CME : ACUTE PANCREATITIS

CASE BASED DISCUSSION

CASE SUMMARY

- initially presented in early November 2025
- Had 1 day history of epigastric pain, fever, vomiting and tea coloured urine
- Amylase was >1500, transaminitis with TB 70
- Other blood parameters was normal
- Admitted to surgical ward for mild acute gallstone pancreatitis 2’ to choledocholithiasis cover for cholangitis

- USG HBS on 4/11/25 noted CBD dilated measuring 1.2cm with multiple calculi seen within (largest measuring 1.1cm)
- CT HBS 5/11/25
  - Gallbladder is partially distended, no hyperdense calculus within.
  - Mild thickening of the gallbladder wall (3mm) with enhancing GB wall, cystic duct as well as pericholecystic fat stranding noted
  - Dilated left and right hepatic ducts(2mm and 6mm respectively), common hepatic (8mm) and proximal common bile duct(10mm), with gradual tapering distally toward the ampulla. No hyperdense calculus seen within the biliary tree.
  - Bulky pancreatic head with mild peripancreatic fat stranding as well as numerous subcentimeter peripancreatic nodes. No pancreatic duct dilatation. No focal pancreatic lesion.

- CT reviewed - no obvious stones features of cholecystitiis concentrated GB and CBD was prominent
- Lft normalised and abdominal pain resolved
- Treated as passed out stone
- In ward, patient was clinically getting well
- Abdominal pain resolved
- completed iv augmentin 1.2g tds for 4 days in ward
- patient was allowed to discharge on 7/11/25

- Patient was reviewed in sopd
- patient still has on and off pain but no fever no jaundice
- recently came to ed for biliary colic - post discharge

- treated with analgesics

- CT scan reviewed - noted gallbladder contracted -> cant go in for surgery now as high risk for CBD injury
- Planned for USG reassessment

- Presented again with abdominal pain and vomiting
- Treated for recurrent pancreatitis

- Done MRCP
  - The right IHD and left IHD measuring 4mm and 6mm respectively. The common bile duct(CBD) measuring 1.1cm in maximal diameter. There is filling defect seen at the mid CBD level measuring 3mm. There is abrupt tapering at the distal CBD(near the ampulla of vater), measuring 4mm in length.
  - Gallbladder is well distended. No filling defect seen within. No GB wall thickening. Pericholecystic fluid seen.
  - Pancreatic duct is not dilated. Pancreas appear bulky, with the widest diameter at the body and tail measuring 2. 1cm and 20cm respectively. 2W hyperintense fluid seen at the peripancreatic region.
  - Ascites seen at the upper abdomen, as well as at the bilateral subcutaneous abdominal wall.
  - Bilateral pleural effusion with adjacent collapsed consolidation at bilateral lower lobes.

- Patient was then transferred to ICU due to persistent metabolic acidosis and tachycardia
- Emergency ERCP done – stented x2
- Subsequently 2 days later in ward patient was complaining of persistent abdominal pain and abdominal distension
- Done CT AP
  - Biliary stent seen with its proximal tip at the porta hepatis region and distal tip at the D2 of the duodenum. Aerobilia noted, predominantly at the left IHD and pericatheter region.
  - Resolved bilateral intrahepatic duct dilatation.
  - Gallbladder is partially distended with non-dependent air locule seen. The GB wall is not thickened, however it appears enhancing. No pericholecystic fluid. No hyperdense calculus within.
  - No extraluminal air locules predominantly at the duodenum. No oral contrast extraluminal leaked. No pneumo intra/retroperitoneal seen.
  - Abdominal fluid seen at the lesser sac, perisplenic, perihepatic, interloop, bilateral paracolic gutter and pelvic region. Some of these fluid shows faint rim-enhancing wall, largest pool at the right abdominal region measuring 7.5 x 17.9 x 18.4cm (AP x W x CC).
  - Bulky homogenous pancreas with mild peripancreatic fluid and fat stranding seen. No pancreatic duct dilatation. No focal pancreatic lesion. Splenic vein is patent.
  - Bilateral mild pleural effusion with adjacent collapse consolidation.

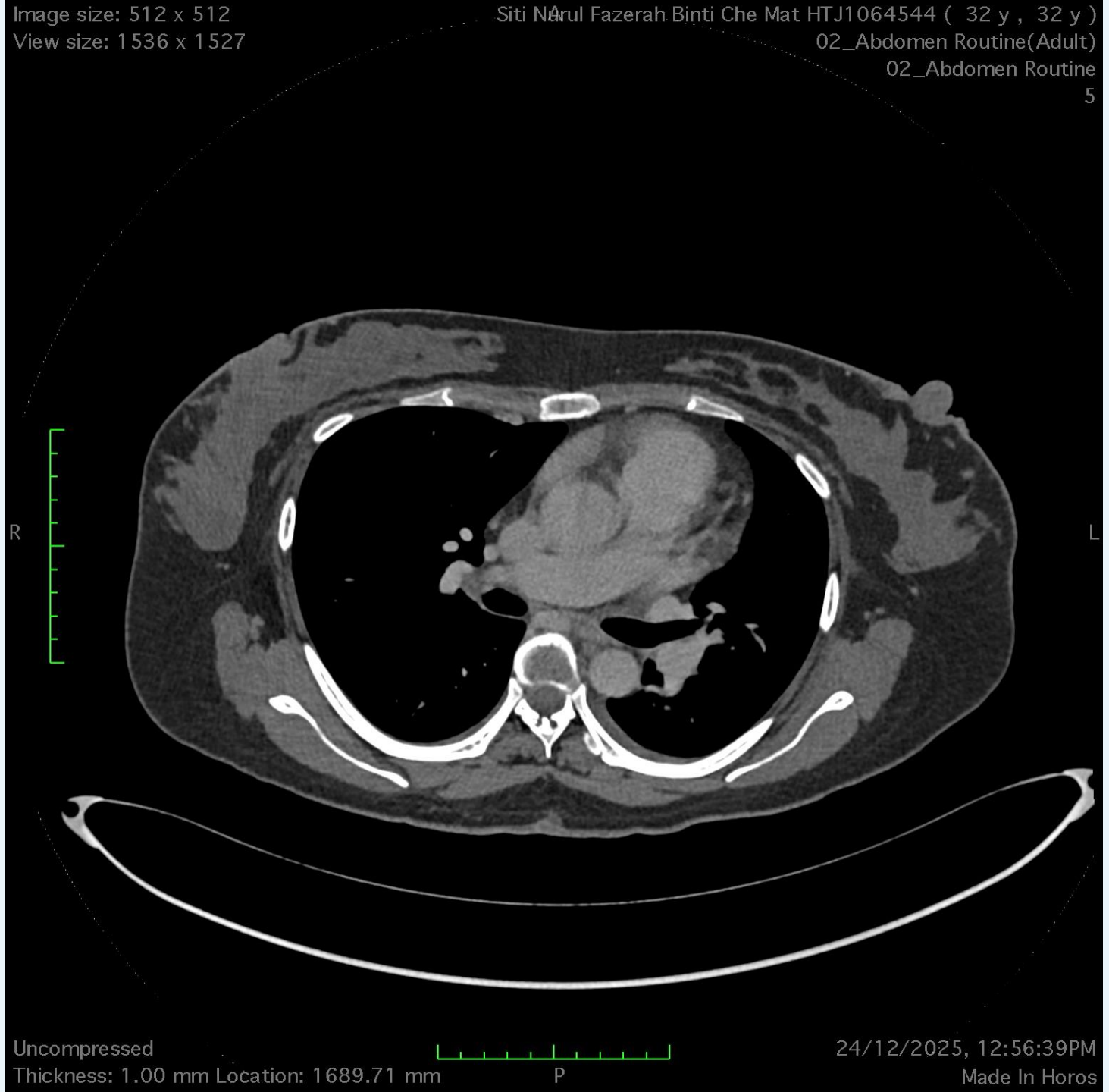


Image size: 512 x 512
View size: 1536 x 1527
Siti Nurul Fazerah Binti Che Mat HTJ1064544 (32 y , 32 y)
02_Abdomen Routine(Adult)
02_Abdomen Routine
5
R
L
Uncompressed
Thickness: 1.00 mm Location: 1689.71 mm
P
24/12/2025, 12:56:39PM
Made In Horos

- done USG Guided Percutaneous Drainage of Intraabdominal Collection on 31/12/2025
- Patient was subsequently discharged with abx

- Patient readmitted with 3 days hx of pain over site of percutaneous drainage over right abdomen
- Treated for Septic shock secondary to severe pancreatitis with lung involvement

-Initially noted in ward having temperature spike and tachycardia
-started on IV Tazosin , then escalated to IV Meropenem
-resuscitated in ward

USG HBS on 7/1/26

- No focal liver lesion.Evidence of biliary stent in situ.
- Intrahepatic ducts are not dilated. Proximal common bile duct is dilated measuring 1.1cm.Distally its obscure by bowel gas. No intraductal stone or lesion seen.
- Gallbladder is well distended. No calculus, gallbladder thickening or pericholecystic fluid seen.
- Visualised head and part of body of pancreas is appear bulky and heterogenous, measuring about 3.2cm. The rest of the pancreas is obscured by bowel gas.
- No peripancreatic free fluid or collection noted.

CT HBS on 9/1/26

- Evidence of double biliary stent in situ.
- Evidence of pigtail drainage catheter at right lumbar region.
- Pancreas is homogenously enhancing, however appearsbulky, with the measurement of head 3.6cm, body 2.0 cm, tail 1.4cm.
- Minimal peripancreatic fluid and fat stranding is noted. No focal pancreatic lesion. Pancreatic duct is not dilated.
- CBD is still dilated measures up to 1.0cm, no hyperdense calculus within.
- Previously seen rim-enhancing collection at the right abdominal region slightly smaller, now measuring approximately 5.2cm x 10.2cm x 16.0cm (previously 7.5 x 17.9 x 18.4cm (AP x W x CC).
- Resolved pelvic free fluid, lesser free fluid in the previously seen location.
- Improving aerobilia and pleural effusion.

Referred ID initially for bile C+S : Candida tropicalis on 12/1/26
-Started on IV Fluconazole and IV and IV Augmentin

Subsequently prior to discharge refer ID back for duration and to oralise antibiotics
-Changed to oral fluconazole 400mg OD
-PO Augmentin 625mg TDS
- To continue both abx till USG reassessment
- TCA ID clinic 3/2/26 at 2pm with blood taking 28/1/26 FBC RP LFT AST CRP

ACUTE PANCREATITIS

Acute pancreatitis is an inflammatory condition of the pancreas most commonly caused by bile stones or excessive use of alcohol

The severe form comprising about 20–30% of the patients is a life-threatening disease with hospital mortality rates of about 15%

Diagnostic Criteria

The Revised Atlanta Classification states **2 or more** of the following 3 criteria should be present in order to diagnose acute pancreatitis[1]:

1) Abdominal **pain** suggestive of pancreatitis,
2) Serum **amylase** or lipase level - ≥ x3 upper normal value, or
3) Characteristic **imaging** findings.

Causes of Pancreatitis

knowmedge

Mnemonic: "I GET SMASHED"

I	IDIOPATHIC	
G	GALLSTONES	2nd most common cause in the US
E	ETHANOL	Most common cause in the US
T	TRAUMA	
S	STEROIDS	
M	MUMPS / MALIGNANCY	
A	AUTOIMMUNE	May have IgG4 antibody present
S	SCORPION STING	
H	HYPERTRIGLYCERIDES OR HYPERCALCEMIA	Usually TG >1000
E	ERCP	
D	DRUGS (e.g. HCTZ, Didanosine, Pentamidine, Bactrim, Azathioprine)	

Disease *Types*

- The Atlanta Classification subdivides acute pancreatitis into 2 broad forms:

INTERSTITIAL
(o)edematous
pancreatitis (*IEP*)

- Swollen, oedematous pancreas
- Still **enhances uniformly**.

NECROTISING
pancreatitis (*NP*)

- Areas of **non-enhancement** or **hypoenhancement** in pancreas.

Subtypes *depending on site of necrosis:*

- ***Parenchymal*** necrosis
- ***Peripancreatic*** necrosis
- ***Combination*** of the above – ***most common***

Disease *Phases*

EARLY

< 1 week

- Imaging appearances may underestimate the disease severity – especially if imaging is obtained in the first 72 hours. Features such as necrosis usually take a few days to become established.
- Poor correlation between imaging findings and clinical severity in this phase – severity should be determined by clinical parameters here (e.g. signs of organ failure).

LATE

> 1 week

(may last weeks to months)

- Generally only occurs in severe cases, mild cases often do not progress to late stage.

Disease *Severity*

MILD	MODERATELY SEVERE ***	SEVERE
✗ **NO** ORGAN FAILURE	✓ **TRANSIENT** ORGAN FAILURE • < 48 hours	✓ ORGAN FAILURE • > 48 hours
✗ **NO** local complications	+/- local or systemic complications	✓ Local or systemic complications
LOW mortality	**LOW** mortality	**HIGH** risk of mortality • up to 30% • ICU support
	*** An addition to the 1992 classification – this was a subset recognised as having complications leading to significant ***morbidity***, but little ***mortality***.	

- '***Local* complications**' = e.g. pancreatic and peripancreatic collections.
- '***Systemic* complications'** = often exacerbation of pre-existing comorbidities secondary to acute pancreatitis – e.g. AKI on CKD.
- '**Organ Failure**' = defined in line with standardised scoring systems, e.g. **MODIFIED MARSHALL Scoring System** – **score of 2 or more**, in at least 1 of 3 organ systems (respiratory, renal, cardiovascular).

Criteria for organ failure based on Marshall scoring system:

Organ system	Score				
	0	1	2	3	4
Respiratory (PaO_2/FiO_2)	>400	301–400	201–300	101–200	<100
Renal (Serum Creatinine md/dl)	≤1.5	>1.5 – ≤ 1.9	>1.9 – ≤ 3.5	>3.5 – ≤ 5.0	> 5.0
Cardiovascular (systolic blood pressure, mm Hg)	>90	<90, fluid responsive	<90, fluid unresponsive	<90, pH<7.3	<90, pH<7.2

Types of Fluid Collection

- 4 distinct types of collection are set out, depending on:
  - **INTERSTITIAL** vs **NECROTISING** disease
  - **TIME** - **≤ 4 weeks** vs **> 4 weeks**

INFECTED COLLECTIONS

- Any collection of the 4 can be sterile or infected.
- Only real imaging feature of infection is presence of **GAS**.**
- …Otherwise clinical features/deterioration.
- Wall enhancement is **NOT** a reliable feature invariably seen in the non-infected collections present at >4 weeks (both pseudocyst and WON, in IEP and NP respectively).

***NB**. If there has been intervention – e.g. percutaneous drainage, then gas may well be present, even if not infected.*

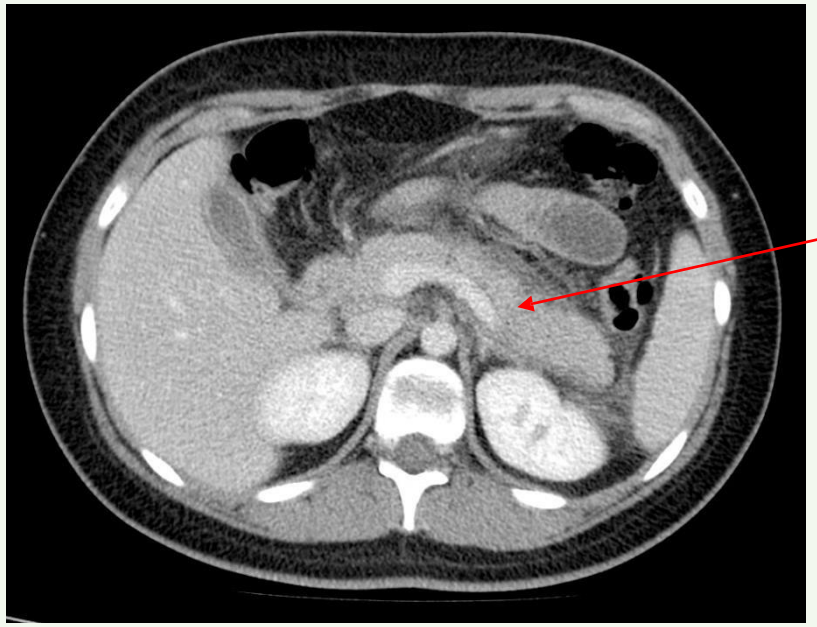
Collections – in INTERSTITIAL Pancreatitis

- **BEFORE 4 weeks** = Acute PERIPANCREATIC Fluid Collections (APFC)
  - Non-encapsulated fluid collections = **NO enhancing capsule**
  - Fluid is **homogenous**

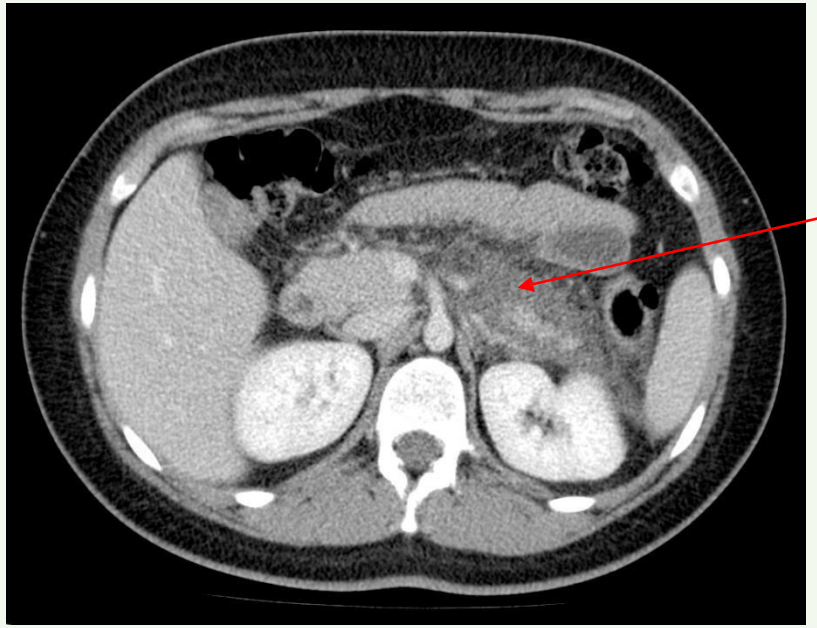
- **AFTER 4 weeks** = PSEUDOCYSTS
  - **Encapsulated** collection
  - Homogenous fluid content only
  - Can be peripancreatic or remote

*** NO NECROSIS! ***

Pancreatic parenchyma may be oedematous but should be uniformly enhancing.



Swollen, oedematous pancreas – but still uniformly enhancing.



Scrolling down, the surrounding fluid is of homogenous density and has no capsule.

≤ 4 WEEKS

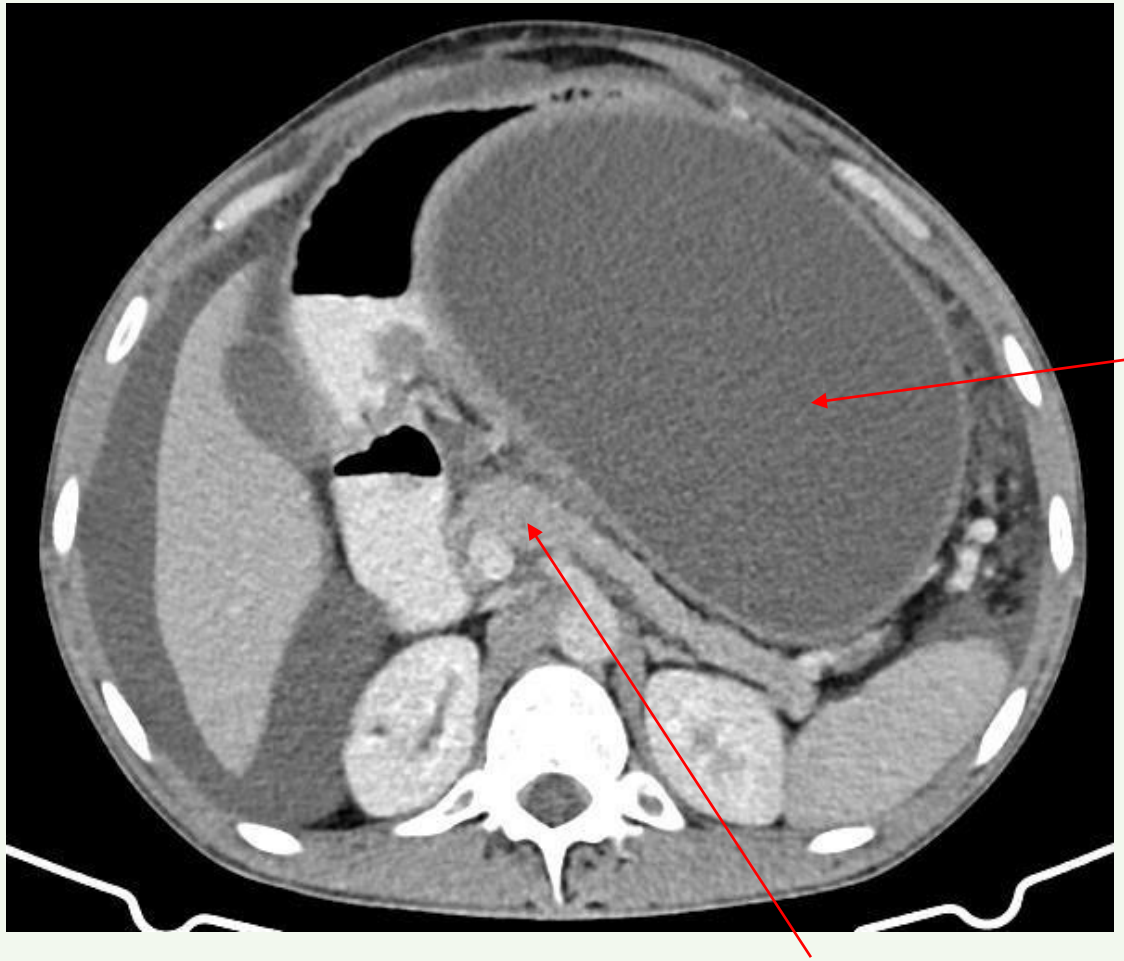
= **INTERSTITIAL**, no necrosis

+

NON-ENCAPSULATED, HOMOGENOUS

↓

ACUTE PERIPANCREATIC COLLECTION



Massive collection, enhancing wall, homogenous fluid content.

No evidence of necrosis on current or previous imaging – normal parenchymal enhancement.

> 4 WEEKS

= **INTERSTITIAL**, no necrosis

+

ENHANCING CAPSULE, HOMOGENOUS CONTENT

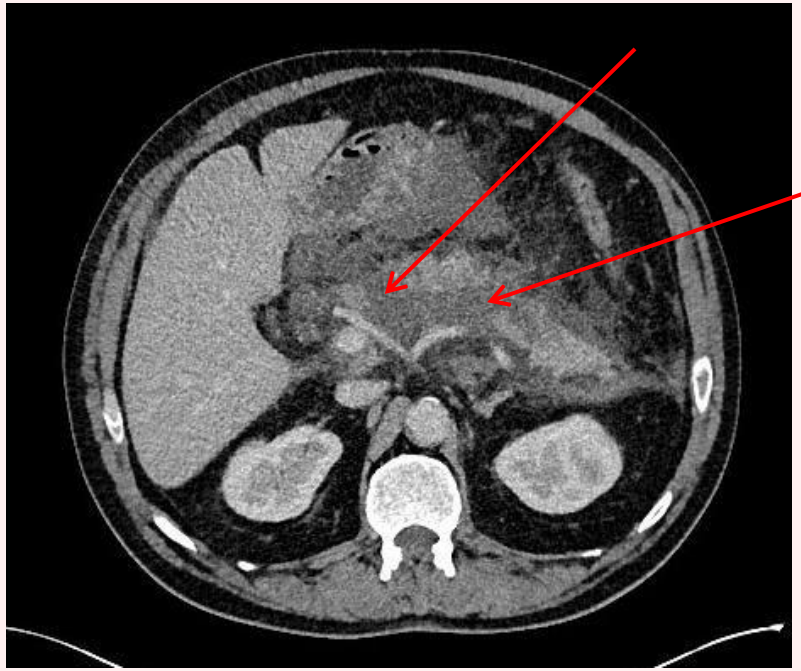
↓

PSEUDOCYST

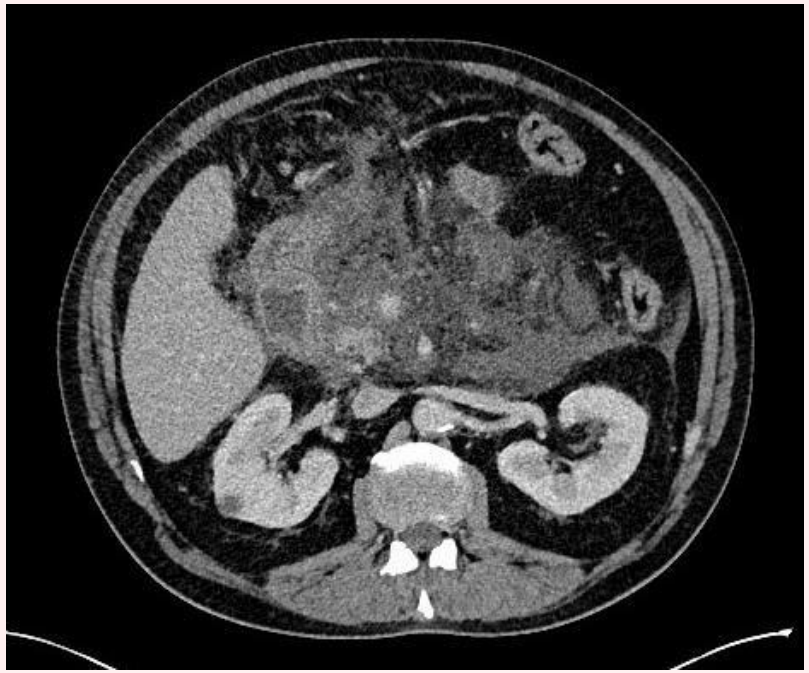
Collections – in NECROTISING Pancreatitis

- **BEFORE 4 weeks** = Acute NECROTIC Collections (ANCs)
  - **Non**-encapsulated fluid collections = **NO enhancing capsule**
  - **Heterogenous**, **non-liquefied** material

- **AFTER 4 weeks** = WALLED-OFF NECROSIS (WON)
  - **Encapsulated** collection
  - **Heterogenous -** solid necrotic debris within



Hypoenhancing areas = **NECROSIS** (*Arrows*)



Scrolling down - **heterogenous** material surrounding – with **no capsule**.

≤ 4 WEEKS

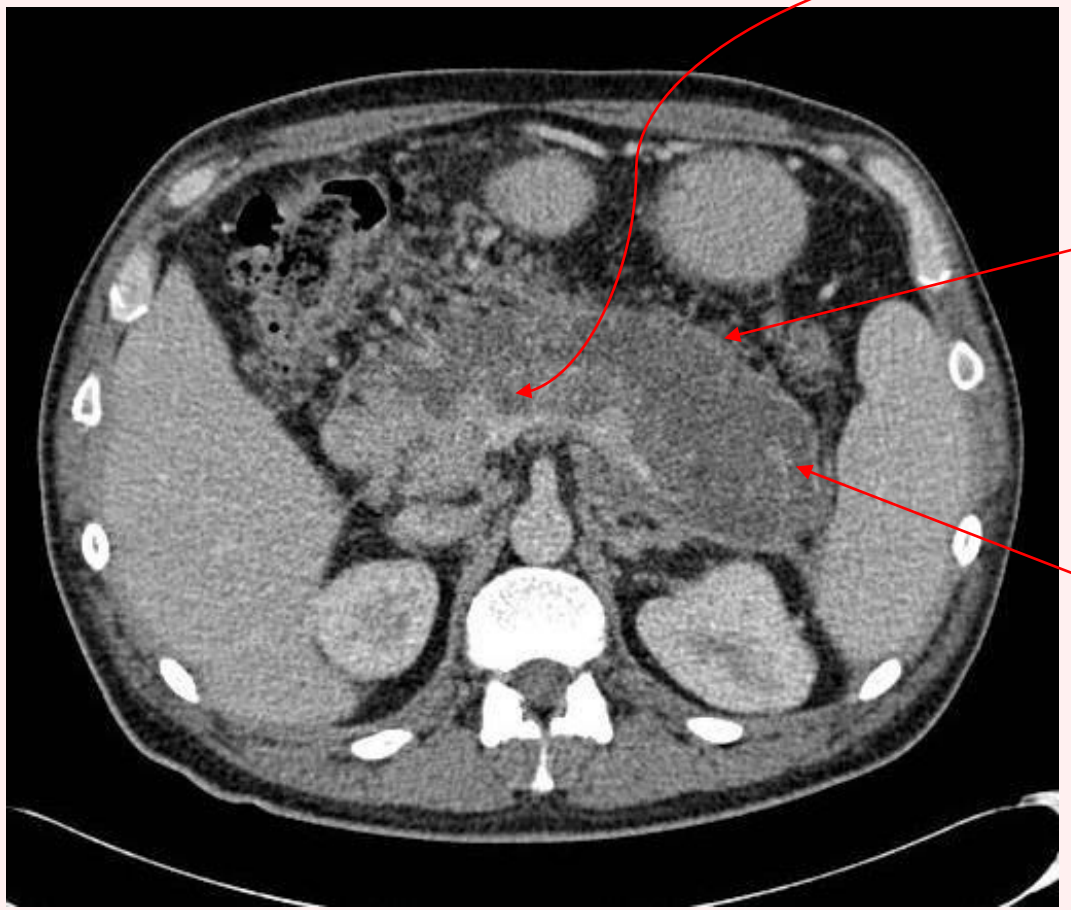
= NECROSIS

+

NON-ENCAPSULATED, HETEROGENOUS

↓

ACUTE NECROTIC COLLECTION



Hypoenhancing areas in pancreas = NECROSIS

Enhancing wall

Heterogenous content of collection – including some solid debris.

A different case – showing progression of features to WON

> 4 WEEKS

= NECROSIS

+

ENHANCING CAPSULE, HETEROGENOUS CONTENT

↓

WALLED-OFF NECROSIS

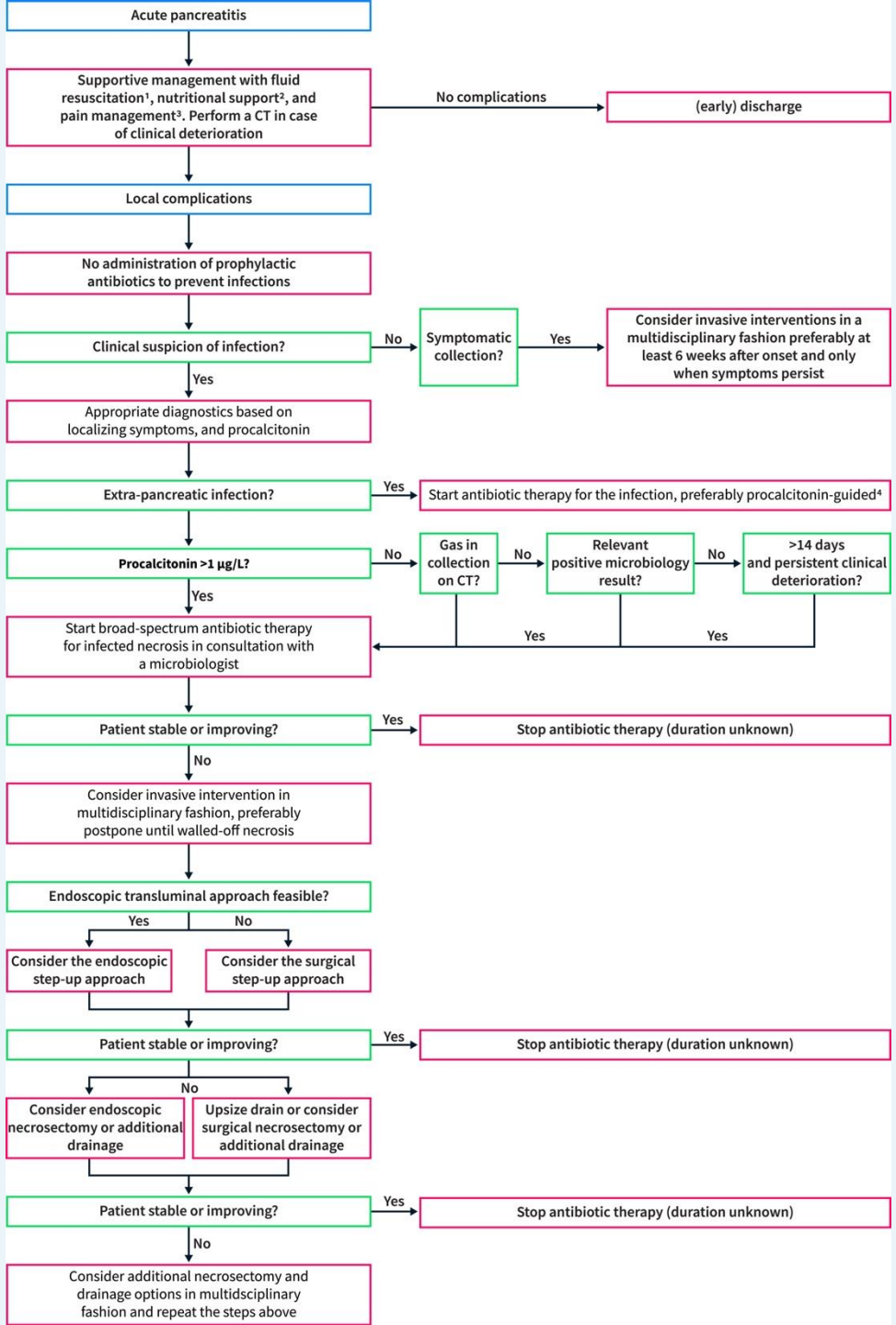
Management

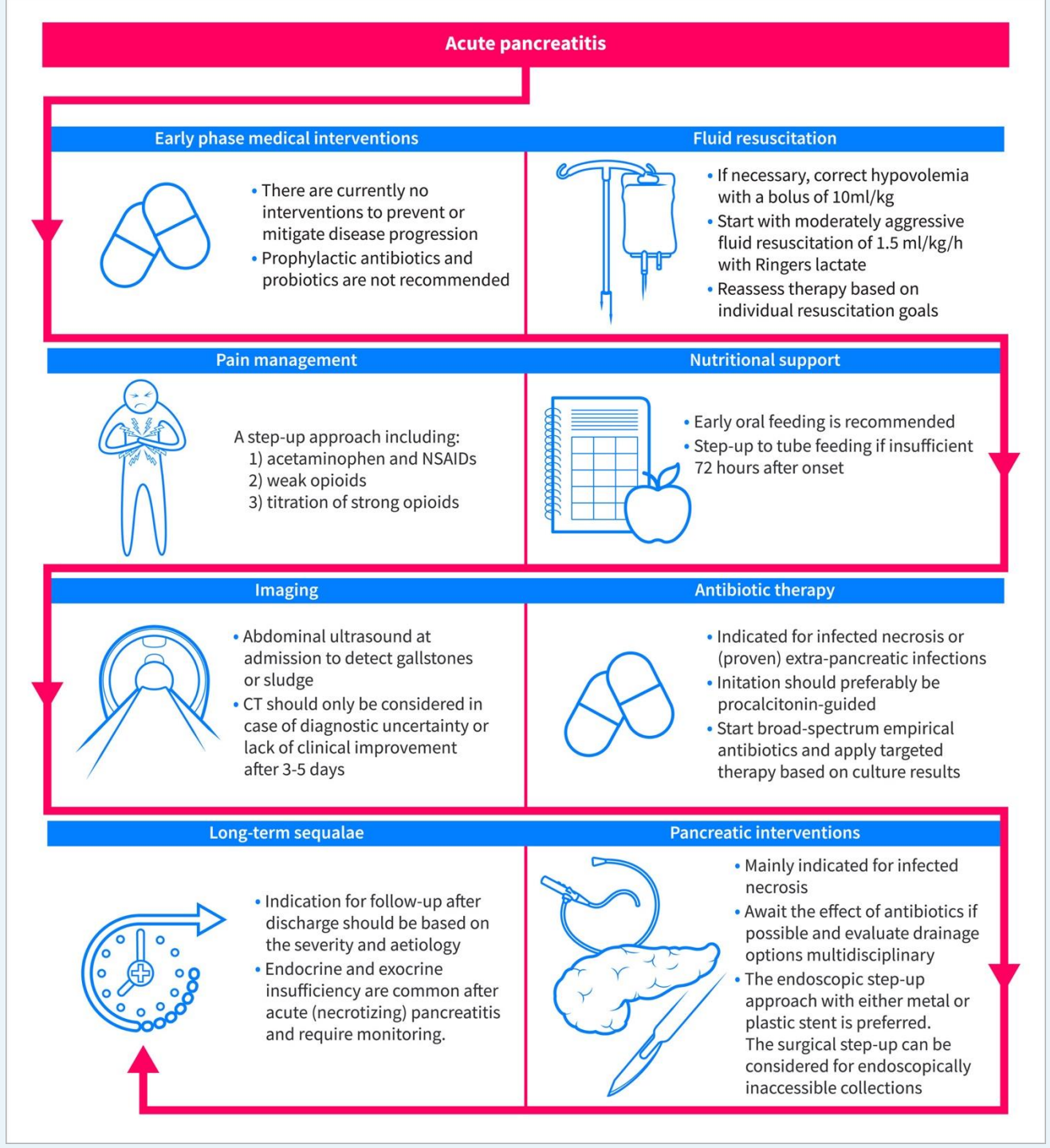
- ≤ 4 weeks = **CONSERVATIVE**
  - Drainage **NOT** advised in early period before fluid walls off.

- > 4 weeks – encapsulated collections = various options:
  1) **Conservative**:
     - Any collection can be managed conservatively if the clinical picture suggests so, many will involute on their own.
     - However, if drainage is required (usually superimposed infection) then the approach is very different in pseuodcyst vs WON
  2) **Non-surgical interventions**:
     a) PSEUDOCYST -> **percutaneous** drainage (radiologically guided) or **endoscopic** (e.g. Axios™), if inaccessible percutaneously. See subsequent section.
     b) WON -> **ENDOSCOPIC** – e.g. Axios™ stent. **NOT SUITABLE FOR PERCUTANOUS DRAINAGE.**
  3) **Surgical options**:
     - Surgical drainage, necrosectomy – performed less commonly now due to endoscopic options currently available. Generally reserved for when collection is not accessible (e.g. in a location distant from stomach).

Prognosis

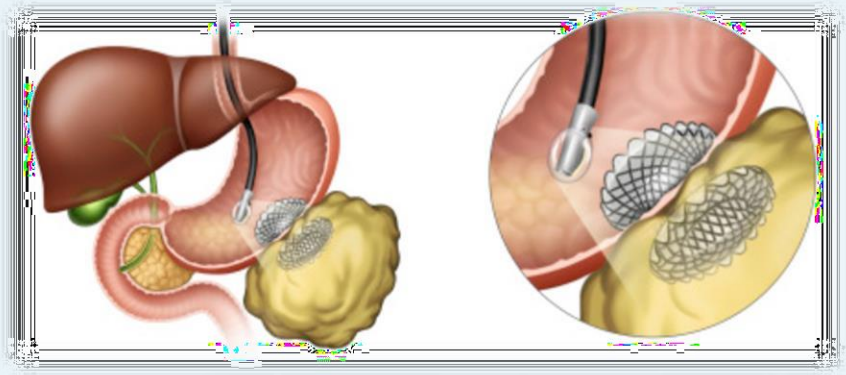
- Worse in necrotising pancreatitis and its associated sequelae (i.e. of the collections at >4 weeks, WON (NP) has a worse prognosis than pseudocyst (IEP)).


Acute pancreatitis
Supportive management with fluid resuscitation[1], nutritional support[2], and pain management[3]. Perform a CT in case of clinical deterioration
No complications
(early) discharge
Local complications
No administration of prophylactic antibiotics to prevent infections
Clinical suspicion of infection?
No
Symptomatic collection?
Yes
Consider invasive interventions in a multidisciplinary fashion preferably at least 6 weeks after onset and only when symptoms persist
Yes
Appropriate diagnostics based on localizing symptoms, and procalcitonin
Extra-pancreatic infection?
Yes
Start antibiotic therapy for the infection, preferably procalcitonin-guided[4]
Procalcitonin >1 µg/L?
No
Gas in collection on CT?
No
Relevant positive microbiology result?
No
>14 days and persistent clinical deterioration?
Yes
Yes
Yes
Start broad-spectrum antibiotic therapy for infected necrosis in consultation with a microbiologist
Patient stable or improving?
Yes
Stop antibiotic therapy (duration unknown)
No
Consider invasive intervention in multidisciplinary fashion, preferably postpone until walled-off necrosis
Endoscopic transluminal approach feasible?
Yes
No
Consider the endoscopic step-up approach
Consider the surgical step-up approach
Patient stable or improving?
Yes
Stop antibiotic therapy (duration unknown)
No
Consider endoscopic necrosectomy or additional drainage
Upsize drain or consider surgical necrosectomy or additional drainage
Patient stable or improving?
Yes
Stop antibiotic therapy (duration unknown)
No
Consider additional necrosectomy and drainage options in multidsciplinary fashion and repeat the steps above

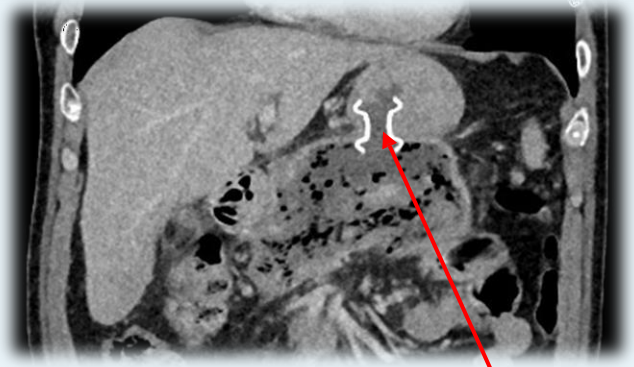

Acute pancreatitis
Early phase medical interventions
There are currently no interventions to prevent or mitigate disease progression
Prophylactic antibiotics and probiotics are not recommended
Fluid resuscitation
If necessary, correct hypovolemia with a bolus of 10ml/kg
Start with moderately aggressive fluid resuscitation of 1.5 ml/kg/h with Ringers lactate
Reassess therapy based on individual resuscitation goals
Pain management
A step-up approach including:
1) acetaminophen and NSAIDs
2) weak opioids
3) titration of strong opioids
Nutritional support
Early oral feeding is recommended
Step-up to tube feeding if insufficient 72 hours after onset
Imaging
Abdominal ultrasound at admission to detect gallstones or sludge
CT should only be considered in case of diagnostic uncertainty or lack of clinical improvement after 3-5 days
Antibiotic therapy
Indicated for infected necrosis or (proven) extra-pancreatic infections
Initation should preferably be procalcitonin-guided
Start broad-spectrum empirical antibiotics and apply targeted therapy based on culture results
Long-term sequalae
Indication for follow-up after discharge should be based on the severity and aetiology
Endocrine and exocrine insufficiency are common after acute (necrotizing) pancreatitis and require monitoring.
Pancreatic interventions
Mainly indicated for infected necrosis
Await the effect of antibiotics if possible and evaluate drainage options multidisciplinary
The endoscopic step-up approach with either metal or plastic stent is preferred.
The surgical step-up can be considered for endoscopically inaccessible collections

‘LAMS’ – Lumen-Apposing Metal Stents

Schematic diagram *(below)* showing a LAMS.[10]

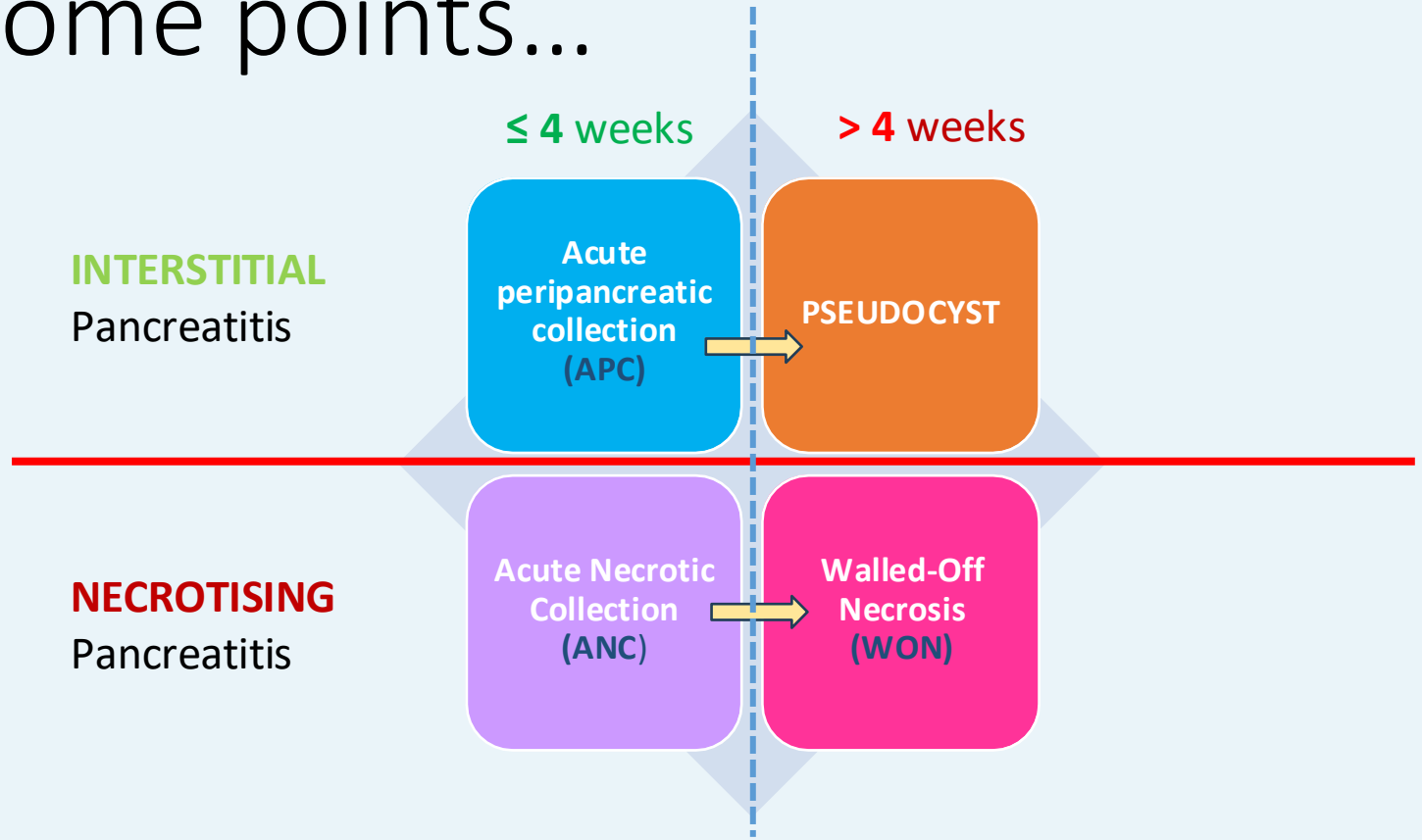


- LAMS (generic name), of which Axios™ (brand name) is most commonly used.
- Such stents are inserted endoscopically and placed between the posterior wall of the stomach and the peripancreatic collection, to allow drainage of material into the stomach (which will from there pass down the GI tract).
- Uses:
  - **Pseudocyst** – e.g. if not accessible percutaneously.
  - **WON** – essentially the only interventional option here, besides surgery.
- Routinely removed 6 weeks following insertion.
- In the case of WON, will invariably become clogged due to nature of material and will therefore require several endoscopies before the routine removal at 6 weeks to ‘manually’ clear the lumen and retrieve as much material as possible.
- **NB**.
  If Axios™ is used for pseudocyst – then can be inserted and left. Content is thin fluid, will drain easily and not block lumen. Routine removal at 6 weeks as above.



Above: A coronal image of patient with Axios™ stent – superior end is seen in stomach and inferior aspect is seen within (infected) WON with air.

Take home points…



- WON and PSEUDOCYSTS have very different content and their management differs. Significant debris means **WON** is **NOT suitable for percutaneous drainage**. If Axios™ stent is used in WON then will invariably require re-endoscopy to clear occluded stent lumen and drain the semisolid material.

.