CLINICAL AND THERAPEUTIC NUTRITION

Actiopathogenesis, clinical features, diagnostic tests, prevention and treatment of malabsorption syndrome



MALABSORPTION SYNDROME

•Major part of the absorption of nutrients take place in the small intestine and the set of enzyme involved in this process are called <u>disaccharidases</u>. In some conditions either <u>genetically, or due to intestinal damage</u> there appears to be deficiency of one of these <u>enzymes</u>, which in turn, leads to malabsorption of the related nutrients.

•The main role of your small intestine is to absorb nutrients from the food you eat into your bloodstream. Malabsorption syndrome refers to a number of disorders in which the small intestine can't absorb enough of certain nutrients and fluids.

•Nutrients that the small intestine often has trouble absorbing can be macronutrients (proteins, carbohydrates, and fats), micronutrients (vitamins and minerals), or both.

•Malabsorption may be due to defects in:

1.The intestinal lumen, resulting in adequate fat hydrolysis or altered bile salt metabolism;

2. The mucosal epithelial cells, affecting absorbing surfaces and interfering with transport function;

3. Intestinal lymphatic obstruction-lymphangiectasia.

MALABSORPTION SYNDROME

Aetiopathogenesis:

□*Anatomical or surgical*: surgical resection, fistula, gastric surgery, blind loop and stricture and diverticulosis.

Enzyme deficiency: pancreatic disease, biliary obstruction and disaccharidases deficiency.

Mucosal defects: celiac disease, tropical sprue, crohn's disease and radiation.

□*Systemic causes*: scleroderma, diabetes, lymphoma, thyroid disease and severe skin disorders

Drugs: cholestyramine, antibiotics, excess laxatives

□*Infections:* giardia and parasitic infestation, tuberculosis and bacterial overgrowth.

Clinical feature: Depending on the nature of the disease process causing **malabsorption** and its extent, gastrointestinal symptoms may range from severe to subtle or may even be totally absent.

•Pale, bulky, frothy, and offensive stools due to abnormally high fat content; •Muscle wasting and progressive weight loss due to steatorrhea, diarrhea and anorexia

Abdominal distension in children, less marked in adults

CLINICAL FEATURE

•Evidence of vitamin and mineral deficiencies, such as macrocytic anemia, hypocalcemic tetany, glossitis and so on.

•General or non-specific symptoms seen are weakness, lassitude and weight loss steatorrhea, and chronic ill health.

•Diarrhea, along with flatulence, mild abdominal pain, anorexia, nausea and vomiting may be present.

• Nutritional deficiencies like anemia and b-group vitamin deficiencies are common and may manifest as glossitis, tetany, bone pain, paresthesia and convulsions.

•Visible signs include smooth tongue, edema, dry skin, bleeding, pigmentation, dermatitis, peripheral neuropathy and proximal muscle atrophy.

Laboratory findings include decrease in serum concentrations of electrolytes, albumin and carotene; impaired absorption of dxylose, glucose, folic acid, and vitamin B-12 and increased fecal fat and nitrogen.

DIAGNOSTIC TESTS

The diagnosis of malabsorption syndrome is based upon findings from absorption tests, intestinal mucosal biopsy, and radiologic studies. Direct tests of absorption involve

- measurement of fecal fat: The balance study method is widely used and involves the chemical analysis of a 72- hour stool collection. The patient is fed a diet containing a known amount of fat, usually 50 to 100 gm, for several days before and during the collection period. Stools are then analyzed for fat. Normal excretion is less than 5 gm per 24 hours. Stool collection are also used to measure fecal radioactivity following administration of a test dose of I-labeled triolein. The triolein is mixed with a marker and stools are collected until the marker is no longer visible. Normal fecal radioactivity is less than 7 per cent of the test dose.
- Serum carotene level: the serum carotene level is a useful screening test, and malabsorption is suspected if levels of less than 60 micrograms per 100 ml are found.
- Oral tolerance tests provide indirect evidence of malabsorption. Most commonly used are d-xylose and lactose. Urinary excretion of d-xylose following ingestion of a 25-gm load is used as an indication of carbohydrate absorption.

DIAGNOSTIC TESTS

- Excretion of less than 4.5 gm in an hours in patients with normal renal function indicates decreased absorptive capacity. The lactose tolerance test is used in suspected lactase deficiency. Administration of lactose, 2 gm per kilogram body weight, or a maximum of 50 gm, is followed by determination of blood glucose levels for 2 hours.
- Lactose malabsorption is indicated if the blood glucose rises less than 26 mg per 100ml. Symptoms of abdominal distension, cramping, and diarrhoea may occur following ingestion of the lactose in persons with lactose intolerance.
- Measurements of breath hydrogen following a lactose load is more sensitive test for lactose malabsorption. Unabsorbed lactose undergoes bacterial fermentation in the colon with production of hydrogen gas, part of which is excreted through the lungs. An increase in breath hydrogen in expired air samples collected at specified intervals indicates lactose malabsorption. In another breath test, excretion of carbon dioxide following administration of certain fats labelled with isotopes is used as a screening test for fat malabsorption.
- Other screening test involve measurement of urinary excretion of 4hydroxyphenyl acetic acid or determination of urinary oxalate following administration of sodium oxalate.
- Schilling test: is frequently used as an index of vitamin B-12 absorption; an oral dose of radioactive vitamin B-12 is administered followed at 2 hours by an intramuscular injection of nonradioactive B-12. urinary excretion of less than 5 to 6 % of the radioactive dose indicates malabsorption.

DIAGNOSTIC TESTS

- The folic acid test consists of assaying urine for 24 hours following injection of the vitamin and again after it is given orally 48 hours later. In malabsorption, excretion of folic acid is less than after an oral dose than after injection.
- Biopsy specimens of the jejunal mucosa showing villous atrophy provide nonspecific evidence of disturbances in absorptive function. Radiologic evidence of intestinal dilatation, altered motility, and bone demineralization may also be seen in malabsorption

TREATMENT

Therapy is directed toward alleviation of symptoms by correction of the basic defect insofar as possible, dietary modification in accordance with the nature of the defect, vitamin and mineral supplements, and prevention or correction of complications by administration of appropriate agents.

Dietary modification:

- The diet in malabsorption syndrome should be high in protein and calories. modification of fat intake is often indicated. Vitamin and mineral supplementation is usually needed. A soft or fibre-restricted diet is useful for patient with persistent diarrhoea.
- Substitution of medium-chain triglycerides(MCT) for longer chain fats (LCT|) is associated with reduced steatorrhea and decreased losses of calcium, sodium, and potassium in many of the disorders comprising the malabsorption syndrome
- The diet should provide for a reduction in long –chain triglycerides by substituting an oil containing medium- chain triglycerides as source of fat. The diet is adjusted to provide 50-70% of the fat kcal as Medium chain-triglycerides.
- Th protein intake may be increased by adding non fat dry milk to fluid skim milk, skim cottage cheese, egg whites, and cereal products.
- The kcal level may be increased by adding high carbohydrate foods such as fruits, sugar, jelly and fat-free desserts.
- Modifications in fibre and consistency may be made by applying restriction concerning the soft diet.
- Initially, small amounts of MCT should be taken with meals and gradually increased according to individual tolerance.

Foods allowed	Foods to avoid
Skim cottage cheese, bread, cooked or dry cereals, macaroni, noodles, rice, spaghetti, egg whites; whole eggs, butter, MCT oil, all fruits except avocado and vegetable, lean meats, fish and poultry , skim milk	Commercial biscuits, coffeecake, corn bread, crackers, doughnuts, muffins, cheese made whole milks, cakes pies, fatty fish , sauces, gravies buttermilk, skim milk, vegetables those with

PREVENTION

- Malabsorption is preventable only when the underlying cause (such as an infection) is preventable.
- Avoid drinking too much alcohol.
- Drink only bottled water ,
- Eat only cooked foods, and avoid fresh salads or other washed produce.
- Medicine to replace intestinal enzymes or reduce spasms,
- □ Vitamin or mineral supplements, such as B12 and iron.
- Pancreatic enzymes are supplemented orally in pancreatic insufficiency.
 - Dietary modification is important in some conditions:
- **Gluten-free** diet in coeliac disease.
- Lactose avoidance in lactose intolerance
- □ Antibiotic therapy to treat Small Bowel Bacterial overgrowth.
- Cholestyramine or other bile acid sequestrants will help reducing diarrhoea in bile acid malabsorption.