**GASTROENTEROLOGY**

Clinical Update 2015
Stephen Mouat

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**Introduction**

- Inflammatory bowel disease
- Coeliac Disease
- Gastro-oesophageal reflux
- Pancreatitis
- Eosinophilic oesophagitis
- Short gut syndrome
- Diarrhoea
- H. pylori

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**Exclusive Enteral Therapy**

- Treating with food!
- 6-8 weeks of formula only
Bacteria

- Bacteria
  - Probably not pathogenic bacteria
  - Target for immune response

Immune system

- Autoimmune disease – immune dysregulation

Crohn’s Vs UC

<table>
<thead>
<tr>
<th></th>
<th>Crohn’s</th>
<th>UC</th>
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<tbody>
<tr>
<td>Distribution</td>
<td>Anywhere</td>
<td>Large bowel</td>
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<tr>
<td>Pattern</td>
<td>Patchy</td>
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<td>Depth of inflammation</td>
<td>Transmural</td>
<td>Mucosal</td>
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Crohn’s Vs UC

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<tr>
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<th>UC</th>
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<tbody>
<tr>
<td>Structuring</td>
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<td>No</td>
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<tr>
<td>Fistulising</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Perianal</td>
<td>Yes</td>
<td>No</td>
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Crohn’s Vs UC

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<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Extra-intestinal</td>
<td>+</td>
<td>++</td>
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<tr>
<td>Surgery</td>
<td>For complications</td>
<td>For cure</td>
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<table>
<thead>
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<th>Crohn’s</th>
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<tbody>
<tr>
<td>Enteral therapy</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>5-asa – remission</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>5-asa- maintenance</td>
<td>+</td>
<td>++</td>
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<tr>
<td>Steroids</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Aza/Mtx</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>TNF mono Ab</td>
<td>++</td>
<td>+</td>
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Early onset IBD

- Immune mediated
- Autoimmune enteropathy
- IPEX
- IL10 receptor
- Multisystem disease
  - 60% genetic abnormality
- Isolated
  - 28% genetic abnormality

Pyoderma gangrenosum

- erythematous papules
- pustules
- haemorrhagic bullae
- indurated ulcer with raised edges and yellowish exudate
- Most common on legs
- May be associated with mild trauma
- UC 50% of cases, CD less common
- Rx – corticosteroid +- cyclosporine
Erythema Nodusum

- Tender, erythematous, poorly circumscribed
- Associated with increasing disease activity
- 4% UC, 2% CD
- Treatment
  - bed rest
  - systemic or intra-lesional steroids
  - NSAIDs
  - potassium iodide,
  - colchicine
  - dapsone

Treatment IBD

- Enteral therapy (Crohn's)
- 5 asa's
- Prednisone
- Azathioprine
- Methotrexate
- TNF monoclonal antibodies
Exclusive Enteral Therapy

- Treating with food!
- 6-8 weeks of formula only

5- amino salicylic acids

- Mesalazine – Pentasa/ Asacol
- Olsalazine – Dipentum
- Sulphasalazine

Prednisone

- Rapid and reliable response to most patients with IBD
Azathioprine

- Monitoring can be done with either genotype or metabolite levels
- 1:300 have low TPMT activity (homozygote)
- 11% intermediate activity (heterozygotes)
- 80% of bone marrow suppression occurs in homozygote with normal activity

Azathioprine

- Levels of 6-TGN (6-thioguanine nucleotide)
  - Bone marrow suppression
  - Increased therapeutic efficacy
- 6-MMP (6-methyl mercaptopurine)
  - increased hepatotoxicity

Azathioprine

- Side effects
  - Dose related –
    - bone marrow suppression
    - hepatotoxicity
    - diarrhoea
    - nausea
  - Idiosyncratic
    - pancreatitis
**Infliximab**
- Chimeric TNF-a monoclonal antibody
- TNF-a is a pro-inflammatory cytokine
- Indications -
  - unresponsiveness to conventional therapy
  - Fistulising disease
  - Moderate/ severe disease
- 50-90% remission rate

**Adalimumab**
- Fully humanised TNF MAB
- Sub cutaneous ever 2 weeks
- Similar efficacy to Infliximab

**TNF mab and lymphoma**
- Background rate: 1:10 000
- Patients with Crohn’s: 1: 8 000
- Patients with Crohn’s on azathioprine + infliximab: 1: 2 000

**Hepatosplenic T cell lymphoma**
- 76 cases – all TNF MAB
- High mortality
- All had also been on azathioprine
- 20 + cases also occurred with azathioprine, before TNF mab used
- Balance of risk/benefit
Infliximab antibodies

<p>| | |</p>
<table>
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<tr>
<td>No meds</td>
<td>24%</td>
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<tr>
<td>Steroids</td>
<td>13%</td>
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<tr>
<td>Immunosuppressives</td>
<td>11%</td>
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<tr>
<td>Steroids and</td>
<td>3.7%</td>
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<tr>
<td>immunosuppressives</td>
<td></td>
</tr>
</tbody>
</table>

But perhaps

- After one year stable anti-TNF-a + immunosuppression
- Withdrawal of adjuvent immunosuppression
- Ab rate went from 6% - to 5%

Next topic!
Coeliac disease

- villous atrophy
- mononuclear infiltrate in the lamina propria
- crypt hyperplasia

Dermatitis herpetiformis

- Painful and intensely pruritic rash
- Associated with CD
- Treatment is gluten avoidance +/- dapsone

Which test?

- Tissue Transglutaminase antibody
- Endomysial antibody
- De-aminated gliaden peptide antibody
  - 90+ sensitive and specific
  - Some IgA, some IgG
  - In at risk population

- Gliaden antibody – rubbish test, don’t do it
Transglutaminase antibody

- TTG abs normalise on treatment after 6-12 months
- false +ve TTG occurs with
  - type 1 diabetes,
  - chronic liver disease,
  - psoriatic
  - rheumatoid arthritis
  - heart failure
- sensitivity of TTG abs > 90%
- titre of TTG correlates with degree of mucosal damage
- sero-ve coeliac disease occurs- patients with mild mucosal changes

HLA DQ2/8 typing

- HLA-DQ2 and/or DQ8 genotypes found in at least 98% of patients.
  - HLA – DQ2 90-95% of Coeliac patients
  - HLA – DQ8 5-10%
- 30-40% of normal Caucasian population have HLA DQ2/8
- 50% of healthy relatives have HLA DQ2/8

Preventing coeliac disease in infants by gluten avoidance

- Last years multi-choice answer
  - C. weaning onto a gluten containing solids between 5-7 months while continuing to breast feed.
- This years multi-choice answer –based on three large prospective trials:
  - Weaning onto a gluten containing diet at 5-7 months while continuing breast feeding delays the onset of coeliac disease but does not alter its incidence.

A mother brings in her 4-month-old infant, whom she has breastfed since birth. Because an older sibling has coeliac disease, the mother wants to have this baby weaned to a gluten-free diet.

Of the following, the BEST advice to offer the mother is that

C. weaning onto a gluten containing solids between 5-7 months while continuing to breast feed.
Role of DQ2/8 typing

- Screening of family members
- Excluding coeliac disease when child on gluten free diet
- Help patients make informed choice about need for endoscopy

Shock horror, babies switched at birth….

- Risk is 1-2%

Coeliac Disease

- Diagnosis
  - Positive small bowel biopsy
  - Complete remission on gluten free diet
  - Some recent recommendations that children
    - With symptoms
    - Repeated highly positive TTg and confirmatory EMA
  - Do not require endoscopy
  - Not validated in NZ and Australian setting

Risk factors for Coeliac disease

- First degree relatives 4-11%
- Down’s syndrome 7-19%
- Turners, Williams
- IgA deficiency 7%
- Type 1 IDDM 5-10%
- Juvenile Chronic arthritis 2-5%
- Autoimmune thyroid disease 8%
New Topic

- 11 year old girl
- Weight 48kg BMI 75% for age
- Acute onset of abdominal pain and vomiting
- On examination
  - pale,
  - tender in RUQ
  - HR 92
  - RR 32

<table>
<thead>
<tr>
<th>CD with classic symptoms</th>
<th>Associated diseases:</th>
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<tbody>
<tr>
<td>Abdominal distension</td>
<td>Type I diabetes</td>
</tr>
<tr>
<td>Anorexia</td>
<td>Thyroiditis</td>
</tr>
<tr>
<td>Chronic or recurrent diarrhea</td>
<td>Sjogren’s syndrome</td>
</tr>
<tr>
<td>Failure to thrive or weight loss</td>
<td>Neurological and psychological disturbances</td>
</tr>
<tr>
<td>Irritability</td>
<td>Ataxia</td>
</tr>
<tr>
<td>Muscle wasting</td>
<td>Autism</td>
</tr>
<tr>
<td>Coeliac crisis (rare)</td>
<td>Depression</td>
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<table>
<thead>
<tr>
<th>CD with non-classic symptoms</th>
<th>Associated diseases:</th>
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<tbody>
<tr>
<td>Arthritis</td>
<td>IgA nephropathy</td>
</tr>
<tr>
<td>Aphthous stomatitis</td>
<td></td>
</tr>
<tr>
<td>Constipation</td>
<td></td>
</tr>
<tr>
<td>Dental enamel defects</td>
<td></td>
</tr>
<tr>
<td>Dermatitis herpetiformis</td>
<td></td>
</tr>
<tr>
<td>Hepatitis</td>
<td></td>
</tr>
<tr>
<td>Iron-deficient anemia</td>
<td></td>
</tr>
<tr>
<td>Pubertal delay</td>
<td></td>
</tr>
<tr>
<td>Recurrent abdominal pain</td>
<td></td>
</tr>
<tr>
<td>Short stature</td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td></td>
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</table>

**Pancreatitis**

- Amylase 3200
- AST 92
- GGT 104
- INR 1.0
- Albumin 48
- FBC normal
- Venous gas normal
- Glucose normal

**Management of acute pancreatitis**

- ABC
- Imaging (USS – looking for stones/ pseudocyst)
- NPO / NJ / NG / IVN?
- Antibiotics?
Pancreatitis

- TIGAR-O
- Toxic/metabolic
- Idiopathic
- Genetic
- Autoimmune
- Recurrent and severe
- Obstructive

Idiopathic

- Accounts for 20% of all large paediatric series

Biliary

- Antibiotics
- Relieve obstruction

Toxic/metabolic

- Drugs (therapeutic and recreational)
- L-asparaginase, valproate, metronidazole, azathioprine, tetracycline, pentamidine
- Alcohol
Metabolic

- Hyperlipidemia
- Hypercalcemia
- Glycogen storage disease
- Organic acidemias
- Burns

Failure to inhibit activation of which enzyme most commonly leads to familial pancreatitis?

- Trypsinogen
- Pepsinogen
- Chymotrypsinogen
- Amylase

75% - trypsinogen related

- SPINK1
  - Trypsinogen activation within the pancreas is inhibited by pancreatic secretory inhibitor (PSTI or SPINK1)
- PRSS1
  - Multiple isoforms of trypsinogen – missense mutation lead to premature activation within pancreas

Genetic

- CFTR mutations (In pancreatic sufficient)
- SPINK1 mutations – family history
- PRSS1 mutations – family history
Anatomical

- Pancreas divisum
- Anomalous junction of the biliary and pancreatic ducts
- Annular pancreas
- Ampullary obstruction
- Crohn’s disease

Chronic Pancreatitis

- May have a sentinel episode leading to pancreatic damage intrapancreatic activation of trypsinogen
- May have normal serum amylase/lipase
- May develop pain syndrome

Investigations

- CF
- Hyperlipidaemia
- Ca/ PTH
- Consider Genetic
- Autoimmune (IgG4)
- Exocrine pancreatic testing
- Image- complications/ Long common channel
You are volunteering with a group of medical students in a small African village. You are asked to examine an apathetic child who refuses to eat. Physical examination reveals thinning hair and thin skin. The child also has oedema of the face, hands, and legs.

A. kwashiorkor
B. marasmus
C. vitamin C deficiency
D. vitamin D deficiency
E. zinc deficiency

Of the following, the condition MOST LIKEY responsible for this child’s findings is

A. kwashiorkor

**Kwashiorkor**
- Insufficient intake of protein
- Soft, pitting, painless oedema, usually involving the feet and legs

**Marasmus**
- Severe calorie malnutrition in a child
- "Wizened old man"
- Hypothermia, bradycardia, and hypotension
Marasmic Kwashiorkor

- Protein-energy malnutrition
- Severe edema of kwashiorkor and cachexia of marasmus

Gastro-oesophageal reflux

- Regurgitation (at least 1 episode daily)
  - 50% in 0- to 3 months,
  - 67% at 4 months,
  - 80% spontaneously resolve at 12-18 months
  - 5% in 24 month-old infants
- Larger volume feeds
- Prone position

My baby spits up frequently and the omeprazole isn’t helping?

- A) It is abnormal for a child to have increasing spills at 4 months of age
- B) This child has GORD
- C) A pH probe should have been performed prior to initiation of therapy
- D) The dose of omeprazole is 0.8mg/kg/day
- E) Omeprazole is the treatment of choice for apnoea associated with GORD

- 140ml/kg/day
- 70kg adult
- = 10 Litre of milk a day
Gastro oesophageal reflux

GOR vs GORD

• GOR – passage of gastric contents into oesophagus
• GORDisease – dysfunction of the gastro-oesophageal reflux barrier resulting in abnormal frequency or duration of stomach contents refluxed into the distal oesophagus or higher and producing symptoms or complications of GOR
GORD

- Poor weight gain
- Excessive crying or irritability
- Feeding problems
- Respiratory problems, including:
  - wheezing
  - stridor
  - recurrent pneumonia
  - Apnea/ALTE

Dose of omeprazole

<table>
<thead>
<tr>
<th>Dose of Omeprazole</th>
<th>% of Patients</th>
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<tr>
<td>Healed with &lt; 0.7 mg/kg/day</td>
<td>44%</td>
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<tr>
<td>Healed with &lt; 1.4 mg/kg/day</td>
<td>72%</td>
</tr>
<tr>
<td>Healed with &lt; 3.5 mg/kg/day</td>
<td>95%</td>
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</table>

N = 65 children with erosive oesophagitis

Apnea/ALTE

- Cause and effect difficult to differentiate
- Differentiation between primary and secondary aspiration difficult
- Vagal stimulation in oesophagus may have a role
- Alkaline / neutral pH refluxate just as damaging as acid

24 hour pH Monitoring

- 50% of children with an abnormal pH probe will have an oesophagitis
- 50% of children with oesophagitis will have an abnormal pH probe
Normal?

24 hour Impedence Monitoring
- Burden of acid and non-acid reflux
  (most reflux is non-acid – particularly after meals)

Investigations
- Bed propping, smaller more frequent feeds, feed thickening, reassurance
- Trial of acid suppression
- 24h pH probe
  - Adequacy of acid suppression
- 24h pH/Impedance study
  - Adequacy of acid suppression
  - Symptom association
- Endoscopy
  - Diagnose Oesophagitis
  - Rule out Eosinophilic oesophagitis
- Barium swallow
  - Hiatal hernia
  - Gastric outlet obstruction/malrotation
- Manometry
  - Adequacy of motor function

Short gut
- Good prognosis
  - >25cm with ileocaecal valve
  - >40cm without ileocaecal valve
- Or put another way
  - <15cm die
  - >40cm live
Short gut syndrome - adaptation

- Estimated length of intestine at birth is 200-300cm small bowel
- Length doubles in the last trimester
- Increase in length max in 1st year of life
- Adaptation could take 3 yrs

Short gut syndrome - stomach

- Lack of duodenal feedback – gastric hypersecretion – treatment proton pump inhibition
- But PPI = increased risk of viral enteral pathogens

Short gut syndrome – role of small bowel

- Jejunum greatest nutrient absorption
- Ileum – B12 and bile acid absorption
- Bile salt depletion leads to fat and fat soluble vitamin malabsorption
- High rate of food allergen sensitivity

Short gut syndrome - colon

- Absence of ileocelecal valve increases risk of sepsis
- Colon important for water and electrolyte reabsorption
Renal stone !!!?

Short gut and the kidney
- Calcium binds to malabsorbed fat, allows an increased absorption of oxalate
- Increased gut losses of sodium / potassium – increased renal avidity for same.

Intestinal failure managements
- Multidisciplinary team
- Maximise enteral feeds
- Cycle IVN
- Omega 3
- Bacterial overgrowth
- Line care
- Consider surgery to maximise usable gut

Small Bowel Transplantation
- Impending or overt liver failure
  - ↑ bilirubin, ↑ liver enzymes, ↑ spleen, ↑ PT, ↑ INR, ↓ platelets varices, stomal bleeding, hepatic fibrosis, cirrhosis
- Thrombosis of central veins
  - ≥ two of subclavian, jugular, or femoral veins
- Frequent central line related sepsis
  - ≥ two episodes per year or ≥ one episode if fungemia, septic shock, or ARDS
- Frequent severe dehydration
Survival

<table>
<thead>
<tr>
<th>Home parenteral nutrition</th>
<th>1-yr</th>
<th>3-yr</th>
<th>5-yr</th>
<th>10-yr</th>
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<tbody>
<tr>
<td>Series of 40 patients excluding malignancy (1986-2003)</td>
<td>97%</td>
<td>82%</td>
<td>67%</td>
<td></td>
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<tr>
<td>Series of 258 patients with SBS and excluding malignancy (1994-2006)</td>
<td>94%</td>
<td>70%</td>
<td>52%</td>
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<tr>
<td>Patients with Crohn’s disease (CD) extracted from multiple series</td>
<td>82%</td>
<td></td>
<td></td>
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<tr>
<td>Series of 60 patients with CD (1979-2003)</td>
<td>87%</td>
<td></td>
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Intestinal transplantation

| Series of 413 patients (1994-2006) | 85%  | 61%  | 42%  |       |
| Series of 687 patients (1987-2009) | 77%  | 61%  | 51%  |       |
| Series of 67 patients with CD (1987-2009) | 79%  | 53%  | 43%  |       |

Chronic diarrhoea

Osmotic gap

- Osmotic gap
- Stool osmolarity = (Na+K) x 2
- Osmotic gap = plasma – stool

Osmotic gap

<table>
<thead>
<tr>
<th>mOsm/kg</th>
<th>Unmeasured osmoles</th>
<th>HCO₃⁻</th>
<th>Cl⁻</th>
<th>K⁺</th>
<th>Na⁺</th>
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</table>

Osmotic gap

\[
290 - 2 \times ([Na^+] + [K^+]) = 230
\]

Secretory diarrhea

Osmotic diarrhea

\[
290 - 2 \times ([Na^+] + [K^+]) = 10
\]
Osmotic diarrhoea

- The osmotic gap is > 50mOsml/kg
- High osmotic gap and stool pH <5 suggests a disaccharidase deficiency
- Can be caused by bacterial overgrowth
- Improves with fasting
- Occurs with exocrine pancreatic insufficiency

Osmotic diarrhoea

- Osmotic gap > 50
- Infectious enteritis, disaccharide deficiency, laxative abuse
- Impaired absorptive states;
  - Coeliac disease
  - pancreatic disease
  - short gut
  - inflammatory processes of the mucosal lining

Secretary diarrhoea

- Osmotic gap <50
- Continues despite fasting
- Causes
  - toxugenic E. coli
  - cholera
  - gut hormone tumours
  - intractable diarrhoea states (microvillous inclusion, tufting enteropathy, autoimmune enteropathy)

Eosinophilic oesophagitis
Eosinophilic oesophagitis
“the facts”

- Incidence 1:10000 / year (0-18 years)
- 1% of oesophagitis
- Atopy occurs in >80%
- Peripheral eosinophilia 50%
- Biopsy demonstrates >20 eosinophils/ hpf cf reflux 5 eosinophils/ hpf

Diagnosis

- Patients with symptoms: dysphagia, vomiting, heartburn, abdo pain, food impaction, FTT
- Failure to respond to PPI

Symptoms by age

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Fraction of Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feeding Disorder</td>
<td>13 %</td>
</tr>
<tr>
<td>Vomiting</td>
<td>26 %</td>
</tr>
<tr>
<td>Abdominal Pain</td>
<td>26 %</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>27 %</td>
</tr>
<tr>
<td>Food Impaction</td>
<td>7 %</td>
</tr>
</tbody>
</table>

Eosinophilic oesophagitis

- Systemic steroids effective but side effect profile unacceptable
- Enteral fluticasone effective
- Elimination diet effective
- Leucotriene receptor modifiers may not be effective
### Eosinophilic oesophagitis and role of allergy
- Amino acid based formula feeding effective, but unpalatable
- Elimination diets (wheat, cow/soy milk, nut, fish, egg) – may be effective
- Skin patch tests often positive

### By the way
- Swedish study 1000 upper endoscopies in asymptomatic adults.
- 1% had eosinophilic oesophagitis by histological criteria.
- Probable relapsing course
- Best treatment/ follow-up yet to be defined
- 10% will respond to high dose PPI

### Ingestions Causing Oesophagitis
- Acidic agents – taste bad, and are rare
- Alkaline agents – usually more significant because they are swallowed
- Observe those with no symptoms and offer clear fluids
  - Drooling, dysphagia, and abdominal pain
- Admit and scope those with clear history
- Do not induce emesis (further oesophageal damage)

### Foreign Body Ingestion
- Most can be diagnosed with chest x-rays (90% are radio-opaque)
- Coins in the coronal plane = oesophagus
- Coins in sagittal plane = trachea
- Endoscopy usually needed for removal
**Pill-Induced Oesophagitis**
- Local irritation due to pill adherence to oesophageal wall
- Most common pills
  - Tetracycline, Doxycycline
  - NSAIDs, aspirin
  - Potassium
- Pill **batteries** are big trouble
- One magnet is OK – two is trouble

**Helicobacter pylori**
**True or False?**
- Common cause of recurrent abdominal pain of childhood
- Transmission is oral to oral
- Infection is commonly acquired in childhood
- Is associated with iron deficiency
- Urea breath test is a useful test to determine eradication

**Magnets**
- One magnet is OK – two is trouble

**Helicobacter pylori**
- Affects 50% of the population
- 10% developed vs 60-80% undeveloped
- Associated with lower socioeconomic status, overcrowding and poor hygiene
- 90% of patients with DU and 70% with GU have H pylori infection
H. pylori

- Infection is usually in childhood
- Spontaneous clearance is uncommon
- Most cases are asymptomatic
- Diagnosis is endoscopic/histological

CLOtest

- Stool antigen
  - Stool antigen test
    - Inexpensive noninvasive
    - Polyclonal test: 93-96% sensitivity and specificity pretreatment, post treatment results variable, sensitivity as low as 63%
    - Newer monoclonal ab test more sensitive (98%), specific (99%), -ve predictive value (99%), but need more studies

So why not screen everyone?

- Yes
  - Causes ulcers and gastric cancer
- No
  - "Commensal role in preventing other autoimmune disorders"
  - Toxicity of treatment
  - Resistance.....
In the meantime

- Screen
  - Family history of gastric carcinoma
  - Refractory iron deficiency anaemia
  - Peptic ulcer disease