NMDC221 Session 4: Gastrointestinal & Alimentary Disease Part IV
Recommended Reading
Pg. 532-539; 541-546 (prescribed text).
Topic Summary

Gastrointestinal and Alimentary Disease: Part IV
Nutritional management and consideration of drug-nutrient interactions

- Inflammatory Bowel Disease
  - Crohn’s disease
  - Ulcerative colitis
- Coeliac disease
- Cholecystitis and cholelithiasis
- Pancreatitis
Inflammatory Bowel Disease
Inflammatory Bowel Disease

- Chronic disorders of the small and/or large intestine characterized by inflammatory changes in the intestinal tissue
- Linked to an immune inflammatory response due to microbial invasion, trauma, toxic agents or an autoimmune response.
- A highly inflammatory response, that leads to ongoing tissue destruction

(Kumar & Clark, 2005, pp.542-543)
Inflammatory Bowel Disease

There are two major types of Inflammatory Bowel Disease:

**Crohn’s Disease**
- May involve anywhere in the GIT but mostly affects (50-60% cases) distal ileum and colon. Crohn’s disease is a poorly understood inflammatory condition
- Affects segments (where as in UC, it is continuous)

**Ulcerative Colitis**
- Ulcerative colitis (UC) is a chronic inflammatory disease of the large intestine and rectum, which is relatively common but remains poorly understood.

(Kumar & Clark, 2005)
# Inflammatory Bowel disease

## Aetiology

<table>
<thead>
<tr>
<th>Genetic predisposition</th>
<th>Leads to -&gt; abnormal activation of the mucosal immune response; secondary systemic response. Further aggravated by dietary factors and stress.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unknown irritant</td>
<td>Vitamin D insufficiency</td>
</tr>
<tr>
<td>(Virus, bacteria,</td>
<td>Food allergies</td>
</tr>
<tr>
<td>autoimmune, vaccine)</td>
<td></td>
</tr>
</tbody>
</table>

## Pathophysiology

<table>
<thead>
<tr>
<th>Inflammatory response</th>
<th>Damage to the cells of the SI and LI -&gt; Malabsorption, ulceration, stricture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sx’s</td>
<td>diarrhoea, weight loss, poor growth, hyperhomocysteinemia; Partial GI obstructions</td>
</tr>
</tbody>
</table>

(Adapted from Mahar & Raymond, 2017, p. 543)
Inflammatory Bowel Disease

Therapeutic Objectives:-

1. Acute
   o Maintain hydration and adequate microbiome status
   o Manage inflammation
   o Include easily digested ‘non reactive’ foods
   o Consider allergenic/elimination diet
   o Modify immune response
   o Support nervous system function during physical and mental stress of an acute flare up.
IBD – Therapeutic objectives cont.

2. Maintenance
- Ensure adequate protein intake & balance calorific demands
- Encourage small, frequent, nutrient dense meals
- Allergenic free diet & / or an elimination diet
- Continue to support healthy immune response (Th1:Th2) and inflammatory responses
- Support nervous system
- Refer to counsellor if necessary

(Osiecki, 2006; Mahan & Escott-Stump, 2008)
# Inflammatory Bowel Disease

<table>
<thead>
<tr>
<th>Nutrient Deficiencies</th>
<th>Crohn’s Disease</th>
<th>Ulcerative Colitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin B12</td>
<td>Moderate</td>
<td>Nil</td>
</tr>
<tr>
<td>Iron</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>Folate</td>
<td>Severe</td>
<td>Moderate</td>
</tr>
<tr>
<td>Magnesium</td>
<td>Mild</td>
<td>Nil</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>Moderate</td>
<td>Nil</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>B-carotene</td>
<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>Mild</td>
<td>Nil</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>Zinc</td>
<td>Moderate</td>
<td>Moderate</td>
</tr>
<tr>
<td>Copper</td>
<td>Mild</td>
<td>Nil</td>
</tr>
<tr>
<td>Selenium</td>
<td>Mild</td>
<td>Mild</td>
</tr>
</tbody>
</table>

(Shils, Shike, Ross, Caballero, & Cousins, 2006, p 1213)
Inflammatory Bowel Disease

Beneficial nutrients.

1. Omega 3 fatty acids
   - Anti-inflammatory action with 2.7g/day of fish oil was found to double the rate of sustained remission in Crohn’s disease (Belluzzi et al, 1996)

2. Fish Oil, FOS, Vitamin E, C & Selenium
   - Supplementation was found to reduce arachidonic acid levels and significantly decreased the use of prednisolone by ulcerative colitis patients compared with placebo (Seidner et al., 2005)
Inflammatory Bowel Disease

3. Vitamin D
   - IBD patients are more likely to be deficient in vitamin D. Activated vitamin D ($1,25(\text{OH})_2\text{D}_3$) has been shown to reduce Th1 driven immune imbalances, including IBD.
     (Cantorna, 2006, p. 61)

4. Probiotics
   - *Lactobacillus rhamnosus* and *L. reuteri* have shown efficacy with IBD. Found to improve the numbers of beneficial T-reg cells, decreasing IL-12 levels and reducing inflammation.
     (Lorea et al., 2007, p. 477)
Inflammatory Bowel Disease

5. Quercetin
   - In vitro evidence supports quercetin’s ability to reduce macrophage induced inflammation, and therefore can play a role in ameliorating IBD
     (Comalada et al., 2005, p. 584)

6. Epigallocatechin Gallate (EGCG)
   - EGCG was able to reduce the extent and severity of damage in an animal model. The proposed mechanism is that ECGC provides an anti-inflammatory effect
     (Mochizuki & Hasegawa, 2005, p. 364)
### IBD – Main Nutrient Considerations

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Omega 3</td>
<td>1000-6000mg</td>
<td>Anti-inflammatory (reduce the production of IL-1 &amp; TNF-α and reduce cellular adherence. Balance Th1 dominance)</td>
</tr>
<tr>
<td>Prebiotics - Inulin, FOS, pectin</td>
<td>1-4gm</td>
<td>Support the growth of bifidobacterium. Fuel source for bacteria. Provide the opportunity to stimulate the adaptive immune response in a direction regulating T-helper cell type (TH1) and TH2 related immunity.</td>
</tr>
<tr>
<td>Probiotics</td>
<td>10-40 billion</td>
<td>Prevent gut dysbiosis. Balance Th1 dominance</td>
</tr>
<tr>
<td>Glutamine</td>
<td>500-3000mg</td>
<td>Tissue repair Anti-inflammatory, Immune support for GALT, Restores gut wall integrity &amp; normal intestinal flora colonization.</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>1000-5000iu</td>
<td>Th1 Immune regulation</td>
</tr>
</tbody>
</table>
### IBD – Nutrients Cont.

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zinc</td>
<td>10-40mg day</td>
<td>Diarrhoea exacerbates losses. Maintains endothelial cell integrity &amp; reduces cytokine induced damage</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>500-6,000mg</td>
<td>Aids tissue repair and collagen Synthesis. Regulates immune function (increasing macrophage activity, lymphocyte production &amp; antibodies IgA, IgG, IgM). Modulates prostaglandin synthesis.</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>100-1,000IU</td>
<td>Malabsorption = deficiency. Balance Th1 dominance. Prevents oxidation of unsaturated fatty acids contained in phospholipids of the cell membrane</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>1000-5000iu</td>
<td>Malabsorption leading to deficiency. Structure and function of epithelial cells, antioxidant</td>
</tr>
</tbody>
</table>
**IBD – Nutrients Cont.**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>N-acetyl-glucosamine</td>
<td>600-3000mg</td>
<td>Defective glycosaminoglycan exacerbating ‘leaky gut’ found in Crohn’s. Precursor to the ground substance/gap junctions for epithelial cells</td>
</tr>
<tr>
<td>Magnesium</td>
<td>300-1000mg</td>
<td>Malabsorption leading to deficiency. Support nervous activity &amp; reduces pain associated with the inflammation</td>
</tr>
<tr>
<td>Iron</td>
<td>15-80mg</td>
<td>Replenish losses if appropriate.</td>
</tr>
</tbody>
</table>

Inflammatory Bowel Disease

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<th>Drug</th>
<th>Action</th>
<th>Side Effects</th>
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<tr>
<td>5-Aminosalicylic Acid: Sulfazaline</td>
<td>Pro-drug that has a local prostaglandin inhibitor action in the colon.</td>
<td>Nausea, abdominal pain, diarrhoea, headache, rash</td>
<td><strong>Folate</strong>: competitive inhibitor of folate transport, absorption and metabolism. Monitor for folate deficiency signs</td>
</tr>
</tbody>
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(Harkness & Bratman, 2003; Bullock et al. 2007; Bryant & Knights, 2007; Kumar & Clark, 2009)
Inflammatory Bowel Disease

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<td><strong>Corticosteroids:</strong></td>
<td>Anti-inflammatory with action of endogenous cortisol.</td>
<td>Sodium retention (potassium loss) = increased blood pressure &amp; fluid retention</td>
<td>Calcium: drug reduces intestinal absorption. Monitor for calcium deficiency</td>
</tr>
<tr>
<td>Prednisolone,</td>
<td></td>
<td>Protein loss (thin skin, hair, reduced wound healing &amp; immune dysregulation)</td>
<td>Chromium: concurrent use may reduce corticosteroid induced diabetes</td>
</tr>
<tr>
<td>Budesonide</td>
<td></td>
<td>Adrenal atrophy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bone thinning</td>
<td></td>
</tr>
</tbody>
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(Harkness & Bratman, 2003; Bullock et al., 2007; Bryant & Knights, 2007; Kumar & Clark, 2009)
## Inflammatory Bowel Disease

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</thead>
<tbody>
<tr>
<td>Cyto-toxic Immuno-suppressant: Azathioprine, Methotrexate, Cyclosporine</td>
<td>Inhibits cell replication &amp; is utilized in this instance to reduce immune system responsiveness</td>
<td>Bone marrow suppression &amp; immuno-suppression Hair loss GIT complaints – nausea, vomiting, ulcers, bleeding</td>
<td><strong>Immuno-stimulants</strong> (Garlic) may reduce drug effectiveness <strong>Peppermint oil &amp; Quercetin:</strong> increase bioavailability of Cyclosporine Folate: Methotrexate is a folate antagonist. <strong>Folate, Vitamin A &amp; vitamin E:</strong> Concurrent supplementation reduces methotrexate side effects</td>
</tr>
</tbody>
</table>

(Harkness & Bratman, 2003; Sarris & Wardle, 2010; Bryant & Knights, 2011)
# Inflammatory Bowel Disease

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<th>Interactions</th>
</tr>
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<tbody>
<tr>
<td>Anti-diarrhoeals:</td>
<td>Used to reduce diarrhoea to sustain hydration &amp; nutrient losses.</td>
<td>Can cause constipation</td>
<td></td>
</tr>
<tr>
<td>Diphenoxylate, Loperamide</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Broad Spectrum Antibiotics:</td>
<td>Used when infection of abscesses and fistulas present</td>
<td></td>
<td>Probiotics: concurrent usage may minimize drug side effects</td>
</tr>
<tr>
<td>Metronidazole</td>
<td></td>
<td></td>
<td>Vitamin B complex: may reduce the absorption of some antibiotics. Separate the dosage by 2 hours. Vitamin B1 &amp; B12 have reduced colonic bacterial</td>
</tr>
</tbody>
</table>

(Kumar & Clark, 2009; Bryant & Knights, 2010)
Coeliac Disease
Coeliac Disease

- Hyperactive immune responses due to incompatibility with antigen (gluten) stimulus leading to chronic intestinal malabsorption
- Can occur anytime from infancy to adulthood, but there’s been an increased prevalence of the condition over the past decade (Mahan and Raymond 2017).
- Peak diagnosis is in the 4th and 6th decade often following GI surgery, pregnancy, stress, viral infection (Mahan and Raymond 2017)

Characterized by:-
- Diarrhoea & malabsorption symptoms, fatigue, weight loss (failure to thrive in children) & anemia

Concurrent conditions include:
- Intestinal dysbiosis diseases, Autoimmune disorders, dermatitis herpetiformis, eczema
  
  (Kumar & Clark, 2009; Mahan & Escott-Stump, 2008)
Pathophysiology

Characterised by:-

1. Genetic susceptibility

2. Exposure to gluten - early exposure of the immature immune system to gliadin is a factor for CD - skewing the immune system toward a T helper (Th) 1 T-cell response (Ventura G, Magazzu, Greco 1999)

3. Environmental ‘trigger’

4. Autoimmune history - high prevalence of various autoimmune disorders, especially type I diabetes, dermatitis herpetiformis, autoimmune thyroiditis, collagen diseases, autoimmune alopecia, and autoimmune hepatitis (Ventura, Magazzu, Greco 1999)

Gluten refers to a specific peptide fractions of proteins (prolamines) found in wheat (glutenin and gliadin), rye (secalin), barley (hordein) and oats (avenin). These peptides are more resistant to complete GI breakdown and may reach SI intact triggering an inflammatory reaction and immune response.

(Mahan and Raymond, 2017, p536-8).
Coeliac Disease

Nutritional Treatment

- Strict avoidance of gluten containing foods is the cornerstone of effective management of coeliac disease.
- It is important to remain sensitive to the patients needs and concerns when initially removing gluten.
- Caution needs to be used with processed foods, as many ingredients are wheat derived and can contain gluten.
- Also, many foods are contaminated and not suitable even though in their ‘natural’ form are suitable.

(Shils et al. 2006, p. 1224)
Coeliac Disease

Nutritional Objectives:

1. Dietary management
   o Identify and remove all Gluten containing foods and implement GF diet (Mahan and Raymond 2017, pg 535); educate on ‘cross contamination.’

2. Correct underlying deficiencies & maintain nutritional status

3. Treat any presenting symptoms & associated conditions to further support
   o Hydration & appropriate bowel flora
   o Immune and inflammatory responses
   o Nervous system and stress management
   o Mucous membrane integrity/health

   (Osiecki, 2006; Mahan & Escott-Stump, 2008)
<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Quantity</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glutamine</td>
<td>500-3,000mg</td>
<td>Glutamine is the main energy source for intestinal cells, and has trophic effects that enhance healing of damaged mucosa (Gropper, Smith &amp; Groff, 2005, p. 185)</td>
</tr>
<tr>
<td>Zinc</td>
<td>25-100mg</td>
<td>Important for the maintenance of the intestinal barrier and reduction of oxidative stress (Ziegler et al, 2003)</td>
</tr>
<tr>
<td>N-Acetyl-D-Glucosamine</td>
<td>1000-3,000mg</td>
<td>Required for the synthesis of mucosal glycoproteins; these are important for protection of the GIT (Ackersand, 1997)</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>5,000-10,000iu</td>
<td>Malabsorption leading to deficiency. Structure and function of epithelial cells, antioxidant</td>
</tr>
<tr>
<td>Supplement</td>
<td>Dosage Range</td>
<td>Description</td>
</tr>
<tr>
<td>---------------</td>
<td>------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Probiotics</td>
<td>10-40 billion org/day</td>
<td>Prevent gut dysbiosis. Balance Th1 dominance</td>
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<tr>
<td>Omega 3</td>
<td>500-6000mg</td>
<td>Anti-inflammatory (reduce the production of IL-1 &amp; TNF-α and reduce cellular adherence. Balance Th1 dominance</td>
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<tr>
<td>Magnesium</td>
<td>300-1000mg</td>
<td>Malabsorption leading to deficiency. Support nervous activity &amp; reduces pain associated with the inflammation</td>
</tr>
<tr>
<td>B Group Vitamins</td>
<td>50mg of all major B’s (exception B12)</td>
<td>Nervous system and to support healthy production of HCl.</td>
</tr>
</tbody>
</table>

(Schlenker & Long, 2007; Mahan et al. 2008, p. 694; Osiecki, 2008; Kumar & Clark, 2009)
### CD – Other Nutrients to Consider

<table>
<thead>
<tr>
<th>Vitamin C</th>
<th>500-5,000mg</th>
<th>Collagen Synthesis. Regulates immune function (increasing macrophage activity, lymphocyte production &amp; antibodies - IgA, IgG, IgM). Modulates prostaglandin synthesis. Heals &amp; rebuilds damaged tissue.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D</td>
<td>1000-5000iu</td>
<td>Malabsorption = deficiency. Th1:Th2</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>100-1000iu</td>
<td>Malabsorption = deficiency. Th1:Th2 Antioxidant for phospholipids of the cell membrane</td>
</tr>
</tbody>
</table>
Dietary & Nutritional Prescription

**Gluten-free Diets**

- Essential for a coeliac’s to follow.
- Poor source of folate, iron, calcium and fibre.
- There is a deficiency of grains which affects the amount of B vitamins consumed particularly thiamine, riboflavin, niacin.
- The addition of three serves of gluten-free alternative grains improves fibre, thiamine, riboflavin, niacin, folate and iron.

(Lee, Ng, Dave, Ciaccio & Green, 2009)
Dietary & Nutritional Prescription

Gluten-free Diets – *Considerations*

- Often low in fructo-oligosaccharides (FOS); specific long-chained carbohydrates that serve as a fuel source for bowel microflora.

Low intake of FOS

↓

Lack of reduced metabolic activity of microflora

↓

Reduced production of SCFAs including butyrate

↓

Lack of energy for colonocytes

↓

Increased pH of colon

↓

Increased risk of colon cancer

(Muir et al. 2013)
Dietary & Nutritional Prescription

Gluten-free Diets – Considerations

- Those adopting a gluten free diet without a specific medical need may negatively be affecting their capacity to produce butyrate and as such, may be increasing their risk to colon cancer.

- Good sources of Gluten-Free FOS
  - Jerusalem artichokes
  - Legumes
  - Guar gum

(Muir et al. 2013)
Dietary & Nutritional Prescription

COX & LOX Inhibitors

- Anti-oxidant agents can alter the production of inflammatory agents that exacerbate and sustain the condition.
- Omega 3 reduce the production of IL-1 & TNF-α and reduce cellular adherence (Coulson et.al. 2001; Mahan & Escott-Stump, 2008)
- High volatile oil agents (garlic, ginger, turmeric) and bioflavonoid agents (pineapple, pawpaw) and cocoa can aid in reducing inflammation
  (Kumakura, 1988; Helmutt et.al. 2005; Osiecki, 2008; Tripathi et.al. 2008)
Dietary & Nutritional Prescription

Gluten Free Diet: Specific Exclusions

- GLUTEN Most ‘normal’ bread, pasta, cereals and most processed foods will all contain gluten.
  - Wheat (and all wheat-derived products)
    - Can be listed a number of ways: durum flour, kamut, semolina, bulgur, cous cous.
  - Rye
  - Barley
  - Oats
  - Triticale and spelt
  - Malt
  - Beer

Mayo Clinic, 2011
Dietary & Nutritional Prescription

Gluten Free Diet: Specific Inclusions

- All fruits and vegetables are naturally gluten free.
- Unprocessed meat, poultry and fish
  - Need to be mindful of coatings, sauces and flavourings that may contain flour or gluten-containing ingredients.
  - Deli meats (e.g.: ham) often contain gluten.
- Beans, pulses, nuts and seeds
- Rice, Corn, Soy
- Quinoa, buckwheat, amaranth, millet
- Arrowroot, Tapioca
- Eggs
- Most dairy products (check label of yoghurts as gluten may be added to thicken)

Mayo Clinic, 2011
Dietary & Nutritional Prescription

Taking a client through a gluten-free diet

- Focus on the things they **CAN** have
- Provide alternatives based on what they generally like to eat
  - E.g. Sandwich for lunch → corn thins with salad and a protein topping
    - Pasta at dinner time → gluten-free pasta or rice noodles
    - Soy sauce → tamari
- Just because something is gluten-free doesn’t mean it’s healthy!
  - Often high in sugar, fat and additives.
Dietary & Nutritional Prescription

- **Soft Diet (Oregon Surgical Specialists)**
  - Focuses on foods that are easy to swallow and digest
    - Soups, stews and smoothies
    - Consider juicing
  - No spicy foods
  - Avoid ‘gas producing’ foods
  - Reduced fibre intake

Oregon surgical specialists, 2012
Dietary & Nutritional Prescription

Optional:
Lactose Free Diet

- Specifically avoid:
  - Cows products (milk, cheese, yoghurt, butter)
  - Goats milks products
  - Sheep milk products

- As a substitute:
  - Nut milks (almond milk)
  - Nut or seed butters or spreads (olive oil spread)
  - Soy products
  - Grain milks (rice or quinoa milk)
# Coeliac Disease

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<td>Cyto-toxic Immuno-suppressant</td>
<td>Supress immune cell replication to reduce inflammatory prostaglandin levels</td>
<td>Bone marrow &amp; immunosuppression Hair loss, GIT symptoms, ulcers</td>
<td>Immuno-stimulants (garlic) may reduce drug effectiveness</td>
</tr>
<tr>
<td>Cyto-toxic Immuno-suppressant: Methotrexate</td>
<td>Folate antagonist to reduce prostaglandin production</td>
<td>As above</td>
<td>Folate, Vitamin A &amp; E: concurrent supplementation reduces drug side effects</td>
</tr>
<tr>
<td>Cyto-toxic Immuno-suppressant: Cyclosporine</td>
<td>Supress the production of pro-inflammatory prostaglandins</td>
<td>As above</td>
<td>Peppermint oil &amp; Quercetin: increase bioavailability of the drug</td>
</tr>
</tbody>
</table>

(Harkness & Bratman, 2003 Bullock et al. 2007; Bryant & Knights, 2007; Kumar & Clark, 2009; Sarris & Wardle, 2010)
# Coeliac Disease

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<td><strong>Corticosteroid:</strong> Prednisolone, Budesonide</td>
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<td><strong>Calcium:</strong> drug reduces intestinal absorption. Monitor for calcium deficiency <strong>Chromium:</strong> concurrent use may reduce corticosteroid induced diabetes</td>
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(Harkness & Bratman, 2003; Bullock et al. 2007; Bryant & Knights, 2007; Kumar & Clark, 2009)
Class Discussion
Case Study

Female, 42 years old

Presenting Symptoms
- Lower abdominal pain
- Diarrhoea, undigested food in stools, pale, float. Mucus in the stools
- Bloating & cramping straight after eating certain food (pasta, pizza, bread) which will last for hours
- Fatigue & an inability to maintain a good weight.
- Initially presented after a severe case of food poisoning 5 years prior. Stool analysis was taken
- Endoscopy revealed coeliac
Case Study

Medications / Supplements
- Iron plus C (bought over the counter at the Chemist)

Pathology Results – from three weeks prior to this consultation

<table>
<thead>
<tr>
<th>Factor Tested</th>
<th>Results</th>
<th>Range (Adult Female)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum iron</td>
<td>32 ug/dL</td>
<td>60-160ug/dL</td>
</tr>
<tr>
<td>Total Iron Binding Capacity</td>
<td>170ug/dL</td>
<td>240-450ug/dL</td>
</tr>
<tr>
<td>Serum ferritin</td>
<td>12 ug/dL</td>
<td>18-200ug/dL</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>10 g/dL</td>
<td>12-16 g/dL</td>
</tr>
<tr>
<td>Red Blood Cell Count</td>
<td>3 million/uL</td>
<td>4.2-6.9 million/uL</td>
</tr>
<tr>
<td>Transferrin</td>
<td>176 mg/dL</td>
<td>191-365 mg/dL</td>
</tr>
</tbody>
</table>
Case Study

Family History
- Mother: IBS, eczema
- Father: Psoriasis, hypertension, Coeliac
- Brother: asthma, IBS

Past Medical History
- Infant: vaginal delivery, bottle fed. Weaned onto solid foods at four months. Lots of colic
- Adolescence: wisdom teeth removed at 19
Case Study

System Presentation

- **Nervous system & Endocrine:** constant fatigue (3/10). Will feel faint if she misses a meal
- **Immune:** wounds heal slowly (1-2 weeks), colds 4-6 per year that will last for two-three weeks and require a couple of doses of antibiotics. Chest infections commonly present as part of the cold.
- **Urinary:** past history of UTI’s (1x year) blood will present in the urine, frequency & urgency.
- **Circulatory:** low blood pressure, postural hypotension. Chronic iron deficient anaemia
Case Study

**Musculo-skeletal**: fingers sometimes swell, get hot and itchy and ache. This lasts for 1-2 weeks and then disappears

**Physical Examination Results**
- **Nails**: spoon shaped nails, pale, poor capillary return
- **Skin**: pale, dry
- **Height**: 165cm  **Weight**: 47kg  **Waist: Hip** 0.70 (Female>0.85)
- **Tongue**: central crack, yellow coating, red spots on tip, quiver
- **Eyes**: brown, yellow lymphatic rosary, white wash, stress rings throughout
- **Zinc tally**: no taste
## Case Study

<table>
<thead>
<tr>
<th>Time</th>
<th>Daily Dietary Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 am</td>
<td>Scrambled eggs or sardines on ‘gluten-free’ toast</td>
</tr>
<tr>
<td></td>
<td>Cup green tea</td>
</tr>
<tr>
<td>11am</td>
<td>Handful nuts (almonds, cashews, macadamia)</td>
</tr>
<tr>
<td></td>
<td>Green tea</td>
</tr>
<tr>
<td>1pm</td>
<td>Grilled salmon &amp; salad: cos lettuce, tomato, onion, carrot, sprouts</td>
</tr>
<tr>
<td></td>
<td>Banana</td>
</tr>
<tr>
<td>3pm</td>
<td>Cheese &amp; ‘gluten-free’ crackers</td>
</tr>
<tr>
<td>6 pm</td>
<td>Casserole: beef, gravy, carrot, potatoes, peas, beans, onion, mushrooms</td>
</tr>
<tr>
<td></td>
<td>Salad: lettuce, tomato, onion, olives feta, salad dressing</td>
</tr>
<tr>
<td>8pm</td>
<td>4 squares of milk chocolate</td>
</tr>
</tbody>
</table>
Discussion

- Devise a holistic treatment plan for this client looking at the contributing factors for this case. Include therapeutic aims for short and long term.

- Consider individual nutrient dosage with clinical decisions, integrative management of each condition giving mechanisms of actions relevant for nutrient-drug interactions.

- Use findings for critical discussion.
Cholecystitis
Cholecystitis

- Inflammation of the gallbladder with the most common cause being gallstone blockage of the bile duct or critically ill patients with decreased gallbladder emptying.
  
  (Mahan & Escott-Stump, 2008, p.729)

- Pain (right upper quadrant, epigastrium, referred to right shoulder tip or interscapular region), fever & leukocytosis present.

  (Walker, Colledge, Ralston, & Penman, 2014)
Cholecystitis

Investigations
- Plain radiographs of the abdomen and chest
- Ultrasonography
- Plasma amylase
- Full blood count

(Walker, Colledge, Ralston, & Penman, 2014)
Cholecystitis

Nutritional Considerations

- During an acute attack, food restriction should be practiced.
- After the attack the patient should consume a low fat diet for a period, with 30-45g fat per day.
- In chronic cases, a long term low fat diet should be followed. Fat should constitute 25-30% of the total daily calories.

(Mahan & Escott-Stump, 2008, p. 729)
Cholecystitis Treatment

Therapeutic Actions
Prevent oxidative stress
- Oxidative stress was found to be an aggravating pathological factor in chronic cholecystitis.
- Beneficial nutrients included vitamins C, E and beta-carotene.

(Zhou et al. 2000)
# Cholecystitis

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Therapeutic Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>5,000-10,000iu</td>
<td>Deficiency may present due to malabsorption. Check for deficiency signs and rectify.</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>1000-5000iu</td>
<td></td>
</tr>
<tr>
<td>Vitamin E</td>
<td>100-1000iu</td>
<td></td>
</tr>
<tr>
<td>Glycine</td>
<td>3,000-30,000mg</td>
<td>Lipotropic factors, liver support for glutathione production</td>
</tr>
<tr>
<td>Cysteine</td>
<td>200-500mg</td>
<td></td>
</tr>
<tr>
<td>Fibre</td>
<td>1-5g/day</td>
<td>Reduces cholesterol saturation</td>
</tr>
<tr>
<td>Ginger</td>
<td>500mg dried root 2-4 times/day</td>
<td>Anti-inflammatory</td>
</tr>
<tr>
<td>Turmeric</td>
<td>100-1500mg/day</td>
<td>Anti-inflammatory</td>
</tr>
<tr>
<td>Vitamin C (Ascorbic Acid)</td>
<td>500-5,000mg</td>
<td>Biliary acid regulation</td>
</tr>
</tbody>
</table>
Cholelithiasis
Cholelithiasis

- The presence of one or more calculi (gallstones) in the gallbladder.
- Bile with excessive cholesterol levels, a deficiency of other substances in bile (bile acids and lecithin), or a combination of these factors lead to calculi formation.
- Calculi are primarily hardened cholesterol.
- Most common symptom is biliary colic. May be asymptomatic.
- Note: Gallstones do not cause dyspepsia,
- Will have fatty food intolerance, steatorrhoea.

(Kumar & Clark, 2008)
Cholelithiasis

Investigations

- Liver function tests
- Ultrasonography
- Gastrointestinal endoscopy
- Barium examination of the small intestine
- Pancreatic function tests,
  Cholescintigraphy
- Liver biopsy.

(Walker, Colledge, Ralston, & Penman, 2014)
Cholelithiasis

Choledocholithiasis

- When calculi slip into the bile duct causing obstruction, pain and cramping.

- Bile flow into the duodenum can be obstructed. This leads to the development of cholecystitis

- Cholelithiasis and choledocholithiasis treatment is the same.

(Mahan & Escott-Stump, 2008, p. 729)
Cholelithiasis

Nutritional considerations
Increase dietary fibre
- Reduces the absorption of deoxycholic acid (compound that reduces the solubility of cholesterol in bile)
Identify food allergens and remove
- There is a link between food allergies and gallstones, an elimination diet may be warranted
Remove foods that cause symptoms

(Pizzorno & Murray, 2006, p. 1690-1693)
Cholelithiasis

Nutritional considerations
Reduce refined carbohydrates
- Diets high in refined foods and fat, and low in fibre lead to lower bile acid concentration in the gallbladder. Mono- and disaccharides are also linked to increased risk of gallstones

Reduce animal proteins
- Vegetarian diets have been found to be protective against gallstones

(Pizzorno & Murray, 2006, p. 1690-1693)
Cholelithiasis

Nutritional considerations
Reduce excessive legume consumption
  o Saponins in legumes increases biliary cholesterol saturation
Include buckwheat
  o Buckwheat has been found to be highly protective against stones, possibly via increasing bile acid synthesis & increasing excretion of steroidal compounds
Coffee
  o Coffee consumption has been shown to be protective against gallstone formation

(Pizzorno & Murray, 2006, p. 1690-1693)
Cholelithiasis

Therapeutic Actions

- Lecithin (100mg- 10g/day)
  - Lecithin has a direct effect on improving cholesterol solubilisation
- Vitamin C (500-4,000mg day and E 100-1000iu day)
  - Deficiencies of these nutrients have caused gallstones in animal models
- Fish Oils (3,000-6,000mg day)
  - Trials with fish oil supplementation and fish oil rich diets have shown to be protective against gallstones

(Pizzorno & Murray, 2006, p. 1690-1693)
Cholelithiasis

**Therapeutic Actions**

- Lipotrophic compounds help to decrease and aid the removal of fat in the liver. Traditionally these have been useful in decreasing stones
  - Choline 1g/day
  - Methionine 1g/day
  - Betaine
  - Folic acid 400-800mcg/day
  - Vitamin B12

(Pizzorno & Murray, 2006, p. 1690-1693)
Cholelithiasis

Post-Cholecystectomy Syndrome

- Dyspeptic symptoms following cholecystectomy occur in about 30% of patients depending on dietary fat intake
- Characterized by complaints of right upper quadrant abdominal pain, flatulence, fatty food intolerance and occasionally jaundice and cholangitis

(Walker, Colledge, Ralston, & Penman, 2014)
Cholelithiasis

Therapeutic Actions
Magnesium

- A small study found patients with Post-Cholecystectomy Syndrome to be magnesium deficient.
- After supplementation with magnesium nearly all cases of PCS were resolved (50/52)
- The dose of magnesium given was 300-600mg/day, and the supplement included calcium (185mg), B1 (60-100mg) and B6 (250-750mg)

(Porr, Szantay & Rusu, 2004)
# Cholelithiasis

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Side Effects</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bile acids:</strong> Ursodeoxycholic acid</td>
<td>Dissolve cholesterol derived bile stones. Only for small stones and long-term (up to 2 years).</td>
<td>Diarrhoea at high doses &amp; hepatotoxicity presents.</td>
<td></td>
</tr>
<tr>
<td><strong>Acid Suppressors:</strong> HMG Co-A Reducatase Inhibitors (simvastatin)</td>
<td>Reduces cholesterol content of bile and blood &amp; the concentration of bile salts. Combined use with Ursodeoxycholic acid to try to limit re-occurrences.</td>
<td>Carnitine &amp; Vitamin B3: may aid in lowering serum LDL levels Coenzyme Q10: depleted by drug. Vitamin A &amp; Vitamin D: levels with statin use</td>
<td></td>
</tr>
</tbody>
</table>

(Kumar & Clark, 2005; Bullock et al. 2007; Braun & Cohen, 2010)
Pancreatitis
Pancreatitis

Acute Pancreatitis
- Temporary condition and can be caused by the following:
  - Gallstones
  - Excessive alcohol consumption
  - High blood triglycerides
  - Abdominal injury
  - Infections
  - Medications and poisons

Chronic Pancreatitis
- Slow, silent process that gradually destroys the pancreas and is most often caused by excessive alcohol consumption (60-80% of cases).
  
(Kumar & Clark, 2005)
Pancreatitis

Concurrent Conditions

- Severe pancreatic insufficiency occurs in cystic fibrosis, chronic pancreatitis, and surgeries of the gastrointestinal system in which portions of the stomach or pancreas are removed.

- Certain gastrointestinal diseases, such as stomach ulcers, celiac disease, Crohn’s disease and autoimmune disorders, such as SLE, may contribute to the development of pancreatic insufficiency

(Kumar & Clark, 2005)
Pancreatitis

Therapeutic Actions
Vitamin B12
  o Deficiency due to malabsorption is common
Avoid Alcohol
  o Studies indicate the refraining from alcohol can decrease or dissipate all symptoms
Macronutrient Requirements
  o Protein 1-1.5g/kg/day
  o Fat 30%
  o Carbohydrate 40-60%

(Shils et al. 2006, p. 1232)
Pancreatitis

Therapeutic Actions

Antioxidants

○ A combination of antioxidants has been found to reduce pain & inflammation in both acute & chronic pancreatitis

  • Selenium 200-600mcg/day*
  • Methionine 2g/day
  • Vitamin A 9000IU/day
  • C 500mg/day
  • E 270IU/day

*Note: selenium dose in Australia is limited to 200mcg/day

(Shils et al. 2006, p. 1232)
# Pancreatitis

<table>
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<tr>
<th>Drug</th>
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(Kumar & Clark, 2007; Braun & Cohen, 2010)
## Pancreatitis

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<th>Side Effects</th>
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</tr>
</thead>
<tbody>
<tr>
<td>NSAIDs</td>
<td>Inhibit synthesis &amp; release of prostaglandin via COX enzymes. Inhibits platelet aggregation.</td>
<td>GIT symptoms, gastritis, skin reactions, may precipitate asthma attacks</td>
<td>Ginger, Turmeric, Glucosamine, Chondroitin, Glutamine, Fish oil, Vitamin E: Additive anti-inflammatory</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Glutamine, Capsaicin, Colostrum: reduced GIT irritation or ulceration</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Policosanol, grapeseed: enhanced antiplatelet activity</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Iron, Potassium, Zinc, Glutathione, Vitamin A, Vitamin C, Tryptophan: depleted by drug</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Calcium, Folic acid: alter drug absorption</td>
</tr>
</tbody>
</table>

(Braun & Cohen, 2010; Sarris & Wardle, 2010)
## Pancreatitis

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</thead>
<tbody>
<tr>
<td><strong>Tricyclic Anti-depressant: Amitriptyline</strong></td>
<td>Noradrenaline reuptake inhibitors, Amitriptyline also inhibits the reuptake of serotonin. Can be utilized to minimize analgesic drug use &amp; manage pain</td>
<td>Sedation, Impaired thinking, concentration &amp; memory, Blurred vision Orthostatic hypotension, Tachycardia Weight gain, Constipation</td>
<td><strong>SAMe, Tyrosine, Tryptophan, %-%-HTP:</strong> can increase serotonin production leading to serotonin syndrome. Don’t use <strong>Zinc:</strong> combined use may increase the efficacy of TCA’s <strong>Coenzyme Q10 &amp; Vitamin B2:</strong> TCA’s deplete levels <strong>B vitamins:</strong> increase drug efficacy <strong>Vitamin B2, Co-Q10:</strong> depleted by drug <strong>Alcohol:</strong> increases drowsiness <strong>Cruciferous Vegetables:</strong> increase phase II liver clearance and reduce drug levels</td>
</tr>
</tbody>
</table>

Pantuck et al, 1984; Threlkeld, 1990; Bryant & Knights, 2007; Braun & Cohen, 2010
# Pancreatitis

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<tr>
<th>Drug</th>
<th>Action</th>
<th>Side Effects</th>
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</thead>
<tbody>
<tr>
<td>Antibiotics</td>
<td>Either Bacteriocidal: cause cell death and lysis</td>
<td>GIT symptoms, dizziness, rash, oral or vaginal thrush</td>
<td><strong>Probiotics</strong>: reduce side effects of the drug</td>
</tr>
<tr>
<td></td>
<td>Bacteriostatic: prevents replication of bacteria</td>
<td></td>
<td><strong>Vitamin B</strong>: taken at same time reduces absorption &amp; bioavailability of some antibiotics. Separate dosages by two hours.</td>
</tr>
<tr>
<td></td>
<td>Antibiotics can be prescribed if there is necrosis present or as a prophylaxis</td>
<td></td>
<td><strong>Vitamins B1 &amp; B12</strong>: destroys bacteria required for production</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Calcium, Iron, Magnesium, Zinc</strong>: taken at the same time block absorption with quinolone &amp; tetracycline antibiotics. Separate by 2 hours.</td>
</tr>
</tbody>
</table>

(Bryant & Knights, 2007; Bullock et al, 2007; Braun & Cohen, 2010)
# Pancreatitis

<table>
<thead>
<tr>
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<th>Side Effects</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pancreatic enzymes:</strong></td>
<td>Used to aid with digestion and minimize pancreatic enzyme output which can minimize pain</td>
<td>Can cause irritation and gastritis if not enteric coated</td>
<td><strong>Folate</strong>: absorption is reduced with concurrent use so separate doses by 2-3 hours</td>
</tr>
<tr>
<td>Pancreatin, Pancrelipase</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>H2-receptor antagonists</strong></td>
<td>Blocks H2 receptors on parietal cells Combined with pancreatic enzymes reduces the effects of extra acid production.</td>
<td>Diarrhoea, nausea, constipation, headache, dizziness, skin rash</td>
<td><strong>Folate, Iron, Vitamin B12</strong>: Reduced absorption. Supplement with long term use</td>
</tr>
</tbody>
</table>

(Bryant et al 2007, p541; Bullock et al. 2007; Kumar & Clark, 2005; Braun & Cohen, 2010)
References


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