**Acute & Chronic Pancreatitis**

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Darryl Mackender
Bathurst & Orange

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**Why the pancreas?**

- Limited access to Gastroenterologist / Subspecialty UGI Surgeon
- Rare illnesses with protean presentations
- Major sequelae if acute pancreatitis diagnosis delayed
- Missed pancreatic cancer not uncommon
- Chronic abdominal pain syndromes
- Emerging Pancreatic entities

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**Why the pancreas?**

- Emerging Pancreatic entities
- Genetic idiopathic chronic pancreatitis (SPINK, CFTR)
- Microlithiasis
- Sphincter of Oddi dysfunction
- Auto-immune pancreatitis

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**“Pan” – All “Kreas” - Flesh**

“When health is alien, wisdom cannot reveal itself, art cannot manifest, strength cannot be exerted, wealth becomes useless & reason powerless.”

Hippocrates of Chacedon 330BC

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**What’s new in Acute Pancreatitis?**

**CAUSES**

- Alcohol: Carbohydrate deficient transferrin level >2:7 U/l 27% specific, 100% sensitive for AP, identified 98% cases

  Perez Mateo JOP 2006

- **Medications**

  - Statins (OR 1.4), COX-II/NSAID’s (OR 1.3)
  - ACEI (OR 1.9) dose related <6mo
  - Ca antagonist (OR 1.5)
  - Idiopathic recurrent Acute Pancreatitis or
  - Idiopathic Chronic Pancreatitis

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**Idiopathic Recurrent acute pancreatitis / ICP**

- Truly idiopathic uncommon
- Majority prove to have
  - Microlithiasis / Choledocholithiasis / Biliary sludge
  - CFTR genetic mutation
  - Hereditary pancreatitis mutation (SPINK)
  - ?? Pancreas divisum
  - ?? Sphincter of Oddi Dysfunction
Microlithiasis

- Long term follow up RAP 75pts mean 1.8mo follow-up
- 50% developed evidence ICP by imaging (CT, ERCP, U/S or EUS)
- 39pts microlithiasis, 2 gallstones
- 10 pts with Microlithiasis – 8/10 persistent RAP or ICP despite cholecystectomy (4), biliary sphincterotomy (4)
- Cohort 2583 pts with gallstones, 3.4% AP
- Post cholecystectomy risk AP similar to general popn

Genetic associations

- CFTR Mutations
  - Incidence depends on cohort, no of mutations tested

Genetic associations

- SPINK mutations
- PRSS1 mutation
- PRSS2 mutation
- Estim. 50%
- Unexplained RAP

Pancreas divisum

- Not thought to cause RAP on its own
- Not thought worthy of surgical or endotherapy

Pancreas Divisum

- Not thought to cause RAP on its own
- Not thought worthy of surgical or endotherapy
Sphincter of Oddi dysfunction:
- Increasingly thought to be secondary to ICP rather than a cause.
- Manometry rarely performed currently.
- HIDA scan not the Gold Standard.
- HIDA GB CCK EF does NOT identify microlithiasis, acalculous cholecystitis or SOD reliably.

Current State of the Art:
- Severe Acute Pancreatitis:
  - Recognition of severity
  - Early post-pyloric enteral feeding
  - "Antibiotic prophylaxis" of infected pancreatic necrosis
  - ERCP
  - Early vs Delayed Cholecystectomy
  - Minimally invasive surgical management complications

Chronic Pancreatitis:
- Chronic pain syndrome / malignancy not an uncommon differential.
- Window of opportunity for resectable pancreatic Ca may be <2 weeks.
- CP relies on imaging – CT / US / MRCP / EUS +/- FNA.
- Pancreatic function testing difficult.
- Secretin-stimulated MRCP may become gold standard.

Auto-immune Pancreatitis:
- First described >40yrs ago.
- New Mayo Clinic classification (29pts between 1999-2005).

Pancreatic function testing difficult:
- Secretin-stimulated MRCP may become gold standard.
IgG4 associated Cholangitis

- Awareness in diffusely “bulky” pancreas
- Steroid responsiveness
- EUS + FNA core may be diagnostic
- Serum IgG4 in 70%, also elevated in some PSC
- Also recognised a steroid responsive IgG4 cholangitis like PSC

Auto-immune Pancreatitis

- Get access good quality CT Cholangiogram & CT Pancreatic Protocol
- EUS growing indications & availability
- Confirmed gallstonepancreatitis / suspected microlithiasis / biliary sludge requires early Cholecystectomy
- Genetic studies in IRAP / ICP available
- Awareness of auto-immune pancreatitis

Conclusion