒.
- 9.**以前看過急診嘛—從來沒有**
- 10..喝酒后會胃痛, 有看過醫師嗎?—2-3 年前看過兩三次, ,醫師勸我少喝酒.我還是想喝.

急診醫師問什麼?(問清楚)

- 1.晚餐吃什麼---海鮮,白斬雞,蝦子,雞湯, --不少
- 2. 喝什麼酒? 喝多少?
- 紅酒, 大家一起喝5-6瓶. 病人說他喝2瓶以上, 比平常多一倍. (12%x2000 cc=240 gm?)
- 3.以前常常喝酒嘛? **最近6年(上班以後)幾乎**
- **每2天就喝一次,量也不少.**
- 4.幾歲開始喝酒? 11歲, 國小五年級就開始喝酒,爸爸愛喝酒,海量, 從小就訓練我喝酒. 大一以後比較常喝.啤酒,紅洒,Whisky,大約一個星期喝一次,量不多(紅洒一瓶,或Whisky半瓶)

醫師詢問: 您為什麼愛喝酒?-關鍵.
問題發生在家庭/在當兵時

- 病人解釋:
- 1. 家學淵源, **我們家有喝酒的基因**, 爸爸愛喝酒,從小就訓練我喝酒.學生時代我喝酒爸爸不會管我.
- 2.當兵的時候,**喝酒**的機會多了.養成常喝酒的習慣.而且越喝越多.
- 3.就業以後,我是產品經理,業務的關係常常要喝酒,太太是我的同事,也不禁止我喝酒.
- 4.我不抽煙,只愛喝酒

醫師詢問: 知道喝酒的壞處嗎-關鍵

- 病人回答?
- 1. 除了花錢以外,有什麼壞處?
- 2.喝酒很舒服呀.可以跟好朋友聊天,開玩笑,很好啊.
  3.可以說喝酒也是工作的一部分

- 醫師再問: 你肝功能怎麼樣,
- 病人回答: 還好
- 醫師問:有沒有得過急性胰臟炎?
- 病人回答:沒有
- 醫師問:過去有得過什麼病嗎.
- 病人回答:沒有,**我很健康**

你知道喝酒會引發一些問題?---(真不知道嘛?)

要他戒酒會有難度,隱約暗示會成為慢性病

醫師詢問(ROS)

- 1.平常大小便怎麼樣? –正常
- 2. 您體重有增加嘛---有, 6年前做過體檢只有60公斤, 現在已經72公斤.
- 3.您身高多少? 164 cm,---BMI 為26.8
- 4.你有運動嘛.---很少,這兩年,孩子大一些我帶孩子去公園走走.一個月2-3 次出去走走.
- 5.你有高血脂嘛– 3年前的檢查說膽固醇高一點(248).沒有特別治療
- 6.您有糖尿病嘛—沒有, 我爸爸媽媽都沒有糖尿病(以後發生糖尿病就是胰臟炎的合併症)

醫師詢問(Drug allergy)
@一定要問

- 您對藥物或食物有過敏嘛?
- ---都沒有過敏.

醫師詢問: 您父母親的健康狀況如何?

爸爸愛喝酒,有沒有得過急性胰臟炎或胰臟癌

- 媽媽現年64, 健康狀況良好
- 爸爸在年輕的時候也得過胰臟炎,(30+) 去年因為胰臟癌過世, 享年69.

醫師詢問:您有兄弟姐妹嗎？
病人回答：我是獨生子. 沒有兄弟姐妹

醫師詢問: 你的上上一代(你的阿公阿嬤)有得到胰臟的疾病嗎?
病人回答: 我不知道
母親補充: 就我所知也沒有, 聽說祖父也很喜歡喝酒

一連串的問題他才會知道喝酒不好
: 病人的爸爸發生胰臟癌可能是慢性胰臟炎的結果.

FH 重要結論

- 1.爸爸跟兒子(病人)都喜歡喝酒
- 2.爸爸跟兒子都發生過胰臟炎，可能都是酒精引起,有沒有家族因素?
- 3. 病人的父親可能有慢性胰臟炎,後來發生胰臟癌，逝世時69歲

或許有家族性胰臟炎(Hereditary pancreatitis)的可能性,
或只是因為愛喝酒引起胰臟炎,環境因素而不是家族因素
值得進行基因的檢查，確認是否有家族因素

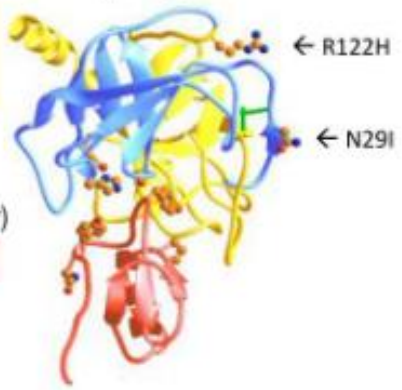
@@趕快查書-hereditary pancreatitis是什么病?

好好問家族病史:often neglected (Family history)

Mendelian: autosomal dominant

- **Hereditary pancreatitis** (HP): multiple large pedigrees
  - **Acute Pancreatitis** in 80% with the gene
  - **Chronic Pancreatitis** in 50% with acute pancreatitis
  - **Pancreatic Cancer** in >40% with chronic pancreatitis.
- Gene: cationic trypsinogen (PRSS1)*
- Variants: "Gain of function"
  - Increase **activation**
  - decreasing **inactivation**.
- HP – illustrated:
  - Acute Pancreatitis (first)
  - Chronic Pancreatitis and complications (later)
  - Suggested a "two hit" CP model (SAPE)**.

Trypsin/SPINK1

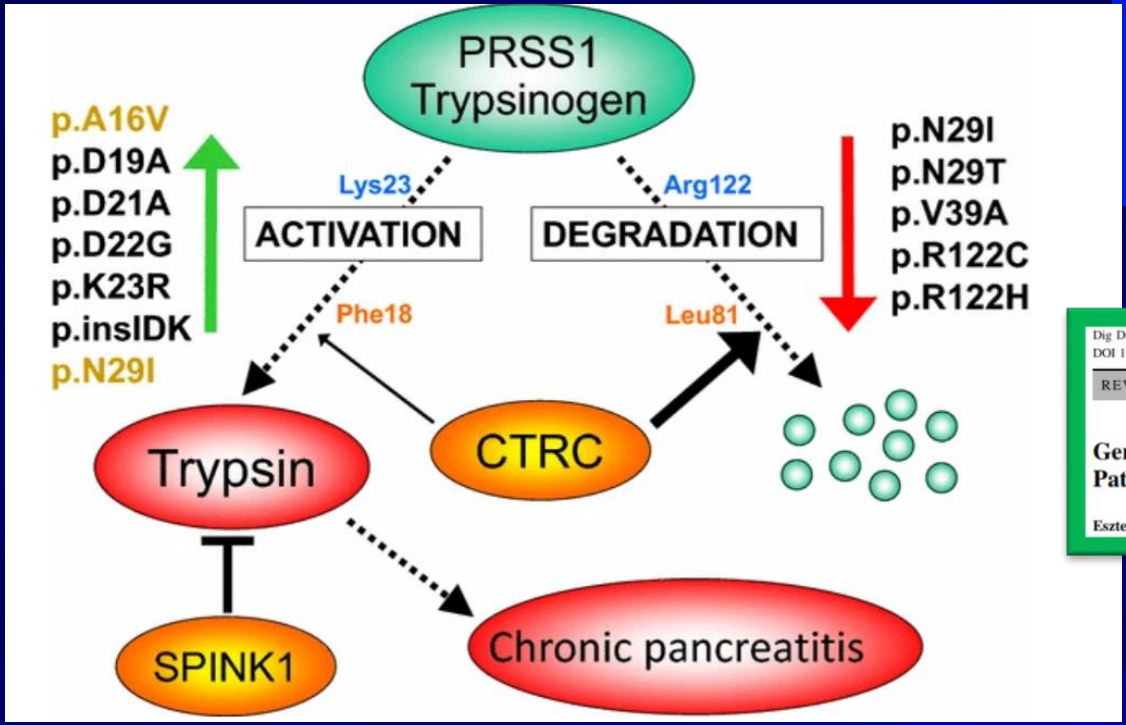


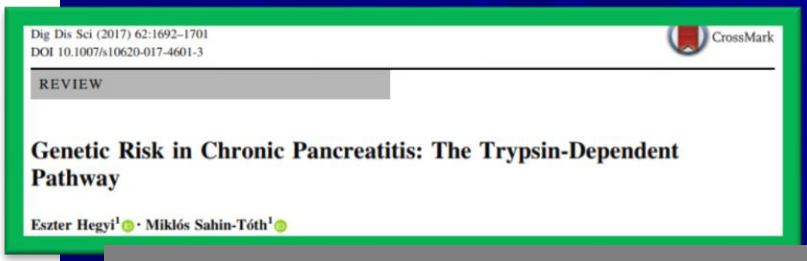
* Whitcomb et al, Nature Genetics, 1996
** Whitcomb, Gut 1999

Hereditary pancreatitis is caused by a mutation in the cationic trypsinogen gene. Whitcomb DC, Gorry MC, Preston RA et al : Nat Genet. 1996 Oct;14(2):141-5. doi: 10.1038/ng1096-141.
(Dept of Medicine, University of Pittsburgh School of Medicine, Pennsylvania)

- 好好問病史
- 至親有多少人得過胰臟炎
- @幾歲發病?
- @有chronic pancreatitis?
- @有pancreatic cancer?
- 三代以內有兩例胰臟炎就要思考家族因素
- 是否是遺傳性的疾病就要考慮做基因檢查來確定

慢性胰臟炎的胰蛋白酶
PRSS1Trypsinogen
→Trypsin
依賴性病理途徑




Dig Dis Sci (2017) 62:1692–1701
DOI 10.1007/s10620-017-4601-3

CrossMark

REVIEW

Genetic Risk in Chronic Pancreatitis: The Trypsin-Dependent Pathway

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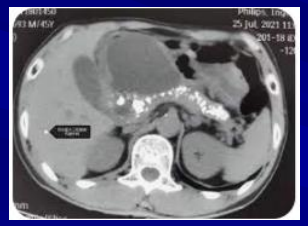
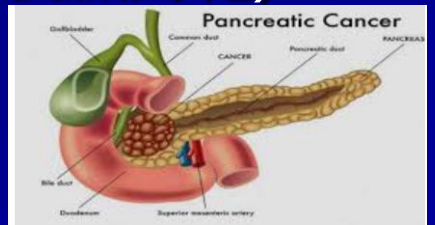
慢性胰臟炎的胰蛋白酶依賴性病理途徑。胰臟中 PRSS1 胰蛋白酶原活化為活性胰蛋白酶是疾病發生和進展的原因。控制胰蛋白酶原活化的保護機制包括 SPINK1 抑制胰蛋白酶以及胰凝乳蛋白酶 C (CTRC) 和胰蛋白酶降解胰蛋白酶原。 CTRC裂解Leu81-Glu82勝肽鍵，胰蛋白酶裂解Arg122-Val123勝肽鍵；這兩種裂解的結合導致不可逆的胰蛋白酶原降解。 CTRC 也透過裂解活化勝肽中的 Phe18-Asp19 肽鍵來刺激陽離子胰蛋白酶原的自活化。縮短的活化勝肽更容易受到胰蛋白酶介導的 Lys23-Ile24 肽鍵活化。所示的遺傳性胰臟炎相關的 PRSS1 突變透過抑制 CTRC 依賴性胰蛋白酶原降解（紅色箭頭）或透過增加 CTRC 依賴性自動活化刺激（綠色箭頭，橘色類型的突變）來增加胰蛋白酶原自動活化。活化勝肽突變直接刺激自激活，與 CTRC 功能無關（綠色箭頭，黑色類型突變）。 SPINK1 的功能缺失突變會降低抑制劑的表達，進而損害胰蛋白酶的抑製作用。 CTRC 的功能喪失突變會減少分泌、阻止酶原活化、降低催化活性或促進胰蛋白酶的降解，從而損害保護性胰蛋白酶原降解

Etiology FRQ: which patients should be tested for pancreatitis-associated genetic abnormalities?

A search for PRSS1 and SPINK1 gene abnormalities should be considered in patients with juvenile pancreatitis or CP of unknown origin and a family history. • Itis hoped that greater consensus will be reached on the genes to be analyzed, gene counseling for mutation positive individuals, and pancreatic cancer screening methods in patients with hereditary pancreatitis. Since the report identifying mutation of the cationic trypsinogen (PRSS1) gene as the cause of hereditary 123 712 J Gastroenterol (2022) 57:709–724 pancreatitis in 1996 [25], abnormalities in various pan creatitis-associated genes, including cystic fibrosis mem brane conductance regulator (CFTR) [26, 27], pancreatic secretory trypsin inhibitor (SPINK1)[28], chymotrypsin C (CTRC) [29], carboxypeptidase A1 (CPA1) [30], and cal cium ion channel TRPV6 [31], have been reported. In the Japan National Survey of Hereditary Pancreatitis [32], mutations in PRSS1 were found in 30 of 73 families (41.1%) and mutations in SPINK1 in 26 (35.6%). Genetic testing for PRSS1 and SPINK1 in patients with hereditary pancreatitis has been reported in Europe and the US [33, 34]. Furthermore, the American College of Gastroen terology guideline for CP [35] recommends that genetic tests for PRSS1, SPINK1, CFTR, and CTRC be performed for CP of unknown origin, especially in young patients. In the 2019 clinical diagnostic criteria for CP [3, 4], mutations in established pancreatitis-associated genes, such as PRSS1 and SPINK1, are included in the diagnostic items for early stage CP, and the role of genetic testing in daily clinical practice is increasing. However, pancreatitis-associated genetic tests are currently not covered by national health insurance in Japan, and it is still unclear how abnormalities in SPINK1 should be handled in the diagnostic criteria for hereditary pancreatitis. It is hoped that greater consensus will be reached by combining the genes to be analyzed in the same genetic test, genetic counseling for mutation positive individuals, and improved screening methods for pancreatic cancer in patients with hereditary pancreatitis.

L1553,L1554: Kyoko Shimizu et al : Evidence-based clinical practice guidelines for chronic pancreatitis. 2021. J Gastroenterol (2022) 57:709–724

疾病的發展不一定按照教科書

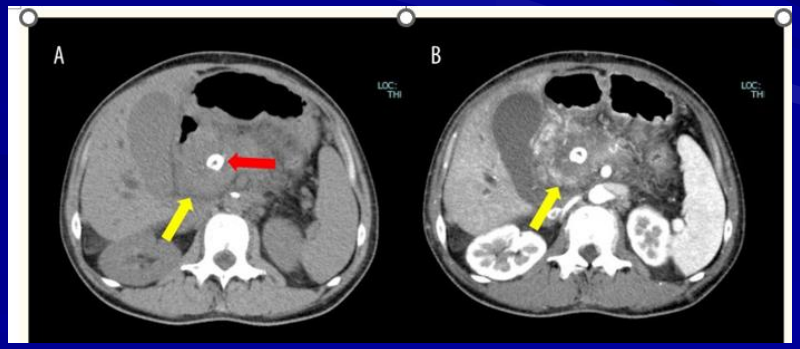
- 1.標準的形態:標準的家族史+
- AP-→CP----------→Pancreatic cancer
- 2.CP 才被診斷(DM?)(Exocrine PI)
- ap(年齡太小發病,沒被診斷)→CP(CT發現胰臟鈣化)
- 
胰臟鈣化 -→ Pancreatic cancer
- 3. Pancreatic cancer才被診斷
- ap-------→cp(出現胰臟鈣化)-→Pancreatic cancer
- 

painless, asymptomatic
- obstructive jaundice

FH(+) Japan(2021): 6歲起上腹痛 18歲出現慢性胰臟炎→DM→39歲胰臟癌

- 一名 39 歲的女性前往診所就診，主訴上腹部疼痛和黃疸。她被懷疑因胰腺腫瘤而患有阻塞性黃疸，並被轉診到我院。
- 她從6歲起就有復發性上腹痛的既往病史。
- 18歲時，她被發現胰腺鈣化，並被診斷出患有慢性胰腺炎。該患者還患有2型糖尿病，並接受藥物治療。她既不喝酒也不抽煙
- FH:她的家族史表明，她的父親在年輕時患上了慢性胰腺炎，並在 56 歲時去世·
- Lab :血液檢查顯示澱粉酶（29 U/L）和脂肪酶（1 U/L）水準下降
- 而肝膽酶水準升高，包括總膽紅素（14.3 mg/dL）、天冬氨酸氨基轉移酶（45 U/L）、丙氨酸轉氨酶（41 U/L）、
- **γ-谷氨醯轉肽酶（837 U/**L）和鹼性磷酸酶（2924 U/L）。
- CEA（10.7 ng/mL）、CA19-9（>12000.0 U/mL）和 DUPAN-2（724 U/m
- $HbA1_c$高達10.2%。

（CT） 掃描顯示胰腺鈣化，胰頭邊緣腫塊直徑約 40 mm（**數位1A–1B**). 紅色箭頭指出鈣化點



Shogo Ota, et al An Autopsy Case of Anaplastic Carcinoma of the Pancreas in a 39-Year-Old Woman that Developed from Hereditary Pancreatitis. Am J Case Rep. 2021; 22: e928993-1–e928993-9.

故事發展很快.

- Hereditary pancreatitis
- 很早發生胰臟炎的癥狀,
- 發展很快-→變成慢性胰臟炎
- 然後就是胰臟癌結束一生
- 39歲結束一生.
- 沒有機會變老

Important and most recent references from the **PubMed**

J Gastrointest Cancer. 2014 Mar;45(1):22-6. doi: 10.1007/s12029-013-9559-6.

Hereditary pancreatitis: dilemmas in differential diagnosis and therapeutic approach.

Mastoraki A[1], Tzortzopoulou A, Tsela S, Danias N, Sakorafas G, Smyrniotis V, Arkadopoulos N.

HP usually appears with an acute, a recurrent acute, and a chronic phase, referring to the inflammation of the pancreas and the symptoms' onset and duration. The clinical features of acute pancreatitis begin in childhood and last less than 6 months. HP carries a 50-70-fold increased risk of pancreatic cancer within 7-30 years of disease onset.

@@
@

J Gastroenterol. 2018 Jan;53(1):152-160. doi: 10.1007/s00535-017-1388-0. Epub 2017 Aug 31.

Nationwide survey of hereditary pancreatitis in Japan.

Masamune A[1], Kikuta K[2], Hamada S[2], Nakano E[2], Kume K[2], Inui A[3], Shimizu T[4], Takeyama Y[5], Nio M[6], Shimosegawa T[2].

271 cases(153 males and 118 females) in 100 families. 41% had the PRSS1 mutations (p.R122H 33%, p.N29I 8%) and 36% had the SPINK1 mutations . DM :5.5% at 20,28.2 % at 40. **pancreatic cancer diagnosis was 2.8% at 40 years old, 10.8% at 60 years, and 22.8% at 70 years**

臺灣有遺傳性胰臟炎的案例嘛?

- 有case report

Lin J T et al : **Hereditary pancreatitis in a Chinese family**

J. Clin Gastroenterology1990 Feb;12(1):81-4.

a 31-year-old man and his 60 year old mother

- 或許我們太不重視家族史的詢問,案例報告不多 FH：non-contributory
- 退休20年參加的案例討論至少有**3個案例**家族史有胰臟炎的病史
- 其它的家族性因素

Metabolic surgery for the treatment of hypertriglyceridemia-related **pancreatitis** due to **familial** lipoprotein lipase deficiency.

Hsu SY, Ser KH, Lee WJ.

Surg Obes Relat Dis. 2014 Sep-Oct;10(5):995-8. doi: 10.1016/j.soard.2013.12.004. Epub 2013 Dec 12.

Pancreatic cancer screening in persons with family history of pancreatic cancer

Ming-Chu Chang et al :Amer. J Cancer Res 2017 Feb 1;7(2):357-369.

- A total of three hundred and three risk individuals in 165 families were enrolled with the mean age of 51.1 years, 38.3% of whom were male. A total of 24 of 303 (7.9%) screened individuals had the PRSS1 mutation, and 7/234 (0.3%) had the SPINK1 mutation. Nineteen (6.3%) risk individuals had pancreatic pathology including seven with pancreatic cancer, and four with pancreatic mucinous neoplasms. neoplasms
- PRSS1 mutation: 24/303 (7.9%)
- SPINK1 mutation: 7/234
- Pancreatic cancer 7/303
- 結論: In summary, pancreatic cancer screening may benefit in risk individuals with family history of pancreatic cancer in our population. The diagnostic yield is similar to prior studies. MRCP as initial screening modality is safe and effective.

間接證明Hereditary pancreatitis
在臺灣還是一個重要的問題.
FH 不可以忽略

酒精性胰臟炎也一樣會變慢性胰臟炎有轉成胰臟癌, 活到60 ,70也不少

- **During alcoholic chronic pancreatitis, the cumulative risk of cancer is estimated at 4% after 15 to 20 years. This cumulative risk is higher in hereditary pancreatitis: 19 and 12% in the case of *PRSS1* and *SPINK1* mutations, respectively, at an age of 60 years.**

Guillaume Le Cosquer [1 2] et al : **Pancreatic Cancer in Chronic Pancreatitis: Pathogenesis and Diagnostic Approach** Cancers (Basel). 2023 Jan 26;15(3):761 (L1027)

不一樣的原因, 不一樣的結局,或許結局相同
但活存的時間不同. 疾病的故事,千變萬化難以捉摸

Inherited pancreatic cancer

家族史是長期以來公認的胰臟癌危險因素，也是疾病風險的重要預測因子。
研究表明，大約 5-10% 的胰腺癌患者報告其近親患有胰腺癌[1,2]。
大多數流行病學研究表明，一級親屬 (FDR) 受影響的個體患胰腺癌的風險增加 2 至 3 倍[3-12]
一些研究表明風險更高。瑞典的一項研究報告稱，如果父母至少有一方患有胰腺導管腺癌 (PDAC)，其後代的胰腺癌標準化發病率 (SIR) 為 1.73 [95%CI: 1.13-2.54] 。在國家家族性胰臟腫瘤登記處(NFPTR) 的一項前瞻性研究中，與SEER（監測、流行病學和最終結果）率相比，胰臟癌的SIR 分別為**6.49(2例)和32.0** (三個 胰臟癌)
Family history, cigarette smoking, chronic pancreatitis, and diabetes are well-established risk factors for pancreatic cancer. Pancreatic cancer is fundamentally a genetic disease caused by both inherited and acquired genetic mutations. Family-based heritability analysis reported 36% of pancreatic cancer was due to genetics. Familial pancreatic cancer kindreds and patients affected by certain genetic syndromes, for example HP, PJS, HBOC, FAMMM, and HNPCC.

Inherited Pancreatic Cancer Fei Chen, Nicholas J. Roberts, and Alison P. Klein
(Department of Epidemiology, The Johns Hopkins Bloomberg School of Public Health, Baltimore, MD 21205, USA 2Department of Pathology, The Sol Goldman Pancreatic Cancer Research Center, The Johns Hopkins Medical Institution, Baltimore, MD 21231, USA 3Department of Oncology, The Sol Goldman Pancreatic Cancer Research Center, The Johns Hopkins Medical Institution, Baltimore, MD 21231, USA)
Chin Clin Oncol. 2017 December ; 6(6): 58. doi:10.21037/cco.2017.12.04.

Family pancreatic cancer

- Hemminki K, Li X. Familial and second primary pancreatic cancers: a nationwide epidemiologic study from Sweden. Int J Cancer. 2003 Feb 10; 103(4):525–30.
- @SIRs for pancreatic cancer (1.68, 95% CI 1.16-2.35) and pancreatic adenocarcinoma (1.73, 95% CI 1.13-2.54)
- were increased when a parent presented with pancreatic cancer.
- @SIRs for pancreatic cancer were 10.01 and 7.96 among offspring who were diagnosed before age 50 years
- when parents were diagnosed with squamous cell and adenocarcinoma of the lung, respectively, before age
- 60 years.
- Klein AP, Brune KA, Petersen GM, Goggins M, Tersmette AC, Offerhaus GJA, et al. Prospective risk of pancreatic cancer in familial pancreatic cancer kindreds. Cancer Res. 2004 Apr 1; 64(7): 2634–8.
- 家族性胰腺癌 （FPC） 親屬被定義為至少有一對患有胰腺癌的一級親屬的親屬，而散發性胰腺癌 （SPC） 親屬則定義為沒有這種受影響對的家庭.
- FPC親屬中，這種風險在 3個（32.0;95%CI，10.2-74.7）
- 2個（6.4;CI，1.8-16.4）
- 或1（4.6;CI，0.5-16.4）患有胰腺癌的一級親屬
- 但SPC親屬則沒有（1.8;95%CI.，0.22-6.4）

Table 2

Risk of pancreatic cancer among unrelated family members and among individuals with a first-degree relative with pancreatic cancer in the familial (FPC) kindreds and sporadic (SPC) kindreds

Family status[a]	No. of individuals	Person-years of follow-up	Observed cases	Expected cases[b]
FPC Kindreds	1993	5273	11	1.22
SPC Kindreds	1964	5265	2	1.12
Unrelated	369	1073	1	0.41

Gastroeneterology Clinic North Amer 2025 Mar;54(1):205-221.
doi: 10.1016/j.gtc.2024.09.006. Epub 2024 Nov 7.

Pancreatic Cancer: Screening and Early Detection

Ghada Mohamed[1], Malak Munir[2], Amar Rai[2], Srinivas Gaddam[3]

• [1]Department of Internal Medicine, Lahey Hospital & Medical Center, 41 Mall Road, Burlington, MA 01805, USA.

hereditary pancreatitis, screening may begin as early as the age of 35 to 40 years.

Endoscopic ultrasound and MRI are acceptable tests for screening for pancreatic cancer.

• • Recent advances in biomarker development offer hope for future screening tests that may be applicable not only to high-risk groups but for the general population.

年青族群飲酒風氣漸盛

- 世界衛生組織（WHO）將「酒精依賴」列為影響全球十大疾病之一。在臺灣因酒品市場開放以及取得容易，啤酒、水果酒價格平易近人，年輕族群飲酒風氣漸盛，人們藉飲酒舒緩壓力，就怕無法控制喝酒過量，長期下來形成酒精成癮，衍生出對個人身體的危害，以及各種社會問題不容小覷。

或許是 自由 風氣：現在的大學生很多都有飲酒的習慣
年青族群飲酒風氣漸盛,是一個重要的課題

酗酒的因素

- **1、基因遺傳：**根據國外研究顯示，酒癮也會有家族史基因遺傳，占比高達5至6成！家族有遺傳基因的人，成因複雜且為多因性，罹患酒癮機率比一般人高出許多。
- **2、環境便利性：**取得酒精管道容易越會有酒癮。舉例來說阿拉伯國家禁酒所以酒癮非常少，由於回教徒戒律，也就是便利性越低，發生酒癮的機會自然降低。
- **3、社會及文化習俗：**成長過程中對酒精的態度，與社會文化習俗有關，像是東方人的敬酒文化以及西方國家，常把飲酒當成生活一部分，提高形成酒癮的機率。

4、個性及酒精風險評估：低估酒精的影響性，沒有想太多，不怕因為喝酒造成各種後果。另外個性的影響則為，喜愛追求刺激、冒險的人，只在乎眼前的樂趣與滿足。而過於逃避、自閉、自卑、社交功能退縮、容易畏懼的人，都與造成酒癮有相關性。

5、精神共病者：情緒困擾、憂鬱症患者，**本身習慣借酒**澆愁；焦慮的人常常借酒放縱，引起酒癮機率與一般人相比，通常高出數倍。

醫師寫下病人的問題
喝酒病史

- CC: Intermittent epigastralgia, in a heavy drinker, from 9;00pm, ABOUT 2 HOURS AFTER heavy drinking WITH meal on 2016.03,03
- Total amount of ethyl alcohol taken :more than 200 gm.
- History **of heavy drinking** : at least 6 years.
- Beginning of drinking from 11,
- Duration of drinking : 24 years.

National Institute on Alcohol Abuse and Alcoholism

- NIAAA 將酗酒(Binge Drinking)
- 定義為一種導致血液酒精濃度 (BAC) 達到 0.08%（或每分升 0.08 克酒精）或更高的飲酒模式。對於典型的成年人來說，這種模式相當於在大約 2 小時內飲用 5 杯或更多飲料（男性），或 4 杯或更多飲料（女性).

Excessive drinking過度飲酒

- The term excessive drinking covers several different groups.
- 1. Binge drinkers: men who have more than five drinks on one occasion or women who have more than four drinks. 男人一次喝5杯, 女人一次喝4杯
- 2.Heavy drinkers: men who have more than 15 drinks in a week or women who have more than eight drinks. 一周喝15杯, 女人一周喝8杯
- 3. Women who drink during pregnancy
- **4. Anyone under age 21 who drinks: 21歲以前喝酒**

Nearly one-third of American adults are “excessive” drinkers,

Dr. Robert Brewer, Alcohol Program Lead at the Centers for Disease Control and Prevention (CDC)（2014.11.21)

Harvard Health Publishing
HARVARD MEDICAL SCHOOL

https://www.health.harvard.edu/blog/heavy-drinkers-arent-necessarily-alcoholics-may-almost-alcoholics-201411217539
Accessed on 2024.06.02.

疾病預防控制中心,藥物濫用和心理健康服務管理局

- 約 70% 的美國成年人至少偶爾飲酒，
- 約 30% 的人報告飲酒過量，
- 3.5% 的人飲酒紊亂。
- 重度飲酒者 (10%)
- 酗酒者的比例較高，從每月酗酒一次或兩次的人中的 4%為重度飲酒者,為到每月酗酒 10 次或以上的人中的 30%為重度飲酒者

SAMHSA
Substance Abuse and Mental Health Services Administration

2014 年 11 月 21 日*作者：* ***Patrick J. Skerrett****，哈佛健康出版社前執行編輯.* *https://www.health.harvard.edu/blog/heavy-drinkers-arent-necessarily-alcoholics-may-almost-alcoholics-201411217539. accessed on 2024.06.02.*

Heavy drinker 的定義
簡單實用

- Heavy drinking : 一次飲酒超過80公克酒精就是heavy drinking
- Heavy drinker : 喝酒超過80公克酒精,**一周五次以上,或是一個月10次以上. 一年有6個月以上有heavy drinking 的紀錄就是heavy drinker.**
- **Heavy drinker 5 years**以上就可以造成顯著的酒精性疾病

喝多少酒才會引發急性胰臟炎?

- 1.過去去報告喝酒引起酒精性肝硬化:大約是每日飲
- 酒100公克以上/ 10年之后就有可能發生肝硬化.
- 2.多少酒才會引發急性胰臟炎,過去並沒有很清楚的
- 敘述或報告.
- 3.個人的經驗如下: 最近2星期完全沒有喝酒, 2 小時以內喝40公克引起急性胰臟炎.*(63 男性平時還是常常喝酒而且,量也不少(30-70 gm, 大約10年).* 另一個案例喝超過65公克(平時倒是不喝酒), 大部分的案例都是超過80公克. 超過120公克的案例達到一半. 喝酒 量 越 多 發生胰臟炎的 機會越 多).

一般成人引發急性胰臟炎的最低量是2小時以內飲酒超過40公克

相對安全飲酒量-
一次飲酒不超過30公克

- 常喝酒的人(喝酒史超過10年以上) 一個晚上,喝超過80公克,有很高的機會發生胰臟炎.
- **不常喝酒的人一次喝120公克即有機會發生胰臟炎**

喝酒不超過30公克應該不至於發生急性胰臟炎
@@喝酒30公克以內應該是相對安全飲酒量

Moderate drinking: (中庸度之飲酒): 2x14=28 gm ethyl alcohol,

For men, moderate drinking is two "standard" drinks or less per day. A standard drink (also called drink-equivalent) is 14 grams of pure alcohol.[1]

[1] https://www.verywellhealth.com/how-much-alcohol-is-it-safe-for-men-to-drink-2328962, accessed on 2024.05.30

Problem list

- P1,(main problem): intermittent epigastralgia noted at 9 pm after taking dinner with a lot of alcohol (heavy intake, more than 200 gm ethyl alcohol) in a 35 year old man with long drinking history, more than 20 years.
- P2, family history of acute pancreatitis, his father also suffered from acute pancreatitis, His father died of pancreatic cancer in 2015(at the age of 69)
- P3.over weight noticed for six years. (BMI was 26.7)
- P4. Hyperlipidemia, doubtful, 3 years ago. No treatment was done.

確定發病的時間 (Time of onset of disease)
: 2016.03.03.21:00

找到診斷的依據

- The diagnosis of acute pancreatitis requires that at least two of the following three criteria are met, based on the Revised Atlanta Classification system:
- 1. Characteristic clinical features
- 2. Laboratory evidence
- 3. Imaging suggestive of pancreatitis

Lab. Data-1

- 1.診斷急性胰臟炎或許比較簡單, 確定發生原因比較複雜.常需要詳細問病史,或進一步抽血和照X光CT, ERCP.找出出急性胰臟炎之原因.
- 2.抽血可以確定診斷: Amylase 689, Lipase 1200.(2016.03.04, at 1:00am)
- 3.抽血可以知道發炎的時間以及嚴重度
- Ca : 7.8 mg/dl , CRP: **0.28 (2016.03.04,1am)**-→5.32(2016.03.07 at 8 am)

Lab. Data-2

- 4. TG was 234 mg/dl (2016/03/04)稍高, 但未>1000, 非急性胰臟炎的原因.

  Total cholesterol : 250, LDL-C: 142 , HDL:35
- 5.Total bilirubin 1.2 mg/dl, GGT :**543**
- **GOT : 44, GPT：51**
- 6. WBC：16.580,
- 7. A/G : 3.9/2.4
- 8.BUN: 12, Cr. 1.2

P1, changed

- P1,(main problem): intermittent **epigastralgia noted at 9pm on 2016.03.03**. after taking dinner with a lot of alcohol(heavy intake, more than 200 gm ethyl alcohol) in a 35 year old man with long drinking history, more than 20 years. Epigastralgia became better on bending. Serum amylase and lipase were abnormally high. Serum calcium was low(7.8 mg/dl), WBC was 16500, all suggested severe disease.

P4 changed

- P4. Hyperlipidemia, **doubtful,** 3 years ago. No treatment was done.
- **Changed to** →P4. Hyperlipidemia, 3 years ago. No treatment was done. Present data on entry : cholesterol :265, TG: 234, LDL-C: 182, HDL-C: 35

Add P5

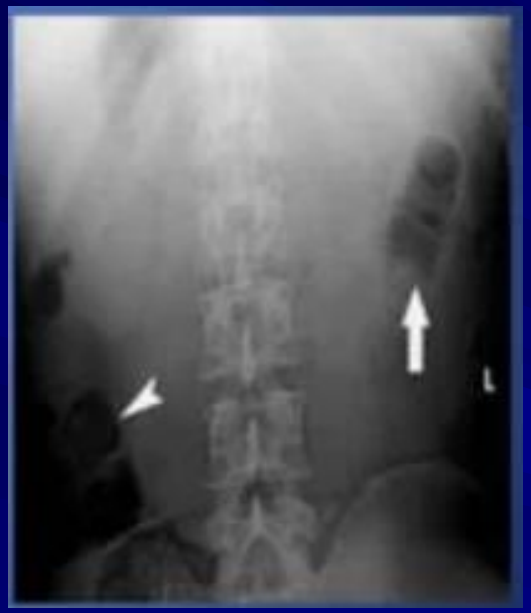
- P5, abnormal liver function **(high GGT)** (slightly abnormal AST and ALT) noticed on entry to emergency.

每一個datum 可能都是關鍵
一定要好好interpretation

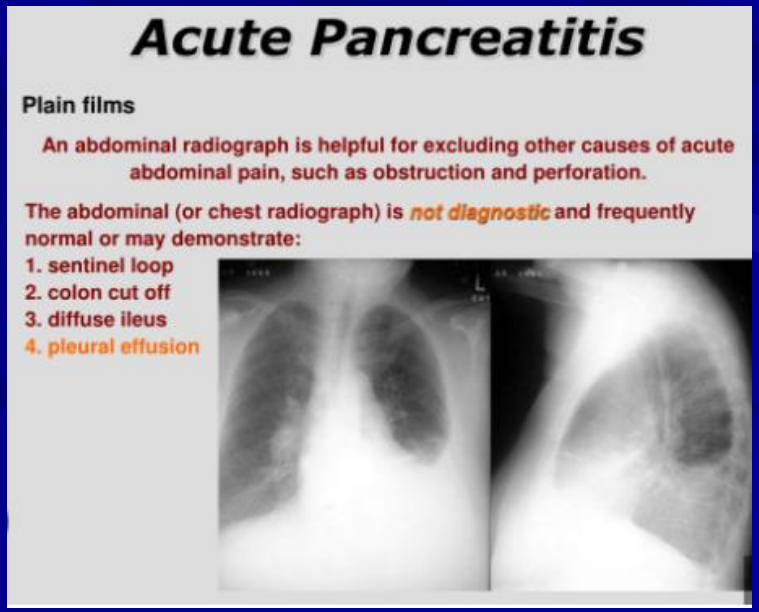
Problem list changed

- P1,(main problem): intermittent epigastralgia noted at 9pm after taking dinner with a lot of alcohol(heavy intake, more than 200 gm ethyl alcohol) in a 35 year old man with long drinking history, more than 20 years. **Serum amylase and lipase were abnormally high. Serum calcium was low(7.8 mg/dl), WBC was 16500, all suggested severe disease.**
- P2, family history of acute pancreatitis, his father also suffered from acute pancreatitis, He was dead of pancreatic cancer in 2015.
- P3.over weight noticed in six years. (BMI was 26.7)
- P4. Hyperlipidemia, 3 years ago. No treatment was done. **Present data on entry : cholesterol :265, TG: 234, LDL-C: 182, HDL-C: 35**
- **P5, abnormal liver function (high GGT)(slightly abnormal AST and ALT) noticed on entry to emergency.**

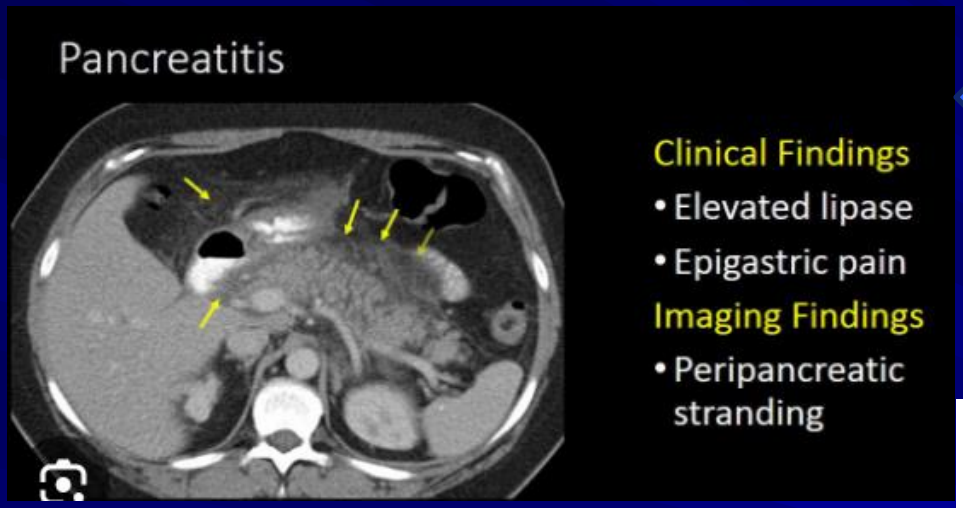
Medical images-1 upright film of abdomen



Sentinel loop.

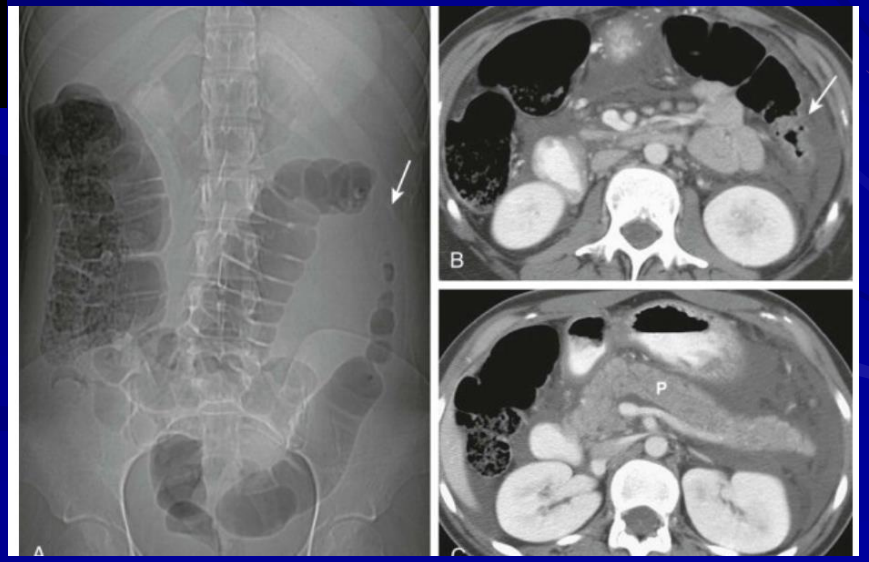


Medical images-2 abdominal CT



Pancreatic stranding

Colon cutoff sign demonstrated by MDCT



Diagnosis @@(要求寫診斷依據)

- Acute severe pancreatitis, probably necrotizing,
- **Dx. Evidence**:.
- 1. Typical history –epigastralgia better on bending, pain
- referred to back
- 2. Abnormal amylase(689) and lipase(1200)
- 3. Radiological evidence_ sentinel loop
- CT showed swollen pancreas and also fluid
- sequestration.—**pancreatic stranding**
- Chest radiography showed left pleural effusion

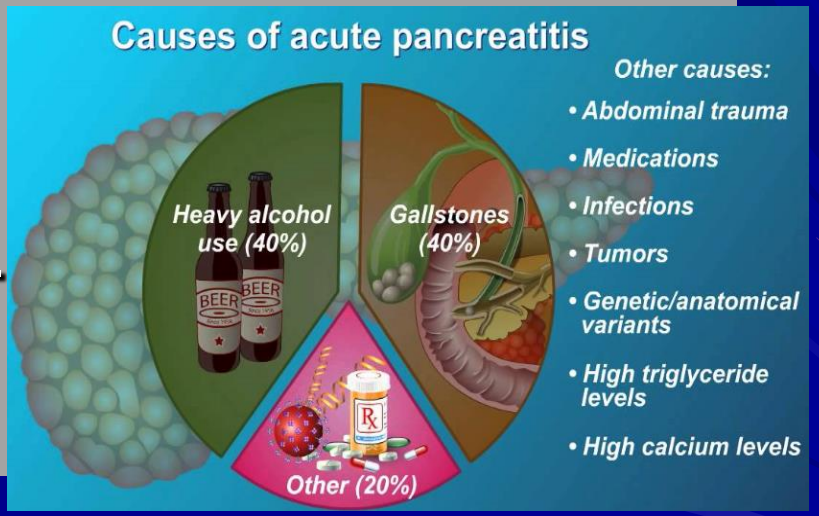
RRSOAP-1

- Roots : alcoholic + HEREDITARY ?
- Risk factors:
  - @prolonged alcohol intake for 20 years or more
  - @ Heavy drinker for at least 6 years
  - @heavy alcohol intake before onset (240 gm or more)
  - @Associated with hyperlipidemia and overweight
  - (? Fatty infiltration)
  - @ low serum calcium,
  - @Evidence of leakage of pancreatic enzyme,-sentinel loop and pleural effusion
  - @familial factors,

RRSOAP-2
Evidence of severe disease

- 1. Low serum calcium,
  2. Evidence of leakage of pancreatic enzyme,
  Sentinel loop and colon cutoff
  Fluid sequestration around the pancreas
  Pleural effusion, left, noticed by chest X-ray

- Causes of acute pancreatitis
- **1. alcohol—by history**
- 2. Gall stone
- 3. Hypertriglyceridemia
- 4. Medicine
- 5. ERCP
- 6. others (FAMILIAL----
- 7. idiopathic



@ **No PRSS1 and SPINK1 gene abnormalities**
@ **Alcoholic pancreatitis, rather than hereditary pancreatitis.**

@好好Review臺灣的報告

前言—1，epidemiology
A. Etiology of acute pancreatitis

- **Etiology of acute pancreatitis--a multi-center study in Taiwan.**
- Chang MC et al (NTUH) :Hepatogastroenterology. 2003 Sep-Oct;50(53):1655-7
  Patients with acute pancreatitis were collected from 8 major leading hospitals located at northern, southern, middle and eastern Taiwan from July 1, 1998 to June 30, 2000.
- 1,193 patients with acute pancreatitis were identified. There were 852 (71.4%) men and 341 (28.6%) women with **a mean age of 52.5 years, ranging from 9 to 100 years**. Etiology was identified as
- **alcohol in 423 (33.6%),**
- **gallstones in 407 (34.1%),**
- **hypertriglyceridemia in 147 (12.3%),**
- **miscellaneous causes in 109 (9.1%), and**
- **idiopathic causes in 107 (9.0%).**
- **Patients with alcohol-related acute pancreatitis were the youngest** (mean age: 41.5 years), while those with gallstone pancreatitis were the eldest (mean age: 64.1 years) ($p < 0.001$). The predominant cause of acute pancreatitis in women is gallstones, while alcohol is the leading cause of acute pancreatitis in Taiwanese males. In northern Taiwan, gallstone is the major cause of acute pancreatitis, while alcohol is the predominant etiology in middle, southern, and eastern Taiwan.

前言—2，epidemiology: Annual incidence in Taiwan ： 36.9/100,000(2000-2009)

- Epidemiology of First-Attack Acute Pancreatitis in Taiwan From 2000 Through 2009: A Nationwide Population-Based Study
- Shen, Hsiu-Nien et al (Pancreas: July 2012 - Volume 41 - Issue 5 - p 696–702.)
- **107,349 patients with first-attack AP** from the Taiwan National Health Insurance Research Database between 2000 and 2009. Severe cases were defined according to a modified Atlanta classification. Incidence rates were standardized by direct method. The averaged annual incidence of first-attack AP was estimated **at 36.9 per 100,000** persons and changed only slightly.
- incidence increased in children (<15 years), elderly people (≥65 years), and patients with biliary cause, but decreased in young to middle-aged men (15–64 years). The prevalence of severe cases increased **from 21.0% to 22.3%,** which was mainly caused by an increase of acute organ dysfunction (from 9.7% to 14.1%). Despite that, **hospital mortality decreased from 4.3% to 3.3**% for all cases and from **18.5% to 13.3% for severe ones.**
- The overall incidence of first-attack AP changed slightly in Taiwan, which differs from the increasing trend observed in most Western countries. Although more patients had severe attacks in recent years, hospital mortality declined.**嚴重的有20 %**

前言—3，epidemiology.
Norway : FAAP:14.6/100,000/year

(First Attack of Acute Pancreatitis)

- **Time trends in incidence, etiology, and case fatality rate of the first attack of acute pancreatitis.** Omdal T et al (U. Bergen, Norway), Scand J Gastroenterol. 2011 Nov;46(11):1389-98
- A total of 874 patients were discharged with a diagnosis of acute pancreatitis from the two hospitals in this region between 1.1.1996 and 31.12.2006. Patient records were reviewed and patients with a verifiable FAAP were identified. Demographic variables, likely etiology, and outcome were registered. FAAP was verified in 567 (65%) of the patients (300 women and 267 men) with a median age of 58 years (range 7-98). **The average yearly incidence rate of FAAP was 14.6/100 000** and the gender-specific incidence rates increased yearly by approx. 6% ($p = 0.006$). There was a decline in diagnoses by s-Amylase from approx. 90% to 62% in 2006 and an increase in diagnoses obtained by CT ($p < 0.001$). **The case fatality rate was low (3.5%), but higher among men (5.8%) than women (2%, $p = 0.037$).** The case fatality rate was lowest among patients with gallstones (0.7%) and higher among patients with alcohol (9%), miscellaneous (10.4%), and non-assessed etiology (6.6%) of FAAP ($p < 0.05$). Male gender, increasing age, and etiology (alcohol, miscellaneous causes, and non-assessed) were associated with increased case fatality rate in an adjusted regression model ($p < 0.001$).
- The incidence rate of FAAP is low and differs from that of official registries. The case fatality rate is low, but related to gender, age, and likely etiology of FAAP.

- 從流行病學看,臺灣喝酒的概況比北歐愛喝酒的國家還厲害.
- _____
- 臺灣:　　36.9/100,000　(2000-2009)
- Norway : 14.6/100,000　(1996-2006)

前言 4:Diabetes Mellitus after First-Attack Acute Pancreatitis (>3 months, 2.5 x)

- **Risk of Diabetes Mellitus after First-Attack Acute Pancreatitis: A National Population-Based Study.** Shen HN[1,2], et al (奇美) Am J Gastroenterol. 2015 Dec;110(12):1698-706.
- Taiwan National Health Insurance.
- 2,966 first-attack AP patients and 11,864 non-AP general controls individually matched on age and sex, with an AP/non-AP ratio of 1:4.
- In the first partition of time (<3 months), the incidences of diabetes were **60.8 and 8.0** per 1,000 person-years in AP and control groups, respectively;
- In the second partition (≥3 months), **the incidences of diabetes were 22.5 and 6.7 per 1,000 person-years in AP and control groups**, respectively (adjusted HR 2.54)
- the risk of diabetes was greater in men than in women (HR 3.21 vs. 1.58)
- 結論:The risk of diabetes increases by twofold after AP.

Causes of acute pancreatitis

- **alcohol in 423 (33.6%),-→History**
- **gallstones in 407 (34.1%),→History, US, 健檢**
- **hypertriglyceridemia in 147 (12.3%), →History and Lab.**
- **miscellaneous causes in 109 (9.1%), and**
- **idiopathic causes in 107 (9.0%). – stone?**

下一步處置

Alcoholic →戒酒

Gallstone

→Laparoscopic cholecystectomy

Hypertriglyceridemia

→Treatment of hyperlipidemia

Diet

Others---?

Idiopathic -→?

History taking 多少可以指出病因

喝酒喝多少才會導致酒精性疾病

- Alcoholic pancreatitis

Personal experience: After taking 40 gm alcohol in a young Man, 50 years ago(1968.)

- Alcoholic liver diseases
- Alcoholic cirrhosis
- Alcoholic dependence

喝酒的量
喝酒的時間

Ans: Ethyl alcohol.>80 gm/drink
Duration of heavy drinking
> 12 years.

喝酒越多,喝酒的時間越長
--Heavy drinking 定義

適量飲酒

- 適量飲酒:
- 根據美國衛生與公共服務部和美國農業部《2020-2025 年美國人飲食指南》，達到法定飲酒年齡的成年人可以選擇不飲酒或適量飲酒，將飲酒量限制在 2 杯或以下(1 杯)，女性每天飲酒1 杯或更少，少飲酒比多飲酒更有利於健康。

- Heavy drinking.
- 重度飲酒
- NIAAA National Institute on Alcohol Abuse and Alcoholism
- 對重度飲酒的定義如下：
  - 對於男性，每天飲酒 5 杯或更多，或每週 15 杯或更多
  - 對於女性，每天飲用 4 杯或更多飲料，或每週飲用 8 杯或更多飲料

某些人應該完全避免飲酒

@**計劃駕駛或操作機器**，或參加需要技能、協調性和警覺性的活動

- 服用某些非處方藥或處方藥
- 有一定的醫療條件
- **正在從酒精使用障礙中恢復或無法控制飲酒量**
- **@年齡小於 21 歲**
- **已懷孕或可能懷孕**

國人幾歲開始飲酒?

- 依據本人2000-2007, 發生酒精性疾病的heavy drinkers 詢問何時開始飲酒.(57 cases)
- Youngest –7歲----------1
- 8-9 (< 10歲)----------4
- 10-14----------26
- 15-19 ---------- 24
- >20---------- 2

半數以上都是15歲以前就開始喝酒

依據國民健康署發布110年青少年健康行為調查之飲酒行為結果2，國中生及高中職生過去30天內飲酒率，分別為14.1%及30.6%，與其他國家相近（西太平洋地區青少年飲酒率約介於15-30%，如澳洲為29.1%、越南為23.7%、韓國為19.4%、日本為15.4%）。

HPA 衛生福利部 國民健康署

發布日：2022/10/06

年輕就開始飲酒是父母親的責任,無意中向朋友炫耀, 從小就教小孩飲酒, 是非常不當的行為.親友絕對不要拍手鼓勵小孩子喝酒.

合法飲酒年齡（Legal drinking age)

- **合法飲酒年齡**（英語：Legal drinking age）是個人可合法飲用酒精飲料的最低年齡。可合法飲酒的最低年齡在不同的國家/地區會有不同。許多法律會包含豁免或是列有特殊情況。
- 大多數法律僅規範在公共場合飲酒，而對在家中飲酒大多未予管制（英國例外，在私人場所進行監督下飲酒的最低合法年齡為5歲）
- 一些國家對不同酒精飲料種類設有不同的年齡限制。[2]大多數國家/地區的最低合法飲酒年齡為18或19歲.
  - 澳洲的研究，對於不滿16歲的未成年人[6]）有關低風險式飲酒和控制飲酒數量的知識，可有效預防日後飲酒風險的發生。[7]

@@美國合法的飲酒年齡是21歲(1984), 臺灣合法的飲酒年齡是18歲,臺灣應該延後一點 (Wang CY, 2025.01.04)

健康的臺灣第一件事情
-當務之急

- 將合法飲酒的年齡延後一點,
- 最好是25歲以後.

Ethn Health. 2018 Apr;23(3):321-328. doi: 10.1080/13557858.2016.1263287. Epub 2016 Dec 1.

Ethnic differences in risk factors of acute pancreatitis.

Ho UC[1], Mu CF[2], Hsu CY[3,4,5,6,7,8,9,10,11].

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原住民的問題
-愛喝酒的習慣產生的疾病

A retrospective study of 622 patients with acute pancreatitis admitted to our hospital (Puli Christian Hospital) from 2006 to 2014. The risk factors and biochemical properties of acute pancreatitis were compared between aborgines and nonaborgines.

RESULTS:

The first episode of acute pancreatitis amongst the aboriginal group was commonly observed in young age groups (39.3 versus 47.8 years, $p < 0.05$), female patients (0.61 versus 0.27, $p < 0.05$), and patients with a habit of drinking alcohol (84% versus 65%, $p < 0.05$). Analysis of the biochemical properties and risk factors demonstrated significantly high uric acid levels (7.63 versus 6.56 mg/dL, $p < 0.05$), and an increased prevalence of alcohol-related pancreatitis (60.0% versus 49.6%, $p < 0.05$) in the aboriginal group.

Table 1: Incidence of acute pancreatitis, chronic pancreatitis and post-pancreatitis diabetes mellitus by ethnicity.

	Incident cases	Population	Crude incidence, %	Incidence rate per 100,000 population per year [95% CI]
Acute pancreatitis				
NZ European	10,063	1,699,938	0.59	60.22 [59.05, 61.39]
Māori	2,872	306,873	0.94	95.21 [91.74, 98.68]
Asian	855	245,523	0.35	35.43 [33.06, 37.80]
Pacific people	736	137,931	0.53	54.28 [50.37, 58.19]
Other	2,227	206,952	1.08	109.47 [104.95, 113.99]

Ethnic and geographic variations in the incidence of pancreatitis and post-pancreatitis diabetes mellitus in New Zealand: a nationwide population-based study

Latest Issue

17th February 2017, Volume 130 Number 1450

Sayali A Pendharkar, Juby Mathew, Jinfeng Zhao, John A Windsor, Daniel J Exeter, Max S Petrov

THE NEW ZEALAND MEDICAL JOURNAL

高脂血症也是引發急性胰臟炎的原因之壹

- 臺灣長庚研究資料庫中確定了 98,819 名在 2007 年 1 月 1 日至 2013 年 12 月 31 日期間被診斷為高脂血症且連續 4 年每年至少進行 1 次甘油三酯測量的患者.
- **平均Follow up 5.9年期間，825例（0.83%）患者新診斷為急性胰腺炎（每10,000人年14.1例).**
- 血清甘油三酯> 500 mg/dL（5.6 mmol/L）時，急性胰腺炎的風險會逐漸增加（8,10）。
- 只有 5% 的血清甘油三酯 >1,000 患者和
- 10%-20% 的血清甘油三酯 >2,000 mg/dL 患者發生急性胰腺炎（11），
- 這表明發生胰腺炎的風險與高甘油三酯血症的關係並不統一

- 在一項針對 17,905 名受試者的研究中，這些受試者於 1976 年至 2007 年在丹麥參加哥本哈根市心臟研究，在平均 20.1 年的隨訪期間，**任何胰腺炎的總體風險為 1.3%[23]**.參與者在確定時完成了一份詳細的問卷，其中包括有關其飲酒和吸煙習慣的資訊。**胰腺炎的風險隨著飲酒量的增加而增加——報告每天飲酒 ≥5 杯的受試者的絕對風險為 ~2.9%，與戒酒者相比，胰腺炎的絕對風險高出 ~3 倍**。

Table 2. Risk of Acute Pancreatitis, Chronic Pancreatitis, and Total Pancreatitis According to Updated Consumption of Alcohol Intake, Copenhagen, Denmark, 1976–2007

Alcohol Intake, drinks/week	Acute Pancreatitis					Chronic I
	No. of Cases	Hazard Ratioa	95% Confidence Interval	Hazard Ratiob	95% Confidence Interval	No. of Cases
0	35	1.0	Referent	1.0	Referent	18
1–6	44	1.2	0.7, 1.8	1.2	0.7, 1.8	25
7–13	36	1.5	0.9, 2.4	1.4	0.9, 2.3	17
14–20	17	1.4	0.8, 2.6	1.3	0.7, 2.4	12
21–34	17	2.0	1.1, 3.6	1.7	0.9, 3.2	9
35–48	13	4.3	2.2, 8.5	3.5	1.8, 7.1	8
>48	9	4.3	1.9, 9.3	3.3	1.5, 7.3	8
		<0.001		<0.001		

23. Kristiansen L， Gronbaek M， Becker U， et al. 飲酒習慣的胰腺炎風險：一項基於人群的佇列研究。 *Am J Epidemiol* 2008;168:932–7

• .Plos One 2016 Oct 11;11(10):e0163984.
doi: 10.1371/journal.pone.0163984. eCollection 2016.

Relationship between Plasma Triglyceride Level and Severity of Hypertriglyceridemic Pancreatitis

Sheng-Huei Wang[1], Yu-Ching Chou[2], Wei-Chuan Shangkuan[3], Kuang-Yu Wei[4], Yu-Han Pan[5], Hung-Che Lin[6]
Affiliations

Affiliations

• [1]Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, Tri-Service General Hospital,
• National Defense Medical Center, Taipei, Taiwan, Republic of China.

1. 66 patients in the low-TG group and 78 patients in the high-TG group
2. The high-TG group had significantly higher levels of glucose (P = 0.022), total cholesterol (P = 0.002), and blood urea nitrogen (P = 0.037), and lower levels of sodium (P = 0.003) and bicarbonate (P = 0.002) than the low-TG group.
3. The incidences of local complication (P = 0.002) and severe and moderate form of pancreatitis (P = 0.004) were significantly higher in the high-TG group than in the low-TG group.
4. The mortality rate was higher in the high-TG group than in the low-TG group (P = 0.07).

急性胰臟炎的後果

- 1.痊癒
- 2.再發
- 3.慢性胰臟炎,糖尿病
- 4慢性胰功能不全(外分泌)
- 5.胰臟癌
- **NEXT:**
- 戒酒是最重要的事,==可是相當困難.
- Pancreatic enzymes 之補充.

2016-2024多次門急診及入院

- 3 次 pancreatitis 入院(2017-2019)
- 1次alcohol withdrawal 入院(2020)
- 1次pulmonary embolism入院(2021) covid?
- 2次hypoglycemic attack --Emergency
- **Regular OPD**
- –GI (~2024)：alcoholic liver disease, chronic pancreatitis
- Psychiatric (~2022) alcohol withdrawal and depression
- Metabolism(~2024), **DM**

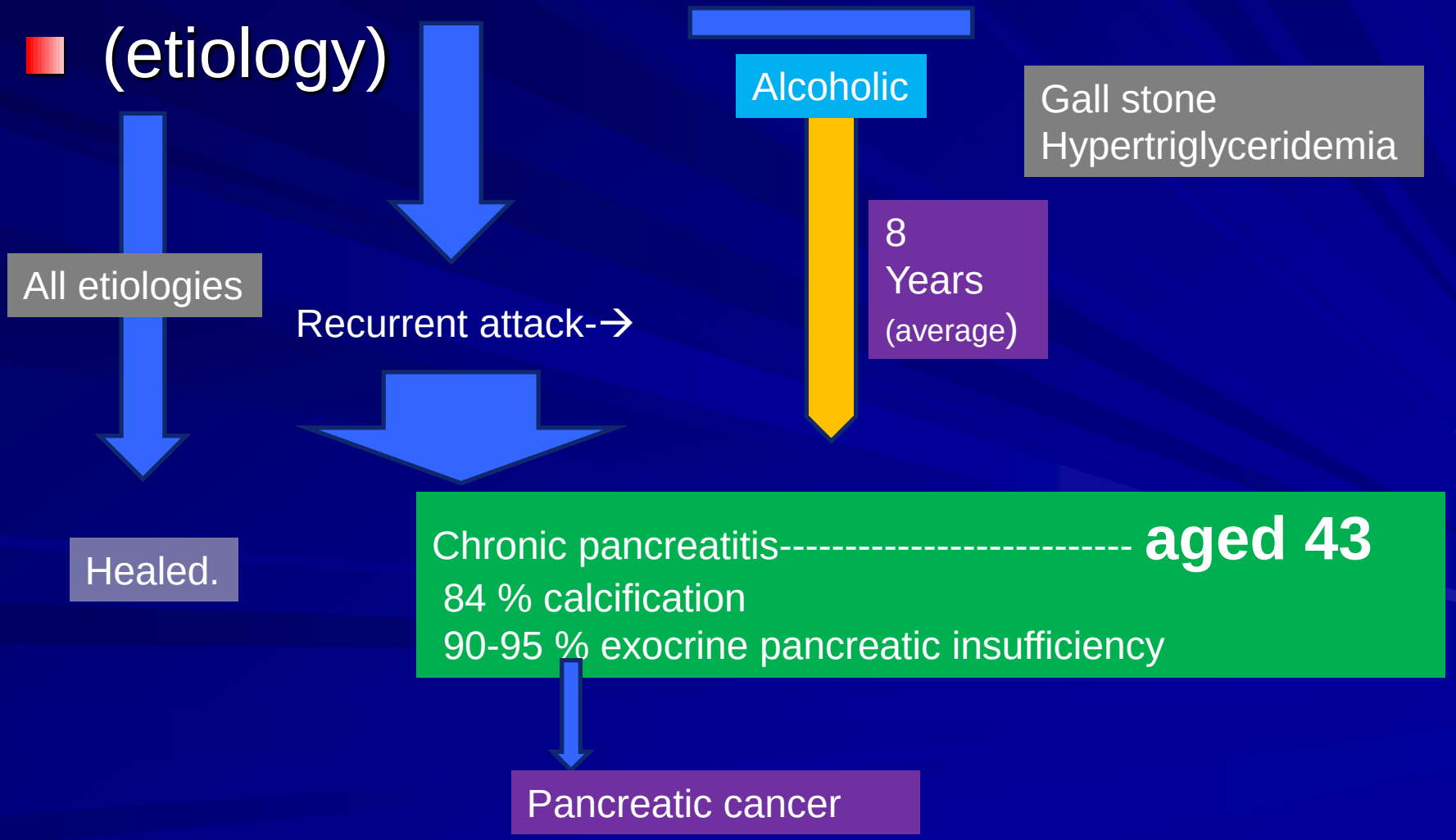
因為沒有及時戒酒,才惹出這些麻煩

Chronic pancreatitis

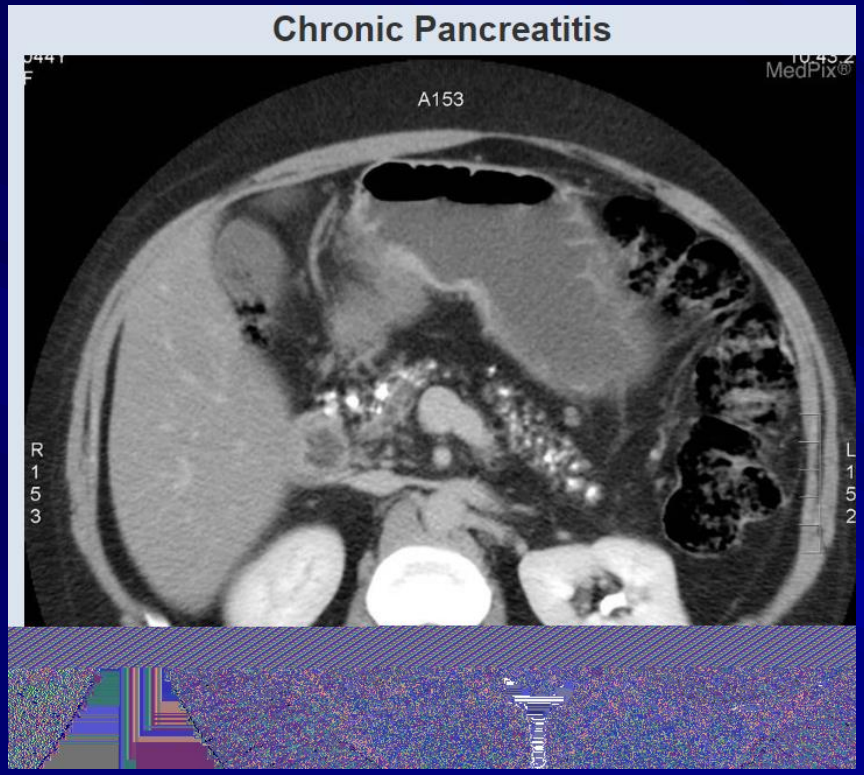
- Chronic alcohol consumption is the single most common cause of chronic pancreatitis, resulting in ~40% to 70% of all cases , and increases an individual's risk of developing pancreatic cancer .
- Recurrent bouts of acute pancreatitis are associated with progression to chronic pancreatitis and are more common in chronic abusers of alcohol.
- Furthermore, most analyses suggest that some degree of chronic pancreatic injury already exists at the time of onset of an AP episode [2].

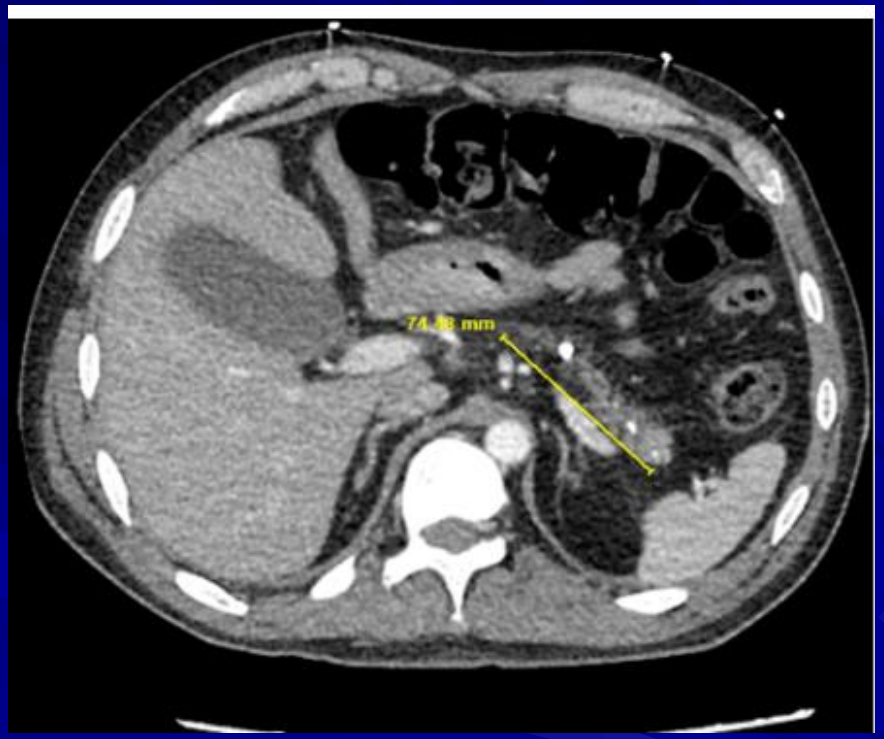
Outcome after acute pancreatitis

- Acute pancreatitis----------aged 35
- (etiology)



Chronic pancreatitis, CT





Findings

Multiple punctate calcifications throughout the uncinate head, body and tail with associated diffuse fatty replacement. No intrapancreatic ductal dilatation is demonstrated. No abnormal areas of enhancement are present. Fatty infiltration of the liver. Calcification is demonstrated within the lower pole of the left kidney.

瀰漫性萎縮的胰腺，伴隨大量實質鈣化

Otero-Colón J、Olivero Y、Virk P 等。（2023 年 5 月 9 日）臨床推理在胰臟功能不全的重要性：病例報告。 Cureus 15(5)：e38760。 doi:10.7759/cureus.38760

exocrine pancreatic insufficiency (EPI)

- 胰腺外分泌功能不全（exocrine pancreatic insufficiency， EPI）定義為由於酶生成、活化或酶降解過早導致的胰酶消化功效不足.
- EPI的發生與多種病理狀況有關，包括自身免疫性胰腺炎、乳糜瀉、炎症性腸病、糖尿病、胰腺腫瘤、囊性纖維化和酒精相關性**慢性胰腺炎.**
- 大多數酒精相關性慢性胰腺炎患者在診斷后10年內出現EPI，幾乎所有患者在20年後出現EPI.

Alcoholic nonprogressive chronic pancreatitis: prospective long-term study of a large cohort with alcoholic acute pancreatitis (1976-1992) Ammann RW, Muellhaupt B, Meyenberger C, Heitz PU. *Pancreas.* 1994;9:365–373. [PubMed]

Outcome: progression to advanced chronic pancreatitis (109/140 ; 77.8%) (84% with calcification, 95% with exocrine insufficiency)

- 140 patients with alcoholic acute (recurrent) pancreatitis were enrolled in a prospective long-term study over the last 16 years.(1976-1992)
- **group A (n = 109; 77.8%) with progression to advanced chronic**
- **pancreatitis (84% with calcification, 95% with exocrine insufficiency)**
group B (n = 31; 22.2%) without progression (no calcification, no
- **exocrine insufficiency).**
- In group B, no pancreatic duct dilatation occurred (in 86% > 8 years from onset). However, 4 of 7 patients with adequate histology showed unequivocal chronic pancreatitis.
- **Our findings indicate that a subgroup of alcoholic acute pancreatitis does not progress to advanced chronic pancreatitis. This subgroup may be identical with "small duct" chronic pancreatitis.**

八年之後:2024.05.又來急診了

- **The patient is a 43-year-old male with a past medical history of polysubstance abuse, alcohol dependence, acute pancreatitis, pulmonary embolism, hypertension, hyperlipidemia and diabetes mellitus type 2 presenting to the Emergency Department with three days of epigastric abdominal pain, nausea and non-bloody, non-bilious vomiting.**

多次胰臟炎住院→CP

- **Past history is significant of multiple admissions for chronic alcohol dependence and various episodes of diagnosed pancreatitis. CT scan of the abdomen and pelvis with contrast was performed and demonstrated a severely atrophic pancreas with numerous parenchymal calcifications which is compatible with sequelae of chronic pancreatitis .**

- The patient's evaluation demonstrated hypokalemia, hypomagnesemia, hypocalcemia, and hypophosphatemia. Repletion was performed through oral and intravenous routes. Despite adequate repletion, **hypomagnesemia and hypokalemia demonstrated to be resistant to treatment.**

- Symptoms were accompanied by occasional nausea, vomiting and diarrhea. Throughout the patient's hospital course, a cyclical pattern emerged of increases in electrolyte concentrations shortly after oral and intravenous administration, with consecutive electrolyte derangement noted thereafter.
- **A trial of pancreatic enzymes was initiated**. The **diagnosis of pancreatic insufficiency** secondary to chronic pancreatitis due to chronic alcohol dependence was achieved.

- Patient was placed on pancreatic enzyme replacement therapy and discharged with continued treatment.
  Unfortunately, the patient was found to be non-compliant with therapy and continues to struggle with alcohol dependence.

- Alcohol dependence,酒精依賴
- Alcohol addition
- **Withdrawal syndrome**, if no alcohol intake

1酒精戒斷症候群（alcoholic withdrawal syndromes）乃酒精成癮者，當減少或完全停止飲酒時所產生的身心的不良反應，屬於急性精神病狀態之一種器質性腦症候群，一般出現在減少或完全停用酒精後48至72小時，且可持續5日。 2.1輕微:失眠、手抖、心悸、輕度焦慮、腸胃系統症候群、頭痛、冒冷汗。 2.2酒精性幻覺：通常在停酒48小時內發生，多為視幻覺或聽幻覺。
2.3抽搐（seizure）：通常在停酒24-48小時出現。
2.4震顫性譫妄（delirium tremens）：約在停酒48-72小時發生

Normal serum Mg:1.7-2.2 mg/dl

- **Undulating potassium and magnesium despite adequate repletion.**
- **Magnesium repletion with Mg sulfate 4-8 gm/day**
- **Serum magnesium**
- **2024.05.03: 1.1 mg/dl**
- **2024.05.04:1.2**
- **2024.05.05. 1.4**
- **2024.05.07: 2.0**
- **2024.05.09: 1.7**
- **2024.05.10: 1.5**
- **2024.05.11: 1.2**
- **2024.05,12: 1.0**
- **2024.05.13: 1.3**
- **2024.05.14: 1.8**

Potassium repletion with oral potassium chloride at 40mEq three to four times daily and IV 10mEq per hour up to 40mEq daily

- Serum potassium

m Eq/L

2024.05.03: 2.0
2024.05.04: 2.1
2924.05.05: 2.6
2024.05.07: 2.7
2024.05.09: 2.4
2024.05.10: 2.8
2024.05.11: 2.3
2024.05.12: 3.6
2024.05.13: 2.8
2024.05.14: 3.6

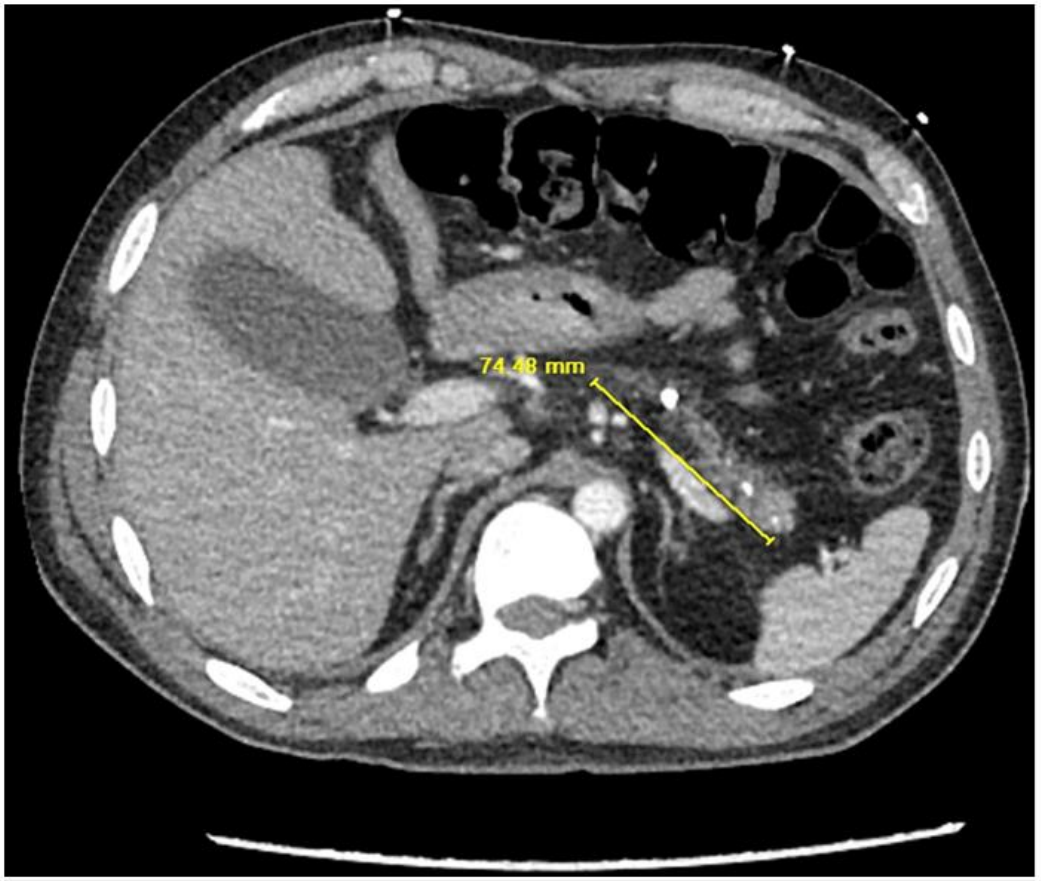


FIGURE 1: Diffusely atrophic pancreas with numerous parenchymal calcifications.

出現很多個鈣化點

急性胰腺炎的各種可能來源

- 1.在急性胰腺炎的各種可能來源中，慢性過
- 量飲酒是最常見的病因.
- 2.慢性過量飲酒會造成慢性胰臟炎

Others : gall stone
Hyper-TG

- 許多與飲酒相關的急性胰腺炎反覆發作的患者會發展為慢性胰腺炎，**持續飲酒是預後的關鍵因素**

- Haber PS, Kortt NC. :Alcohol use disorder and the gut.
- *Addiction.* 2021;116:658–667.

Acute and chronic gastrointestinal problems are common in the setting of excessive alcohol use, and excessive alcohol use is associated with injury to all parts of the gastrointestinal tract. There is mounting evidence of gastrointestinal injury and increased cancer risk even from moderate alcohol consumption. The major causes of alcohol-related morbidity and mortality within the gastrointestinal system are liver disease, pancreatitis and gastrointestinal cancer. Other alcohol-related intestinal dysfunction is common but not life-threatening, leading to diarrhoea, malabsorption and nutritional deficiencies.

胰腺外分泌功能不全(EPI)是慢性胰腺炎的長期後果之一

- 胰腺外分泌功能不全是慢性胰腺炎的長期後果之一[9]
- Advanced EPI會導致脂肪和蛋白質消化不良，導致多種維生素和微量營養素缺乏。此外，整體吸收不良是由廣泛黏膜受累或吸收面減少相關的疾病引起的，例如慢性胰腺功能不全嚴重[4]。吸收不良狀態可能會降低血清鈣、鎂和鋅的濃度。
- 在**長期酗酒繼發胰腺功能不全的病例中，實驗室檢查結果顯示重度、難治性低鎂血症、低鉀血症、低磷血症和低鈣血症。**

Hypomagnesemia

- 鎂已被確定為識別慢性胰腺炎患者胰腺功能不全的有希望的標誌物.
- Papazachariou 等人於 2013 年進行的一項前瞻性研究。顯示，13例患者慢性胰腺炎與鎂缺乏呈正相關。
- Magnesium deficiency in patients with chronic pancreatitis identified by an intravenous loading test. Papazachariou IM, Martinez-Isla A, Efthimiou E, et al. *Clinica Chimica Acta.* 2000;302:145–154.
- Since serum levels of magnesium are a poor indicator of magnesium deficiency, the retention of a low-dose intravenous magnesium load
- (0.1 mmol/kg body weight) was determined in 13 patients with chronic pancreatitis (10 due to alcoholism) and eight healthy controls.
- Percentage magnesium retention was greater in patients with chronic pancreatitis than controls (59.8+/-37.3% S.D. versus 22.0+/-38.2% S.
- D.: P=0.038), and 10 of 13 patients showed evidence of magnesium deficiency
- 2021年一項納入胰腺功能不全患者的研究也表明，鎂缺乏與胰腺外分泌功能不全顯著相關.
- Yield of testing for micronutrient deficiencies associated with pancreatic exocrine insufficiency in a clinical setting: an observational study. Jalal M, Campbell JA, Tesfaye S, Al-Mukhtar A, Hopper AD. *World J Clin Cases.* 2021;9:11320–11329.
- 112名接受FEL-1 (糞便彈性蛋白酶-1） 和PEI-MD (PEI相關的微量營養素缺乏症)檢測的患者。41/112 （36.6%） 患者發現PEI，82例患者進行胰腺 CT檢查。總體而言，在 21/112 （18.8%） 患者中發現了 PEI-MD。如果存在PEI，**PEI-MD的產量**為17/41（41.5%），顯著高於無PEI的4/71（5.6%）（*P*=0.0001）。當PEI和CP同時出現13/22時，PEI-MD的產量顯著高於沒有PEI的CP和沒有CP的PEI（*P*< 0.03）。個體微量營養素評估顯示，當存在PEI時，前白蛋白8/41（19.5%）、硒6/41（14.6%）和鎂5/41（12.2%）缺乏症的發生率更高（<0.02）。使用我們佇列中確定的重要微量營養素作為 PEI 預測因數的準確性顯示陽性預測值為 80%-85.7% [95% 置信區間 （CI）：38%-100%]，低敏感性為 9.8%-19.5% [95% CI：3.3%-34.9%]。
- PEI-MD 檢測應包括硒、鎂和前白蛋白(prealbumin)。

低鎂血症

- 鎂水準可作為營養狀況的標誌物，胰腺功能不全患者補充鎂可預防嚴重不良事件，如住院治療。鎂可以通過口服和靜脈注射補充，確切的途徑可能取決於整體臨床情況。鑒於鎂缺乏的潛在嚴重後遺症，監測胰腺功能不全患者的鎂水準可能是合理的.
- 低鎂血症應包括在胰腺功能不全患者的鑒別診斷中，這些患者報告了神經系統癥狀，如刺痛、麻木、疲勞或肌肉痙攣

低鎂血症發生在急性胰臟炎之后. an alcoholic patient admitted to our hospital with alcohol induced acute pancreatitis who developed severe hypomagnesemia (serum magnesium 0.36 mmol/l) during hospitalization.

[1] G Liamis[1], C Gianoutsos, M Elisaf **Acute pancreatitis-induced hypomagnesemia**

Department of Internal Medicine, Medical School, University of Ioannina,Greece

Pancreatology 2001;1(1):74-6

- 鎂是一種重要的電解質，是人體內各種生理過程的關鍵組成部分，影響細胞功能、神經傳導和整體健康。儘管鎂在生理過程中發揮著至關重要的作用，但臨床醫生可能忽略了維持 1.46 至 2.68 mg/dL 正常血清水平的重要性。
- **低鎂血症的特徵是血清鎂水平**
- **低於 1.46 mg/dL**，會帶來各種挑戰和併發症，需要全面了解其評估、治療和潛在後果。

- In multiple cohort case studies, the relationship between EPI and alcohol abuse was exemplified, as nearly half the cases were shown to have a direct correlation. Furthermore, it was found that these patients also developed **alcohol-associated severe electrolyte imbalances due to malabsorption as a result of EPI and surface area disease.**
- Retrospective analysis directly showed a positive correlation of EPI with magnesium imbalance. However, it was recommended that routine monitoring was not necessary.

Magnesium should be used as a marker and used to prevent adverse events in patients with EPI.

L1553,L1554: Kyoko Shimizu et al : Evidence-based clinical practice guidelines for chronic pancreatitis. 2021. J Gastroenterol (2022) 57:709–724

Gastrointestinal Causes of Hypomagnesemia :
The mechanisms involved in the gastrointestinal tract as a cause of hypomagnesemia involve poor oral intake, malabsorption of magnesium, and increased losses through gastrointestinal secretions. Multiple mechanisms often coexist in the same patient.
Hypomagnesemia is common in patients with pancreatitis. Similar to hypocalcemia, the mechanism involves saponification of magnesium in a milieu of pancreatic fat. Additionally, patients with pancreatitis often have pre existing negative magnesium balance.

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L1563, L1564

CQ: is pancrelipase recommended for treatment of pancreatic exocrine insufficiency?

- • Pancrelipase is a high-titer pancreatic enzyme preparation that can be recommended for treatment of pancreatic exocrine insufficiency with steatorrhea and weight loss.
- Strength of recommendation; strong,
- evidence level: A
- Significant improvements in fat absorption, nitrogen absorption, and fecal fat content have been reported in multiple randomized-controlled trials of pancrelipase in patients with pancreatic insufficiency due to CP or following pancreatic surgery. Furthermore, a multicenter questionnaire-based survey found that pancrelipase improved quality of life, for example, by improving weight loss and steatorrhea.

L1553,L1554: Kyoko Shimizu et al : Evidence-based clinical practice guidelines for chronic pancreatitis. 2021. J Gastroenterol (2022) 57:709–724

CQ: are agents that suppress gastric acid recommended for treatment of pancreatic exocrine insufficiency?

- • If the therapeutic effect of pancreatic enzyme replacement therapy is inadequate in patients with pancreatic exocrine insufficiency, an **H2-receptor antagonist or proton pump inhibitor can be used. Strength of recommendation: weak, evidence level: C**
- In cases of pancreatic exocrine insufficiency, the pH in the upper small intestine is lower because of a decreased bicarbonate concentration in pancreatic juice. A pH of\4 in the small intestine inactivates pancreatic lipase, and precipitation of bile acid leads to poor formation of micelles. Furthermore, enteric-coated digestive enzyme preparations are not released at a pH\5. Treatment with a proton pump inhibitor or H2-receptor antagonist has been shown to be effective when combined with a pancreatic digestive enzyme agent, whether enteric-coated or not, in patients with steatorrhea [99–101]. Combined use of a gastric acid-suppressing agent has been reported to be effective

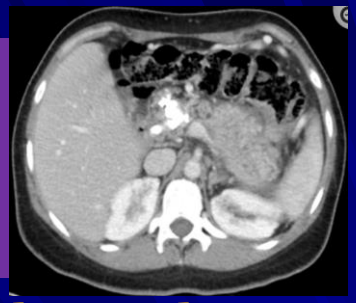
L1553,L1554: Kyoko Shimizu et al : Evidence-based clinical practice guidelines for chronic pancreatitis. 2021. J Gastroenterol (2022) 57:709–724

CQ: is a fat-restricted diet recommended for treatment of pancreatic exocrine insufficiency?

- • A uniform fat-restricted diet is not recommended in the decompensated stage of CP with exocrine pancreatic insufficiency. Strength of recommendation: strong, evidence level: D
- A short-term low-fat diet (fat 30–35 g/day; fat B 10 g/ meal) is recommended for patients with compensatory abdominal pain and back pain. However, **in the decompensated stage with pancreatic exocrine insufficiency, a daily fat intake of 40–70 g or 30%–40% of total calories is recommended in combination with pancreatic enzyme replacement therapy to prevent malnutrition** [95].

L1553,L1554: Kyoko Shimizu et al : Evidence-based clinical practice guidelines for chronic pancreatitis. 2021. J Gastroenterol (2022) 57:709–724

Gitelman syndrome
associated with CP



- Gitelman syndrome (GS) is a rare **autosomal recessively inherited** disease and salt-losing tubulopathy, also refers as familial hypokalemia-hypomagnesemia, characterized by hypokalemia, hypomagnesemia, hypocalcemia, hyperreninemia, and hyperaldosteronism, It is caused by mutations of genes encoding the sodium, chloride, and magnesium carriers in the apical membrane of the distal convoluted tubule. The mutations involve 1 - *SLC12A3* gene .

.Nourah ALSaleh,et al :Gitelman syndrome: A first published clinical association with chronic pancreatitis, a case report and review of literature Int J Surg Case Rep. 2022 Feb; 91: 106779. Published online 2022 Jan 17

A case of a 39-year-old female with Gitelman syndrome and chronic pancreatitis in the absence of well-known causes of CP. Her clinical and radiographic profile constituted an indication for surgical intervention, namely pancreatic head and body coring and pancreaticojejunostomy (Frey's procedure) (FP). On follow up 3 month later, the patient is pain-free and is satisfied.

1. multiple attacks of acute on top of chronic pancreatitis, despite the negative history of predisposing factors of chronic pancreatitis.
2. multiple intraductal stones the largest measured 1.2 cm and located within the uncinate process.
3. Hypomagnesemia, with successful resuscitation, heart failure, diabetes mellitus, hypothyroidism, latent tuberculosis, and pulmonary embolism 16 years ago
4. Exploratory laparotomy, removal of main pancreatic duct (MPD) stones, and pancreaticojejunostomy (Frey's procedure). Pathology report confirmed chronic pancreatitis.

Gitelman syndrome

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胰臟炎會變胰臟癌嗎？ **胰臟炎並不一定會導致胰臟癌**。但研究顯示慢性胰臟發炎的病人，罹患胰臟癌的機率的確比較高

- 胰臟癌在早期幾乎沒有明顯的症狀，但隨著腫瘤的逐漸擴大，一些症狀可能出現。以下是胰臟癌的一些常見症狀[1]：

1. **黃疸**：眼睛和皮膚變黃，通常是因為胰臟腫瘤壓迫到總膽管，或者腫瘤轉移到肝臟所致。
2. **腹部和背部疼痛**：隨著腫瘤增大，它可能開始壓迫附近的器官，引起疼痛。
3. **體重減輕和食慾不振**：患者通常胃口不好，這可能與進食不佳以及消化、吸收異常有關。
4. **噁心和嘔吐**：腫瘤也可能壓迫周圍的器官，阻礙消化道並造成胃排空困難。
5. **糖尿病**：胰臟癌可能導致血糖出現變化，可以通過血液檢查來確定。

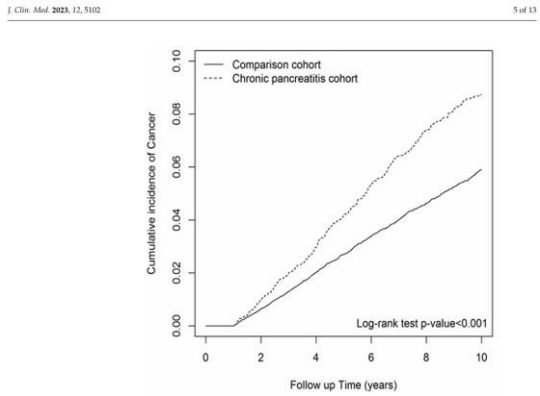
- 此外，胰臟癌的危險因子包括肥胖、抽菸、糖尿病、工作接觸特殊化學品等[1]。如果您有相關症狀或擔心自己的風險，建議您儘早就醫進行檢查。[2]

- **從慢性胰臟炎變為胰臟癌總要20年(不少案例是2-3年)**

2011 年一篇分析台灣糖尿病患與胰臟炎、胰臟癌兩者間關係的論文指出，糖尿病合併慢性胰臟炎的病人，罹患胰臟癌的機率是一般身體健康者的 33.5 倍；研究同時發現，糖友確診糖尿病 2 年內發現胰臟癌的機率也會增加，建議將糖尿病作為胰臟癌的早期徵兆，提早追蹤以預防。

https://blog.health2sync.com/diabetes_care_pancreas/#:~:text=2011%20%E5%B9%B4, accessed on 2024.05.27

Higher risk of pancreatic cancer in patients with CP



Among men, individuals with CP exhibited a significantly higher risk of stomach cancer (aHR=2.19)and other cancers(aHR=2.45;95%CI=2.03,2.96).
Irrespective of gender, CP patients had elevated risks of liver cancer (women:aHR=2.01;95%CI=1.09,3.72/ men:aHR=1.54;95%CI=1.06,3.79) and pancreatic cancer(women: aHR=16.20;95%CI=1.46,179.70/men:aHR=3.17;95%CI=1.12,9.04) Compared to the non-CP group

Table 4. The crude and adjusted incidence rates of various cancers for the subjects with and without chronic pancreatitis.

	CP (−)			CP (+)			Crude			Adjusted		
	N	PYs	IR	N	PYs	IR	cHR	95% CI	*p*-Value	aHR	95% CI	*p*-Value
Female												
Liver	32	55,954.01	0.57	15	13,552.22	1.11	1.94	1.05~3.59	0.034	2.01	1.09~3.72	0.025
Breast	17	55,797.93	0.30	6	13,532.12	0.44	1.45	0.57~3.67	0.435	1.45	0.57~3.68	0.434
Lung	28	55,941.09	0.50	9	13,561.11	0.66	1.34	0.63~2.83	0.448	1.33	0.63~2.81	0.462
Thyroid	13	55,807.78	0.23	4	13,522.32	0.30	1.27	0.41~3.89	0.678	1.28	0.42~3.92	0.667
Colorectal	44	55,992.41	0.79	10	13,566.08	0.74	0.94	0.47~1.86	0.856	0.94	0.47~1.87	0.859
Kidney + Bladder	21	55,861.04	0.38	4	13,512.90	0.30	0.79	0.27~2.29	0.662	0.80	0.27~2.33	0.681
Stomach	10	55,783.60	0.18	4	13,525.42	0.30	1.65	0.52~5.27	0.397	1.66	0.52~5.30	0.390
Pancreatic	3	25.25	118.83	10	56.66	176.49	2.31	0.50~10.67	0.285	16.20	1.46~179.70	0.023
Other + NP	136	56,585.40	2.40	35	13,686.26	2.56	1.07	0.73~1.54	0.739	1.07	0.74~1.55	0.732
Male												
Liver	167	126,522.20	1.320	57	28,791.8	1.98	1.50	1.11~2.03	0.008	1.54	1.14~2.08	0.005
Lung	88	126,131.09	0.698	21	28,541.08	0.74	1.05	0.66~1.70	0.826	1.05	0.65~1.68	0.855
Thyroid	8	125,666.5	0.064	3	28,440.52	0.11	1.67	0.44~6.31	0.447	1.69	0.45~6.36	0.440
Colorectal	104	126,271.97	0.824	24	28,555.55	0.84	1.03	0.66~1.60	0.904	1.03	0.66~1.61	0.882
Prostate	66	126,027.67	0.524	12	28,502.86	0.42	0.81	0.44~1.51	0.512	0.77	0.41~1.42	0.400
Kidney + Bladder	46	125,852.34	0.366	11	28,478.92	0.39	1.05	0.54~2.03	0.887	1.07	0.55~2.06	0.845
Stomach	20	125,732.778	0.159	10	28,477.04	0.35	2.21	1.04~4.73	0.040	2.19	1.02~4.68	0.043
Pancreatic	12	80.55	148.97	16	56.08	285.30	2.35	1.06~5.22	0.036	3.17	1.12~9.04	0.031
Other + NP	293	127,539.21	2.30	154	29497.62	5.22	2.29	1.88~2.79	<0.001	2.32	1.90~2.82	<0.001

PYs: person-years; NP: nasopharyngeal.

Hsieh, C.-C.; Fu, Y.-H.; Ku, N.-E.; Hsia, C.-C.; Hung, Y.-T.; Hsu, T.-J.; Chen, S.-H.; Kuo, S.-J. The Impact of Chronic Pancreatitis on the Occurrences of Human Cancers: Real-World Data. J. Clin. Med. 2023, 12, 5102. https://doi.org/10.3390/jcm12155102 (L1557, L1558)

CP: 5394
Non-CP: 21576

- 1. CP patients exhibited a significantly **higher cancer risk** (adjusted hazard ratio (aHR) of 1.32 for females and 1.68 for males) and cumulative incidence ($p <$ 0.001) compared to non-CP individuals.
- 2. CP showed notable associations with pancreatic (aHR = 3.51), liver (aHR = 1.62), stomach (aHR = 2.01), and other cancers (aHR = 2.09). In terms of liver cancer, CP was significantly associated with patients without viral hepatitis, regardless of gender (aHR = 2.01 for women; aHR = 1.54 for men). No significant cancer occurrences were observed within the first year following CP diagnosis. Pancreatic or liver cancer developed in approximately half of CP patients within 2-3 years, while gastric cancer in male CP patients predominantly occurred around the fifth year after diagnosis. These findings inform potential cancer-screening plans for CP patients.

如何預防胰臟受損？

Health 2 Sync

戒掉菸酒和高糖飲食

養成規律運動的習慣

定時定量的均衡飲食

選擇植物性的蛋白質

少吃油炸煎炒的食物

攝取葉酸及維他命B6

https://blog.health2sync.com/diabetes_care_pancreas/ 2020.04.26
Accessed on 2024.06.10

結論: chronic alcohol use and EPI

- There is a relationship between chronic alcohol use and EPI, with magnesium deficiency as a strong positive marker, which should be repleted adequately to reduce the number of adverse events and hospitalizations.

酗酒者今後的20年充滿了挑戰與困擾 (-43+20=63)

- Multiple diseases
- DM, Chronic pancreatitis, alcoholic liver diseases, alcoholic dependence. hypoglycemia, psychiatric problems.
- 小心胰臟癌的發生.
- 2-20 years 都有可能
- biomarkers
- Abdominal CT, Abdominal sono.
- GI follow up.定期追蹤檢查

結論(2025.03.28)

- 飲酒過量,對健康有很大之影響.足可以引起肝臟疾病及胰臟炎.—住院是一時的問題,但影響卻是整個人生.
- 一旦飲酒過量發生各種健康上的問題,即應該完全戒絕飲酒.
- 一次飲酒40-80 公克以上,即有可能發生急性胰臟炎.
- 發生急性胰臟炎之後如果沒有解決飲酒短時間內可能就發生慢性胰臟炎(平均8年,95%發生胰臟炎)
- 發生慢性胰臟炎之后,2-20 年有機會發生(轉變為)胰臟癌
- **臺灣法律放鬆喝酒的年齡至18歲,並不恰當.應該延後至25歲為合法之飲酒年齡.**
- 協助飲酒者**戒絕喝酒**是社會大眾的責任,醫師應努力勸導病人戒酒. 責無旁貸.