SO, WAT'S THERE INSIDE THE PANDORAS BOX?

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- Dr Kalpana
- Dr Sarath Balaji
- Dr Babu

- Dr Pon divya

Case history

- 10/F
- No significant previous medical/surgical illness
- Fever x 20days
  - High grade, intermittent, no chills/rigors
- No cough/breathlessness
- No vomiting/abdominal pain/jaundice
- No joint pain/swelling/skin rashes

Started on ATT elsewhere based on Mantoux positivity.

2 days later, she developed

- Abdominal pain- severe
- vomiting- 3-4 episodes, non projectile, non bilious, contained food particles
- Continued to have low grade fever spikes

- She was taken to another private hospital for the above symptoms
- Amylase- 244
- Lipase- 126

USG ABDOMEN:

- Bulky pancreas
- Well defined cystic lesion in the head of pancreas with few enlarged adjacent LN- ?pseudocyst

- ATT was stopped
- Managed symptomatically with IV Fluids, PPIs and antibiotics
- Her fever spikes and abdominal pain settled and was discharged

3 days later…..

- Again developed severe epigastric pain+
- Low grade fever spikes+
- Vomiting+
- Amylase -418, Lipase-940

- Admitted 4 times in 2 months at the same hospital for pancreatitis

- As she had recurrent pancreatitis, CT abdomen was taken

It showed:

- Pancreatic divisum
- Small thick walled collapsed pseudocyst communicating with the main pancreatic duct
- Multiple enlarged peripancreatic, left gastric and portocaval lymphnodes

- Managed symptomatically
- Abdominal pain decreased and vomiting settled
- Continued to have low grade fever(2 months)
- Not on ATT

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A week later, came to ICH,

- With c/o low grade fever spikes+
- Epigastric pain
- Loss of weight
- poor appetite

EXAMINATION

- Conscious, oriented, illnourished
- Febrile- 101'f
- No pallor, icterus, edema
- BCG scar +

Systemic examination

- Abdomen:
  - Tenderness + over the rt hypochondrium and the epigastrium
  - Liver palpable 3cm below rt costal margin
  - Spleen 2cm below lt costal margin

Workup was done

- CBC
- Pathologist smear- normal
- RFT,LFT- TB<1, sgot and sgpt normal
- Amylase- 244, lipase- 366
- Usg abdomen – peripancreatic lymphnodes with pseudocyst+
- Multiple paraaortic nodes+
- Hepatospenomegaly+

Summary – 10/F

Persistent fever spikes – 3 months **ATT 2 days**	**Hepatosplenomegaly**	**Mantoux positive**
Peripancreatic and paraaortic LN	**Recurrent pancreatitis**	**Pancreatic divisum**

POSSIBILITIES

TB	**Drug induced pancreatitis**
Pancreatic divisum	**Lymphoma**

- **Mantoux – positive 24mm**
- CXR- normal
- RGJ- CBNAAT negative
- MGIT BAL – negative
- UGI Scopy- Normal
- Retroviral – negative
- ANA, Anti-ds Dna – negative
- Bone marrow – normal, no blasts
- CT Guided biopsy – planned, deferred due to fear of injury to great vessels.

POSSIBILITIES

TB	**Drug induced pancreatitis**
Pancreatic divisum	**Lymphoma**

- **ATT was restarted**
- INH replaced
- ATT Regimen-
  - Ofloxacin+ Rifampicin+Pyrazinamide+Ethambutol
- Not given any steroids

On follow up

Within a week…..

- Well being+
- Appetite improved, wt gain+
- Asymptomatic -No abdominal pain/ fever
- Lipase and amylase - normal

- Rpt usg- peripancreatic nodes decreased, pseudocyst+ size reduced
- Completed ATT

- **A CASE OF (SUSPECTED) PANCREATIC TUBERCULOSIS**

- First case of pancreatic tuberculosis - ICH

DISCUSSION - PANCREATITIS

The predominant causes include

- Abdominal trauma(23%),
- **Anomalies of the pancreaticobiliary system (15%),**
- **Multisystem disease (14%),**
- Drugs and toxins (12%),
- Viral infections (10%),
- Hereditary disorders (2%)
- Metabolic disorders (2%).
- 25% Idiopathic

- **Pancreatic tuberculosis** - extremely rare form of extrapulmonary tuberculosis even in endemic areas that masquerades as a mass or inflammation
- It accounts for less than **5% of all tuberculosis** in the developing world.
- Usually misdiagnosed as a pancreatic malignancy or mere pancreatitis.
- However, the excellent response to anti-tuberculous therapy means that these patients should not be misdiagnosed and mismanaged.

Why pancreatic TB is rare?

- It is not easy for tubercular pathogens to survive and progress in human pancreas.
- **Resistance provided by the pancreatic enzymes**.
- Pancreatic enzymes, including lipases and deoxyribonucleases, have **antimycobacterial effects** to interfere with the seeding of Mtb.
- That is the reason why pancreatic TB usually occurs in immunocompromised individuals or as a complication of miliary TB.

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ULTRASOUND

- CBD and PD dilatation
- Enlarged pancreas (focal or diffuse)
- Focal hypoechoic lesions
- Peri-pancreatic lymphadenopathy
- Other abdominal findings of extra-pancreatic tuberculosis like ascites,bowel thickening, enlarged abdominal lymph nodes

COMPUTED TOMOGRAPHY

- Cystic or mixed lesions
- Calcifications
- Vascular encasement
- Peri-pancreatic lymphadenopathy, mediastinal lymphadenopathy
- Other abdominal findings of extra-pancreatic tuberculosis like ascites, peritoneal enhancement, bowel thickening, and abdominal lymph nodes

MAGNETIC RESONANCE IMAGING

- Ductal dilatation (CBD and PD)

  T1: hypointense lesions

  T2: hyperintense lesions
- Post contrast: rim enhancement

ENDOSCOPIC ULTRASOUND

- Ductal dilatation (CBD and PD)
- Hypoechoic lesions
- Vascular involvement
- Calcifications
- Peri-pancreatic, mediastinal lymphadenopathy
- Ascites, pleural effusion, and other extra-pancreatic findings

CONFIRMATORY

- Ultrasound- or CT-guided FNAC may provide the diagnosis
- Microscopic features are those of **caseating granulomatous inflammation with signs of necrosis.**

- Polymerase chain reaction (PCR)-based assay- detects *Mycobacterium tuberculosis* DNA in resected specimens.

- Endoscopic stenting
  - to relieve obstructive jaundice
  - cholangitis

INH PANCREATITIS

Fatal if not withdrawn

Estimated incidence **0.1 - 2%.**

complete recovery on drug withdrawal

TAKE HOME

- The diagnostic possibility of pancreatic TB must be considered in patients with a pancreatic mass especially in individuals, residing in endemic countries

- INH induced pancreatitis is not uncommon

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THANK YOU!!!!!!!!