Morbidity & Mortality Conference

Arun Chaudhury MBBS, MD

Geisinger

Privileged, nonpunitive, learning exercise to improve patient care

6-28-2022

12:27 44 y/o male (BW: 109 Kg Ht 5'11" BMI 33.52) presented to ED with abdominal pain, vomiting since AM, chills x 2 hrs

133/90, 90, 18, 96%, 98°F

PMH T2DM, HTN, Lumbar DDD, asthma, medication non-compliance, hypertriglyceridemia, (Adx March 2022 for pancreatitis), splenic vein thrombosis, migraine, diabetic polyneuropathy

PSH EGD 7-26-2016

Social History : No tobacco, current alcohol (1-2 wine coolers), previous marijuana

Family History : Paternal grandmother diabetes, maternal grandmother diabetes, father HTN, mother cancer, high TG in multiple family members.

ROS SOB, N/V, diarrhea, chills; No CP or recent alcohol use

Labs : Glucose 259, CO2 20, AG 19, Lipase 1170, Trop <6, Lactate 2, WBC 15.6 (N 84%), Hgb 16.7, Urinary Ketone 80, ABG pH 7.32, EtOH –ve, P 1.7

Home Meds : Insulin Aspart 20U meal time + SS, Lantus 50U, empagliflozin, fenofibrate, atorvastatin 80, fish oil, amlodipine, lisinopril, aspirin, spironolactone, sildenafil, topiramate, magnesium oxide, gabapentin, albuterol prn

6-28-2022

H&P **19:58**

HPI :Went to work (home health aide), experienced diaphoresis, central abdominal pain; was fine last night, had M&M  for snack, had pizza 1 day ago, 8/10 epigastric pain dull, constant; constipation x 2 days, compliant with medications and diet "for most part", wants medication to manage food cravings. Cannot tolerate metformin and GLP1 inhibitor due to pancreatitis, insurance coverage for Contrave limited, has not followed up OP with GI nutritionist

Initial ED management : DKA protocol, 2L NS bolus, 1L Isolyte, continuous Isolyte infusion, insulin drip, morphine, NPO, BG Q2H, VTE Px Lovenox

21:45 Report to RN PCU

22:35 Arrived to PCU, (2L NC, AAO x 4)

6-29-2022

Overnight no issue; diffuse abdominal pain and tender to palpation, WBC 12.54, Lactate 2, TG >4425
Lipase 490, Na 133, Glucose 203, Ionized Ca 1.1

10:13 **CT A/P** with IV contrast; peripancreatic fat stranding extending to transverse mesocolon and omentum, thickening of left anterior pararenal fascia, hypodensity in pancreatic tail : necrosis, ? pseudocyst, ? dilated pancreatic duct, splenic vein thrombosis, basal atelectasis, no pneumonia on lung window

Initiated on therapeutic Lovenox, GI consult, clear liquid diet, AG closed x 2, recommendation to continue insulin column for 2 hrs and then transition to carb coverage, AM Lantus

Working diagnosis : acute moderate to severe interstitial edematous pancreatitis with focal necrosis with fluid collection with new onset splenic vein thrombosis (**BISAP score** 1)

transferred to floor

13:50 GI consult: insulin gtt, TG goal<500, clear diet, advance diet with dietary fat <5% of total calories, continue fenofibrate, no endoscopic evaluation

15:34 TG 3121, insulin column, 11u/h, Accucheck Q2H

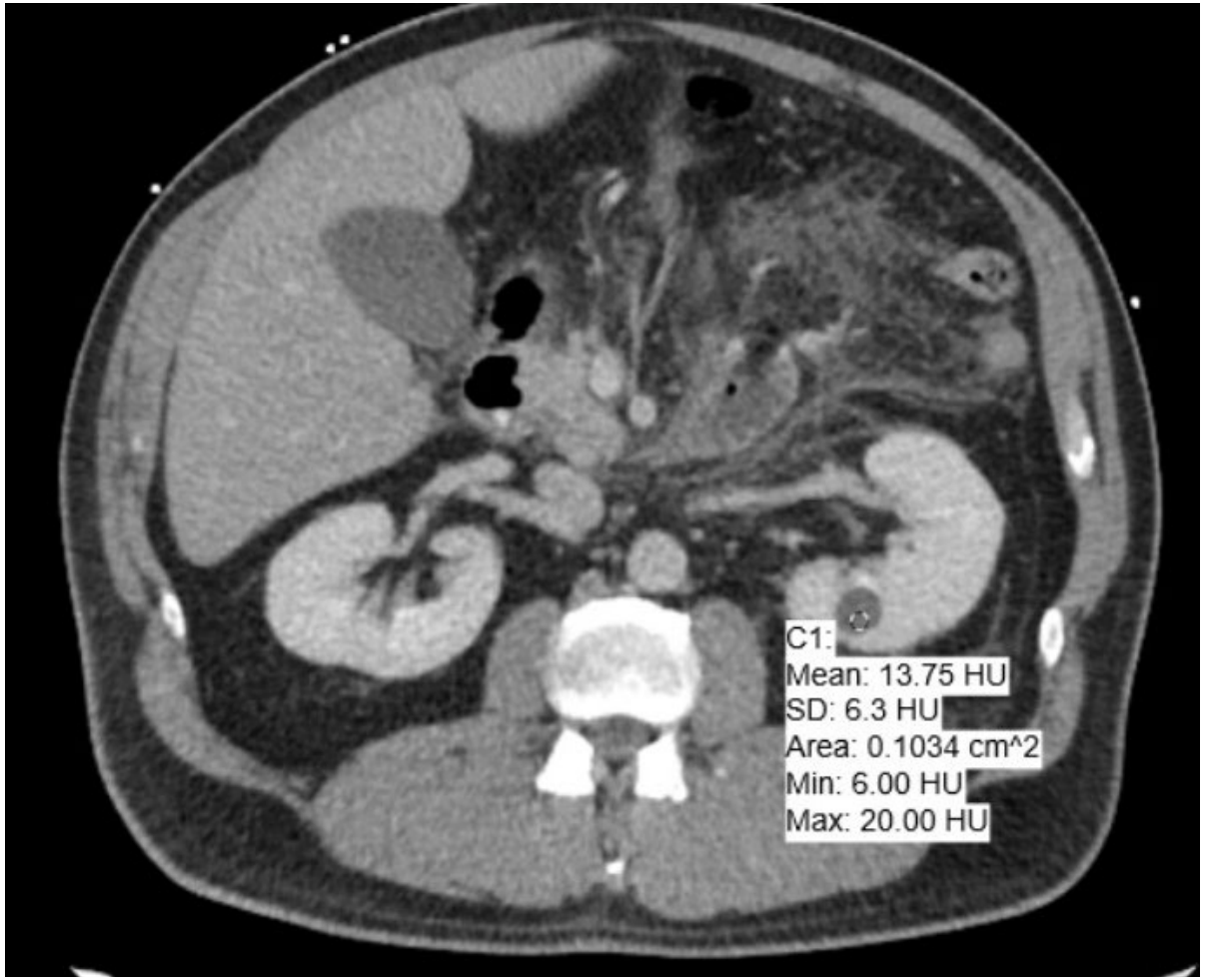
17:07 Left sided shoulder pain

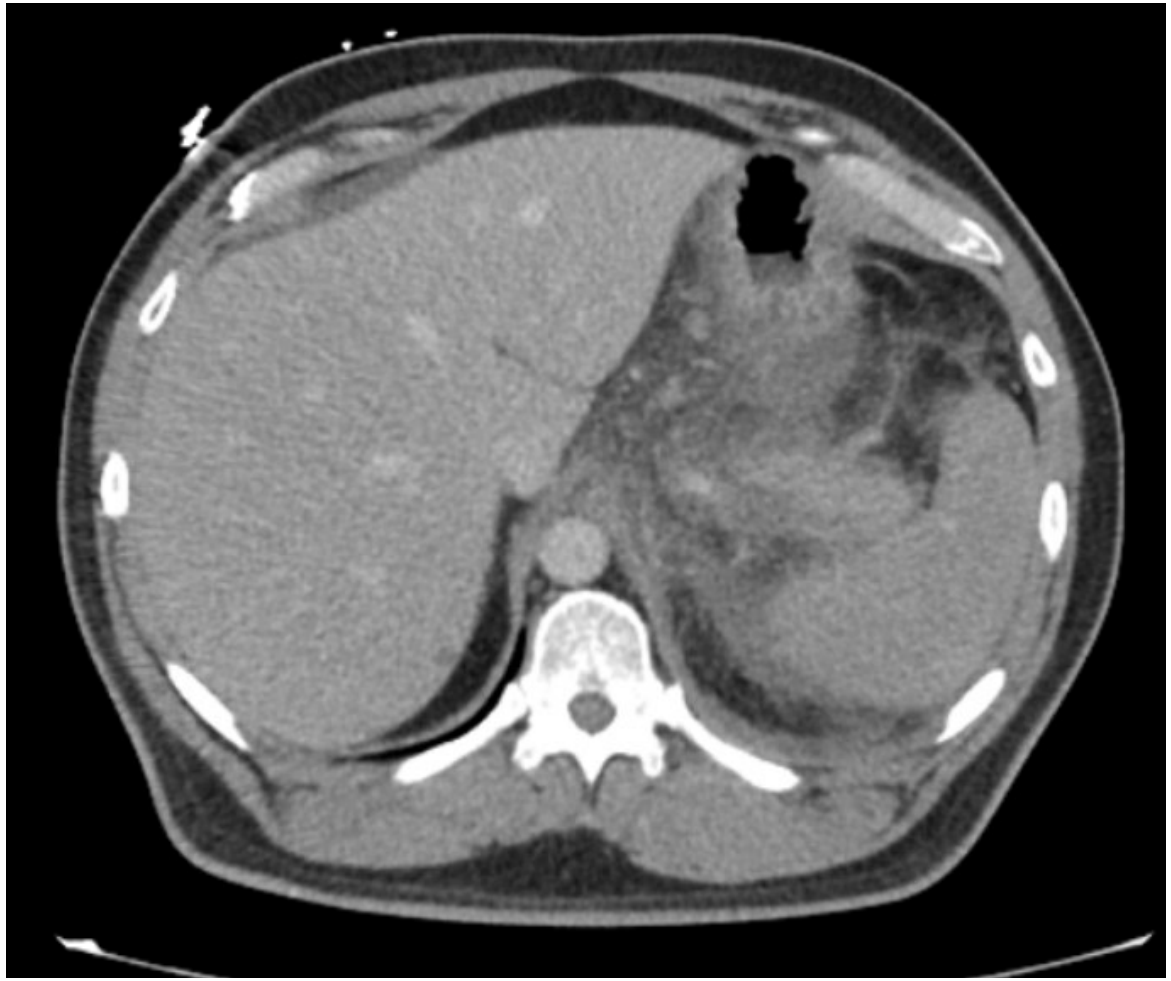
22:31 Insulin gtt, 1L O_2 NC

23:35 **Night Team** LLQ tenderness, BS+, 100.7 - 102.5F, 97/64, 114, 20, NSS Bolus 30 ml/Kg, Vanc/Zosyn

6-29-2022

10:13





6-30-2022

01:08 : Feeling weak, c/o decreased sensation in hands, more lethargic

02: 41 difficulty breathing, RR 14, 94%, 1L O2 NC, breathing treatment

03: 06 88/53, 85% on 1L O_2 NC, O_2 increased to 2L.

03: 57 86/62 manual, AO x 3, 3L O_2 NC, **transferred to PCU**

06: 26 Was started on Levo, now d/ced as MAP >65, mild diffuse abdominal pain, 89/60, 93, 13, 95%, glucose 127, WBC 10.18, Hgb 10.9, TG 1493, Cr 2.2, continuing insulin drip, continuing Zosyn, **WILL ASK GEN SURG FOR RECS** no **CXR?**

06: 30 GI same recommendations as before

08: 34 Antimicrobial stewardship program (ASP) generated ID consult: sepsis – mimic, no abscess, no positive culture, recs to monitor off antibiotics, supportive management

16: 13 Surgery agree with plan, no current role for surgical intervention, 107/68, 109, 16, 93%

MRI: no organized fluid, no gas, small area of necrosis

Surgery sign off, remain in background and to contact for change in clinical status

7-01-2022

09:28 137/88,99,17, 94%, TG 1493, Cr 2 to 4.9, gastroenterology consult, abdomen exam benign, insulin, fenofibrate, heparin, 1L bolus plasmalyte, infusion 150 to 250 ml/h, monitor fluid overload **CONCERN FOR SEVERE ACUTE NECROTISING PANCREATITIS**, prognosis guarded,

recommended PCU for at least next 48 hrs

10:29 Repeat **CT A/P** without IV and oral contrast, no interval changes

12:53 Nephrology consult, AKI 2/2 acute pancreatitis, fluid (if worsening renal function, therapeutic plasma exchange, ASFA 2019), avoid sodium bicarb for metabolic acidosis to prevent hypocalcaemia

15: 30 ID Recs to monitor off antibiotics because no current indication, AKI, and high risk of MDRO (Klebsiella pneumoniae Carbapenemase resistant)

14: 50 Suddenly unable to answer orientation questions or follow commands, "my body feels weak", stat head CT, no acute abnormality

17: 30 Bladder scan 612 ml, ambulated to bathroom but refused to void in presence of RN, neuro check intact, left bathroom at 18:20

7-02-2022

08:58 Cr 6.5, plasmalyte 150cc/hr, nausea, Tmax 100.5, **PCU status**

11: 13 Nephro no acute indication RRT

7-03-2022

04:15 136/84, 85, 20, 99%, **transferred to floor**

07:17 UOP +, abdominal distention, loose stool, Nephro recs : ? Compartment syndrome, check KUB to rule out any worsening ileus, (6:48, segment of small bowel in the left abdomen measures 3.8cm, mildly dilated, non specific mild gaseous distention of stomach and segments of colon), TG 476

7-04-2022

07:35 Abdominal pain improved, flatus, liquid stools, Cr 7.6, Hgb 8, IV heparin to Eliquis, insulin drip d/ced, Lantus and ISS, significant improvement of urine output, early sign of renal recovery

13:48 Gastroenterology, no plans for current drainage, + GPC blood culture, unclear source, intermittent fever, consider repeat CT scan to evaluate collection for gas that would be concerning for infected necrotic pancreatitis

7-05-2022

10:39 no acute overnight events, denies V/chills, good UOP, oral intake limited, particularly bad because of abdominal discomfort and nausea

11:41 Nutrition consult

14:59 Abdominal pain 6/10, relieved by Dilaudid, HD stable, 2L O2, "examines much improves today distention is down" continuing eliquis, continued fever spikes, Staph epi, assumed contaminant, repeat blood culture

21:52 Patient refusing Senokot, House Officer alerted unsure of why patient has 2 orders for Senokot (despite diarrhea)

22:00 10/10 pain on left side

23:19 0.5 mg IV Dilaudid

7-06-2022

AM rounds: Tired and fatigue, did not sleep well, left shoulder pain and generalized abdominal pain

12:30 RRT, Intolerable left shoulder pain, reproducible pain on palpation, hemodynamically stable, EKG NSR

13:45 dizzy and light headed during using the bedside commode, (loose BM, void , assisted back to bed), 3L O2 via NC

14:00 Patient sleeping

14:43 repeat labs K3.5, Cr 6.5, INR 1.43, WBC 9.87, Hgb 7.8 (HCT 23.6, MCV 88.4), TG 406

Attending note: 8/10 left sided chest pain
Chest pain assessed as musculoskeletal etiology, troponin –ve. Fever curve trending down, hold on Abx

20:42 Nephrology assessment, presyncopal episode may be due to developing intravascular volume depletion, related to acute pancreatitis and post ATN diuresis, consider NS 500-1000ml IV x 1

7-07-2022

04:32 : House Officer's Note: "Overnight patient hypotensive 97/67 held narcotics, gave 500ml bolus, rechecked pressure, went to 80/42, went to bedside, patient states he wants to sleep and wants pain to be in control, gave additional 100 ml bolus, Melatonin, BP improved to 116/72, but now having abdominal bloat, bladder scan done and showed 528 ml, will check a UA/Urine Cx, Prn straight cath"

01:47 Nursing Note: Post Bolus VS 80/42, 103, 20, 97% on 3L, 97.2, additional 1000 ml isolyte bolus

02:43 Isolyte Bolus Initiated

04:20 116/72, 98, 18, 99% on 3L, 98.5, patient reports bloated, per House Officer Ok to administer pain medication as needed, abdomen examination distended and firm, patient not voided since approximately 17:00, bladder scan 528ml, patient encouraged to void in urinal

04:45 : Patient reports he is unable to void, agreeable to straight cath

05:30 : Straight cathed 450 ml clear yellow urine, urine culture and UA sent, patient continues to report bloating, gas, abdomen remains firm/distended, House Officer updated

7-07-2022

08:30 WBC 19.13, Hgb 4.6, Cr 7.2

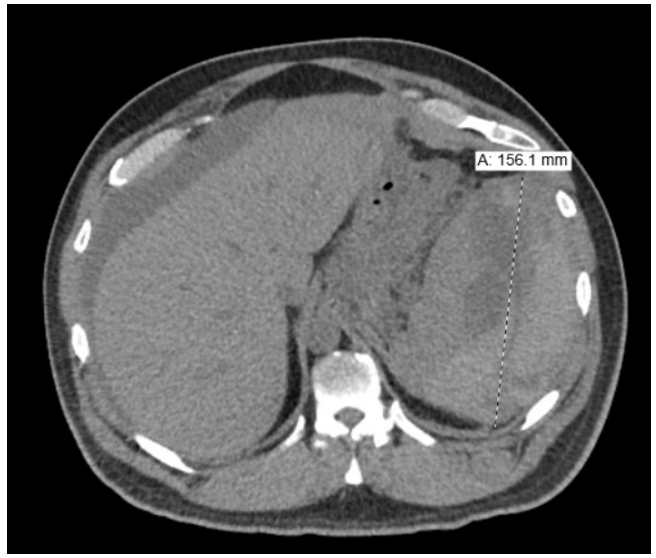
10:41 **CT A/P** splenic rupture with large amount of blood in the left upper quadrant surrounding an indistinct spleen, large hematoma adjacent to liver and moderate amount of diffuse hemoperitoneum; acute pancreatitis of the body and tail, with worsening ill-defined soft tissue density in the mesentery inferior to the pancreas representing either worsening inflammation or additional blood products

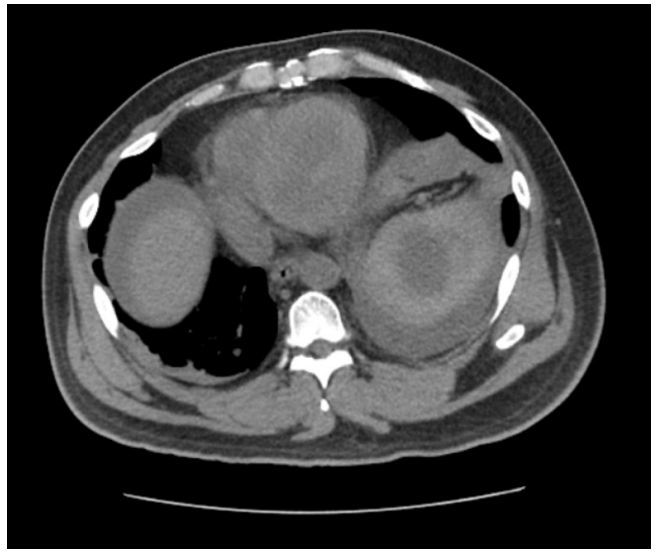
11:40 Attending Note: 8/10 sharp abdominal pain, worse with movement, alleviated with pain medications, patient did report some abdominal distention; abdominal examination: normoactive bowel sound, distended, tenderness in all 4 quadrants

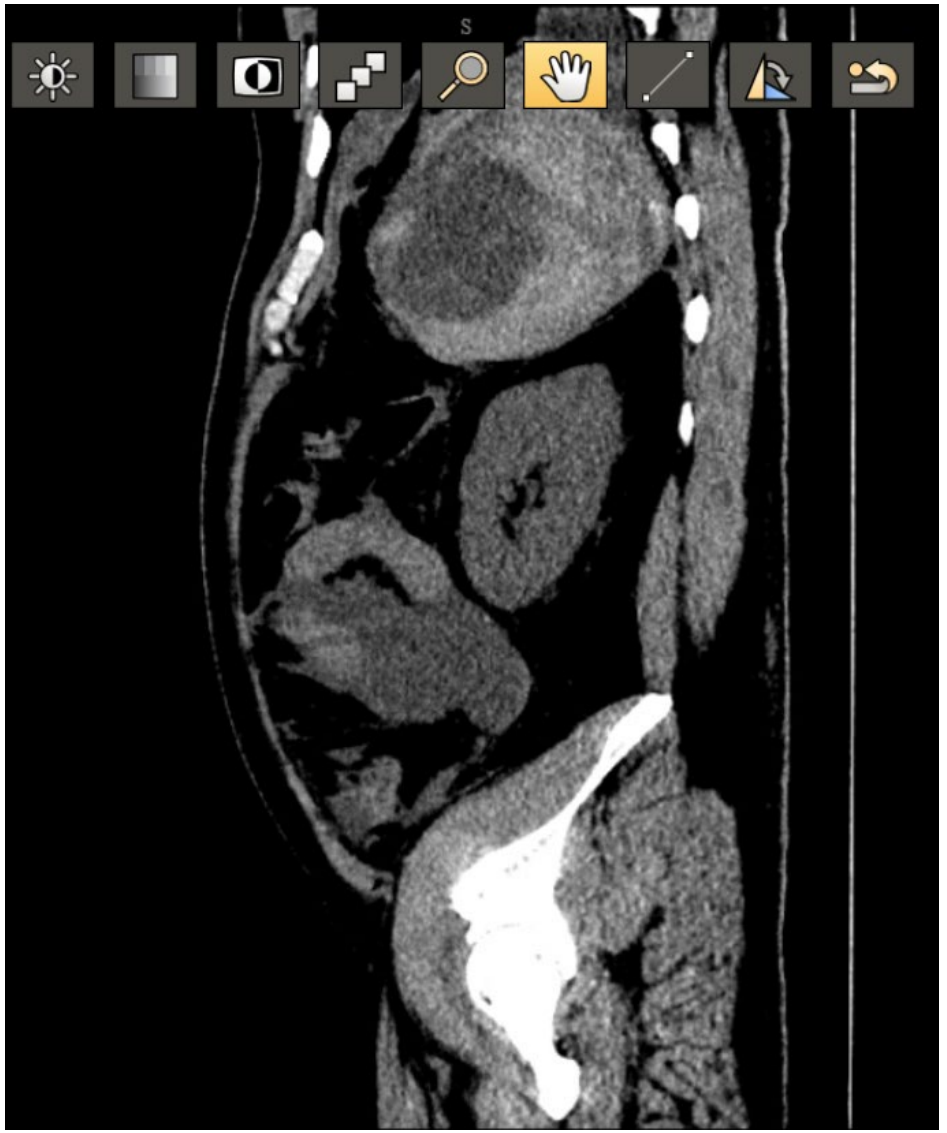
Acute blood loss anemia 2/2 splenic vs splenic artery aneurysm rupture D/C eliquis, Kcentra, 3U PRBC, IV fluids, CTX and flagyl, H/H Q6H, Hold all antihypertensives, **STAT SURGICAL CONSULT**

7-07-2022

10:41





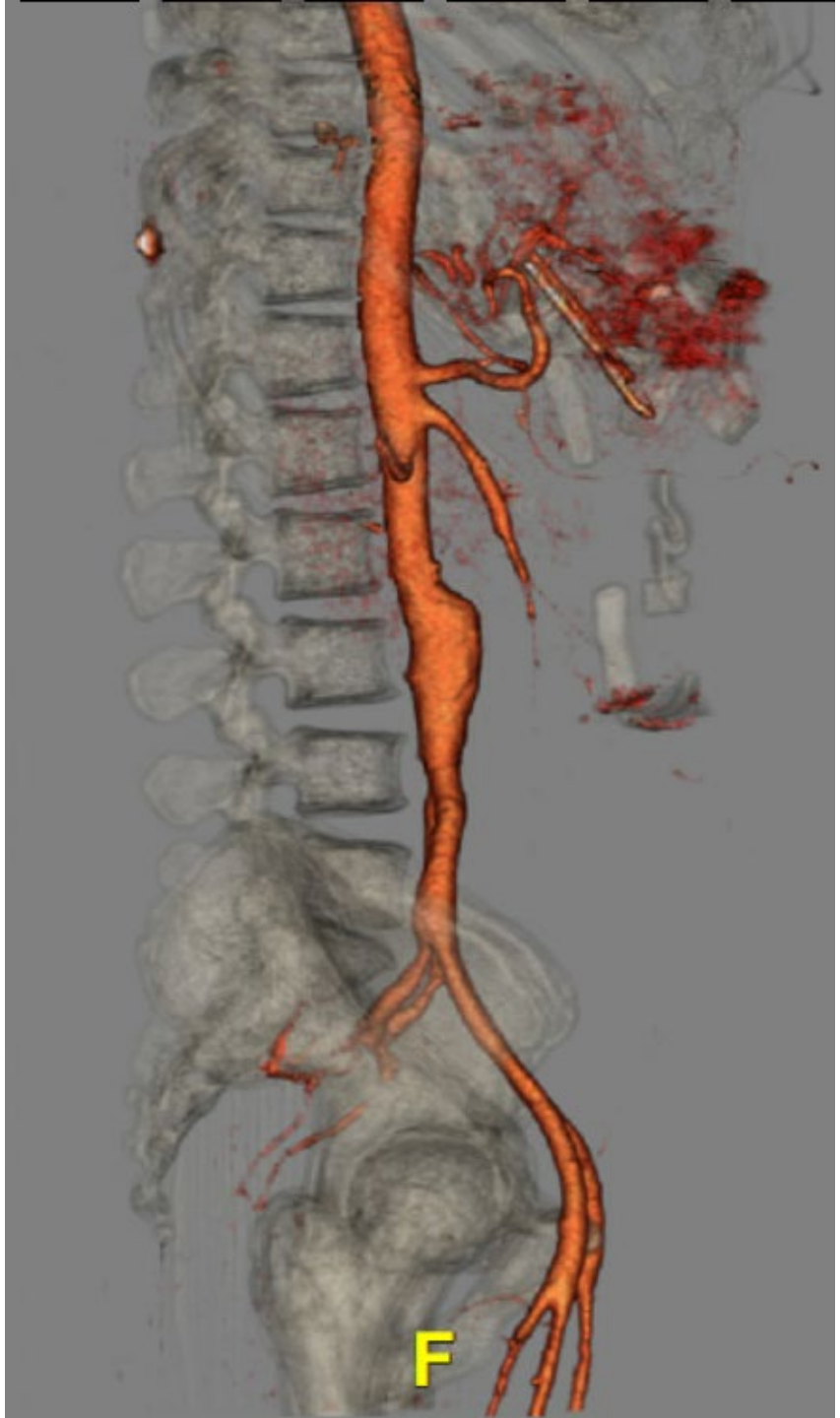


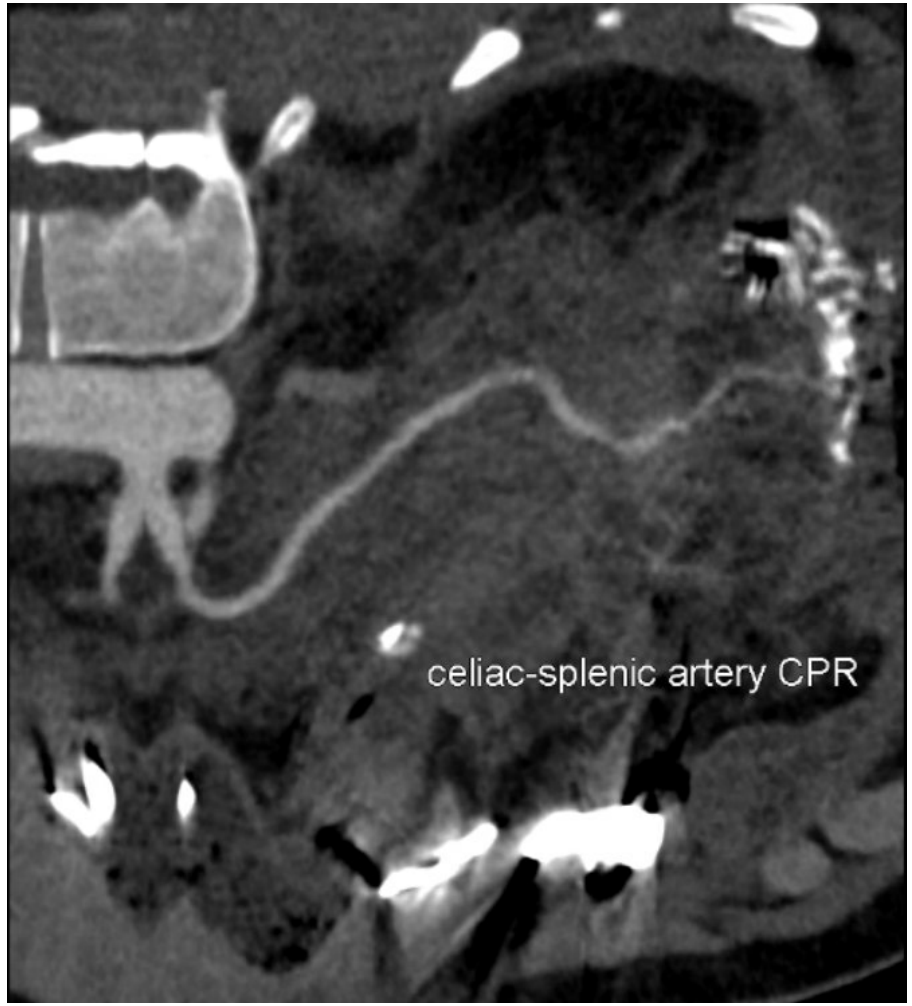
7-07-2022

10:30 patient taken for **CT**
11:21 Results communicated to primary team by phone
11:23 Trauma/Emergency Surgery Consult
11:45 Patient seen by Trauma/Acute care Surgery, diffuse abdominal tenderness with focal point tenderness in LUQ Patient planned for Emergency OR within next 2 hrs for ex lap and splenectomy. Discussion with IR regarding perisplenic hematoma with unclear splenic anatomy, possible rupture, no obvious pseudoaneurysm, no obvious source of bleeding, given the degree of hemoperitoneum, endovascular intervention thought to be of more limited utility
13:03 Anesthesia pre Op assessment
13:26 tried to reach spouse multiple times, left VM
13:53 updated wife regarding medical condition on phone
15:00 Central venous access, right IJ
15:51 Booked for OR and will be transported momentarily
18:27 Exploratory Laparotomy, splenectomy, -ve pressure wound therapy ABThera (NPWT DME > 50sq cm), estimated blood loss 2500 ml, 1600ml crystalloid, 3u whole blood , 2u PRBC ; no exploration of lesser sac intra op, unable to rule out proximal lesion of artery, concern for rebleeding from splenic hilum, possible splenic artery aneurysm or other vascular anomalies, CTA to evaluate feasibility of embolization, Discussed with IR at 23:07

7-07-2022

23:54





7-08-2022

ICU Night time events

102.6, Packed in ice, Tylenol, 500 ml wound vac, pink tissue protruding from wound vac, patient agitated, fentanyl 200mcg/hr, Versed drip, levo 18, 1unit RBC

23:36 Exploratory Lap, removal of Laps, Zfolds, washout, suture ligation of splenic artery, distal pancreatic necrosectomy, wound vac, packing with 5 laps, blood loss 300ml, 500ml 5% albumin, 1 Unit whole blood

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7-09-2022

8:32 Plans to return to OR for abdominal closure in 24-48 hrs, Hgb 7.2, levo 8, NPO, making urine, no acute CRRT, Leukocytosis but Abx d/ced, SQH

7-10-2022

12:20 No acute overnight event, low dose Levo, robust urine output, 2 blood transfusion, trickle tube feeds, plan for repeat OR on 7/11 (Hgb 6.7 & 6.4)

7-11-2022

Cr 4.3, low dose Levo, wound vac elevated but appropriate suction, bowel good peristalsis, hold trickle feed

Operative Note : removal of device, abdominal washout, 19F Blake drains in inferior sac and adjacent to stomach, 32F malecot drain at splenic hilum, wound vac in subcutaneous tissue, abdominal wall closure

7-12-2022

Cr improved, independence from vasopressor, agitated, precedex, (propofol cannot be used due to elevated TG), Levo restarted due to precedex, Hgb 9 (3 unit plasma, 7 unit PRBC, 4 unit whole blood in all)

Patient spouse raised concern for depression prior to admission, request for outpatient Psychiatry consult

Successful ventilator liberation to 6L NC

21:21 Failed bedside swallow, kept NPO

7-13-2022

7:38 Medically stable for transfer to PCU
09:21 Started on oral diet
Transferred to PCU on 7/14 at 00:25

7-15-2022

20:15 **Transferred to Floor**

7-20-2022

16:29 **Discharged** home with home health and wound vac management

What, if any, gaps occurred in care?

- Thorough assessment for level of care
- Assessing deterioration index
- Appreciate that acute pancreatitis can escalate
- Scoring to assess severity of acute pancreatitis
- Addressing gaps in primary care, including med compliance, insurance, GI nutrition consult, endocrinology referral
- Long term care of these individuals

Acute Pancreatitis (2/3)

Abdominal pain (acute onset, persistent, severe, epigastric pain often radiating to the back)

 Lipase>3 ULN

CECT demonstration of characteristic findings

Temporal classification of pancreatitis

Acute pancreatitis
Gallstone
Alcohol (>5 years, > 50-100gms/day)
HTG (11.3 mn)
Post ERCP
Hypercalcemia
Pancreas divisum
Tumor
Genetic polymorphism
Drugs
Infection
Idiopathic

Recurrent acute pancreatitis

36%

Alcohol, smoking, male

Chronic pancreatitis

5%

Pancreatic Ca

MILD	MODERATE	SEVERE
No OF	Transient OF	Persistent OF

Why Prognosticate acute pancreatitis ?

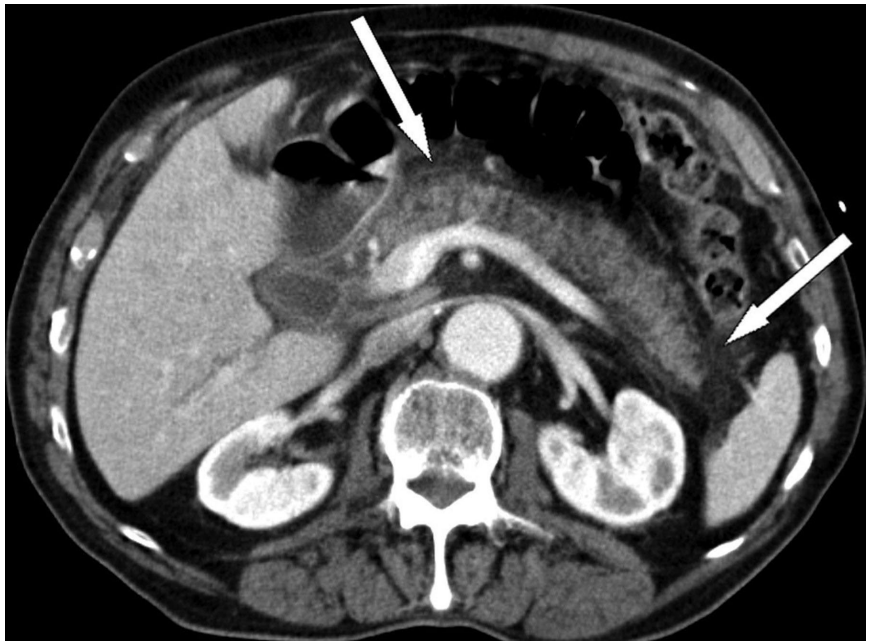
- Commonest Gastrointestinal Cause of Hospitalization
- Arrival to ER but without organ failure
- OF develops in next 1-3 days
- Clinical appraisal scoring needed to screen out population at risk

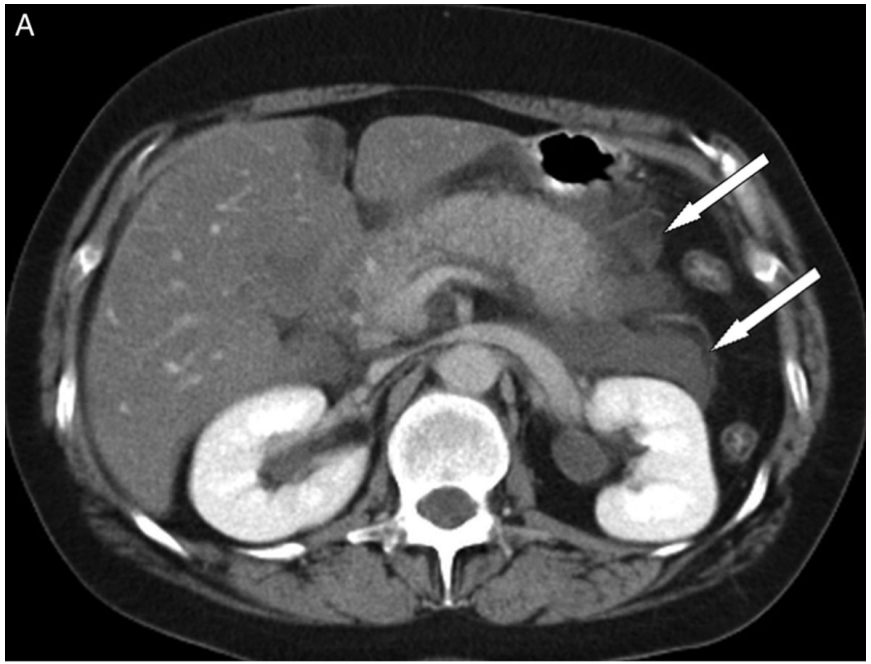
- APACHE II Score
- BISAP Score
- Balthazar Computed Tomography Severity Index
- Ranson Score

Bedside Index of Severity in Acute Pancreatitis (BISAP)

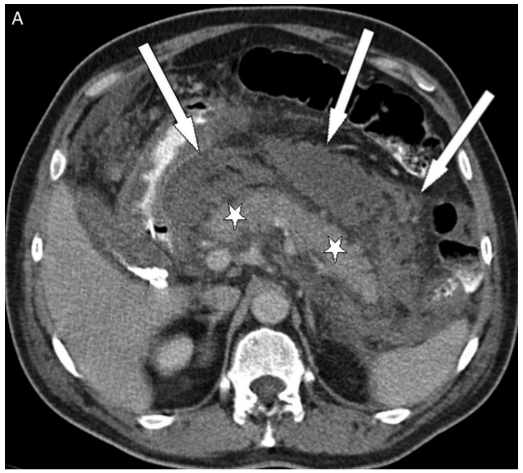
- Blood urea nitrogen > 25 mg/dl
- Abnormal mental status (Glasgow coma score <15)
- Evidence of systemic inflammatory response syndrome
- Greater than or equal to 60 years old
- Pleural effusion
- To calculate the BISAP, sum the number of positive variables (0–5)

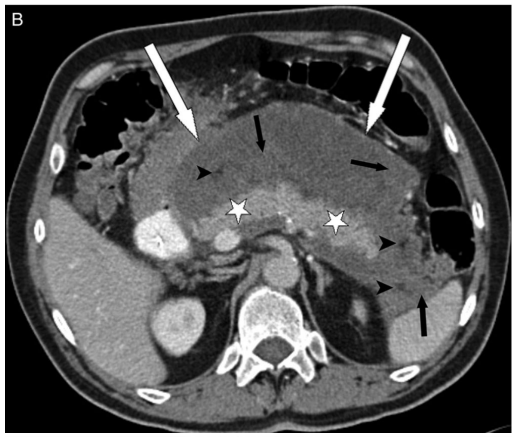
Interstitial edematous pancreatitis

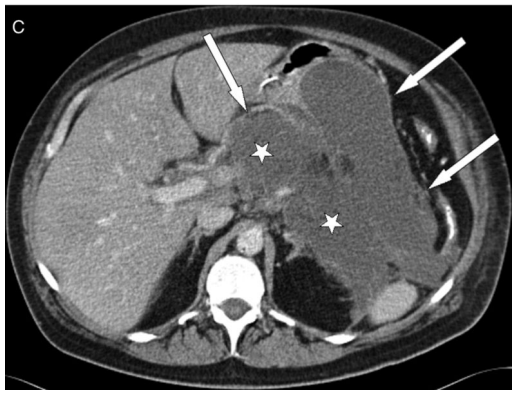




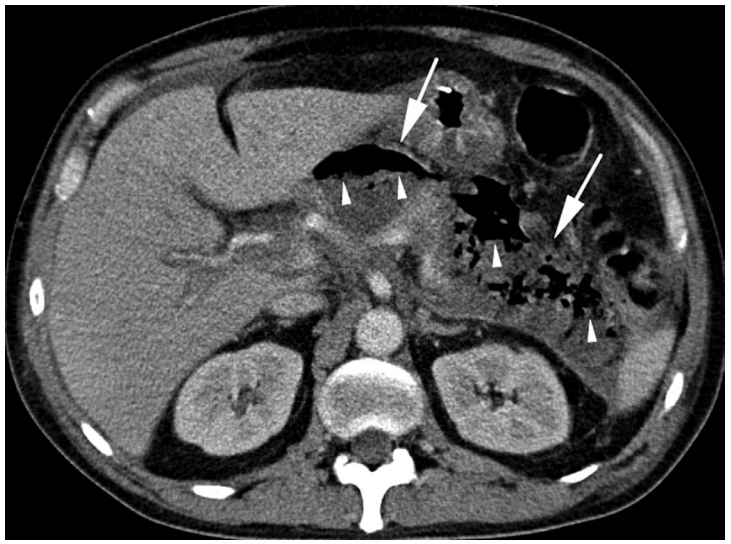
Necrotising pancreatitis







Infected pancreatic necrosis

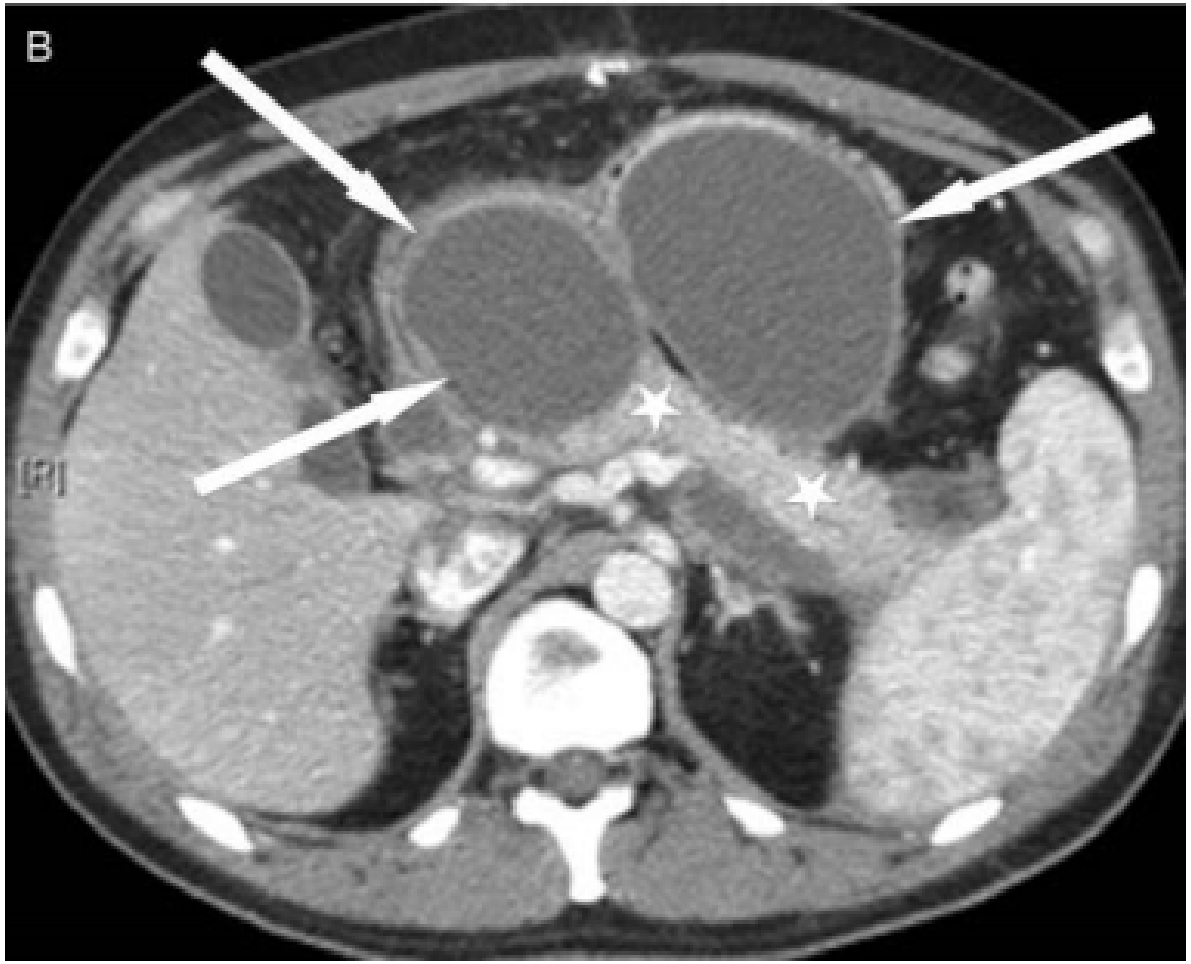


Atlanta Classification

Banks, Sarr et.al Gut 2013

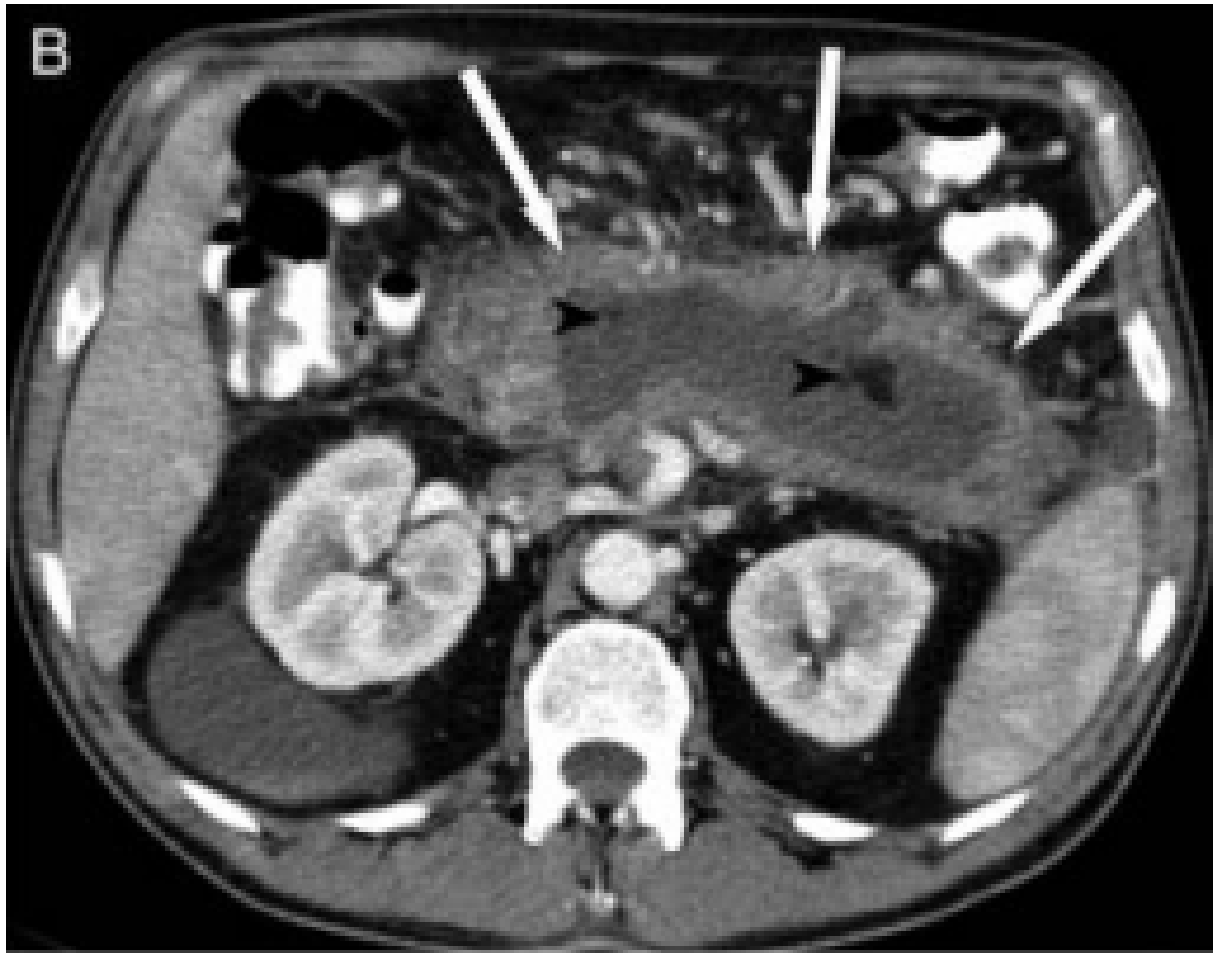
Sequelae

Pseudocyst



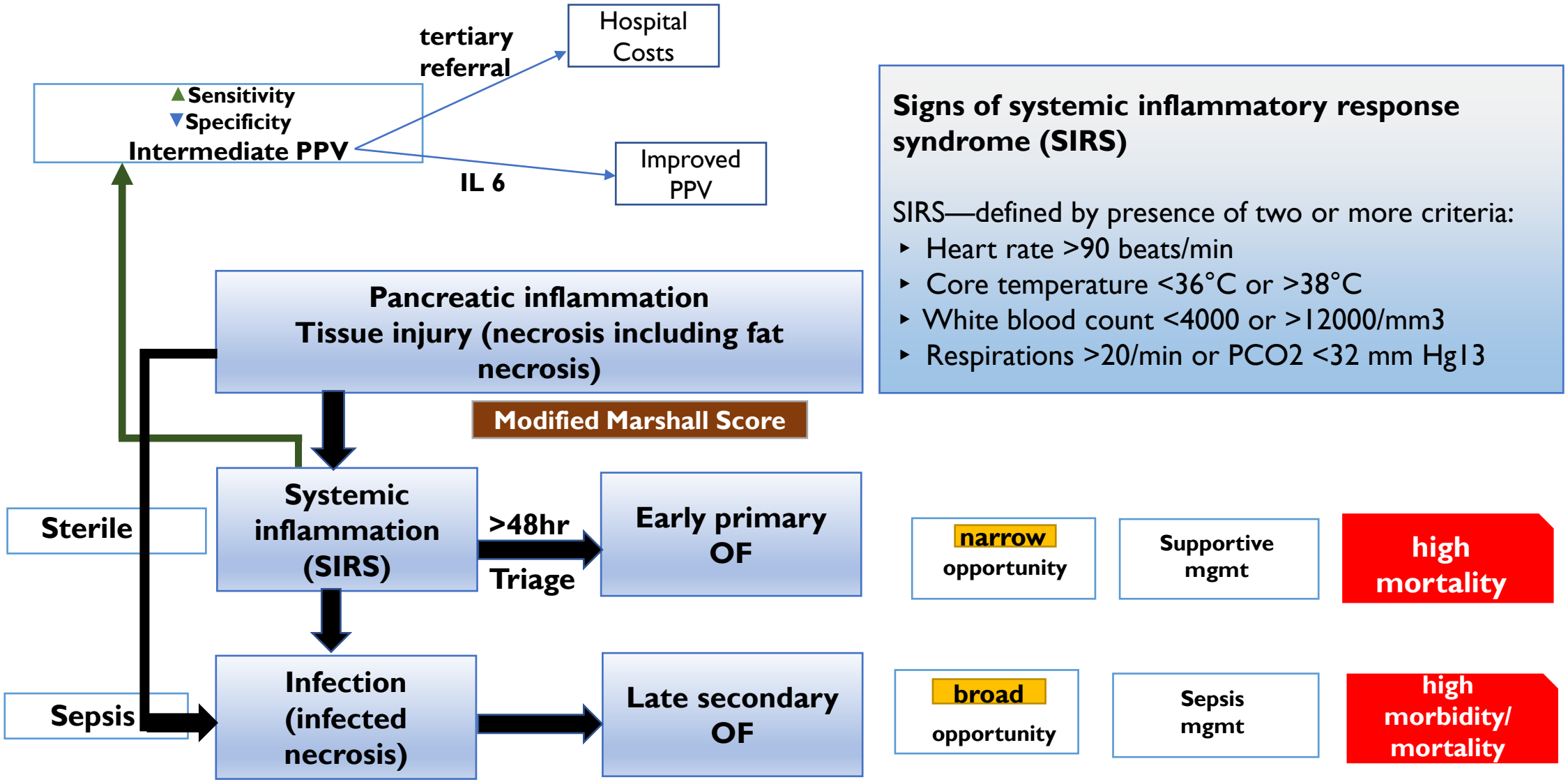
> 4 weeks

Walled off necrosis (WON/necroma)



several weeks

Banks, Sarr et.al Gut 2013



Signs of systemic inflammatory response syndrome (SIRS)

SIRS—defined by presence of two or more criteria:

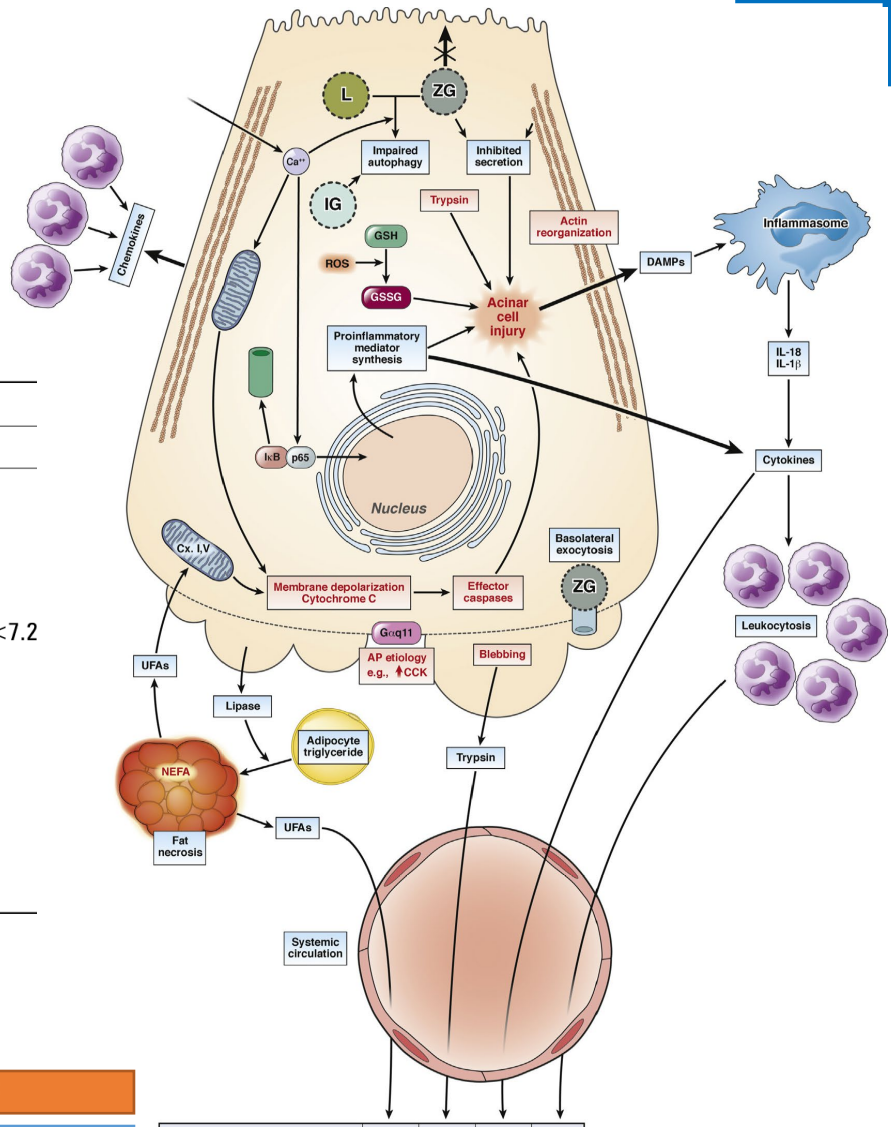
- Heart rate >90 beats/min
- Core temperature <36°C or >38°C
- White blood count <4000 or >12000/mm3
- Respirations >20/min or PCO2 <32 mm Hg13

Marshall Scoring

Table 1 Modified Marshall scoring system for organ dysfunction

	Score				
Organ system	**0**	**1**	**2**	**3**	**4**
Respiratory (PaO_2/FiO_2)	>400	301–400	201–300	101–200	≤101
Renal*					
(serum creatinine, μmol/l)	≤134	134–169	170–310	311–439	>439
(serum creatinine, mg/dl)	<1.4	1.4–1.8	1.9–3.6	3.6–4.9	>4.9
Cardiovascular (systolic blood pressure, mm Hg)†	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH<7.3	<90, pH<7.2
For non-ventilated patients, the FiO_2 can be estimated from below:					
Supplemental oxygen (l/min)	**FiO_2 (%)**				
Room air	21				
2	25				
4	30				
6–8	40				
9–10	50				

A score of 2 or more in any system defines the presence of organ failure.
*A score for patients with pre-existing chronic renal failure depends on the extent of further deterioration of baseline renal function. No formal correction exists for a baseline serum creatinine ≥134 μmol/l or ≥1.4 mg/dl.
†Off inotropic support.



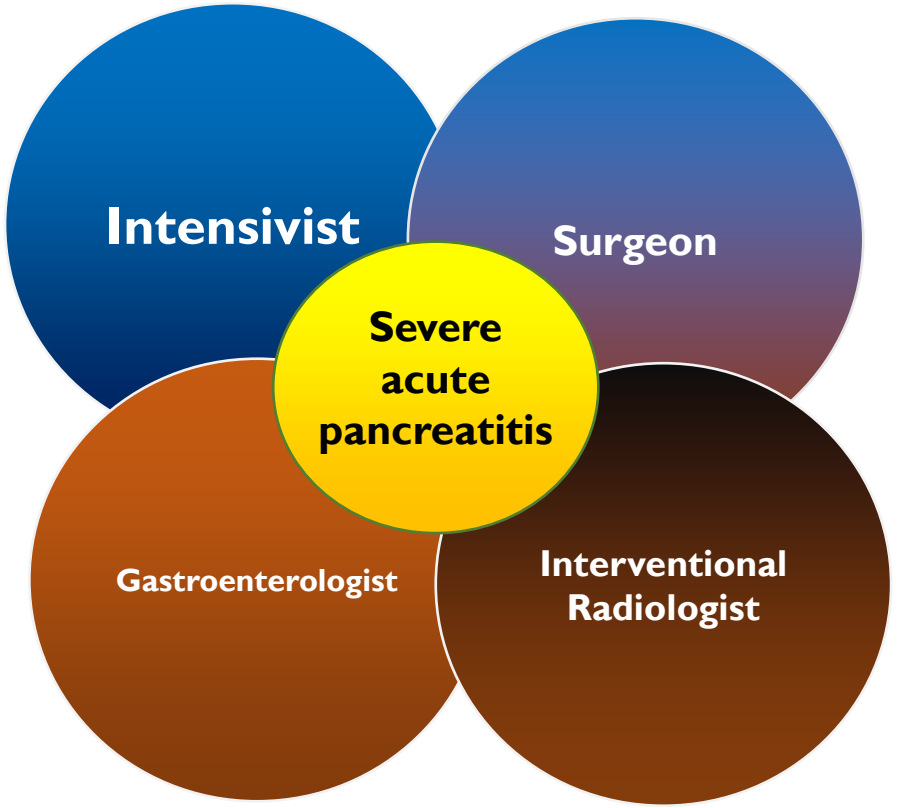
Management
Mechanical ventilation
Fluids, RL vs NSS, Aggressive vs adequate, Vasopressor(s)
HD/CRRT

OF
Respiratory ARDS
Cardiovascular
ARF

Lung MPO increase	+	+	+	+
Lung TUNEL increase	+	±	-	-
Microvascular leak	+	+	±	-
Hypotension	+	+	±	-
Renal tubular injury	+	-	-	-
BUN increase	+	-	±	-

Garg and Singh, Gastroenterology, 2020

Multidisciplinary management of the critical illnesses



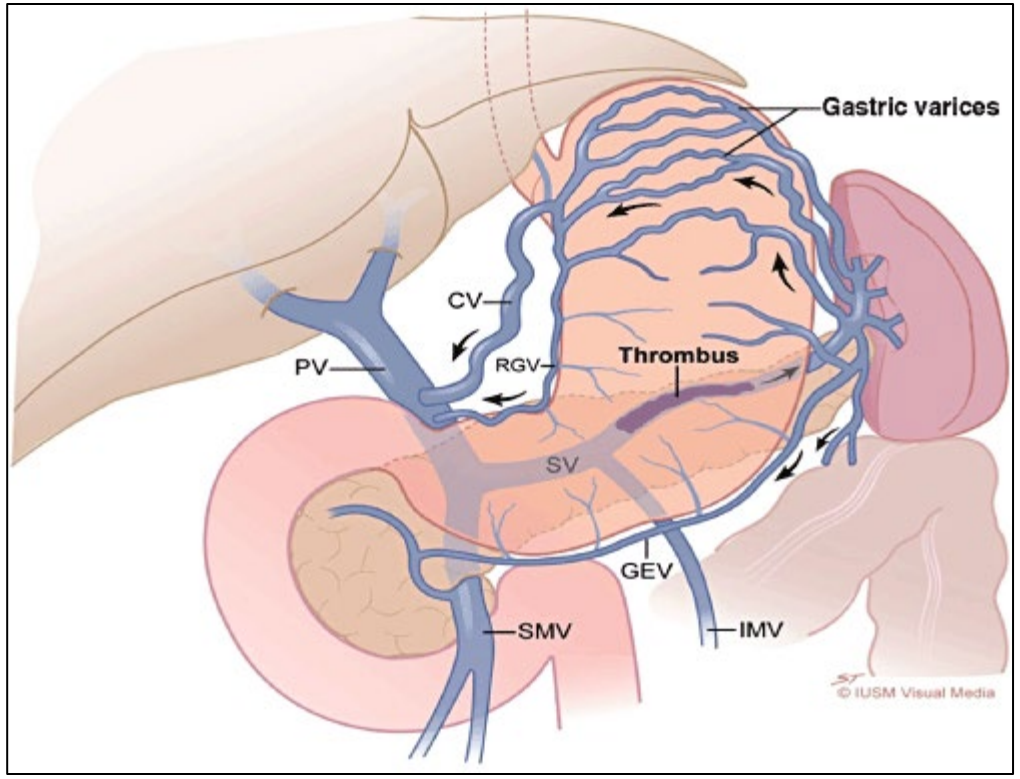
ICU

Complete clinical stability

Deescalate levels of care to PCU/ Med-Surg

Complications of acute pancreatitis

PISVT



Sinistral/left sided portal hypertension

Abdominal Compartment Syndrome

Diabetes

Chronic Pain

Butler et.al, HPB 2011

Reginald Huber Fitz

John H.C. Ranson

VOL. CXX., No. 8.] BOSTON MEDICAL A.

Original Articles.

ACUTE PANCREATITIS.

A CONSIDERATION OF PANCREATIC HEMORRHAGE, HEMORRHAGIC, SUPPURATIVE, AND GANGRENOUS PANCREATITIS, AND OF DISSEMINATED FAT-NECROSIS.[1]

BY REGINALD H. FITZ, M.D.,

Shattuck Professor of Pathological Anatomy in Harvard University and Physician to the Massachusetts General Hospital.

UNTIL the time of Clässen[2] the evidence of an

FIG. 2. Title page of Fitz's initial paper on pancreatitis, published on February 21, 1889 in the *Boston Medical and Surgical Journal.*

docyst. Fitz further suggested a causal relationship between pancreatitis and disseminated fat necrosis, and provided the first clear link between pancreatic hemorrhage and pancreatitis. In light of these progressive observations, Fitz's extraordinary claim that 'the dark-brown pancreas may lie nearly free in the omental cavity [and] . . . may be discharged as a slough from the bowels' may be easily disregarded.

On the basis of his exhaustive review, Fitz concluded 'Acute inflammation of the pancreas is both a well characterized disease, and one which is much more frequent than is generally thought. It is an important cause of peritonitis, and one readily overlooked.' In contrast to his recommendations of prompt laparotomy in acute appendicitis, Fitz observes that 'an operation . . . in the early stages of this disease, is extremely hazardous.' It is interesting to note that Fitz subsequently waivered from this conservative stance. Thus in 1903 he proposed that 'In cases of acute pancreatitis . . . laparotomy in an increasing number of cases has proven the most satisfactory method of treatment, and, like most abdominal operations for the relief of acute symptoms, is the more helpful the earlier in the course of the disease it is performed.'[14]

The impact of Fitz's 1889 treatise was clear. For the first time the varied manifestations of acute pancreatitis were organized into a systematic framework that allowed for its eventual antemortem diagnosis. In 1913 the surgeon W. W. Keen from Philadelphia wrote, 'In another fruitful field, pancreatitis, [Fitz] laid the foundations of our present knowledge of the pathology and symptomatology and therefore, of the correct treatment of this so frequently and suddenly fatal disease.'[15] Fitz's work contributed greatly to the progress of other turn-of-the-century investigators, including Opie in the field of gallstone pancreatitis,[16] and Mayo-Robson in the arena of surgical therapy.[17] Thus Fitz's contributions to the subject of pancreatitis were recognized for their breadth and vision even before his death in 1913. Given his primary role in the description of pancreatitis, it is ironic that Fitz's death was related to another autodigestive phenomenon: he died after hemorrhage from a gastric ulcer.[3]

In the immediate years following Fitz's death, several other physicians provided significant contributions regarding the pathophysiology and diagnosis of acute pancreatitis. Of particular significance were Moynihan's further delineation of the surgical approach to pancreatitis in 1925,[18] and the demonstration of elevated serum amylase levels by Elman, Arneson, and Graham in 1929.[19]

In considering the current status of acute pancreatitis, it is particularly distressing that Fitz's comments, now 100 years old, continue to approximate our current understanding of this disease. Fitz's classifications of hem-