

Acute Pancreatitis

“Acute Pancreatitis is the most terrible of all the calamities that occur in connection with the abdominal viscera.”

Sir Berkeley Moynihan *Ann Surg* 1925

Outline

- Def
- Clinical presentation/Differential
- Epidemiol
- Pathophys
- Causes
- Complications
- Radiology
- Scoring systems/prediction
- Management: fluids, antibiotics, nutrition, ERCP
- Long term consequences (CP, DM, cysts, bleeds)

Acute Pancreatitis

- Inflammation of the pancreas and associated adjacent organs without evidence of chronic pancreatitis
- Atlanta Symposium in 1992 defined acute pancreatitis clinically as **2 of 3 of the following**
 - Typical pancreatic type pain
 - Radiographic findings of acute pancreatitis
 - Elevations in blood chemistries (typically amylase and/or lipase $>3x$ ULN)

Causes of elevated:

Amylase

- Renal insuff
- Salivary inflammation
 - Ile vomiting, parotiditis
- Macroamylasemia
 - Hereditary or from hatta starch
- Intestinal infarct/peritonitis
 - Through transperit absorpt
- Chole'itis, Salpingitis, ectopic preg
- Ovarian cysts, lung inflamm
- Acidosis, ESLD
- Intest radiation, obstruction
- Colon, ovar, panc, brst, prst, lung, esoph CA
- MM, pheo, appendicitis, gastroenteritis
- Burns, normal pregnancy, FHF

Lipase

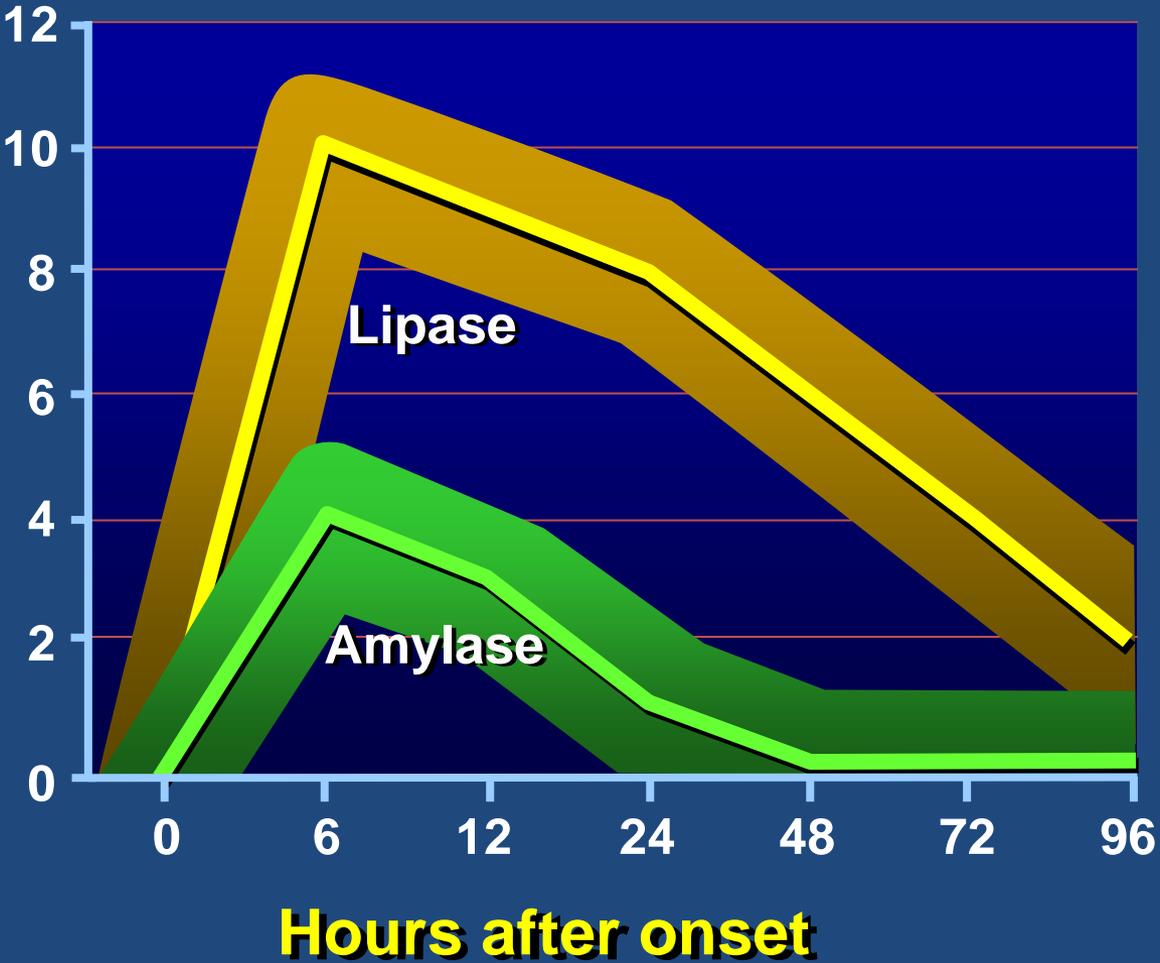
- DU, renal insuff
- Small Intestinal ischemia/obstr
- Tuboovarian abscess
- Macrolipasemia (nonhodg, cirrh)
- Tylenol OD
- Hypotension/sepsis/DKA
- HIV, panc CA
- Gulló's syndrome
- Cholecystitis

False negative amylase/lipase: Hypertrigs, stoic person (vets) who presents late

Working up false pos amylase/lipase

- If obvious other cause (vomiting, tubo-ov abscess) then no further w/u needed
- Serum isoamylase (35-50% of serum amylase usually pancreatic)
- Urinary amylase (beware of spitting in cup, munchausen)
- Serum trypsin (RIA, UF, NEJM 1984).
- Barely high levels: repeat the measurement in 6-12hr
- True elevations require workup, malign, CP, etc

Acute Pancreatitis: Time course of enzyme elevations

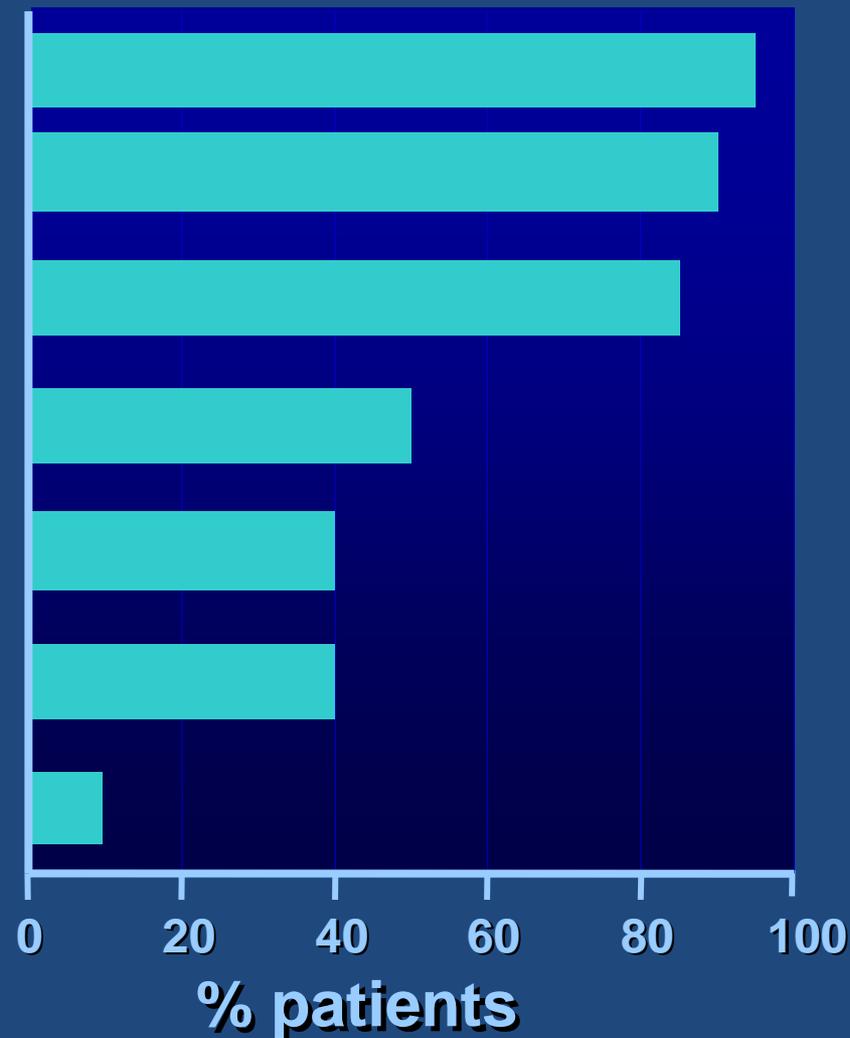


Amylase half life 10 hrs

Acute Pancreatitis

Presenting features

Abdominal pain
Nausea / vomiting
Tachycardia
Low grade fever
Abdominal guarding
Loss of bowel sounds
Jaundice



Syncope! Rare

Painless: post op, legionaire's,
DM, perit dialys

Extraintestinal manifestations

- Arthritis (lipase laden fluid with leuks)
- Serositis (pericarditis, pleuritis)
- Panniculitis, subcutaneous fat necrosis, can look like e nodosum (1% of all cases, 10% have it at autopsy)
- Purtscher's retinopathy (rare)
 - Sudden blindness, post retinal artery occlusion

Pain, Oh the pain

- “Worse than childbirth” “Worse than being shot”
- Starts fast within 10-20min reaches peak
 - Third fastest pain onset in GI after perf and SMA thromb
- Does not usually undulate (not colicy)
- Lasts days (if no underlying chronic damage)
 - Longer than biliary colic which is hours
- Radiate to back in 50%
- Sometimes diagnosed at autopsy (painless)
- Almost always causes ER visit/admission
- Capsaicin, glutamate, vanilloid, ppar-gamma

Acute Pancreatitis: Epidem

- 5-35/100,000
- Increasing incidence (detection?meds?iatrog?)
- Increases with increasing age
- Onset before 14-15 yrs unusual
 - unless hereditary, traumatic, anatomic anomaly
- 250,000 admissions per year in U.S. (2nd GI)
- \$2 billion in direct costs per year
- 6th costliest GI disease behind ESLD, cancers, IBD
- NIDDK funding is 11 out of 17 GI illnesses

Acute Pancreatitis Pathophysiology

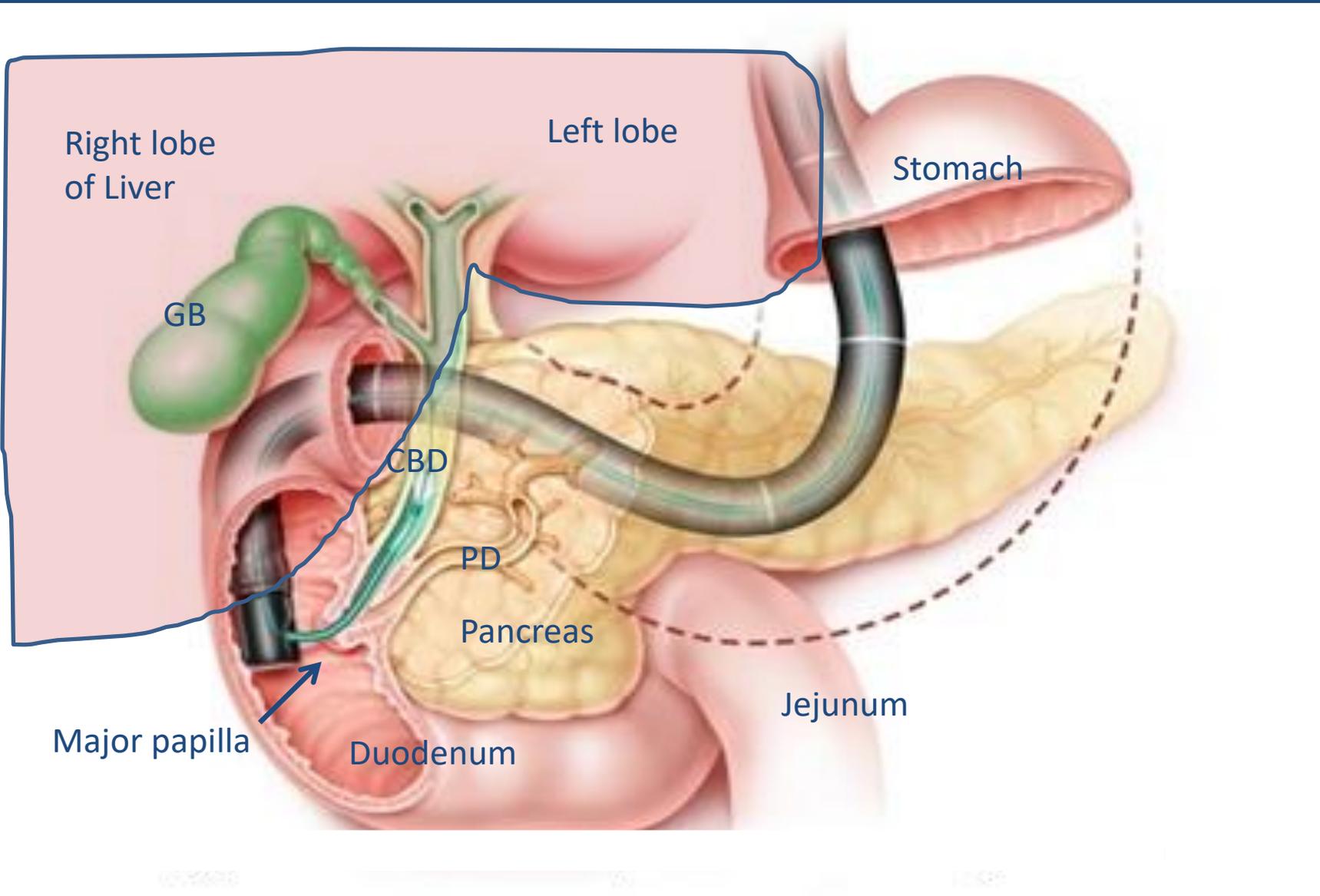
- Since starch and triglycerides are not stored in the pancreas, trypsin is the major catalyst for pancreatic autodigestion, not amylase or lipase (but later lipase gets to abd fat....)
- PREMATURE/INTRACELLULAR activation of trypsin.
- Leads to activation of chymotrypsinogen, more trypsinogen, elastase, phospholipase A2, complement, kinins ->>**AUTODIGESTION!!**

Etiologies of Acute Pancreatitis

- Biliary (gallstones) ***
- Alcohol****
- Triglycerides***
- pERCP,* post surgical
- Drugs
 - (except byetta and L-asparagenase and trigs **)
- Tumors/obstruction
- Trauma**
- Ischemia/embolic***
- Infection (except mumps **)
- Hypercalcemia (hypPTH)
- Autoimmune/Sprue
- Hereditary
- Controversial (divisum/SOD)
- Scorpions ***
- Chemical: insecticide/MeOH
- Idiopathic: 30%!!

Number of *'s denotes tendency to be severe

Biliary anatomy



Biliary

- Gallstones or sludge, Microcrystals?
- Most common etiology in world. Still 35% in US.
- More in women
- Usually small ones that don't obstruct cystic duct or most of CBD until right at major pap
- Usually pass on own, but don't be complacent!
 - Can be Necrotizing!!
- Biliary duct dil/LFT can occur late! (insensitive!)
- If fever, bili over 2, SIRS, (ie **cholangitis**) call adv endo immediately.
- ALT 3X ULN (>150) 50% sens and 90% specif.
- First ALT then bili then ductal dilation.
- ALT/AST can be 1000!
- **NOTE MUST BE ON CHART FROM SURGERY BEFORE D/C !**

Biliary: who has extant CBD stone?

- Cholangitis—call even at 2am if look unwell, septic
- TBili over 3, esp if over 5
- LFT not improve, esp if pt still has pain
- Pt looks unwell
- High (ERCP), moderate (MRCP), low risk (watch)
- Very personalized decision. Depends on local MRCP quality, surgical expertise in intraop cholangiogram, etc
- Call even on weekend
- MRCP can have false pos>>>>false neg

Biliary panc'itis Scenarios

- PT with fever, tbili 1.8, ALT 500, AP 250, tachy, WBC 20 with 20% bands, duct dil on US, looks unwell
- PT looks well but ALT still 100, AP 200 TB normal—sat nite at VA
- PT was very ill when they called you, fever, tachy, tachypnic, bili 3, AP 250, ALT 500, duct dil, but when you arrive suddenly pt feels great, looks better, stat labs bili now 6, AP300, ALT 640, WBC still 15 with left shift

Biliary panc'itis scenarios

- Pt with pain of 6 hours duration now, bili 4.4, WBC 15, ALT 340, AP 300, t 100F, CT with mild AP and ductal dil
- No radiologist in house Sat 9pm at the VA
- You look at CT and inform the rad PGY2 at HUP that there is a CBD filling defect, likely stone, about 6mm in size. He agrees he must have missed it.
- Pt still not feeling well, writhing in pain

Biliary panc'itis scenarios

- Pt with fever, WBC 11, bili 2.2, ALT 300, AP 300, AP, ER RUQ US shows gallstones, acute chole with duct dil 2am, pt does not look bad, feeling better than when first arrived, but still signif RUQ pain
- 2am surg PGY 2 says, “consult GI for urgent ERCP for cholangitis, discussed with surgery attdg” and that quote is on the chart.
- ER calls you at 230am.

Alcohol

- TAKE A CAREFUL HISTORY
- Often after pt stops drinking (CCK is upregul and pts start to eat more fat/protein).
- “The night of the day after” a binge
- Typically a lot: >50g/day for years
- *But no amount of ETOH is safe*
- More in men; lipase 2X amylase?
- 1st or 2nd most common in US (31-40%)
- Mitochondrial toxin, lysosome instability
- Reactive oxygen species, proinflamm
- Increased lysosome and enzyme production
- Decrease panc blood flow, precipitate panc proteins
- Why only 10% of alcoholics get panc'tis? SPINK?
- Often have CP

Triglycerides

- Usually >1000 : an endocrine emergency!
- Can occur in 500 range
- In Children it is known that keeping trigs <200 prevents AP
- Alcohol raises trigs usually to 400-500 range, can be higher
- *Can have normal amylase and lipase.*
- What about post prandial trigs?
- Uncontrolled hyperglycemia can lead to high trigs
- Often have CP
- IV insulin works faster/better than SQ

Trauma

- Disrupt PD as pancreas crosses spine in mid body
- ERCP needed once stabilized to “bridge” duct disruption with stent to prevent apoptosis of tail.

pERCP

- Often mild/interstitial
- 5% of all ERCP
- But only 1/1000 of those are necrotizing
- pH of contrast dye? Osmolarity of contrast? Stent fell out?
- Mechanical swelling of papilla? Wire in duct?
- Bacterial reflux? Thermal effect of sphincterotomy?
- RFs: Pt, procedure (SOD, nondil ducts, no cancer, no stones, more cannul attempts, more panc dye injection (body, tail, acinarizat), pt with nml panc, pdivisum/ampullectomy, spincterotomy esp precut, dilating biliary orifice without sphincterotomy, <50 cases/year, <200 lifetime cases) Prophy: indocin PR? PD stents, wire guided cannulation

Other endoscopic causes

- Diagnostic or therapeutic EUS
- Deep enteroscopy
- Duodenal adenoma resection even if lateral wall

Post surgical

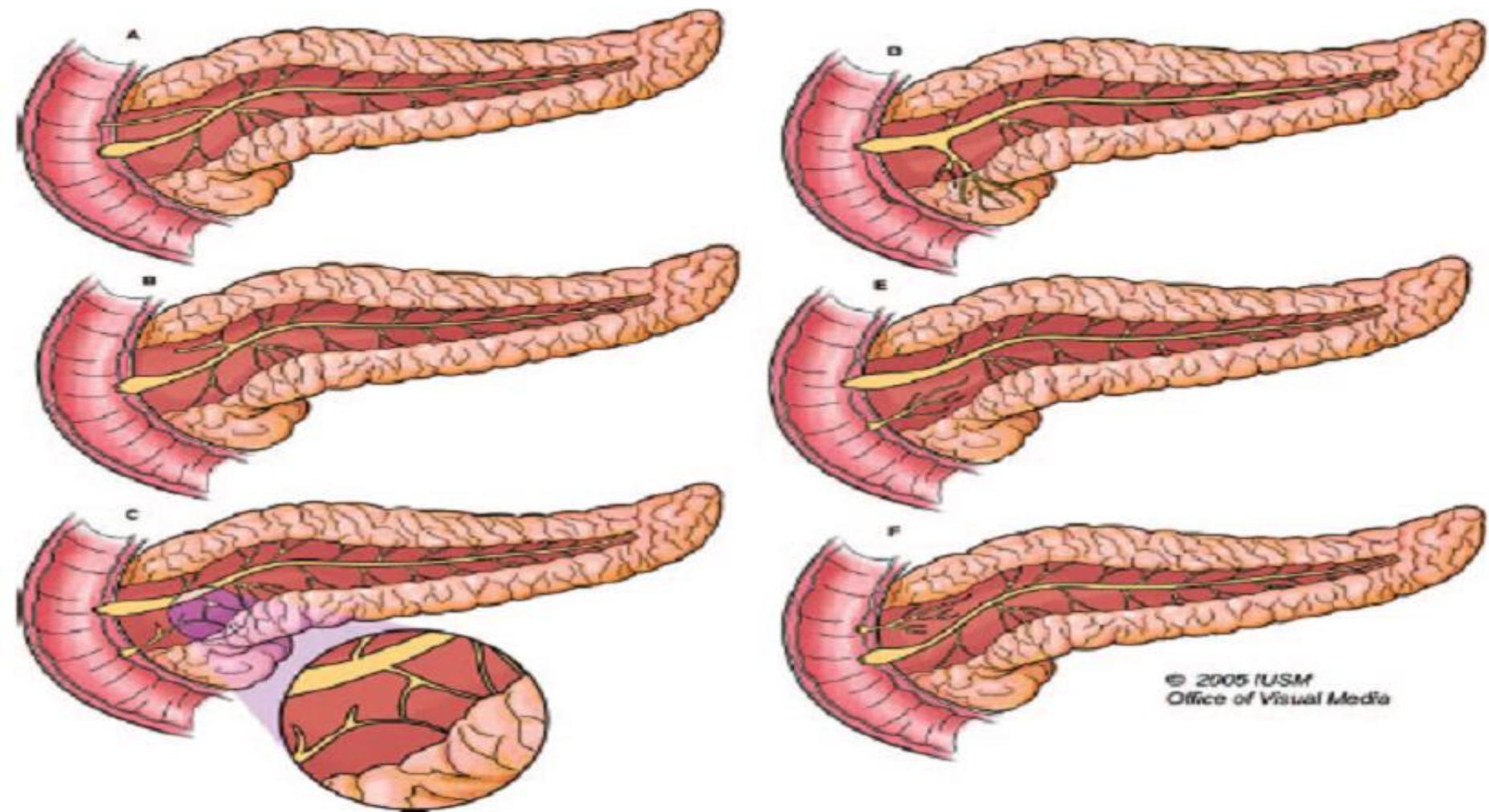
- 25% post CABG have high amylase
- 1% of CABG have necrotizing pancreatitis
- Mechanical stretch—Kocher maneuver?
- Ischemia?
- Anesthetics (propofol/trigs)?
- Cardioplegia? (CaCl during CABG)
- Note: amylase/lipase elevations in ICUs are common, most often not clinical pancreatitis

Obstructive/Tumor

- Adenocarcinoma of pancreas/Acinar cell Ca
- P divisum? SOD? Long Common channel, Caroli
- IPMN, neuroendocrine, mets, lymphoma
- Ascariasis
- Ampullary tumors/diverticula (latter, controver.)
- Post acute pancreatitis with panc duct stricture
- Sprue or Crohns of duodenum
- **ALL UNEXPLAINED PANCREATITIS PTS OVER AGE 50 OR EARLIER IF FHx OF RELEVANT CA's (RCC, Breast CA, brain CA, uterine, etc) SHOULD HAVE A CT 6 week or so after the AP.**

IPMN





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Figure 3. Diagrams show normal and abnormal variants of ductal anatomy. In a normal variant (A), the main pancreatic duct drains via the major papilla, and a patent accessory duct drains via the minor papilla. In the first abnormal variant (B), the accessory duct is not patent at the minor papilla. In incomplete pancreas divisum (C), a filamentous ductal branch connects the ventral and dorsal pancreas. In another variant of pancreas divisum (D), the entire pancreatic ductal system drains via the minor papilla. In typical pancreas divisum (E), a small ventral duct drains half of the pancreas via the major papilla, and a large dorsal duct drains the other half via the minor papilla. In reversed pancreas divisum (F), the accessory ductal system drains a small portion of pancreatic parenchyma via the minor papilla, and the main pancreatic duct drains the major part of the pancreas via the major papilla. (Reproduced with permission from the Office of Visual Media, Indiana University School of Medicine.)

Drugs

- AIDS— didanosine, pentamidine
- Antimicrobial— metronidazole, sulfonamides, tetracycline , INH, dapson, nitrofurantoin
- Diuretics— furosemide, thiazides (sulfa?)
- Immunosuppressive/antimetabolite— L-asparaginase, azathioprine , 6MP, $\text{INF}\alpha$?, sorafinib*
- Neuropsychiatric— valproic acid
- Antiinflammatory— sulindac, sulfasalazine, 5-ASA, salicylates
- Others— calcium, estrogen, tamoxifen, Byetta, TPN, propofol, accutane, vit A
- *sorafinib can raise lipase/amylase without p'itis

Drug Induced AP by timing

- Early
 - Within 30 days
 - Reflects hypersensitivity or direct damage
 - Rash, eosinophilia
 - Azathiapriner/6MP, Sulfa, flagyl, ACE, salicylates
- Late
 - Often after several months
 - IgG or T cell related?
 - Buildup of toxic metabolites?
 - Didanosine, pentamidine, valproic acid

Drug induced AP by mechanism

- **HYPERTRIGLYCERIDEMIA**

Tamoxifen, estrogen, finasteride, beta blockers, vit A, thiazides

- **ANGIOEDEMA**

ACE-I's—Bradykinin?

- **DIRECT TOXIC**

Sulfa, diuretics

- **IMMUNOLOGIC**

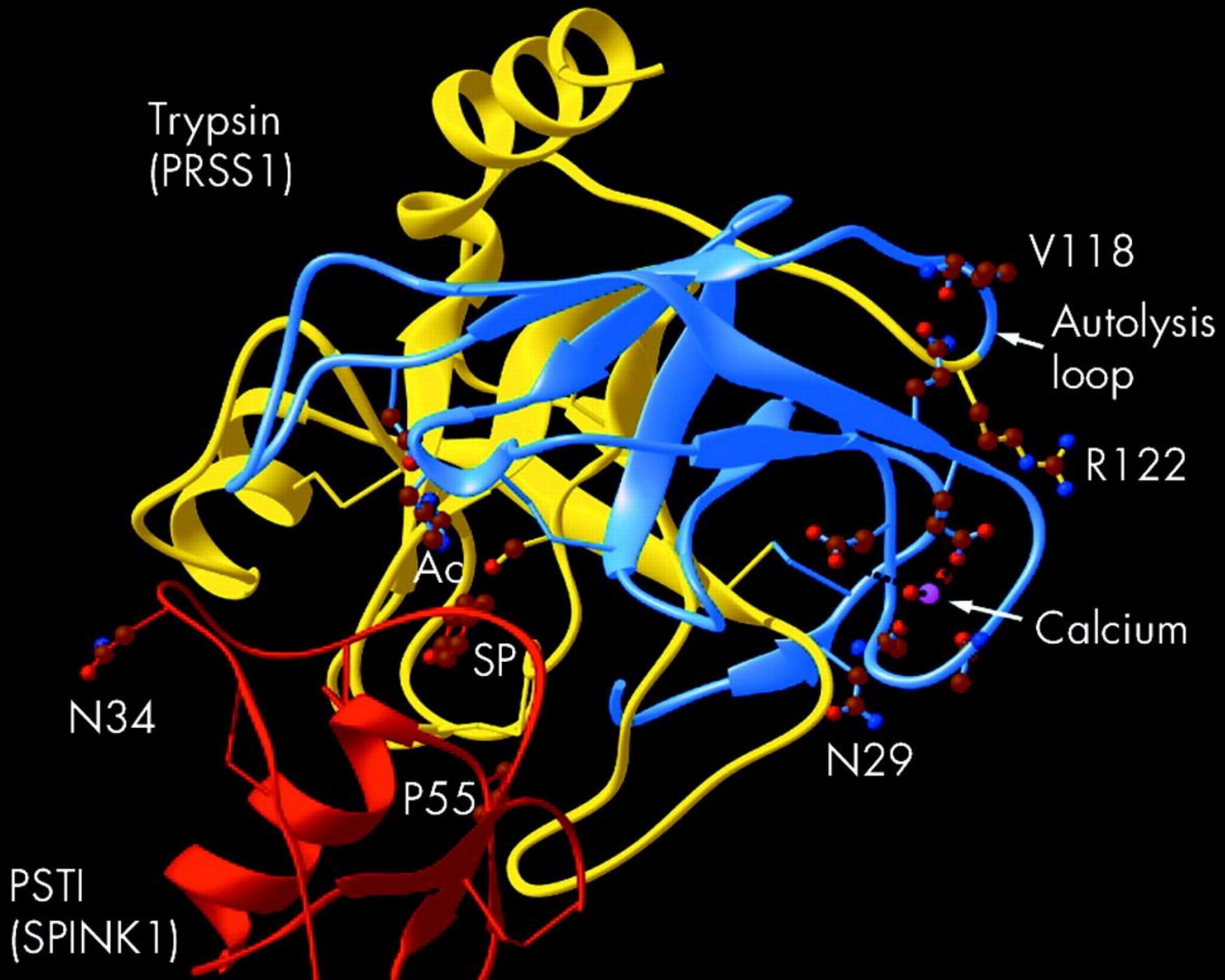
Sulfa, 6MP/Imuran

Infectious

- Viruses – Mumps, coxsackievirus, hepatitis B, cytomegalovirus, varicella-zoster, herpes simplex, HIV, atypical dengue
- Bacteria – Mycoplasma, Legionella, Leptospira, Salmonella, MAI/TB
- Fungi – Aspergillus, cryptococcus
- Parasites – Toxoplasma, Cryptosporidium, Ascaris, Pneumocystis carinii, leshmaniasis

Exotics

- Scorpions (Trinidad/Mexico/India/Israeli)
- Brown Recluse
 - (Sorry Santhi V, I cannot corroborate, maybe dapsone?)
- Snakes (very rare)
- African “killer” bees (very rare)
- Gila Monster



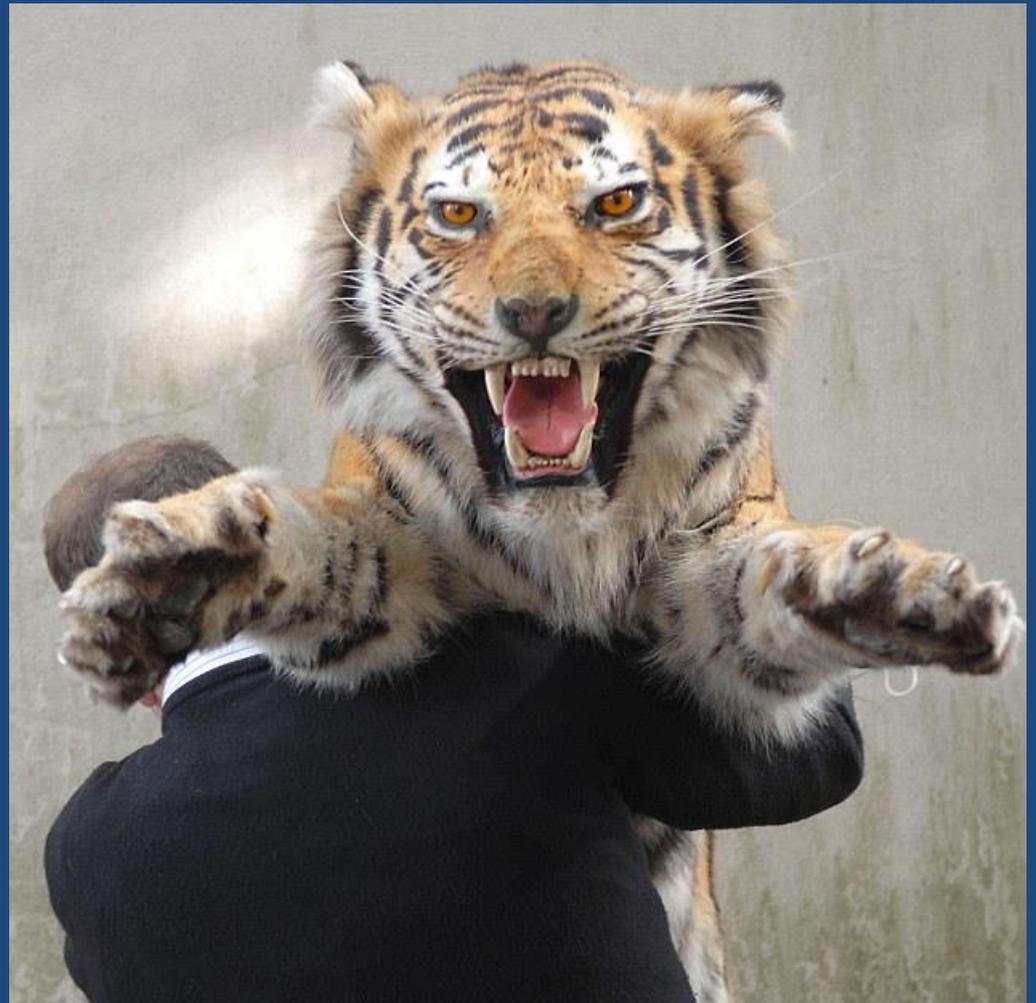
Hereditary

- PRSS1
 - Auto dom. Incomplete pen. First attack by teens
 - Calcif CP is inevitable. No Tx. 20% lifetime CA risk
 - Islet cell autotransplant?
- SPINK –recessive. Not a cause, but a modifier
- CFTR—“atypical CF” “panc sufficient CF”
 - Explains many “idiopathic” cases. Recess.
 - 2 CF’s with two SPINKS: marked increase risk
- Chymotrypsin C (rare)

Interstitial/edematous pancreatitis



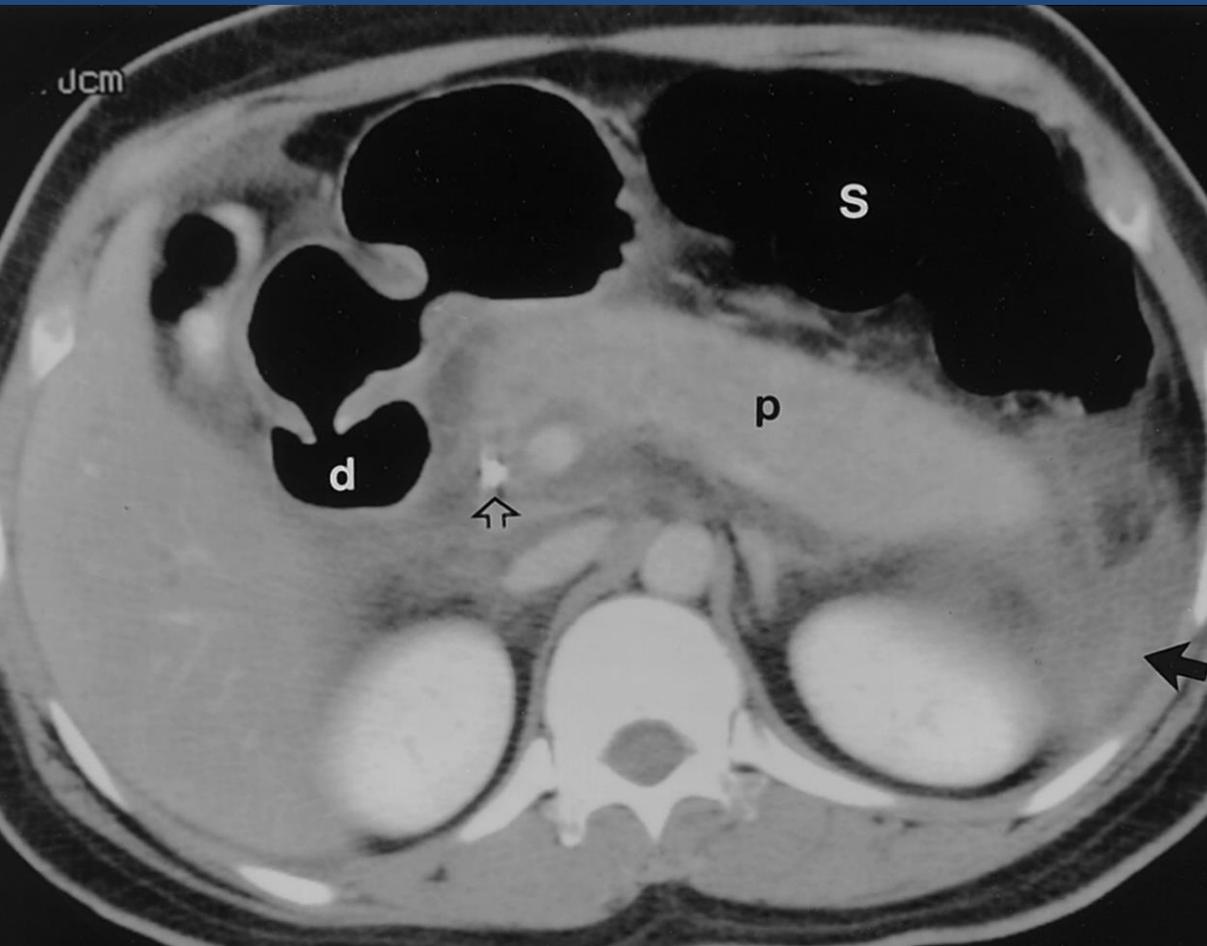
Necrotizing Pancreatitis



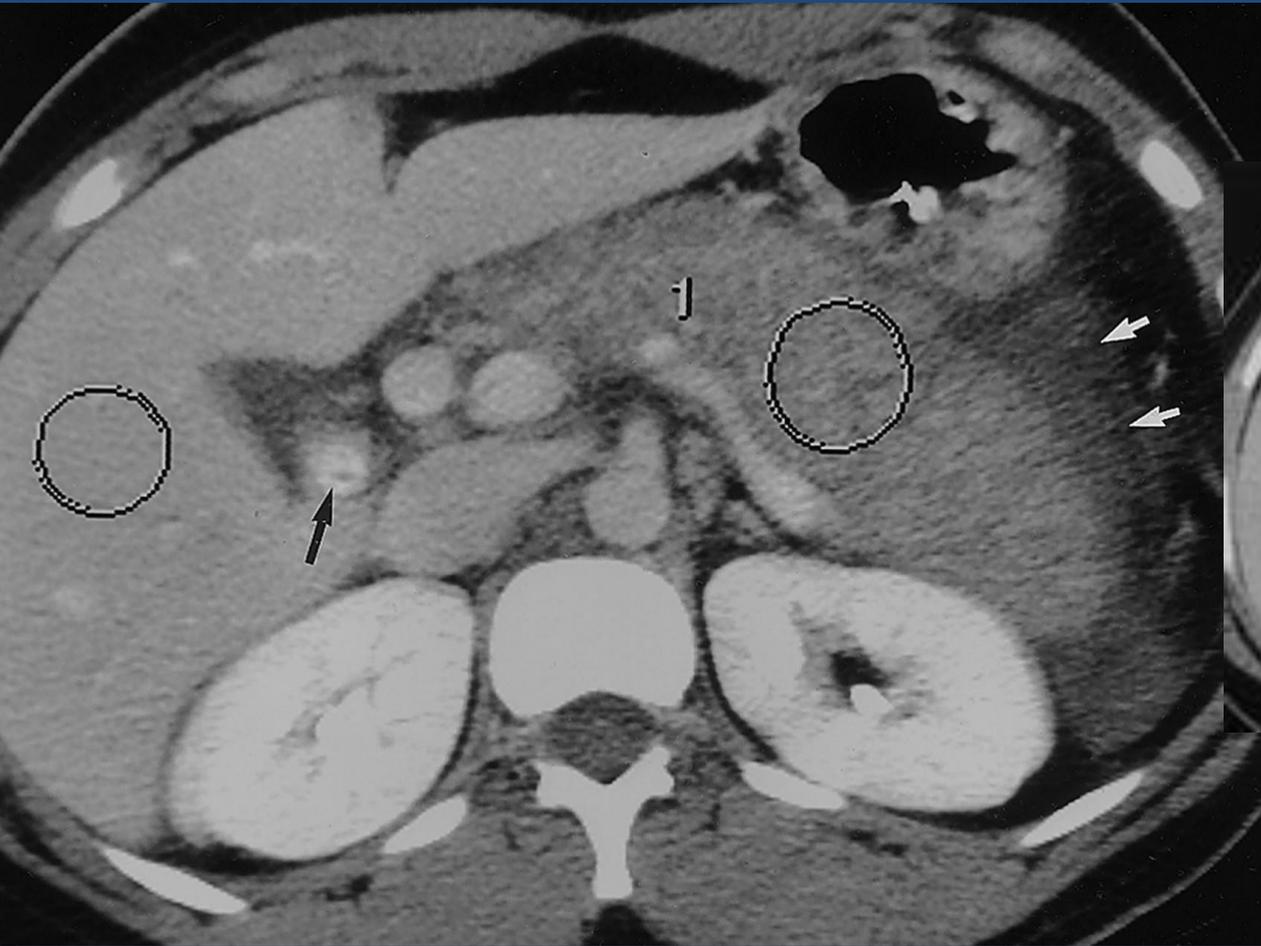
Mortality

- Overall 2-5% and decreasing slightly
- Interstitial/mild pancreatitis (80% of all cases)
 - $\leq 1\%$ mortality
- Necrotizing/severe pancreatitis (20% of all cases)
 - 20% mortality, long ICU stays (1-3 months)
- Infected necrotizing pancreatitis (occurs late)
 - 50% mortality

Interstitial pancreatitis



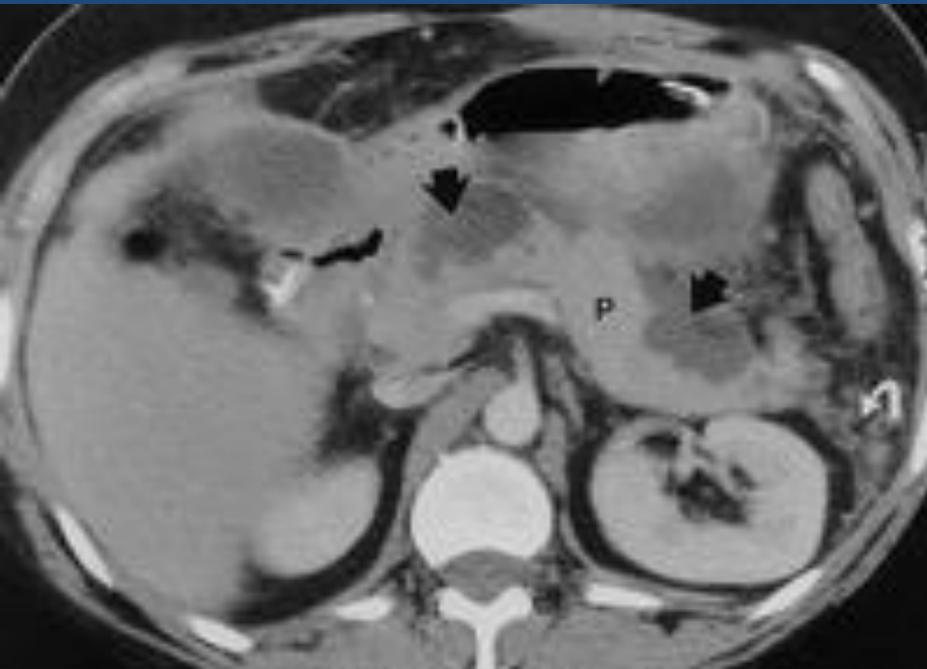
Interstitial pancreatitis

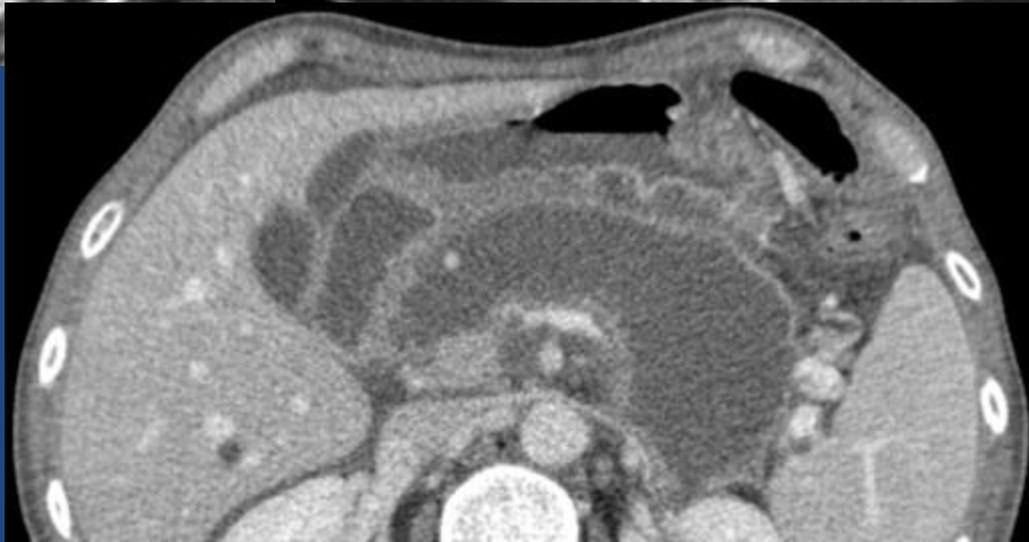


Necrosis

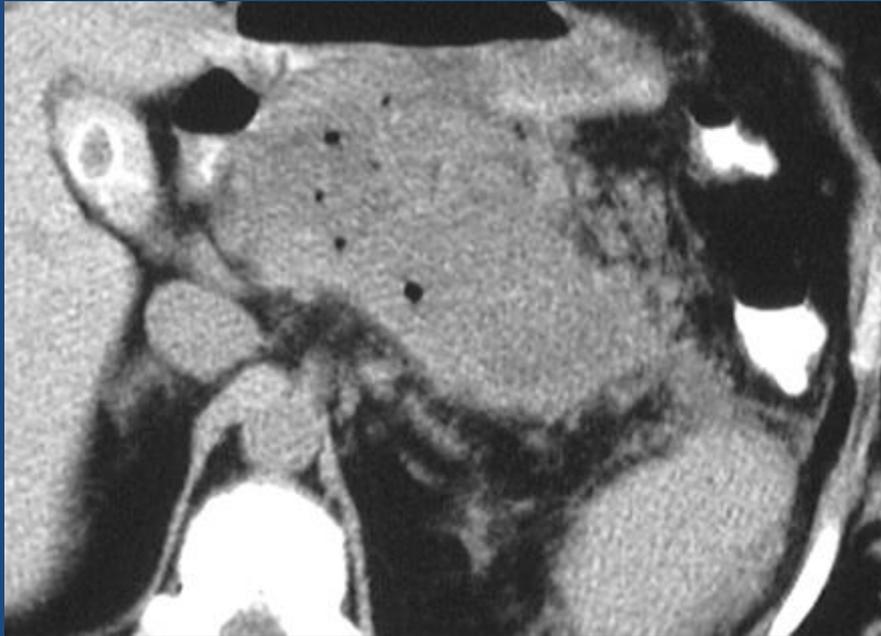


Necrosis





Pancreatic abscess/infected necrosis



How pts die with AP?—Two Peaks

- **Early** within 1-2 weeks and often within 72 hours: multisystem organ failure (kidneys, lungs with ARDS) (can't be ventilated/oxygenated even on vent), DIC, hypocalcemia, shock/hypotension, abd compartment synd, aspiration, cholangitis, acidosis, hemorrhagic pancreatitis, intest ischemia (clot in SV->SMV))
- **Late**: pancreatic abscess/infected necrosis, usually by 2 weeks, secondary biliary obstruction, hypoalbuminemia, Hospital acquired (VRE, MRSA, line infect, aspirations), PE, gastric variceal bleeding, gut failure, neg nitrogen balance.

Hemosuccus Pancreaticus





Clues to send to ICU



- Tachypnea
- Oliguria $<50\text{ml/hr}$
- Hypotension/orthostasis
- Tachycardia >130
- Tense, distended abd
- Grey-Turner's/Cullen's
- Pallor, cold extremities
- Jaundice esp if febrile
- Azotemia, hypoalbumin
- Age > 55 , high fluid req'm
- First attack
- Mental status changes
- Uncontrol hyperglyc/hypo
- Cardiac ectopy (recurrent runs of NSVT/PVCs)
- QTc $>440\text{msec}$
- Obesity BMI >30 , "apple"
- Baseline dec card/pulm fn
 - Diastolic dysf
- Hemoconcentration
- WBC >15 , bands/myelos
- Pleural effusion

Clinical indices of severity

- RANSON
- APACHE
- ATLANTA
- BISAP
- Glasgow
- Delta HCT and/or Delta BUN

Ranson

At presentation

- Age >55
- White blood cell count >16
- Blood glucose >200 mg/dL
- LDH >350 U/L
- AST >250 U/L

At 48 hours

- Hematocrit Fall by $\geq 10\%$
- BUN Increase by ≥ 5 mg/dL despite fluids
- Serum calcium <8 mg/dL
- pO₂ <60 mmHg
- Base deficit >4 mEq/L
- Fluid sequestration >6 L

1-2 criteria - > <1% mortal

3-5 criteria - > 15% mortal

6-8 criteria - > 60% mortal

9-11 -> >75% mortal

APACHE II

- Temp high or low
- MAP high or low
- HR high or low
 - (HR 60 gets 2pts!)
- Na high or low
- K high or low
- Creat elev
- Age over 44
- APACHE-O
 - BMI>25 1 pt
 - BMI>30 2pts
- WBC high or low
- Glasgow coma (low)
- pH or HCo₃
 - High or low
- PaO₂
- Nonsurgical and emergency surgery
 - More points

Score <8 Mortal <4%

Score >8 8-18%

ATLANTA (1992)

- Mild vs severe (necrosis or organ failure)
- APACHE \geq 8 or RANSON \geq 3
- Organ failure
- Systolic blood pressure <90 mmHg
- Pulmonary insufficiency $PaO_2 \leq 60$ mmHg
- Renal failure Creatinine ≥ 2 mg/dl after rehydration
- Gastrointestinal bleeding 500 ml in 24 h
- DIC: Platelets ≤ 100 fibrinogen <1.0 g/l and fibrin-split products >80 μ g/l
- Calcium ≤ 7.5 mg/dl

ATLANTA REVISED (2008)

- Early severity->organs fail
- Late severity->Structural (necrosis), esp infect
- PERSISTANT ORGAN FAILURE (>48 hrs)
- NEW DEFs of Radiographic/structural features of severity

ATLANTA

1992

- Interstitial vs necrotic
- Pseudocyst vs abscess

Revised, 2008

- Interstitial edematous panc
- Sterile necrosis
- Infected necrosis
- Acute
 - Necrosis vs fluid, sterile vs infected
- Chronic
 - Pseudocyst vs walled off necrosis
 - Sterile vs infected

BISAP

- SIRS
 - T >38.5°C or <35.0°C, HR>90,
 - RR >20 or PaCO₂ <32 mm Hg
 - WBC >12,000, <4000 or >10 percent immature (band) forms
- BUN>25
- Age>60
 - 0-2 pts: <2% mortal
 - 3-5pts: 22% mortal
- Pleural effusion
- Altered mental status (glasgow CS < 15)

Glasgow

- Age >55
- WBC >15
- LDH>600
- Glucose >180
- Album <3.2
- Calcium <8
- PaO₂<60
- BUN>45

At admission and at 48hr
Score 0 to 2: 2% mortality
Score 3 to 4: 15% mortality
Score 5 to 6: 40% mortality
Score 7 to 8: 100% mortality

Grading based upon findings on unenhanced CT BALTHAZAR SCORING

Grade	Findings	Score
A	Normal pancreas - normal size, sharply defined, smooth contour, homogeneous enhancement, retroperitoneal peripancreatic fat without enhancement	0
B	Focal or diffuse enlargement of the pancreas, contour may show irregularity, enhancement may be inhomogeneous but there is no peripancreatic inflammation	1
C	Peripancreatic inflammation with intrinsic pancreatic abnormalities	2
D	Intrapancreatic or extrapancreatic fluid collections	3
E	Two or more large collections of gas in the pancreas or retroperitoneum	4

Necrosis score based upon contrast enhanced CT

Necrosis, percent	Score
0	0
<33	2
33-50	4
≥50	6

Lieb's Orderset

- Call for temp >100.5 or <97, HR >100 or <50, U/O <10cc/kg/hr, MAP <60, RR >12
- FS q4hr. Call if >200 or <60
- I/O check q4hr
- Dilaudid PCA, zofran/phenergan for N
- IV H2 blocker if not already on something. Good for first few dys, after that probably would not use esp if not on it on presentat.
- Hold ACEI
- NPO except comfort swab
- If neighbor not NPO, get private room.
- Repeat BMP/CBC in 4-6hrs after last one, consider LFT, mag, phos, cal/album repeat then too.

Fluids: Theory and Data

- Principles/theory:
 - Fluid prevents capillary microthrombi (necrosis?). Improves renal perfusion
 - Can we prevent necrosis? First 12 hours maybe the key
 - Once necrosis occurs, be careful with over resuscitation
 - Often these pts have low albumins and have near abd compartment anyway
 - Bolus, frequently. A pulse jet, not a hose! Think sepsis goal directed therapy
- Data: retrospective –more fluids are bad, classic causality bias
 - Goal directed no better: classic referral bias (post hoc, LR better)
 - China RCT: Colloid like heta starch (HESPAN) better? Really?
 - But Large studies in all takers with shock show increased ARF, coagulopathy and death with heta starch

Fluids, practical

- MD Stay in house until pt makes urine.
- Have night person in house check U/O, glucoses and f/u repeat BUN/creat/CBC
- Send to ICU NOW! if hemoconcentration, question of abd compartment, elev BUN, oliguria
- LR for first 1-2 L's unless hypercal/kal. If no U/O may have to switch to NS AND BOLUS another 1-2L. Once second lytes back and OK and if urinating can do LR again
- Typically 250cc/hr or more for first 12 hr.
- Care with old age >75, diastolic dysf, CRI, baseline edema/hypoalbum, known necrosis (ie late presentation)
- When in doubt give more fluids, consider CVP monitoring

Imaging now or later?

- RUQ US STAT anyone with no ETOH Hx and with intact GB and with elev transaminases/bili.
- CT: only those with questionable dx or maybe very ill about to be intubated or whisked to OR or >72 hrs into course.
 - Is it a perf DU? But a KUB will help
 - No CT post ERCP.
- MRCP can be done without gad!
- Future: $^{13}\text{NH}_3$ PET

Nutrition in acute pancreatitis—ideal world

- Goals: rest the pancreas/avoid bile flow
 - Post Lig o Treitz, small volumes
 - Can use elemental/MCT's (medium chain trig)
 - Avoid aspiration
 - Give with enzymes
- RCT comparing stomach feeds to TPN found stomach feeds superior if started within 48hr.
- RCT comparing post LOT feeds superior to TPN.
- Try to avoid TPN-- cost, infection, proinflam
- Dobhoff feed everyone with AP (DDW 2012)?!!

Nutrition in acute pancreatitis—real world

- Most mild AP resolves in a few days, don't need nutrition suppl.
- Most necrotizing panc have partial duodenal sweep obstruction so tough to pass dohoff.
- Sedation for GI guided nasojej feed tube may require general due to aspiration risk. Long 1.5hour procedures at least.
- Most necrot panc are hypoalbum, hypoprotein.—no oncotic pressure to absorb enteral
- Ileus often present in necrotizing panc.
- Pts are high risk of aspiration of feeds.
- MCT's, elemental feeds expensive, almost like TPN

When to use antibiotics?

- Never for prophylaxis?
- If use antibiotics should be broad.
- Use if cholangitis suspected (amp/sul is all that is needed if no pancreatic necrosis)
- Meropenem (MRSA, anaerobes, GNR) or pip/tazo
- If PCN all, can use Vanco, levo and/or azactam, flagyl
- Don't forget fungi if recent surg/intervention or if failing a carbapenem.
- Don't forget VRE.
- Ecoli 51%, enterococcus 19%, staph 18%, klebs10%, proteus 10%, pseudomonas 10%, bacteriodes 6%

When should IR drain a collection?

- Never?—Should only tap and send for culture, if grow a bug/bugs?
- In practice, IR tends to leave a drain—maybe bad!
- Typically surgery team should be aware of pt before you send to IR (you infect the surg bed!)
- If IR drain, should be retro approach

Infected necrosis, new reality

- NEJM april 2010, Dutch group, 88pts, RCT, “PANTER”
- “Step up approach”
- Open laparotomy (bilateral subcostal) vs IR (12 Fr, multiple drains, only 2 endoscopic followed by retroperit debride (VARD))
- No mortal diff. 10% less cost in IR approach.
- Combined end point “death or major comlx” reduced from 69% to 40%.
- Open pts had more reoperations for sepsis
- 50% were not university hospitals, but were “tertiary” so were surgeons not as good? Why open approach, bilater subcostal?
- More of a test of VARD vs open not IR vs open

Infected necrosis principles of mngmt

- Wait as long as you can before intervening
- Ideally at least 3-4 weeks
- Even if that means intubation, dohoff feeds
- Let the bed mature/get walled off—this allows for IR/Endoscopic management
- New data suggest conserv mngmt for all necrosis has a 7% mortal
- Surgery series, 25-50% mortal (old studies)
 - Newer surg series say 50% mortal in first 14 dy, 15% at 30 or more days

Endoscopic necrosectomy

- Must be within 1cm of stomach, walled off and mature
- Avoid if resp unstable, OK if intubated already
- If pseudoaneurism, must embolize in IR first.
- Endoscopic series
- 0 -7.5% mortality (2/96)—but these are sick pts, many “too sick” for surgery
- Median scopes: 5
- Morbidity 25%, particularly bleeding, aspiration, abscess
- 80-90% successful
- F/u 43 mo
- Old teaching “not for infected necrosis”—no longer!

When to call a surgeon?

- Pt unstable.
- Infected necrosis proven or suspected. They may not intervene until later, but let them know
- Abdominal compartment syndrome (ck foley pres)
- Pt with multiple poor prognostic signs, age, WBC, oliguria, SIRS
- **ELECTIVELY WITH ANYONE WITH STRONG SUSPICION FOR GALLSTONE ETIOLOGY WITH GB STILL IN SITU. MUST HAVE CONSULT ON CHART AND F/U ARRANGED—HIGH RECURRENCE RATE W/O CHOLE**
- Pt in ICU, can't eat/tolerate enteral, not improving

Scenarios

- Alcoholic, 3 attacks of AP from Sept to Feb with very similar CT findings, ER visit in April for same, but no CT done. Pt insists feels much worse sense april, early satiety, nausea
- Alcoholic presents to ED in extremis with acute onset abd pain 2 hours ago, AP, tachypnic, distended, not tense, ileus BS's, hypotensive, creat 1.5 (was 0.8), BUN 50, was 5, afebrile—CT or no CT? what else to do? VA?
- 50 yo main line lawyer with unexplained AP neg US, nml trigs “I don't want a CT, too many rads. MRI, cost ineffective”

Scenarios

- 50 yo poorly controlled DM, obese, with 24 hrs abd pain, AP, after eating BarBQ/fries/cheesecake and two beers
- WBC 15, glucose 400, creat 2.2, on admit up from 1.2, trigs 400 after 36 hr fast
- Doing well on med floor, feels better, FS 250
- CT now shows 10% necrosis and fluid collection about 3cm
- Pt wants to eat and “get outa here”
- Enthusiastic med resident calls, “hey GI/IR, please Drain collection?”

Scenarios

- Alcoholic, severe presentation with SIRS, hypoxia, oliguria, gets intubated and then few days later, generally better. Initial CT on admit revealed “mild AP” with peripancreatic fluid
- Febrile on admit, deferved after 2-3 days, now has few temps 100F in last 48 hours. You are called b/c ICU wonders why pt still distended, not tolerating dohoff feeds, not weaning. You note initial WBC of 25, currently 15.
- What do you do next?

Scenarios

- OSH doc (GI or gen surg) calls you at 2am on Sat nite from Poconos or S Jersey (200bed hosp)
- Pt with severe AP in their ICU same scenario as last one, with CT showing 50% gland necrosis with extensive peritoneal/retroperit necrosis, but doc asks—“this area has to be drained, I’m going to call in IR for perc drain and then we will send pt to you, OK? Do you accept pt?”

Scenario

- You are the PGY4 on call
- New PGY6 starting an ERCP rotation (20th ERCP)
- ERCP done for intermittent dil CBD and RUQ pain with normal LFT, biliary sphincter done in this young female, age 20 with no alcohol history. Never had pancreatitis. Never had DM No stones found, no stent used, She smokes. She is 5'6" and weighs 190lbs, pt admitted for pain/AP post procedure.
- Floor nurse calls you for glucose on panel is 300.
- Worried? Not worried? What other questions should you ask? How to best manage this pt

Scenario

- Same pt calls you from home, has pain, thinks it is gas pains. What should you do?
- Different pt just had “a small intestine polyp” removed by Dr. Ginsberg with PGY5 at 1pm, calling you with same at 8pm. She also “saw a plastic straw” in her BM just now. “Is that OK doc?” Ran out of her pec’s last week. “can you just get me a few more pec’s? I will be OK.” What do you do?
- Dr. Katzka’s IBS pt had a colonoscopy today and calling you with same. You note 20 calls in EPIC in the last month for various things. You note 3 CTs in the last 2 years in EPIC What do you do?

Scenario

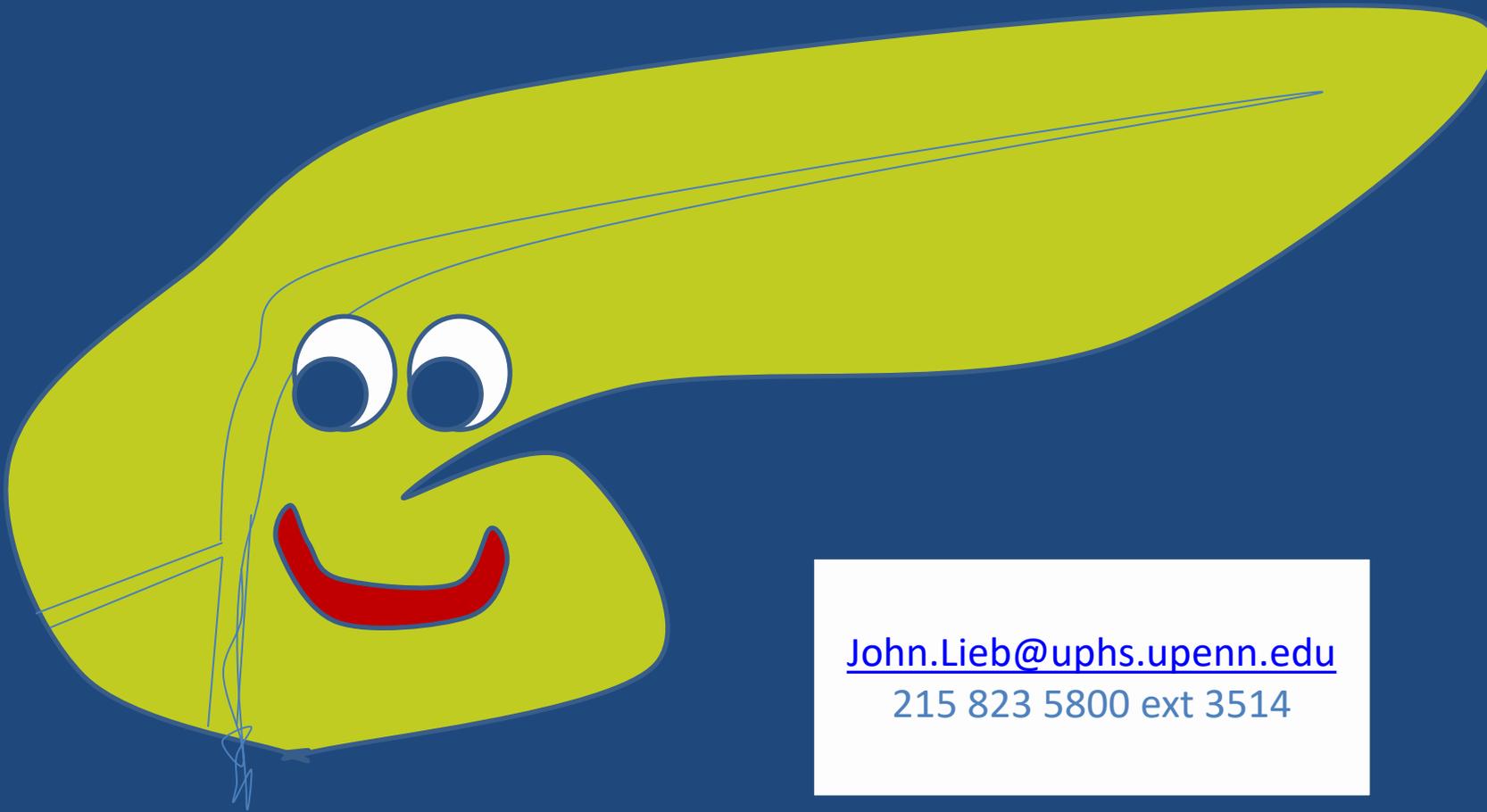
- 44 yo Main line lawyer with idiopathic Acute relapsing panc. Uncle had bad attacks of abd pain.
- Thin build, jogger, no other med problems.
- Calls you in ER “I’m waiting too long” “Can’t you do something?! I feel terrible. The pain is unbelievable”
- First attack was 9 months ago. Was in ICU with necrosis, ARF, tachypnea, but resolved. Two more attacks about every 3 mo since but not in ICU. Last CT 3 mo ago without necrosis or collections
- Worried? Not worried? Why? What do to next?

Take home points AP

- Causes, presentation, labs (incl trigs!) CAN BE PAINLESS!!!
- Causes of false pos/false neg amylase/lipase (>3X ULN!!!)
- Predict the problem pt:
 - age, abd obesity, SIRS, WBC, BUN, glucose, first attack etc
 - Know the most severe etiologies, GET TRIGS EARLY/ADD ON!!!!
- Triage (scoring systems)
 - Tiger or kitty (ie necrotic vs interstit)? Time will tell
- Hydrate hydrate hydrate for early disease by boluses, LR, consider colloid/heta starch if already necrosis
- Monitor Urine output, glucoses get 6 hr post CBC/BUN
- The first 12-24 hours should feel labor intensive—that means you are doing it right!
- Never hesitate to send to ICU if your gut is concerned
- Know how to recognize severe AP by CT criteria
- RUQ US first test of choice for unexplained
 - (non con CT or noncon MR when US not available or when no GB)
- CT with contrast useful later (48 hrs or more) or if other diff dxs

Take home points in AP

- CT more liberally later on (cyst or not)
- Know what a pseudoaneurism is and how to deal
- LFT abnml (esp ALT) predict biliary source (LABS are 12-24hr behind the pt!)
- ERCP for cholangitis in biliary pancreatitis
- Know when to MRCP (noncont if nec) vs ERCP
- Know who gets most severe pERCP pancreatitis (needle knife, SOD, ampullectomy, p divisum, etc) and RF's for such
- Know 2 mortality peaks and the causes
- Nutrition (early doh hoff jejunal feeds if necro)
- Antibiotics only when infected necrosis or cholangitis/biliary pancreatitis
- Call Surgery for instability, abd compart, biliary, suspected abscess, pt failing "to fly"
- Do not call IR until after surgeon involved (ideally panc surg)
- If call IR, encourage VARD if possible (left retroperiton)
- If no clear cause, check panc protocol CT in 6wks



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