Gastroenterology Case

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Case

- 56 year old woman
- PC: 3 months history of change of bowel habit

- > 3/12 history of watery diarrhoea
- Intermittent fresh PR bleed
- Weight loss 5kg
- No associated with eating or drink
- No associated with food choice
- Some abdominal pain: non specific

What else do you want know?

- No new recent medication
- No regular medication
- No family of colon cancer, inflammatory bowel disease or coeliac

- Previous pancreatic operation : cyst removal

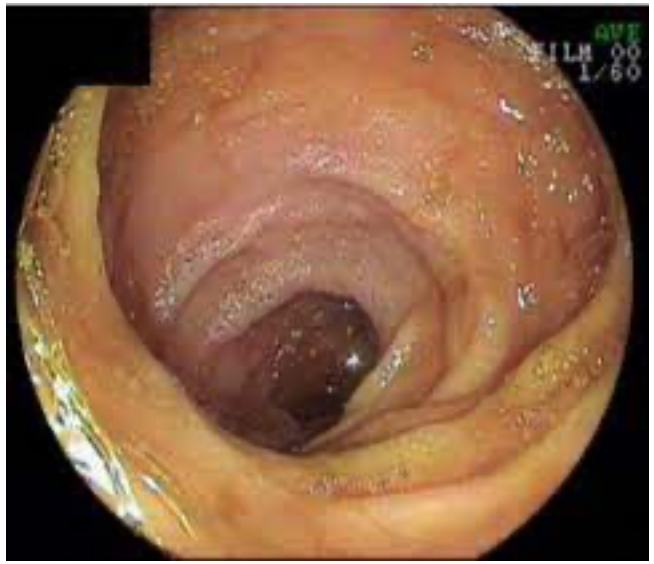
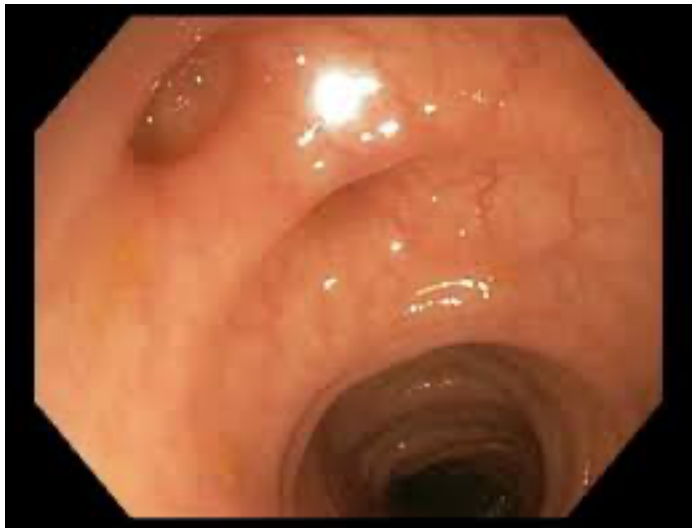
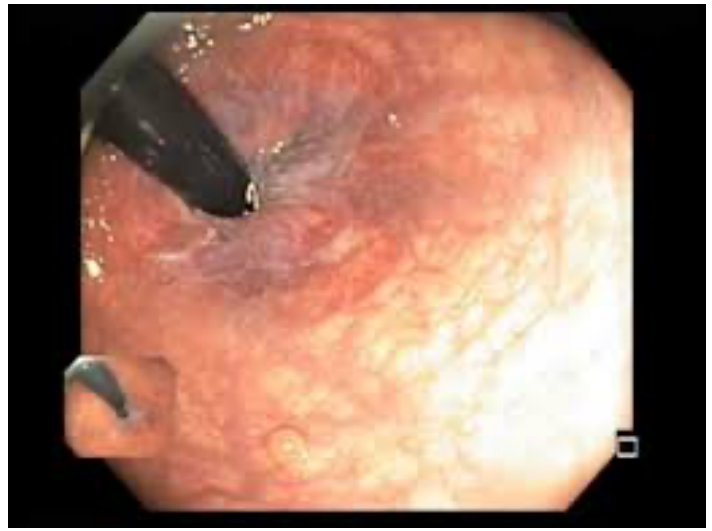
Investigation

- Liver functions: normal
- U+E/Cr: normal
- FBC/ferritin: normal
- Coealiac serology: normal
- TFTs: normal

- Faecal calprotectin: 70 (<50)
- Faecal spec: no parasite no bacteria or virus

Next step?

- Colonoscopy

- Histology: normal terminal ileum, right colon and left colon

Next step?

- ? Food diary
- ? Konsyl-D
- ? Dietician review for D-IBS
- ? Pancreatic elastase
- ? Faecal steatocrit
- ? Gastrin, VIP, chromogranin A, urinary catecholamines

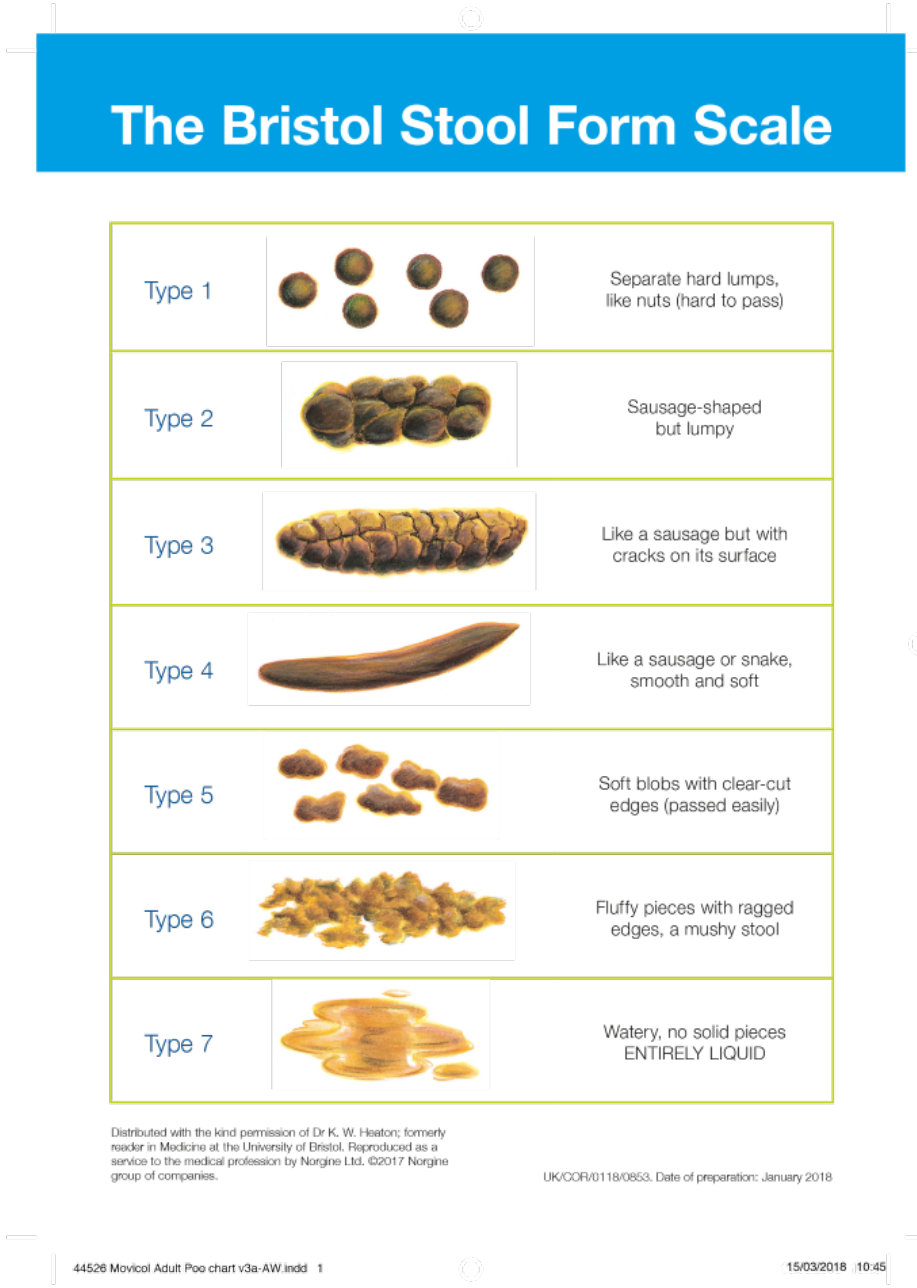
- Faecal steatocrit: 10%

- Pancreatic elastase: 120
  - Mild to moderate exocrine pancreatic insufficiency

Diarrhoea

- “Ask the patient”
- Patients vary in what they call diarrohea
  - Consistency
  - Quantity –
  - Frequency
  - Fecal incontinence
- 3 or more
  - loose or liquid stools in one day, or more frequently than normal

- Acute: ≤ 14 days
- Persistent: > 14 days
- Chronic: > 30 days



History

- Clear understanding of what the patient means by diarrhea •
- Stool characteristics
- Duration and onset
- Alarm symptoms
- Nocturnal symptoms
- Extra intestinal symptoms
- Risk factors – Travel, contacts, family history

Colon physiology

- Normal physiology
  - Absorption and secretion of water and electrolytes occur throughout intestine
  - In the non fasting state, approximately 9 litres of fluid pass into small bowel (salivary, gastric, pancreatic and biliary secretions; intake of fluid)
  - Water and electrolytes are simultaneously absorbed by the villi and secreted by the crypts
  - Net result is absorption
  - 1-2 litres of liquid reach the colon, with only 100 to 200 ml being excreted in the faeces
  - Colon has an absorptive capacity of 3-5 litres
  - Water is absorbed passively by osmotic gradients that are created by an active sodium absorption
  - Co transport of glucose and sodium has been documented
  - Oral rehydration solutions containing both sodium and glucose enhance water absorption in patients with diarrhoea

Acute diarrhoea: ≤ 14 days

- Infective Diarrhoea
  - Bacterial
    - E.coli (most common), Salmonella, Shigella
  - Viral
    - Rota virus –MOST COMMON cause
    - Noro virus
  - Parasitic
    - Giardia, cryptosporidium
  - Acquired abroad
    - Cholera, Amoebic dysentry

Acute diarrhoea: ≤ 14 days

- Drugs
  - Antibiotics- 5 ways in which antibiotics can lead to diarrhoea
    - Clindamycin (and others) – proliferation of C diff as most other bacteria killed
    - Erythromycin- increases motility
    - Penicillins- breakdown products act as osmotic agent
    - Tetra cycline- inhibits fat absorption
    - Neomycin- affects bile salt re-absorption, osmotic agent
  - Laxatives
  - Others- digoxin, magnesium salts, PPI esp omeprazole, NSAIDs
- Ischaemic colitis (elderly, AF)

Table 3. Drugs Associated with Diarrhea

Osmotic

Citrates, phosphates, sulfates

Magnesium-containing antacids and laxatives

Sugar alcohols (e.g., mannitol, sorbitol, xylitol)

Secretory

Antiarrhythmics (e.g., quinine)

Antibiotics (e.g., amoxicillin/clavulanate [Augmentin])

Antineoplastics

Biguanides

Calcitonin

Cardiac glycosides (e.g., digitalis)

Colchicine

Nonsteroidal anti-inflammatory drugs (may contribute to microscopic colitis)

Prostaglandins (e.g., misoprostol [Cytotec])

Ticlopidine

Motility

Macrolides (e.g., erythromycin)

Metoclopramide (Reglan)

Stimulant laxatives (e.g., bisacodyl [Dulcolax], senna)

Malabsorption

Acarbose (Precose; carbohydrate malabsorption)

Aminoglycosides

Orlistat (Xenical; fat malabsorption)

Thyroid supplements

Ticlopidine

Pseudomembranous colitis (*Clostridium difficile*)

Antibiotics (e.g., amoxicillin, cephalosporins, clindamycin, fluoroquinolones)

Antineoplastics

Immunosuppressants

Information from reference 2.

Chronic diarrhoea investigation

- 3 stool specimens for culture and sensitivity include C difficile – Giardia, amoebiasis, C difficile
- Check FBC, U&E, ESR, CRP, Albumin, TTG
- Faecal calprotectin
- Faecal steatocrit
- Pancreatic elastase
- Colonoscopy if over age 45 or alarm features
  - Inflammatory
  - Diverticular disease
  - Cancer
  - Normal ? Irritable bowel syndrome, non-GI cause

Chronic diarrhoea

Colon	Colonic neoplasia
	Inflammatory bowel disease (ulcerative colitis, Crohn's colitis)
	Microscopic colitis
Small bowel	Celiac disease
	Crohn disease
	Other small bowel enteropathies
	Bile acid malabsorption
	Disaccharidase deficiency
	Small bowel bacterial overgrowth
	Mesenteric ischemia
	Radiation enteritis
	Lymphoma
	Chronic infection (eg, giardiasis)
Pancreas	Chronic pancreatitis
	Pancreatic carcinoma
	Cystic fibrosis
Endocrine	Hyperthyroidism
	Diabetes
	Hypoparathyroidism
	Addison disease
	Hormone-secreting tumours (eg, VIPoma, carcinoid, gastrinoma)
Other	Factitious diarrhea
	Surgery (eg, small bowel resection, internal fistulas)
	Drugs (eg, nonsteroidal anti-inflammatory drugs, antihypertensives, antibiotics, antiarrhythmics, antineoplastics, drugs containing magnesium)
	Food additives (eg, sorbitol, fructose)
	Alcohol abuse
	Autonomic neuropathy

Adapted from reference 1

Exocrine Pancreatic Insufficiency (EPI)

- Exocrine pancreatic insufficiency (EPI) is a condition caused by reduced or inappropriate secretion or activity of pancreatic enzyme.

- In a German-based study, one of the most common causes of EPI had an age-adjusted prevalence of 8 per 100,000 for males and 2 per 100,000 for women; these numbers are probably relatively close to the prevalence of EPI in most developed countries. No other reliable data are currently available.

- The natural history and progression of EPI depend on the underlying etiology

Table 1 Prevalence of EPI in different clinical conditions

EPI caused by pancreatic disorders		
Disease	EPI prevalence	Factors associated with EPI occurrence
Chronic pancreatitis	30%–90%	• Long disease duration • Alcoholic etiology • Extensive calcifications • Ductal obstruction
Acute pancreatitis	Mild pancreatitis: 15%–20% Severe pancreatitis: 30%–40%	• Necrosis extent (>30%) • Alcoholic etiology
Autoimmune pancreatitis	30%–60%	Extensive mass/calcification
Unresectable pancreatic cancer	20%–60%	• Head localization • Large size • Ductal obstruction • Coexistent chronic pancreatitis
Pancreatic neoplasms after surgery	Pancreaticoduodenectomy: 80%–90% Distal pancreatectomy: 20%–50%	• Whipple intervention* • Gastropancreatic anastomosis*
Benign pancreatic tumor (before surgery)	30%–60%	• Head localization • Large size • Ductal obstruction • Coexistent chronic pancreatitis
Cystic fibrosis	80%–90%	Classes I, II, III, VI CFTR mutations
Shwachman–Diamond syndrome	80%–90%	–

The natural history and progression of EPI depend on the underlying etiology

EPI caused by extrapancreatic disorders		
Type I diabetes	30%–50%	• High insulin requirement • Poor glycemic control • Early diabetes onset
Type II diabetes	20%–30%	• Insulin requirement • Poor glycemic control • Long diabetes duration
Inflammatory bowel disease	Ulcerative colitis: 10% Crohn's disease: 4%	• Disease reactivation (only for temporary EPI) • Long disease duration • Surgical patients
Celiac disease	5%–80%	Untreated disease (no gluten-free diet)
Pediatric intestinal transplantation	10%	
HIV syndrome	10%–50%	Retroviral therapy
Gastrointestinal surgery	Total/subtotal gastrectomy: 40%–80% Esophagectomy: 16%	• Extensive intestinal resection • Vagal denervation
Sjogren's syndrome	10%–30%	
Aging	15%–30%	Age >80 years
Tobacco usage	10%–20%	Alcohol usage
Somatostatin analogs therapy	20%	

- Chronic Pancreatis is the most common pancreatic disease associated with EPI. In CP, progressive loss of acinar cells and fibrosis reduce lipase secretion.
- Clinically significant EPI in CP requires a reduction of almost 90% of pancreatic enzymes and is reported in 60%–90% of CP patients within 10–12 years.
- EPI can also be the consequence of a previous AP episode with significant loss of parenchyma

EPI symptoms

- Gas, bloating, dirrhoea
- Oily, greasy, foul smell stool
- Weight loss
- Abdominal pain
- Malnutrition can sometimes occur: fatigue, dizziness, dry skin, brittle nails, hair loss, depression, irritability

Diagnosis

- Pancreatic elastase: Little (or no) elastase can indicate EPI.
- Faecal steatocrit: A high fat volume may be a sign of EPI.

Management

- Replace pancreatic enzyme
  - Creon capsules contain digestive enzymes (amylase, lipase, protease) extracted from the pancreas of pigs.
  - Restore the normal digestive action
  - With meals and snacks

- High calories and high fat diet
- Vitamin replacements

Management

- Treatment depends on the cause of the EPI
  - ? Chronic pancreatitis
  - ? Autoimmune pancreatitis
  - ? Alcohol ? Drug
  - ? Cancer
  - ? Previous surgery
- Amylase, lipase, IgG4, Autoimmune screening
- CA19-9
- CT pancreas: cyst, calcification, cancer, IPMN…etc
- MRI
- Endoscopic ultrasound

Case continue

- 56 year old woman
- Exocrine pancreatic insufficiency
- Previous pancreatic cyst resected overseas: no note
- Alcohol use with likely chronic pancreatitis on CT

- Improved with creon 300mg with meals and creon 150mg with snacks
- Alcohol abstinent

Take home message

- Diarrhoea
  - History
  - Risk factors and alarm symptoms
    - GI related
    - Non-GI related
  - Refer if chronic diarrhoea, risk factor or alarm symptoms
  - Colonoscopy
  - Think outside the box