Treatment of IBS vs IBD

Lecture 5

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Irritable bowel syndrome (IBS)

A very common chronic functional idiopathic GIT syndrome presenting with abdominal pain, altered or fluctuating bowel frequency & stool consistency, abdominal gas distension and varying degrees of anxiety or depression.

Often worsen after meals, with flare up for several days.

Associated manifestations may include mild ↑mucus, defecation urgency, frequency of micturition & irregular menstruation.

Also headache & fatigue.

Diagnosed by exclusion.

DD: IBD & cancer colon.

Etiology & associated mechanisms

Genetic & environmental.

Mainly by life style change and minimal genetic.

Diet: intolerance to some foods.

Intestinal dysbiosis.

Stress, anxiety or depression.

Sleep disorders, obesity & lack of exercise.

Altered 5-HT metabolism, autonomic imbalance & hormonal changes e.g. around menstruation.

Pelvic floor dysfunction specially in constipation, malfunction in GIT muscles, low grade inflammation & ↑colon sensitivity to pain.

Further investigations if

Age: > 50 years, with recent changes in bowel habit.

Family history of IBD or colorectal cancer.

Nocturnal pain or defecation.

GIT bleeding, fecal occult blood or fecal calprotectin test.

New blood test is depending on intestinal dysbiosis.

Anemia or weight loss.

N.B. cancer colon is 3rd common cancer after lung & breast.

LIFESTYLE MODIFICATION

A) Diet:

1. Small frequent meals, chewing, slow eating, in the same time daily. Unprocessed & organic food. ↑fluids.

2. ↑soluble fibers e.g. vegetables, fruits, legumes & cereals specially in constipation. Except in acute flare up.

3. Probiotics & prebiotics for few weeks.

4. Avoid or ↓trigger foods:

1. Milk products: lactose intolerance → alter gut flora.

2. Insoluble fibers in.....

3. Flatulogenic foods e.g. legumes, cabbage, cauliflower, onion, eggs, fermented carbohydrates & carbonated drinks.

4. To less extent, trigger foods in ulcerative colitis (.....).

B) Exercise, ↓weight. , ↓stress. Sleep.

Drug therapy

A) Antispasmodic drugs: for abdominal pain. e.g.

1. Anticholinergic drugs as atropine & hyoscine substitutes.
2. Direct smooth muscle relaxants as mebeverine.
3. Peppermint: Ca++ channel blocker in smooth muscles.

B) Antidepressants for depression:

Low dose daily for 4 weeks.

Mechanism: mainly for neuropathic pain modulation > depression.

TCA for diarrhea, SSRIs for constipation.

C) Antidepressants or **anxiolytics** for anxiety.

C) Antiflatulent drugs in patients with gas distension due to IBS, PU, functional dyspepsia, postoperative and air swallowing.

Orally with or after meals and at bed time.

Simethicone: It changes surface tension of gas bubbles,

→ coalesce → freeing of gas to be eliminated easily.

Charcoal: adsorbent, detoxicant, and soothing agent.

D) Prokinetics: ↓accumulation of food in colon with gas formation.

E)Treatment of constipation or diarrhea.

Rifaximin is poorly absorbed antibiotic related to rifampin.

Against E coli. Used as tablets 200 mg tds 14 days in IBS if main symptom is diarrhea.

F) In hyperacidity: short course of acid – suppressants & antacids.

Inflammatory bowel disease (IBD)

It includes **ulcerative colitis** & Crohn,s disease.

A chronic disease characterized by periods of inflammation & ulcers in colon.
*↑↑mucus or bloody stools, diarrhea, defecation urgency & abdominal pain .

Investigations:

1. Stools tests for:
a. Occult blood.

b. Calprotectin: a protein marker secreted by inflamed colon to monitor level of intestinal inflammation, effectiveness of ttt & relapse. False positive by NSAIDs.

2. Colonoscopy.

Etiology & associated mechanisms:

Genetic & environmental.

Overactive immune system.

Diet: intolerance to some foods. Intestinal dysbiosis.

Stress, anxiety or depression.

Sleep disorders, obesity & lack of exercise

A) LIFESTYLE MODIFICATION

Similar to IBS +:

1. Folic acid (large dose, daily), omega 3 fatty acids & antioxidants.

Curcumin (low amounts, → bleeding & renal stones) is anti - oxidative, anti - inflammatory & anticancer used in IBD, GIT cancer & liver cirrhosis.

3) Avoid or ↓trigger foods:

1. Fats.
2. Excess meats: specially fatty & red .... hydrogen sulfide →....& DNA....
3. Milk products: lactose intolerance → alter gut flora.
4. Insoluble fibers →…...
5. Flatulogenic foods e.g.
6. Caffeine intolerance: coffee, tea, energy drinks, chocolate.

4) Avoid or ↓UC trigger drugs:

NSAIDs (→ enteropathy) & antibiotics (→ intestinal dysbiosis).

Drug therapy

A) Aminosalicylates

They contain 5- amiosalicylic acid (5-ASA) which acts only topically.

80% is absorbed in small intestine and does not reach distal small intestine & colon in sufficient amount.

To deliver it in these sites different formulations are used.

1. Azo compounds:

They contain 5-ASA bound to an azo (N=N) bond to another 5-ASA (olsalazine) or another compound as sulfapyridine (sulfasalazine).

Active 5-ASA is released in terminal ileum & colon by bacteria.

2. Mesalamine compounds:

Contain timed- release microgranules releasing 5-ASA throughout intestine. Suppositories & enemas are given in affection of rectum & sigmoid colon respectively.

Mechanism:

1. Modulate COX & LOX- derived inflammatory mediators.
2. ↓production of inflammatory cytokines.
3. ↓cellular functions of lymphocytes & macrophages.

Uses: For treatment & maintenance of remission.

Adverse effects:

Mainly by sulfasalazine (40% intolerance), more in slow acetylators, dose related. Rare in other formulations. e.g. GIT upset, headache, malaise, arthralgia. Sulfasalazine (only) causes hypersensitivity, oligospermia & ↓folic acid absorption.

B) Glucocorticoids

Only in acute cases. Not in remission.

Mechanism is similar to aminosalicylates and ↓gene transcription of NO synthase.

C) Immunosuppressants

D) Anti TNF monoclonal antibodies

Infliximab (IV infusion).

Adalimumab (top selling drug) & golimumab (SC).

Vedolizumab is new, gut - specific.