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Persistent diarrhea

Watery diarrhea continue more than two weeks and the cause is infectious in origin .

10% of acut diarrhea become persistent , it cause 30% of all diarrhea death .

Causes :

Shigella , salmonella ,enter aggregative E coli ,pathogenic E coli ,cryptosporidium (immune compromised patient) and giardia.

Risk factor of persistent diarrhea :

-**malnutrition**: due to delay intestinal repair and lowering immune response

Younger age: highly incidence of death in age less than 18 months

Immune deficiency.

Previous history of persistent diarrhea

Bottle feed : this due to

Bacterial contamination

Hypersensitivity to cow milk

Lactose intolerance

Investigation :

GSE : macroscopic and microscopic (RBS .pus cell, cysts, trophozoite , PH , reducing substance)

Stool for culture and sensitivity

CBC

Serum electrolytes

GUE , blood urea , serum creatinine,

Immunological assay

Treatment :

1-Treat dehydration

2-Antibiotic according to stool culture and sensitivity.

3- Nutritional therapy by :

Reduce the amount of bottle and lactose milke

Provide sufficient vitamin , minerals , protein , and calories

Avoid giving food and drink that aggressive of diarrhea

Continue breast feeding or Lactose free milk

Giving small frequent meal containing cereals , meat , fish….etc

Dysentery

Diarrhea with visible blood in stool .

Causes : Shigella ,H. pylori , salmonella ,enter invasive E coli(0157) ,enter hemorrhagic E coli ,cryptosporidium, plisiomonas, Aeromonas,

Cause 10% of diarrhea under 5 years .

It most sever type lead to loss of protein from large bowel and anorexia due to distraction of large bowel .

Clinical manifestation :

Frequent diarrhea with blood and pus cell associate with tenesmus , fever , abdominal pain ,rectal pain , dehydration.

Complication :

Intestinal perforation , toxic megacolon , rectal prolapse , convulsion, septicemia , and HUS .

Shigella cause 60 % of dysentery .

Diagnosis :

GSE, Stool C&S., blood C&S for septicemia,

CBC(show leukocytosis with neutrophilia in bacterial dysentery and eosinophilia in amoebiasis).

The diagnosis of amoebiasis is by finding the vegetative from E histolytica in stool engulfing RBC in it vacuoles and not only finding the cyst or trophozoite.

Treatment :

Fluid and antibiotics and feeding

Antibiotics used for 5 days better giving orally or IV ampicillin or 3rd generation cephalosporin .

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Chronic diarrhea

Diarrhea continues more than 2 weeks, usually started semiliquid or watery stool and not infectious in origin .

Causes

Parenteral infection *(UTI,OTITS MEDIA , MALARIA)*

Dilatory factors *: over feeding cow milk and soy protein intolerance .*

Carbohydrate malabsorption *:*

Congenital : congenital sucrose isomaltase deficiency , congenital glucose –galactose malabsorption

Acquired : acquired glucose lactose intolerance .

Pancreatic disorder *:CF ,schwachman diamond syndrome , chronic pancreatitis*

Liver disorder *: chronic cholestasis*

Celiac disease *.*

Abetalipoproteinemia

Functional tumor *: zollimger –ellison syndrome , neuroblastoma*

Inborn error of metabolism *, galactosemia ,tyrosinmia.*

GIT anomalies *:malrotation ,familial polyposis , blind loop syndrome*

Chronic inflammatory bowel disease *:ulcerative colitis ,crohn disease ,eosinophilic gastroenteritis .*

Severe malnutrition

Toxic : *chemotherapy or radiation*

Drugs *: iron*

Acrodermatitis enteropathica *(zinc deficiency)*

Endocrine *:thyrotoxicosis , congenital adrenal hyperplasia*

Protracted diarrhea start in the neonatal period *: congenital lactase deficiency congenital chloride losing diarrhea …etc*

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Investigation *:*

GSE ,GUE , CBC ,sweat test ,serum zinc level , barium mail and fallow throw ,intestinal biopsy,.sigmoidoscopy,T3,T4,TSH,seum and urine chromatography

Malabsorption

Definition :

Condition that result from defective digestion or absorption ,which lead to defective assimilationof one or more of the food material.

Types :

---- generalized malabsorption : affected more than one food material , presented with common signe and symptom as abdominal distention ,pale foul bulky stool ,wasted with growth retardation so called **malabsorption syndrome.**

-----specific or individual malabsorption.

1----Generalized malabsorption causes:

Pancreatic : CF, chronic pancreatitis----etc

Sever protein colary malnutrition.

Intestinal :massive resection ,short loop syndrome---etc

Infection: giardiasis ,immune deficiency

Miscellaneous :chemotherapy ,lymphoma

2----Specific malabsorption causes :

Fat :Abetalipoproteinemia

Proteins :inborn error of metabolism

Carbohydrate : *Congenital : congenital sucrose isomaltase deficiency , congenital glucose –galactose malabsorption*

Acquired : acquired glucose lactose intolerance

Pancreatic : congenital lipase deficiency , congenital trypsinogen deficiency

Drug induce ; sulfasalazine , cholestyramine ,phenytoin

Vitamin **: vit.B12** (juvenile pernicious anemia , trans cobalamin deficiency) , **ions and trace elements** (chloride losing diarrhea , Acrodermatitis enteropathica ,menkyes syndrome).

Clinical manifestation :

Generalized malabsorption presented with failure to thieve, abdominal distention , passing of pale bulky foul stools ,muscles wasted ,loss subcutaneous fat ,edema , anemia clubbing ,depigmentation of skin and hair , liability to infection , growth failure , jaundice in Alagille syndrome , diarrhea in celiac disease ,

Investigation :

1-Stool examination : **macroscopic** for color ,volume , Oder and constancy

Microscopic : RBC , pus cell , cyst or trophozoite ,fat globules ,fatty acid crystals and undigested meat fiber

In pancreatic malabsorption the stool contained undigested fatty globules and meat fibber .

In intestinal malabsorption ,stool contain crystalline aggregates of monoglyceriodes and fatty acid .

Chemical analysis : Ph , reducing substance(clinisticks for detection of glucose and clinitest reducing substance)
, 72 hr fecal fat fecal α antitrypsin

2-Biochemical test : blood urea ,electrolytes serum iron ,vit.D assay ,and B12 level.

3-GUE.

4-sweat test : if >60mEq/L of chloride indicate celiac disease.

5-Haematological test: CBC, PT. BLOOD FILEM

6-ultrasound : pancreatic or biliary anomalies .

7-Radiological image :

X-ray for rickets , barium study show dilated loop or thickening ,.

8- small bowel biopsy for :pancreatic enzyme and foe histology

Diagnosis of fat malabsorption :

1-micreoscopic examination for fat drops >6-8 drops /lower pwer field is abnormal .

2- 72hrs. stool for fat ,if >7% it abnormal (steatorrhea).

3-fasting serum carotene :normally >100mg/dl ,<50mg/dl indicate for fat malabsorption.

4- breath carbon -13 lipid

Diagnosis of carbohydrate malabsorption :

1- decrease stool PH (less than 5.6)

2- +ve reducing substance

3-(-ve) oral tolerate test

4- increase breath hydrogen concetration >20ppm

5-increase stool osmotic gap .

Diagnosis of protein malabsorption

1-low serum albumin

2-increase nitrogen loss.

3-increase fecal α antitrypsin

4-increase excretion of CrCL labeled albumin

Diagnosis of pancreatic function :

Direct test by duodenal aspiration and measured of pancreatic enzyme

Blood test : serum trypsinogen.

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disaccharides deficiency :

Carbohydrate digestion start at mouth by salivary amylase , in duodenum by amylase. This converted starches to polysaccharides, oligosaccharides and disaccharides, and in small intestine converted to monosaccharides ready for absorption by action of Disacrharidase secreted from villi of brush border.

Lactase : converted lactose to glucose and galactose .

Sucrose : converted sucrose to fructose and glucose .

Maltase :converted maltose to two glucose molecules and maltotriose to three glucose molecules.

Lactose secreted from tip of villi while sucrose, maltase , isomaltase from side of villi.

Two type of deficiency :

Congenital : congenital sucrase – isomaltase deficiency , congenital lactase deficiency.

Acquired : acquired Disacrharidase deficiency , acquired lactose intolerance.

Pathophysiology :

Absence of disaccharides lead to accumulation of of sugar in small intestine lead to increase of intraluminal osmolality , with water inside the intestine. In The colon the bacterial flora converd the sugar to lactic acidosis, hydrogen , methane and carbon monoxide. Unabsorbed sugar and lactic acidosis lead to osmotic watery diarrhea, acidic stool that cause skin excoriation of buttocks and +ve reducing substance. The hydrogen excreted by lung that give +ve breath hydrogen test.

Clinical manifestation :

Primary lactase deficiency : present after birth when start feeding by watery diarrhea and vomiting , abdominal distention ,colic , irritability and skin excoriation of buttocks.

Treatment by lactose free milk.

Patient with sucrase-isomaltase deficiency (autosomal recessive), present after start solid food contain sucrase.

Treatment : by given sacrosidase enzyme before meal.

Primary lactase intolerance is autosomal recessive that occurs at preschool age and school children with attach of recurrent abdominal pain with or without diarrhea after milk intake.

Treatment : lactase tablet given with meal .

Secondary disaccharides deficiency: occur fallowing damage of villi and brush border after gastroenteritis , treated by temporary feeding with lactose free milk .

Diagnosis :

Stool ph<5.6 , +ve reducing substance , - ve oral tolerance test , increase breath hydrogen , normal intestinal biopsy enzymes deficiency.

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Cow milk allergic reaction :

Due to beta lacto globulin and casein protiens

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