Acute & chronic pancreatitis

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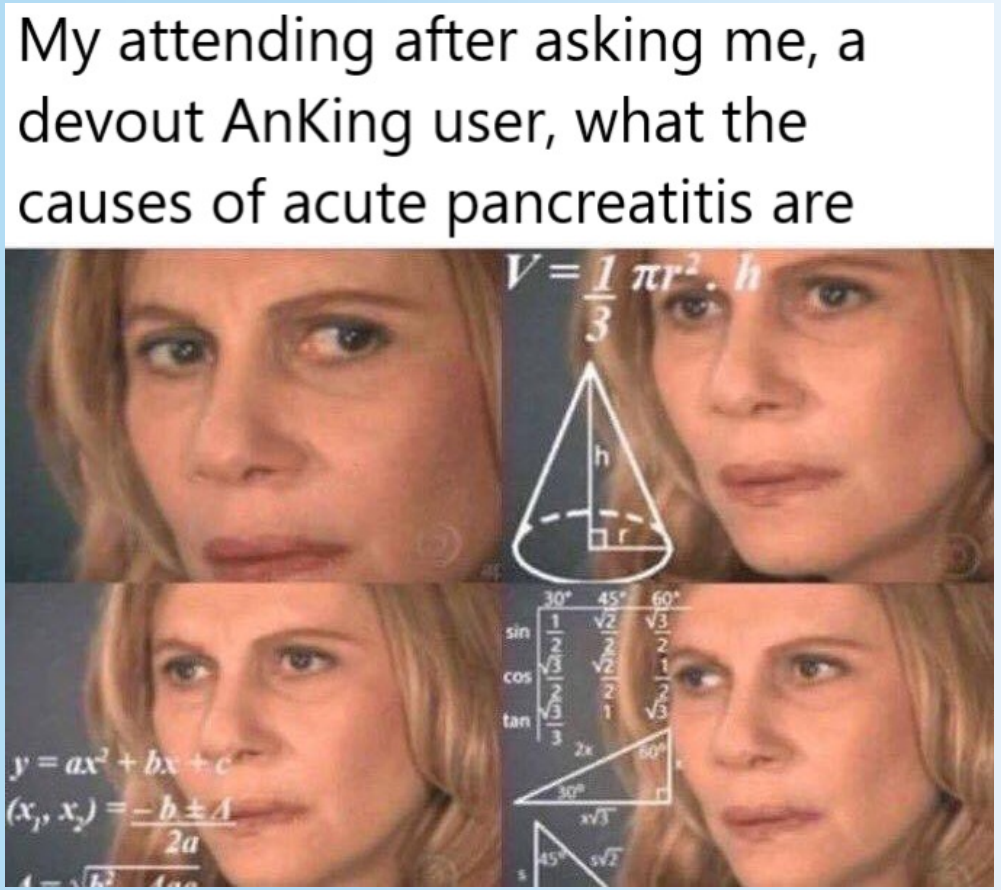
Topics

Function of pancreas

Acute pancreatitis

Case

Chronic pancreatitis



Function of pancreas

Exocrine function

Acinar cells: Produce digestive enzymes and bicarbonate

Lipase

Amylase

Protease

Trypsin

Endocrine function

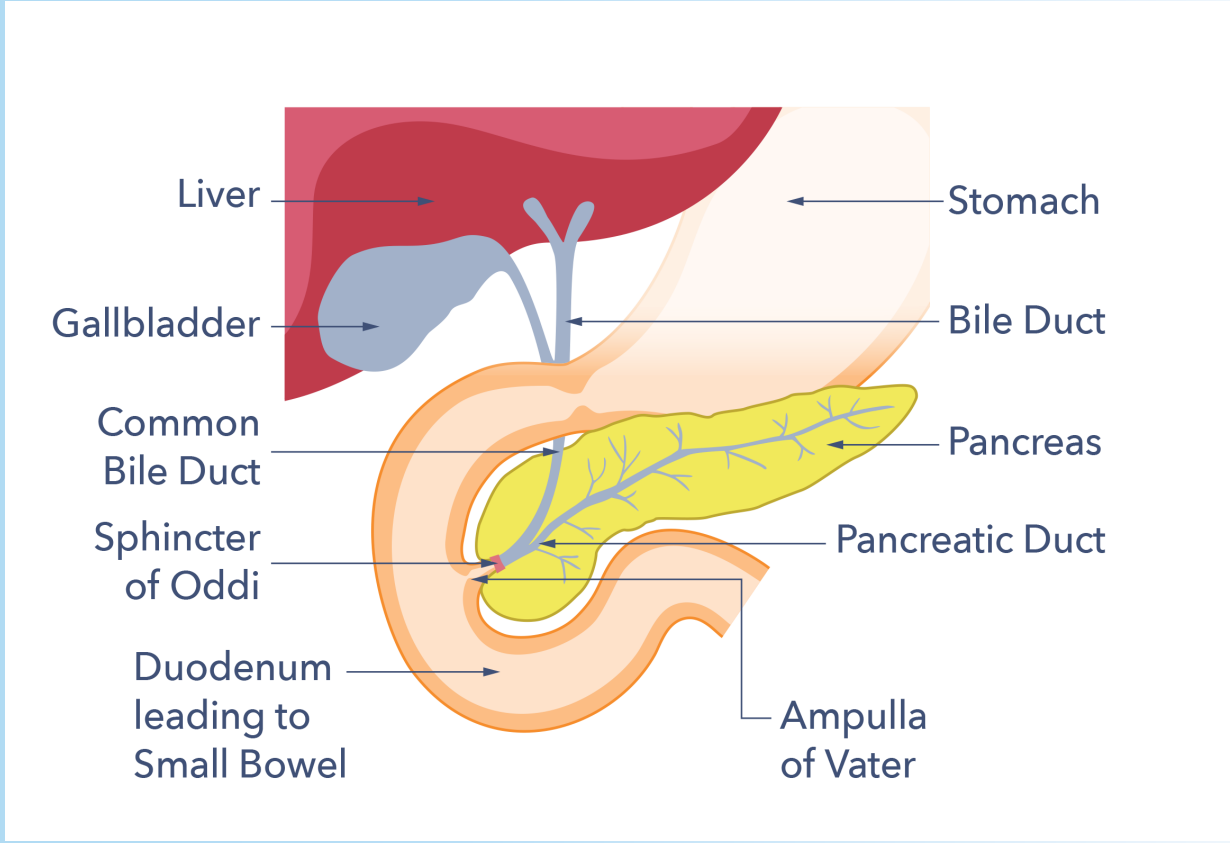
Islets of Langerhans with its 4 cell types:

Alpha cells - glucagon

Beta cells - insulin

Delta cells - somatostatin

PP cells - pancreatic polypeptide

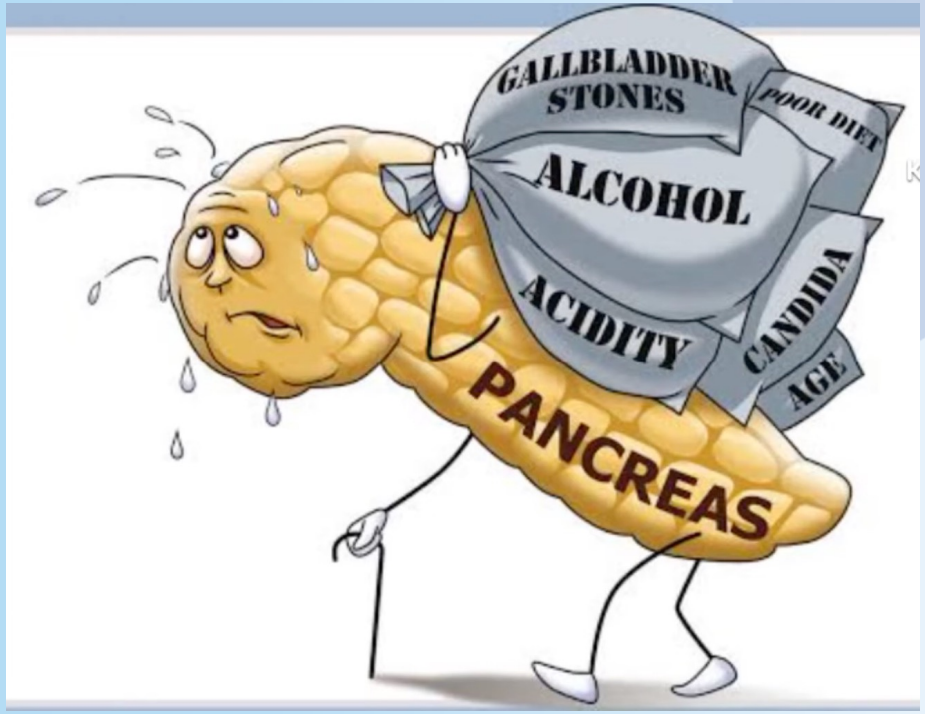


Causes of acute pancreatitis

I – Idiopathic

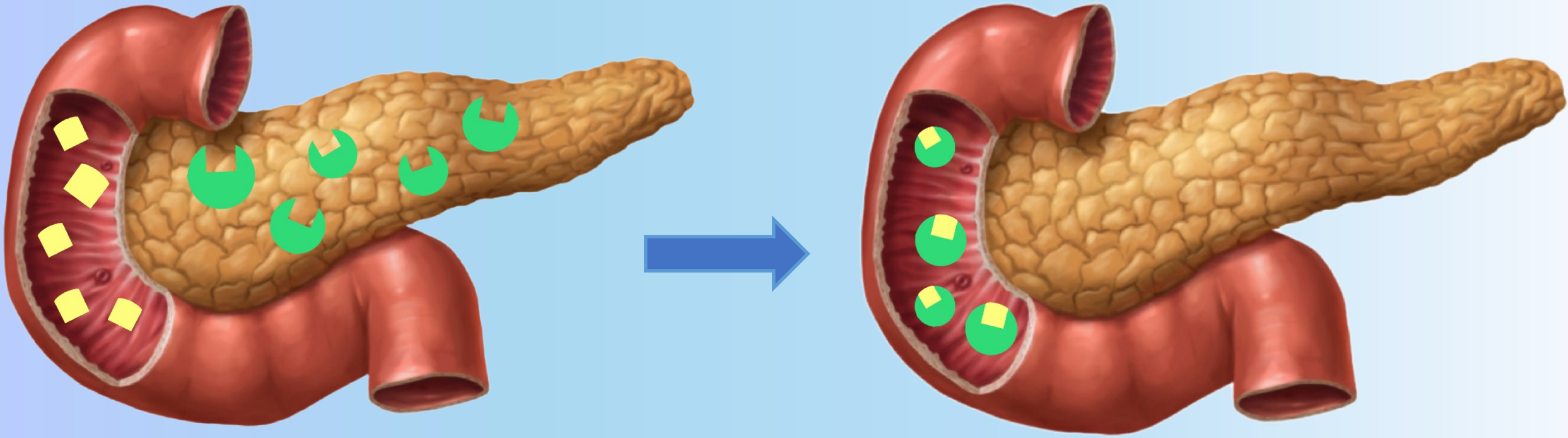
G – Gallstones
E – Ethanol abuse
T – Trauma

S – Steroids
M – Mumps virus
A – Autoimmune disorders
S – Scorpion stings
H – Hypertriglyceridemia and hypercalcemia
E – ERCP
D – Drugs



+ Genetic predisposition: mutation of **cationic trypsinogen gene (PRSS1)** on chromosome 7

Process of enzymatic activation



Trypsinogen + Enteropeptidase (protease) → Trypsin

Pathogenesis of acute pancreatitis

Alcohol-induced

1. **Increased zymogen production**

2. **Decreased production of fluid and bicarbonate in ducts**

   Pancreatic juices become thick and sticky, easily producing plugs

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Gallstone-induced

3. **Pancreatic juices back up causing increased pressure**

   Distending pancreatic ducts

   Membrane traffic becomes chaotic

4. **Zymogen granules fuse with lysosomes**

   Early activation of trypsinogen

5. **Autodigestion of pancreas**

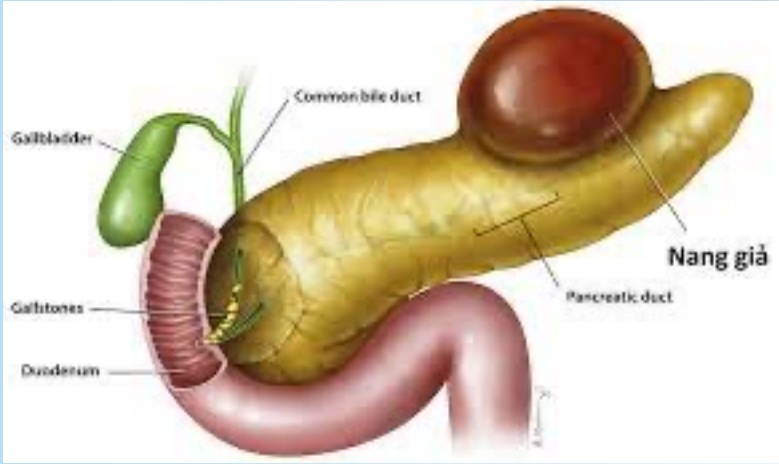
Acinar cell injury lead to:

(1) Interstitial inflammation and edema

(2) Proteolysis

Action of proteases: increased vascular permeability

Blood vessels leak and rupture → ARDS



(3) Fat necrosis

Action of lipase and phospholipase

Destruction of peripancreatic fat

(4) Hemorrhage

→ Hypovolemia & septic shock

Action of elastase: destroys elastic tissue of blood vessels

Digestion and bleeding can liquefy tissue: liquefactive hemorrhagic necrosis and pancreatic pseudocyst

Diagnostic measures

Clinical picture

Pain in epigastric region radiating to the back.

Cullen`s sign

Grey Turners`s sign

CT scan

Show inflammation, necrosis and pseudocyst.

Ultrasound

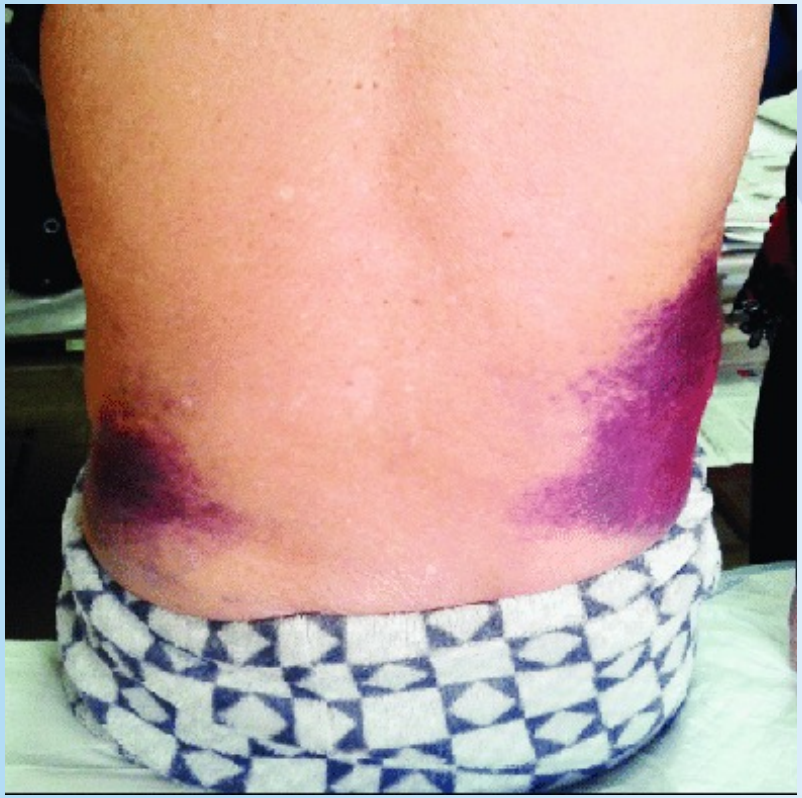
May show gallstones.

Lab values →

Lab values:	Explanation:
⇑ Lipase (3x normal value)	Rise within 8 hours Return to normal within 14 days Autodigestion of pancreas (consequence of acute pancreatitis) results in release of lipase and amylase.
⇑ Amylase (3x normal value)	Rise within 12 hours Return to normal within 5 days Autodigestion of pancreas (consequence of acute pancreatitis) results in release of lipase and amylase.
⇑ Leukocytes & hematocrit	Increased due to dehydration or hemorrhaging
⇑ CRP and LDH	Increased due to inflammation
⇑ BUN & Creatinine	Renal insufficiency, pancreatic necrosis and dehydration
⇓ Calcium	Decreased due to that fat necrosis consume Ca^{2+}

Cullen`s sign

Grey Turner`s sign



Both caused by bleeding due to pancreatic necrosis

A regular day at the ER in Prokocim

65 years old **Mrs. Pani Dyducha** comes into the ER.

She presents with:

Comatose state

GCS-score: 3 -> only respond to pain-stimulation.

You find her somehow familiar, and you realize you have seen this lady drunk around the parks of Planty so many times.

Arterial blood gas

Sodium (mmol/L)	142	135-145
Potassium (mmol/L)	3.2	3.5-5.0
Urea (mmol/L)	27	2.5-7.5
Creatinine (umol/L)	140	30-120
Chloride (mmol/L)	104	95-108
pH	7.30	7.35-7.45
PaO2 (mmHg) at FiO2 0.5	100	
PaCO2 (mmHg)	28	
HCO3 (mmol/L)	8	22-28
Glucose (mmol/L)	50	3.5-6.5

What is significant with this blood gas?

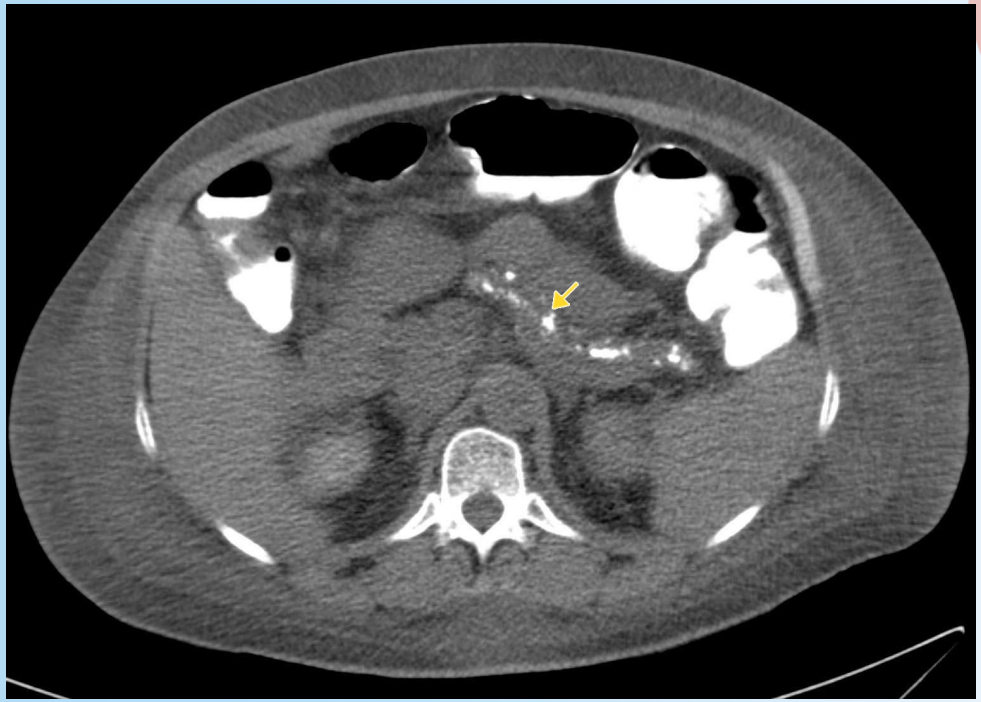
How can we get Mrs. Pani Dyducha out of this comatose state?

Diagnostic measures

Clinical picture

NO Gray Turner or Cullen sign

Chronic pain in epigastric region radiating to the back



Transabdominal CT

→ Show calcifications

Lab Values

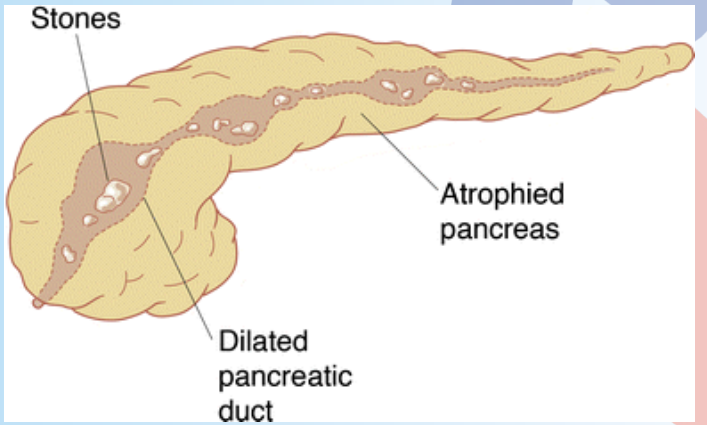
↑ Bilirubin

↑ ALT

↑ HbA1c

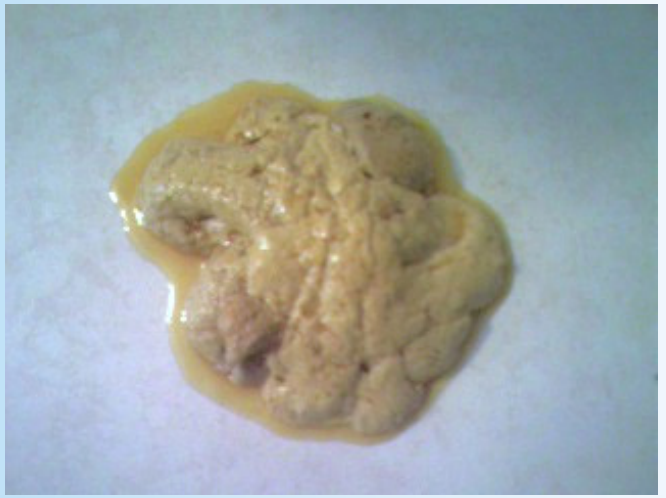
What is wrong with Mrs. Pani Dyducha?
&
why has this happened to her?

Chronic pancreatitis



(1) Repeated bounces of acute pancreatitis

(2) Persistent inflammation cause changes in structure



Ductal dilation

Stellate cells produce fibrotic tissue causing stenosis

Calcium deposition → Plugs

(3) Pancreatic insufficiency

Destruction of pancreatic b-cells → Diabetes mellitus

Acinar cell atrophy → Decreased production of digestive enzymes → ADEK deficiency & Steatorrhea

Thank you for your attention ;)

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