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GASTROENTEROLOGY
DEHYDRATION

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Children, especially infants, are more susceptible than adults to dehydration because of the greater basal fluid and electrolyte requirements per kg and because they are dependent on others to meet these demands.

TYPES OF DEHYDRATION

- According to the degree of dehydration:

1- Minimal or no dehydration (wt loss is less than 3%): normal examination, but urine output may be decreased.

2- Mild-moderate dehydration (wt loss is 3-9%): the child looks normal, fatigued, irritable; thirsty, eager to drink; heart rate is normal to increased; breathing is normal or fast; eyes slightly sunken; decreased tears; dry mouth and tongue; skin turgour is prolonged but less than 2 seconds; cold extremities; decreased urine output; and prolonged capillary refilling.

3- Severe dehydration (weight loss is >9%): Lethargic, unconscious; drinks poorly; tachycardia, with bradycardia in most severe cases; pulses are weak, thready, or impalpable; deep breathing; deeply sunken eyes; absent tears; parched tongue; skin turgour recoils in >2 sec; capillary refilling is prolonged and minimal; extremities are cold, mottled, cyanotic; minimal urine output.

- According to the serum osmolarity & sodium level:

1- Isotonic dehydration: account about 70 % of cases, normal serum sodium & osmolarity, occur when the deficit of Na & water are equal.

2- Hypertonic dehydration: S. Na is more than 150 mg/ dl. Occur when the loss of water is more, 20 % of cases. Plasma & interstitial hypernatraemia lead to shift of water from cells to the interstitial tissue causing intracellular dehydration & collapse of the brain cells. Children are often lethargic, but irritable when touched. Hypernatremia may cause fever, hypertonicity, and hyperreflexia; more severe neurologic symptoms may develop if cerebral bleeding occurs. The skin is doughy in nature, signs of dehydration are less severe, but the tongue is parched, shriveled & small. The condition is dangerous & may lead to permanent brain damage.

3- Hypotonic dehydration: about 10 %, Na level is below 130 mg/ dl with low serum osmolarity, occur when the sodium deficit is more than water deficit. Shift of water from interstitial tissue to the cells causing cellular distension, extracellular dehydration with more hypotension and more severe signs of dehydrations; severe dryness of

mucous membrane, severe loss of skin turgor, sunken eyes, & more liability to shock & renal shutdown.

TREATMENT

In general:
Mild dehydration: treated at home by ORS (Dextrolyte)
Moderate dehydration: treated by ORS at hospital (ORS center), I. V. fluid in certain situation.
Severe dehydration: by I. V. fluid at hospital, & by ORS in certain situations.

ORS (Dextrolyte)

Standard ORS (WHO): It contains 3.5 gm NaCl, 2.9 gm Na citrate, 1.5 KCl, & 20 gm glucose, after dissolution in one liter of water: 90 mEq sodium, 80 mEq Cl, 20 mEq K, 30 mEq HCo3, & 111 mmol glucose.

The low-osmolarity World Health Organization (WHO) oral rehydration solution (ORS): It contains 75 mEq of sodium and 75 mmol of glucose per liter is now the global standard of care.

It is given by spoon every 2-3 minutes, the baby fed in sitting position, & the rest of solution is discarded after 24 hr of dissolution. Vomiting may occur during the first 2 hr of administration of ORS, but it usually does not prevent successful oral rehydration if the ORS is given in small amounts at short intervals (a teaspoon every 1 to 2 min) & the emesis usually lessens over time.

Minimal or no dehydration

1- Deficit (the lost fluid);
There is no need for replacement.

2- Ongoing loss (The fluid will be lost by diarrhea & vomiting):
it is recommended to be replaced
- if <10 kg body weight: 60-120 mL ORS for each diarrheal stool or vomiting episode
-if >10 kg body weight: 120-240 mL ORS for each diarrheal stool or vomiting episode.

3- Nutrition:
Continue breastfeeding, foods with complex carbohydrates (rice, wheat, potatoes), lean meats, yogurt, fruits, and vegetables are also recommended. Fatty foods or foods high in simple sugars (juices, carbonated sodas) should be avoided.

Mild to moderate dehydration

1- Deficit: ORS 50-100 mL/kg body weight over 3-4 hr

2- Ongoing loss: same as above

3- Nutrition: same as above
- limitations of ORS are:
1- severe emesis.
2- an ileus.
3- intussusceptions.
4- shock.
5- Carbohydrate intolerance (rare).
6- High stool output (>10 mL/kg/hr).
In such conditions, I. V. fluid can be given.

Severe dehydration

Treated by I.V. fluid as soon as possible

Approach to a child with severe dehydration:
- Initially:
. Admit the patient to the emergency unite and measure the body weight, insert cannula.
. Rapid assessment of the level of consciousness, PR, BP, RR, urine output, & skin perfusion.
. Aspirate blood for urea, creatinine, and serum electrolytes.

- Start Rehydration:
. Rapid restoring intravascular volume as soon as possible by **Initial Rehydration Therapy (IRT)**: 20 ml/ kg Normal Saline or Ringer Lactate within 20 min or faster, you may need to repeat this dose 3 times until perfusion and mental status improve, hypovolemic patients generally void within 2 hr. After volume resuscitation;

- administer 100 mL/kg body weight ORS over 4 hr or 5% dextrose normal saline IV according to the following equation and serum Na:

Total fluid requirement = deficit + maintenance

Deficit:

= wt (gm) * % of body loss.

e. g. 10 % dehydration in 8 kg wt baby= 8 × 1000× 10/100=800 ml.

Maintenance fluid:

The daily requirement of fluid which is physiologically lost through metabolism, urine, skin, lung, & stool. It is calculated according to the weight of the child:

1st 10 kg= 100 ml/kg/ day.

50 ml/kg /day for every kg above 10 kg

20 ml /kg /day for every kg above 20 kg body wt.

Result of S. Na;

- if S. Na 130- 150 mEq/ l : continue the same fluid, give one half of the total fluid during the1st 8 hr and give the rest within the next 16 hr.

- If S. Na was more than 150: there will be generation of osmoles within the brain to increase the osmolality within the cells of the brain to protect the brain cells from shrinkage. These osmoles will dissipate slowly during correction of hypernatremia. Rapid lowering of the extracellular osmolality during correction of hypernatremia will lead to rapid water movement into the cells of the brain, producing cerebral edema, which can lead to seizures, brain herniation, and death; therefore, it is important to replace the deficit slowly within more than 36 hr and monitoring of S. Na (decrease of s. Na of no more than 12 mEq/ l/ day).

- If S. Na less than 130 mEq/l: a rapid increase of S. Na will precipitate central pontine damage, s. Na must not be increased more than 12 mEq/l/day.
  Continue the same regimen for isonatremic dehydration, but take your care with s. Na monitoring. Patients with neurologic symptoms (e.g. seizures) from hyponatraemia need to receive an acute infusion of hypertonic (3%) saline (5 ml/ kg) to rapidly increase the serum sodium concentration.

- This plan of rehydration is only at beginning of management and all calculations are approximated, especially the assessment of percent dehydration. It is important to monitor the patient during treatment and to modify therapy based on the clinical situation.

- If the urine didn't pass after the adequacy of the circulating blood volume, give a diuretic (furosemide 2-4 mg/kg), if there is no response with deranged renal function tests, discontinue diuretic and start management of acute renal failure.

Ongoing loss:
same as above; if unable to drink, administer through nasogastric tube or administer 5% dextrose in normal saline 10ml/ kg/ motion or vomiting, with 20 mEq/L potassium chloride IV

3- Nutrition: same as above.

WHO METHOD

More easy, more applicable, money saving, the patient classified into:
Group A: called Diarrhea with no dehydration.
Group B: called D. with some dehydration, mild - moderate dehydration.
Group C: called D. with severe dehydration.

Group C:
Give the initial rehydration therapy 30 ml/kg Normal saline (for less than 1 yr within 1 hr, for more than 1 yr within 1/2 hr) , then continue the therapy with Ringer lactate 70 ml/ kg (for child less than 1 yr within 5 hr, for more than 1 yr within 2.5 hr), then assess the state, if no improvement repeat the dose till improvement, if improved, shift to group B.

Gluten-Sensitive Enteropathy
(Celiac Disease)

Clinical disorder results from gluten sensitivity of the intestine, it predominantly affects the proximal part of small intestine results in malabsorption. It is a permanent intolerance to gluten & withdrawal of gluten results in complete remission.

It doesn't present until gluten products have been introduced into the diet, it may present in infancy, children & adult, but the most common period of presentation is between 6 mo and 2 yr of age, and

Etiology

Celiac disease is a T-cell–mediated chronic inflammatory disorder with an autoimmune component. A genetic predisposition is suggested by the family aggregation and the concordance in monozygotic twins, which approaches 100%. There is strongest association with human leukocyte antigen in more than 90%. Type 1 diabetes and other autoimmune diseases are highly associated.

The gluten present in wheat, barley, rye, & possibly oat, the activity of gluten resides in the gliadin fraction, immunodominant epitopes from gliadin are highly resistant to intraluminal and mucosal digestion; incomplete degradation favor the immunostimulatory effect by sensitization of the lymphocytes of the lamina propria and activation of innate immunity mechanisms before activation of the adaptive immune response.

Pathophysiology

Long term dietary exposure to gluten cause sensitization of the lamina propria lymphocytes lead to inflammatory process & damage of the mucosa with characteristic microscopical changes showing villous atrophy, crypt hyperplasia, irregular vacuolated surface epithelium with increased numbers of lymphocytes in the epithelial layer.

The lesion takes few wk – 2 yr of exposure to gluten to develop, & few month- 2 yr to change to normal on gluten free diet.

Screening & incidence

Screening test by serological markers show the incidence of celiac disease varies with population, e.g. 1/ 4000 in Denmark & 1/ 150 in Ireland.

Screening tests indicate the asymptomatic cases form 5 -7 times the symptomatic patients.

Clinical features

The mode of presentation is vary considerably;

- Most patients present with **diarrhea** (**constipation** in some).
- Children can have **failure to thrive or vomiting** as the only manifestation, 10% of children referred to endocrinologists for growth

retardation without an endocrine or overt gastrointestinal disorder have gluten sensitivity.

- **Anorexia** is common (in contrast to infants with cystic fibrosis).
- Infants is often **clingy, irritable**, **and unhappy**.
- **Pallor and abdominal distention**.
- **Digital clubbing** can occur.
- **Other presentation** like alopecia, bone pain, lymphocyte gastritis, rectal prolapse, asymptomatic hepatitis, dermatitis herpitiformis, & neurological manifestation (like ataxia, developmental delay or epileptic convulsion).
- It is more commonly associated with **other conditions** like type 1 D M, autoimmune thyroiditis, rheumatoid arthritis, pernicious anemia, isolated IgA deficiency, Addison disease, & Down syndrome.

Investigation

- Hb, blood film (mostly show microcytic or dimorphic anemia).
- serum protein and albumin.
- stool pH & reducing substance.
- Serological markers:

  . **Anti-gliadin IgA and IgG antibody** tests are no longer recommended tests due to lack of specificity.

  . The **anti-endomysiel IgA** and **anti-tissue transglutaminase IgA antibody** tests are highly sensitive and specific. The anti-endomysiel IgA antibody test is relatively expensive; interpretation is operator dependent and prone to errors so that it has largely been replaced by anti-tissue transglutaminase IgA antibody tests, which are simpler to perform and have similar sensitivity and specificity.

- Intestinal biopsy is the gold standard for diagnosis.

Many diseases may cause flat intestinal mucosa, e.g. Infections such as rotavirus enteritis & Giardia lamblia, undernutrition, cow's milk protein or soy protein intolerance.

Treatment

Long life gluten free diet, response occurs within a week with change in mood, increase appetite & wt & improvement of diarrhea. No long-term complications from a gluten-free diet have been recognized.

Patient is more liable to develop malignancy of the intestine (lymphoma), and other forms of cancer. The long life strict GFD will reduce the risk of all these cancers.