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Review Article

Malabsorption Management

Causes and Management of Malabsorption, A Review

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Abstract: Maldigestion refers to poor nutritional digestion within the intestinal lumen or at the brush boundary, whereas malabsorption refers to decreased nutrient absorption at any point where nutrients are absorbed. Any flaw in the digestion/absorption process might cause malabsorption. Reduced absorption of certain nutrients, impaired GI motility (decreased peristalsis and stasis), altered bacterial flora, infection, or limited blood flow or weakened lymphatics are all possible causes for these problems. Protozoal infections of the small intestine are very frequent and may be linked to malabsorption. The majority of these infections are self-limiting. For the diagnosis of malabsorption, there is no gold standard. Additional tests should be done based on the clinical situation and the probable underlying condition. Nutritional assistance is provided by enteral or parenteral feeding, as well as screening for and treatment of vitamin and mineral deficiencies. In this article we will be looking at malabsorption causes and management.

Keywords: Malabsorption, GIT, Nutrition, Digestion and Lipid Malabsorption

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1. INTRODUCTION

Fats, carbs, proteins, vitamins, minerals, and trace elements are all absorbed through the gastrointestinal system. Maldigestion refers to poor nutritional digestion within the intestinal lumen or at the brush boundary, whereas malabsorption refers to decreased nutrient absorption at any point where nutrients are absorbed.[1] Due to its huge surface area supplied by the many microvilli covering intestinal villi and the digestive enzymes actively released on its surface to maximise uptake of food components, the small intestine is the region of the gastrointestinal system where majority of the absorption occurs. Adequate absorption occurs when the digestive organs are mechanically intact and have enough blood flow. Also they most have the proper motility, and microbiota to create the required enzymes[2]. Any flaw in the digestion/absorption process might cause malabsorption. Reduced absorption of certain nutrients, impaired GI motility (decreased peristalsis and stasis), altered bacterial flora, infection, or limited blood flow or weakened lymphatics are all possible causes for these problems. The consequence is either a general impairment in nutrient absorption or a particular impairment in nutrient absorption.[1,3,4]Within the gastrointestinal system, physiological digestion and absorption of nutrients necessitates considerable interaction between secretory, motor, and absorptive activities. The sophistication of this mechanism explains why malabsorption can be caused by a variety of disorders and illnesses. While it is well understood that severe pancreatic insufficiency causes malabsorption, it is less well understood that coordinated gastrointestinal motility is also one of the fundamental prerequisites for undisturbed and effective meal digestion and absorption. Milder motility abnormalities, on the other hand, might cause or contribute to symptoms like diarrhoea, constipation, and stomach discomfort, while severe motility disturbances can limit nutritional absorption[5]. Protozoal infections of the small intestine are very frequent and may be linked to malabsorption. The majority of these infections are self-limiting. Traveller's diarrhoea is frequently caused by protozoa. A tiny percentage of infected patients get chronic diarrhoea and malabsorption.[6] SIBO (small intestine bacterial overgrowth) is caused by a disturbance of the small bowel's typical, established ecology. Certain bacteria overgrow and deconjugate bile acids, leaving them incapableof fat absorption. Bacterial overgrowth may occur alongside atrophic gastritis or the use of proton pump inhibitors (PPIs). Vitamin B12 absorption may be hampered by PPIs (rarely to a clinically significant degree). SIBO can also be caused by long-term lactose deprivation, blind loops established by inflammatory processes like IBD, any cause of GI stasis, or medical diseases that promote gastric dumping of food with an acidic pH. SIBO bacterial overgrowth is spotty, unlike celiac disease, which has a broad distribution[1,7]. The process of digestion begins in the mouth, with the mechanical disruption of food caused by chewing and combining with salivary enzymes. In the stomach, mechanical digestion proceeds until the semiliquid meal, also known as chyme, is created. When chyme is transported into the duodenum and treated with pancreatic enzymes and bile salts, digestion is complete. When normal gastric function is compromised as a result of surgery such as partial or total gastrectomy, partly digested food flows quickly into the duodenum, generating osmotic pressure and diarrhoea. Enzymatic activity is also important for nutrient absorption.[2] The proximal small intestine absorbs nutrients extremely well: up to 80% of triglycerides, 60% of carbohydrates, and 50% of proteins have been shown to be absorbed before reaching the distal duodenum 20 cm apart after nutrients are perfused into the proximal duodenum at physiological postprandial rates. [5,8,9] Small quantities of nutrients are not absorbed during small intestinal transit even in healthy adults, they reach the terminal ileum and are transported to the colon. As a result, most complex carbs may have roughly 10% malabsorption, with lipid malabsorption accounting for up to 5% of the amount supplied.[5] Physiologically malabsorbed nutrients provide a vital source of energy for colonic bacteria and regulate, mostly inhibit, upper gastrointestinal activities. This ileal brake mechanism is thought to have a role in the restoration of the interdigestive secretory and motor pattern at the end of the digestive phase.[10,11]

2. CAUSES OF MALABSORPTION

Nutrient digestion and absorption are essential for living creatures' existence and have developed into the gastrointestinal (GI) system's complicated and particular responsibilities. The GI system will thus operate correctly to consume nutrients, create energy, and eliminate wastes in healthy conditions. Numerous diseases, on the other hand, might disrupt the physiological systems that ensure correct digestion and absorption of nutrients (including macro- and micronutrients), resulting in a wide range of symptoms and nutritional repercussions[12]. Focusing on the following 3 stages of digestion and nutrition absorption is crucial when considering the various reasons of acquired malabsorption: Processing at the luminal and brush borders, intestinal mucosal absorption, and circulatory transfer are the first three steps. Abnormalities at any of these three steps can lead to malabsorption, and there may be multiple concurrent defects. The acquired reasons of malabsorption are described in the next sections of this article in relation to the aforementioned stages of the nutritional absorption process[13].

2.1 Processing at The Luminal and Brush Borders

Most acquired causes of malabsorption are caused by illnesses that disrupt the luminal phase of intestinal absorption. You can categorise them as having an effect on the absorptive surface or having an effect on the digestive enzymes or substrates. Reduced absorptive surface as a result of inadequate intestinal length brought on, for the most part, by surgical resections, is a significant clinical factor in malabsorption. In affluent nations, gastroschisis, volvulus, necrotizing enterocolitis, intestinal atresia, and significant aganglionosis in Hirschsprung's disease are the most common causes of major intestinal surgery in children.[14,15] Mesenteric infarction (arterial or venous thrombosis), Crohn's disease, radiation enteritis, surgical complications, intestinal volvulus, familial polyposis, abdominal trauma, intestinal angiomatosis, and complex intussusception are the leading causes of intestinal resections in adults[16]. Short bowel syndrome can result from intestinal resections. Short bowel syndrome is characterized by inadequate small intestinal length, which results in suboptimal nutrient absorption.[17] In general, if fewer than 200 cm of the small intestine is still present, nutritional/fluid supplements may be required. The following are some causes of small bowel syndrome in kids:

2.2 Resection of The Small Bowel Beginning at The Treitz Ligament or Less Than 70% of The Entire Length

75 cm with a term delivery, 50 cm with a preterm birth (36 weeks) and 100 cm for children older than one year[18] and in

adults: Duodenostomy, Jejunoileal Anastomosis, Jejunocolic or Ileocolic Anastomosis, and End Jejunostomy, with a remnant small intestine of approximately 35 cm to 60 cm to 115 cm[19,20]. After resection, continuity with the residual colon is crucial. The colon's main purposes are to absorb short-chain fatty acids as well as water and salts. These acids are created when dietary fibres are digested by intestinal bacteria and play a significant role in the intestinal energy metabolism. The absence of Bauhin's valve is a crucial component in the emergence of short bowel syndrome. It is possible for colonic bacteria to overgrow in the small intestine after ileocaecal valve resection. Bacterial overgrowth may affect digestion and nutrient absorption because bacteria and enterocytes fight for resources[19,21]. While luminal lesions that might produce fixed blockages are absent, intestinal dysmotility disorders can cause the luminal process of absorption to become dysregulated, leading to disordered propulsion of the intestinal contents. Therefore, even if the intestine is the right length, the abnormality causes it to become dysfunctional. Malabsorption can result from dysmotility problems that affect multiple areas of the intestine, notably the small intestines. Postoperative or acute critical illness-related ileus, which comes from reduced gastrointestinal motility linked to systemic or intra-abdominal inflammation, is an example of an acute intestinal dysmotility condition. Permanent intestinal dysmotility is a symptom of chronic intestinal pseudo-obstruction, which can be brought on by a number of illnesses, including infections, autoimmune conditions, mitochondrial malfunction, and drug side effects[16]. Diabetes, systemic scleroderma, amyloidosis, or intestinal pseudo obstruction are examples of specific reasons. However, the root cause is typically unknown. Histologically, there are three subcategories of chronic intestinal pseudo-obstruction: neuropathies, myopathies, or mesenchymopathies (involving the interstitial cells of Cajal). Due to bouts of non-mechanical intestinal obstruction or feeding-related worsening of digestive symptoms, which restricts oral and enteral nutrition, these illnesses might result in malabsorption.[22,23]

2.3 Intestinal Mucosal Absorption

The subsequent section goes over acquired causes of malabsorption during the mucosal absorptive stage of intestinal absorption. A brush boundary transport, an enterocyte, or an enzyme deficit can all be the cause of the mucosal abnormality. Lactase deficits are the sole cases of acquired enzyme deficiencies that are clinically significant. Lactose, a carbohydrate found in dairy products, must be digested by the enzyme lactase[24]. Diffuse small intestine illness results in acquired abnormalities of brush border protein transporters and enterocyte function. Inflammation in celiac disease results in mucosal dysfunction, which can cause aberrant lipid uptake and re-esterification. Enterocyte dysfunction can also result from Crohn's disease.[25]

2.4 Circulatory Transfer

Transporting the absorbed nutrients into the bloodstream is the last step in the absorption process. A healthy lymphatic system is especially important for the transfer of lipids. Lymphatic blockage can cause steatorrhoea, chylous ascites, a protein-losing enteropathy, impaired chylomicron and lipoprotein absorption, and fat malabsorption. Primary intestinal lymphangiectasia (Waldmann's disease) and secondary blockage brought on by neoplasia like lymphoma or infections like Whipple's disease, tuberculous enteritis, or filariasis are the two main causes of lymphatic obstruction.[26]

3. CHEWING PROBLEMS

Digestion of food breaks down ingested nutrients in the mouth, combining them with saliva to produce a meal bolus that may be swallowed easily. People who have difficulty chewing, particularly those who are edentulous and do not wear dentures, lose weight and have a higher death rate, which is likely related to nutritional deficiency. However, efficient absorption of nutrients from some vital, tougher dietary components like meat and vegetables appears to be dependent on the masticatory process. [5]

4. LIPID MALABSORPTION

Lipid digestion can start in the mouth with lingual lipase generated by tongue glands and continue in the stomach with lingual and gastric lipase produced by main cells. However, because only 15% of fat digestion happens by the time food exits the stomach, most fat arrives in the duodenum intact in adult humans. Cooking aids emulsification of dietary fat, which continues with chewing and concludes in the stomach with churning and peristalsis. In the duodenum, polar lipids, phospholipids, fatty acids, cholesterol, triglycerides, denatured dietary proteins, dietary oligosaccharides, and bile salts cover the emulsion droplets arriving from the stomach, which contain practically all of the dietary triglycerides and diglycerides in their cores.[12,27,28] Clinical symptoms (steatorrhea, indicators of reduced availability of lipid-soluble vitamins) usually appear only when the pancreatic secretory capacity is reduced to less than 5-10% of normal activity. The deleterious impact of milder abnormalities on bone metabolism, on the other hand, may have been overlooked. Furthermore, dyspeptic symptoms and diarrhoea may be exacerbated by moderately compromised pancreatic exocrine insufficiency, especially in individuals with other gastrointestinal problems.[5,29,30]

4.1 Intestinal lymphangiectasia

Decreased lymphatic flow affecting fat digestion; this is one of the most prevalent yet underappreciated causes of persistent, non-infectious infantile diarrhoea. Cow's milk protein allergy and cystic fibrosis are two of the most prevalent causes of infantile diarrhoea. Whipple illness is a systemic infection caused by Tropheryma whipplei. Symptoms include diarrhoea and weight loss, which might indicate malabsorption. Fever, arthralgias, and stomach discomfort are common side effects. Lymphadenopathy, endocarditis, lung disease, and CNS infection are all possible signs. Whipple disease may be indistinguishable from the symptoms of Mycobacterium avium on biopsy in certain circumstances. Acid-fast stains can tell the difference between them. [1,31,32] Crohn's disease is a chronic inflammatory bowel illness that affects In tropical places, Crohn's disease is becoming more common, and it's a crucial differential diagnostic for TB. Malabsorption in Crohn's disease can be caused by a variety of reasons. Small intestine involvement affects around a third of individuals, reducing the absorptive surface area. Similarly, extensive small bowel resections would have the same result. Vitamin B12 deficiency and bile salt malabsorption can develop from terminal ileal resections, whereas bacterial overgrowth causes malabsorption after ileocaecal valve resections. In an unselected case series from northern India, almost 9% of

individuals with malabsorption were found to have Crohn's disease. [6,33,37] Bacterial overgrowth can reduce the availability of substrates in the luminal space (carbohydrates, protein, and vitamins).To transfer digested items from the lumen into the cells, the mucosal phase relies on the integrity of the brush boundary membrane of intestinal epithelial cells. Lactose intolerance and sucrase–isomaltase insufficiency can be caused by reduced brush border enzyme activity.[6] Gastric bypass for the treatment of obesity, small intestine tumours, adhesions from earlier surgery, strictures due to radiation or inflammatory bowel disease, blind intestinal loops, and small intestinal diverticulosis are examples of anatomic diseases. Intestinal stasis occurs in all of these situations, encouraging the development of bacteria such as E. coli, Klebsiella spp., and Aeromonas, among others[12]. Deficiencies in nutrition absorption can be inherited or acquired. Glucose–galactose malabsorption and abetalipoproteinemia are two inherited disorders.

4.1 Transit time

Due to a high osmotic load, increased bacterial metabolism in the colon, and disrupted regulatory processes, substantial acceleration of intestinal transit can cause nutritional malabsorption and symptoms. Delay in small intestine transit, on the other hand, has no effect on the small bowel's absorption ability. Delay in transit, on the other hand, may encourage small intestine bacterial overgrowth by allowing ascending colonic germs to colonise the small bowel. These bacteria eat a portion of the macro- and micronutrients consumed, potentially leading to malnutrition. Furthermore, bacterial enzymes that deconjugate bile acids limit bile acid absorption in the terminal ileum, diminish the bile acid pool, and disrupt lipid absorption. [5]

4.2 Decreased Intrinsic Factor

The production of chlorhydropeptic and intrinsic factors by parietal cells is required for vitamin B12 absorption. Vitamin B12 is protein-bound in meals and is dissociated in the stomach's acid environment with the aid of pepsin. R-binders, which are vitamin B12-binding proteins released in the saliva, bind to vitamin B12 in the stomach. Intrinsic factor is produced by parietal cells, and R-binders are cleaved by pancreatic proteases released into the higher pH duodenum, allowing vitamin B12 to bind to the intrinsic factor. These disorders cause a significant reduction in stomach acid production, as well as a state of selective vitamin B12 malabsorption due to intrinsic factor secretion. this is the situation in the case of autoimmune atrophic chronic gastritis. [12,38]

4.3 Tropical Sprue

Acute or persistent diarrhoea characterises tropical sprue, a malabsorption illness. In the absence of any specific reason of malabsorption, it is found in persons from the tropical zone. Its aetiology is assumed to be infectious, with environmental variables playing a role. It affects the small intestine and causes malabsorption as well as a variety of nutritional deficiencies, including vitamin B12 and folic acid. [39]

5. DEFINITION

The symptoms of tropical sprue include malabsorption, various nutritional deficiencies, and abnormalities of the small intestinal mucosa. The cause of this acquired disease is unknown. There is ongoing debate over how to define the different clinical disorders together referred to as tropical sprue. The diagnosis of tropical sprue, according to Baker and Klipstein, should only be established when there is malabsorption of two or more unrelated nutrient categories (for example, fat and carbohydrate), and only after other known causes of malabsorption have been ruled out[40]. To describe a state of malabsorption in travellers returning to the UK in whom the sickness was preceded by an acute diarrheal illness and who had signs of jejunal colonisation with aerobic and anaerobic bacteria, Cook introduced the phrase "post-infectious tropical malabsorption."[41]

5.1 Epidemiology

Tropical sprue has not been reported in Jamaica or sub-Saharan Africa, but it has been reported in South and South East Asia, Central America, Venezuela, Colombia, and portions of Mexico and the Caribbean islands[42,43]. In Puerto Rico, it was estimated that 8% of North Americans had endemic sprue[44]. In the late 1960s, British troops in Malaysia and Hong Kong, as well as American personnel in Vietnam, both suffered significantly from it.[45] Even while endemic tropical sprue is far less common than it formerly was, it nevertheless causes approximately 40% of malabsorption in south Asia, affecting both adults and children[46]. During the Second World War, outbreaks of tropical sprue were documented among soldiers and POWs in the Indo-Burma region as well as among American military personnel stationed in the Philippines[47,48]. The disappearance of epidemic tropical sprue and the decline of sporadic tropical sprue may be related to the widespread use of antibiotics as well as improvements in hygiene and water quality. [49]Tropical sprue epidemics affecting villagers in southern India were reported between the 1960s and early 1980s but have not been detected since then.[50]

6. PATHOPHYSIOLOGY

The involvement of both the proximal and distal small intestines contributes to nutritional malabsorption in tropical sprue. The small intestine's crypts have degenerating cells, which suggests stem cell injury, according to ultrastructural research[51]. Reduced xylose absorption and malabsorptive fat and fat-soluble vitamin absorption are the outcomes of decreased overall absorptive surface area caused by villus atrophy and loss of epithelial cell microvilli. While vitamin B12 malabsorption suggests terminal ileal involvement, iron and folate deficiencies indicate proximal small-bowel involvement. Because of terminal ileal involvement, there is bile acid malabsorption, which can cause diarrhoea. Colonic malabsorption of water and electrolytes, which may be caused by unabsorbed bile acids and free unsaturated fatty acids, significantly contributes to diarrhoea in sprue patients.[52] Additionally, the colonic mucosa exhibits lymphocytic infiltration.[53]

7. DIAGNOSIS

The three assays most frequently used to examine absorption are stool fat estimation, D-xylose absorption, and vitamin B12 absorption. If there are no other reasons of malabsorption, two abnormal tests in the proper environment are consistent with tropical sprue.[54] The most accurate test for malabsorption in the tropics is quantitative stool fat measurement. Steatorrhoea is frequently evaluated semiquantitatively utilising Sudan staining of oil (triglyceride)

droplets in stool due to the difficulty of performing this test. Sudan stain is useful for identifying increased faeces fat (triglycerides) in people with chronic pancreatitis, but it is not sensitive enough to diagnose tropical sprue, a condition in which faeces fat is present in the form of fatty acids rather than triglycerides[55]. The physical features of the stool's fat are measured by the acid steatocrit, which has not been examined in tropical sprue. About 99% of patients have D-xylose malabsorption, 90% have steatorrhoea, and 60-90% have vitamin B12 malabsorption. Endoscopically acquired duodenal mucosal biopsy has taken the position of peroral capsule biopsy of the jejunal mucosa. As villi in the second half of the duodenum may be shorter than they are more distally in the duodenum and in the jejunum, it is crucial to collect biopsy samples from further into the duodenum.[56]

8. TREATMENT

Dehydration patients require the restoration of fluid and electrolyte balance, while chronically sick patients require the correction of magnesium and potassium deficiency. Supplements administered parenterally or orally can be used to treat specific vitamin deficiencies, including those in the B complex, vitamins A, and D. Parenteral vitamin B12, oral folate, and iron supplementation all quickly relieve anaemia, glossitis, and anorexia symptoms and cause weight gain even before intestinal absorption improves. Both bacterial absorption and injury to the host epithelium can reduce folate levels. Both villous atrophy and macrocytic anaemia are improved by folate administration. Tetracycline 250 mg four times per day (or doxycycline 100 mg once per day) for 3-6 months is the preferred antibiotic. Antimicrobials are frequently utilised in treatment. Diarrhoea is one of the main symptoms, and limiting long-chain fatty acids in the diet can assist. Long-chain fatty acids can be replaced by medium-chain triglycerides. [57]

9. CONSEQUENCES

The severity and persistence of the underlying reasons directly affect the effects of malabsorption. This may result in growth retardation, osteopenia, anaemia (iron, folate, and vitamin B12), abdominal pain and bloating (caused by bacterial gas production and bacterial overgrowth), diarrhoea and steatorrhoea, fluid and electrolyte losses, and anaemia (caused by bacterial gas production and bacterial overgrowth)[58,59]. Malnutrition is the main result of malabsorption. Malabsorption is a problem that needs to be diagnosed early and treated promptly because malnutrition can be considered an independent risk factor for morbidity and mortality. Malnourished hospitalised patients had a much higher chance of suffering infection complications, respiratory failure, cardiac arrest, cardiac failure, arrhythmias, and wound dehiscence, according to a recent research by Richard et al. outlining the effects of malnutrition. No of the underlying condition or the method of therapy, the hospital stays for the malnourished patients were similarly noticeably longer. Furthermore, malnourished individuals are four to five times more likely to require a stay in the hospital longer than 12 days.[60]

9.1 Malabsorption Syndrome

Fats, carbs, proteins, vitamins, minerals, and trace elements are just a few of the nutrients that the gastrointestinal system is involved in absorbing. Malabsorption describes a problem with nutrition absorption at any point of the nutrient absorption process. Any flaw in the digestion or absorption process might lead to malabsorptive conditions. Congenital flaws in the intestinal membrane transport systems, impaired nutrient absorption, impaired GI motility (decreased peristalsis and stasis), disrupted bacterial flora, infection, impaired blood flow, or impaired lymphatics can all cause these defects. They can also result from innate mucosal diseases, conditions that cause acquired mucosal damage, acquired mucosal diseases, impaired blood flow, or impaired lymphatics. The end outcome is either a general impairment of nutrient absorption or a specific impairment of nutrient absorption.[3,4]

9.2 Fat Malabsorption

One of the most prevalent malabsorption syndromes is fat malabsorption, which is caused by problems with fat digestion and absorption. The act of suspending fat molecules in aqueous solution to expose their surfaces to hydrolytic enzymes is known as emulsification, or lipid processing.[27]

9.2.1. Causes

- Duodenal pH decreased; the ideal pH is 6.5.
- Reduced transit time and less exposure to digestive enzymatic activity are the results of decreased absorptive intestinal surface area and diminished functioning small intestine mucosa.
- Small intestinal bacterial overgrowth (SIBO) is brought on by a disruption of the small bowel's regular, established ecology.
- Certain bacteria that are overgrown deconjugate bile acids, making them useless for absorbing fat[61].
- Pancreatic lipase, colipase, and bicarbonate generation is compromised in pancreatic exocrine insufficiency.[62] Chylomicron/lipoprotein secretion dysfunction. problems of the lymphatic system.

9.3 Carbohydrate Malabsorption

The terms "carbohydrate digestion and absorption" frequently apply to the human diet's starch, lactose, and sucrose. The human small intestine cannot digest cellulose. For effective absorption, the monosaccharides must be well digested. Salivary and pancreatic amylase are the first enzymes that break down carbohydrates. The microvillus membrane continues to process the end products. Brush border enzymes over the carbohydrate mixture, and then hydrolyze it to produce monosaccharides. Monosaccharides can either be actively or passively absorbed. The colon begins to ferment any leftover carbs that are not absorbed, including non-absorbable cellulose (i.e., degraded by bacteria). Colonic epithelial cells can absorb fatty acids that are generated as a result of bacterial fermentation and use them as fuel. Acidic faeces, gas, and bloating are signs of increased bacterial fermentation in carbohydrate malabsorption.[3]

9.3.1 Causes

A lack of pancreatic amylase, disaccharidase activity that is insufficient, decreased intestinal absorption surface area.[63,64]

9.4 Protein Malabsorption

Proenzymes that automatically activate at low pH levels start the process of protein digestion and absorption as proteolysis in the stomach (i.e., an acidic environment). The degree of

proteolysis is influenced by gastric motility for mixing, pH levels, and other food components present at the time. For instance, the release of amino acids from the stomach affects the release of cholecystokinin (CCK) in the duodenum and jejunum. CCK is released in response to amino acids, and CCK in turn increases the release of pancreatic enzymes.[3]

9.4.1 Causes

- Decreased secretion and/or action of pancreatic bicarbonate and proteases
- Decreased intestinal absorption surface area

9.5 Malabsorption of Vitamins, Minerals, And Trace Elements

The absorption of vitamins, minerals, and trace elements is accomplished through a variety of intestinal transport systems. Any vitamin, mineral, trace element, or nutrient that depends on one of these levels to be properly absorbed is malabsorbed when one of these levels is dysfunctional. Vitamin B12, calcium, iron, folate, vitamin D, magnesium, carotenoids, thiamin, copper, selenium, and other shortages are only a few examples of those that exist.

9.5.1 Causes

- Stomach or proximal small intestine pathology (e.g., vitamin B12 deficiency).
- Fat malabsorption is brought on by the binding of calcium, magnesium, and other divalent cations by fatty acids.
- Decreased intestinal absorption surface area.[4]
- General evaluation of malabsorption syndrome:
- Blood test
- Electrolyte abnormalities, hepatic function, and renal function are all included in the comprehensive metabolic panel.
- A complete blood count is one tool for assessing anaemia.
- Albumin, Magnesium, Zinc.
- Vitamins phosphorus (e.g., vitamin B12, folate, vitamin D).
- Metal panel (includes serum iron, total iron-binding capacity, ferritin).
- Faecal test is Tthe most accurate test for fat malabsorption syndromes :
- Fecal fat - a single specimen is used to measure faecal fat.
- The gold standard for the diagnosis of steatorrhea is faecal fat excretion after 72 hours.
- Sudan III stain: a sensitive procedure used on a spot stool sample.
- Steatocrit acid.
- NIRA, or near-infrared reflectance analysis.

10. EPIDEMIOLOGY

Millions of individuals throughout the world suffer from malabsorption. The frequency and incidence of malabsorption syndromes are obscured by the fact that they have numerous etiologies. To estimate various malabsorption syndromes, the epidemiology of subgroups is discussed[2]. The prevalence of gluten-sensitive enteropathy (GSE) is higher in Europeans and North Americans. GSE can occur in some areas of India and is most uncommon in people with Asian, Caribbean, and African ancestry. Residents and visitors to Puerto Rico, the Caribbean, West Africa, northern South America, south-east Asia, and India are known to be impacted by tropical sprue. Although the precise frequency of pancreatic exocrine insufficiency in the general population is unknown, its prevalence in particular subgroups with predisposing factors can be appreciated. The incidence of severe chronic pancreatitis is 85% in those who have it, 30% in those who have milder cases, and once more, 85% in neonates with cystic fibrosis. The prevalence of diabetes varies, with diabetes type I having a greater prevalence (26% to 44%), as do the prevalences of HIV/AIDS (26% to 45%), inoperable pancreatic cancer (50–100%), and operations (19% to 98%). (distal pancreatectomy, Whipple). Other populations, however, exhibit a lower prevalence (IBS, diabetes type 2).[6]

10.1 Prognosis

Typically, malabsorption syndromes don't pose a life-threatening concern. However, some malabsorption syndromes can be deadly or even life-threatening due to their severity and length. Examples include life-threatening electrolyte imbalances from protracted, intractable diarrhoea, severe malnutrition from prolonged pancreatic exocrine insufficiency, and intestinal perforation[6]. Other malabsorption syndromes, such lactose intolerance, are less likely to dramatically worsen a patient's health. The effectiveness of illness management and the progression of the disease both contribute to this (e.g., avoidance, supplementation, supportive care).

10.2 Complications

Signs of the digestive system (such as persistent diarrhoea, bloating, and flatulence), Malnutrition , poor weight gain/weight loss, deficits in vitamins, minerals, and trace elements (e.g., vitamin D, B12, iron, folate). Musculoskeletal dysfunctions, Dermatologic symptoms, Visual impairment, and Hematologic diseases. alterations in the electrolyte, Diseases of the cardiovascular system, the nervous system, and the endocrine system.[6]

Causes of malabsorption :[58,65]
Premucosal
Impaired digestion
Bile acid/enzyme deficiencies
Mucosal
Reduced absorption
Bowel resection
Diseases affecting absorption
Postmucosal
Altered nutrient transport
Vascular or lymphatic abnormalities

Diagnostic tests:[58,66-68]
Premucosal
Impaired digestion
Serum electrolyte, mineral and vitamin values
Faecal fat excretion, hydrogen breath test
Ultrasound for obstructions/calcifications
Mucosal
Reduced absorption
Bowel resection
Endoscopy and histology
Xylose test, Schilling test
Postmucosal
Altered nutrient transport
Ultrasound/contrast for fistulae

Treatment
Premucosal
Impaired digestion
Partially digested food
Pancreatic enzyme supplementation
Surgery for obstruction
Mucosal
Reduced absorption
Partially digested food
Disease-specific treatment
Postmucosal
Altered nutrient transport
Surgery for obstruction

11. MANAGEMENT

Correcting deficiencies, addressing the underlying cause, avoiding triggers (usually food), and treating symptoms are all part of malabsorption syndrome treatment (e,g often diarrhea). A malabsorption syndrome misdiagnosis or missing diagnosis might cause harm or have no impact. As a result, treatment should focus on addressing the underlying cause, which is determined by the diagnosis since malabsorption syndromes are caused by deficiencies in the digestive system. Treatment options include dietary adjustments such as food avoidance or supplementation, as well as more intrusive procedures such as surgery (e.g., transplants, resections). A multimodal approach to tropical sprue treatment is used. The majority of the time, the therapy is done in an outpatient environment. It focuses on correcting the underlying aetiology and addressing malnutrition with mineral and vitamin supplements. Only a few people with dehydration and weight loss report to the emergency department. These individuals require immediate examination and hydration and electrolyte supplementation. In some situations, the patients will need to be admitted to the hospital, such as: Symptomatic anaemia, severe dehydration, and electrolyte imbalance[39]. Regardless of the diagnosis, assessing and improving nutritional status should be part of any therapy approach. Lactose intolerance treatment, regardless of the reason, involves avoiding dairy, using lactase supplements, and making a strategy to replenish calcium if a shortfall occurs[1]. The use of disease-modifying antirheumatic medications (DMARDs), anti-necrosis factor-alpha, or glucocorticoids to treat rheumatism might promote Tropheryma Whipple infection, which could be lethal in Whipple illness[40]. When removing an obstructing stone in pancreatitis, endoscopic retrograde cholangiopan creatography (ERCP) might be curative, and pancreatic enzyme replacement would be recommended for exocrine pancreatic insufficiency. Because gastrointestinal illnesses are relatively easy to identify and treat, stool microscopy should be used to check for protozoan and helminth parasites initially. When needed, testing for HIV infection is done following counselling. In a juvenile, it is prudent to inquire about gluten sensitivity and do a celiac sprue screening. The next phase in the process is to confirm malabsorption, which includes

estimating faecal fat, dxylose absorption, and vitamin B12 absorption. A small bowel biopsy series and a deep duodenal biopsy are recommended if two of these tests are abnormal. [6]

12. CONCLUSION

Since digestion and absorption are very complicated processes, a malfunction in any of their steps can lead to deficiency in the absorption of any nutrients. Reduced absorption of certain nutrients, impaired GI motility (decreased peristalsis and stasis), altered bacterial flora, infection, or limited blood flow or weakened lymphatics are all possible causes for these problems. In most cases malabsorption can have little to no threat on the person health. However, in severe cases especially in infected cases, diarrhea may cause severe dehydration which may be life-threatening. Therefore, it is important to assess these cases probably for adequate management.

13. AUTHOR CONTRIBUTION STATEMENT

All the authors read and approved the final version of the manuscript.

14. CONFLICT OF INTEREST

Conflict of interest declared none.

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