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- **Impact of Type 2 Diabetes Mellitus on Nutritional Status**
- **Use of home based naso-jejunal feeding for long term nutritional support in a chronically ill patient A case report**

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To Our Readers

Dear Readers,

We, at the CRNSS, once again thank you for your valuable support and patronage as the Update Series"Nutrition in Disease Management" over the past 11 years as the series would now be completing 50 issues(the current issue is No. 50)!

This issue carries a lead review article which discusses the potential impact of nutritional status on overall management of individuals with Type 2 Diabetes Mellitus. It also draws attention to the fact that appropriate and timely nutrition intervention and maintaining an optimum nutritional status can positively influence pharmacological management in these individuals.

The second article is a case report which describes how prolonged nasojejunal feeding in the specific clinical situation which was nutritionally challenging is a safe and effective route of providing nutritional support in sick patients, even in the home setting.

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Impact Of Type 2 Diabetes Mellitus On Nutritional Status

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INTRODUCTION

Diabetes mellitus, a metabolic disorder, is primarily a disorder of carbohydrate metabolism. Pathophysiology is related to "insulin resistance" means that body cells do not respond appropriately when insulin is present. Type 2 diabetes mellitus is a more complex problem than type 1 DM, but is sometimes easier to treat, especially in the early years when insulin is often still being produced internally. Diabetes mellitus with a known etiology, such as secondary to other diseases, known gene defects, trauma or surgery, or the effects of drugs, is more appropriately called secondary diabetes mellitus or diabetes due to a specific cause. Examples include diabetes mellitus such as MODY [increasingly seen in adolescents] or those caused by hemochromatosis, pancreatic insufficiencies, or certain types of medications (e.g., long-term steroid use).

The onset of type 2 diabetes has been most common in middle age and later life, although it is being more frequently seen in adolescents and young adults due to an increase in child obesity and inactivity. Excess energy intake and excess body weight are risk factors strongly associated with diabetes. Severe complications can result from improperly managed type 2 diabetes, including renal failure, erectile dysfunction, blindness, slow healing wounds (including surgical incisions), and arterial disease, including coronary artery disease.

According to WHO International Classification of Diseases, "Malnutrition is a real diagnosis" and is defined as "comprising a group of clinical conditions, with

many causes and manifestations and arising from abnormalities associated with nutrient intake, digestion, absorption, metabolism and excretion". The evaluation, prognosis, and treatment depend on its underlying mechanism and additional clinical factors. Diagnosis obligates to consider potential therapies by: 1. Putting a label on a pathogenic process; 2. Forcing consideration of the etiology of that process; 3. Suggesting the prognosis of that process & 4. Mandating consideration for therapy. Paradoxical situations need to be assembled and integrated on four planes, as to how

1. Nutrition Effects on Islets of Langerhans, both in terms of the progression of development of the organ per se and its function
2. Malnutrition Effects on Islets of Langerhans, both in terms of the limitations in the development of organ per se and its resultant malfunction
3. Effect of Diabetes mellitus disease on nutrient metabolism and hence nutrient requirements
4. Effect of Treatment Modality on nutritional requirements

Malnutrition Related Diabetes Mellitus MRDM, presenting in two forms fibrocalculous pancreatic diabetes [FCPD] and protein-deficiency diabetes mellitus [PDDM] accepted as a separate entity in 1985 by the World Health Organization [WHO] Study Group on Diabetes Mellitus [1] answers the question "how is malnutrition a causative agent for Diabetes Mellitus?", but a literature search to answer the question "how does Diabetes Mellitus cause Malnutrition?" reveals a lacunae and paucity of evidence-based documentation [2,3].

A total of 117 diabetic patients [50% each, male and female] aged 20-65 years suffering from diabetes for at least one year were enrolled in this cross sectional study conducted in Nawabganj Diabetic Center, a branch of Bangladesh Diabetic Association. Subjects were categorized as overweight and obese. The conclusions drawn from this study were that health education should be aimed to enhance awareness of particularly rural and illiterate people for regular visit to nearby diabetic center and to strictly adhere to dieticians' advice [2].

Nutritional status of 115 subjects [56 male and 59 females] suffering with NIDDM and attending a diabetic clinic in Mysore, was assessed using anthropometrics, biochemical parameters, diagnostic symptoms, food intake and nutrient adequacy. 34% males and 36% females were overweight, 5% males and 13% females were obese. 49% and 34% of female subjects and 41% and 17% of male subjects had higher than ideal tricept skin fold and waist hip ratio respectively. Diet survey showed the intake of carotene, iron, vitamin C, niacin, and riboflavin to be inadequate and that of calcium, fat and thiamine to be adequate. Among associated risk factors of diabetes, heart disease and eye problems were the most prominent [3].

Diabetes Mellitus The Indian Scenario

Despite, Diabetes being a major health problem worldwide and India, in particular, with a larger number of people with diabetes than any other country in the world [4] this *aspect of understanding as to how Diabetes mellitus causes Malnutrition? that is imperative to formulate nutrition support prescriptions and management strategies is grossly lacking.* With WHO estimates, that India had 32 million diabetic subjects in the year 2000 and this number would increase to 80 million by the year 2030 [5], and even the International Diabetes Federation also reporting that the total number of diabetic subjects in India would rise from 41 million in 2006 to 70 million by the year 2025 [6], it is *mandatory that such an exercise is undertaken on a war footing.*

It has been indicated and reported that the Type 2 Diabetes epidemic in India is a result of affluence and changing lifestyle. Gene- environment interactions also appear to be responsible for this rapid increase in the prevalence of Diabetes. Indians have a younger age of onset of diabetes compared to other ethnic groups [7]. Despite having lower BMI, Indians tend to have greater waist circumference thus having a greater degree of central obesity, have more visceral fat and greater insulin resistance [8, 9]. The above risk factors tend to develop early in Indian subjects. Many studies demonstrated that life style factors such as urbanization, socio economic status, stress, sedentary life style, excess energy consumption, generalized and central obesity to be the important diabetes risk factors [10, 11]. A population- based approach to prevent or control Diabetes includes enhanced physical activity and regulated energy intake. There is an urgent need to modify lifestyle, especially in the urban areas of India, by promoting physical activity and *healthy dietary choices.*

Nutrition/Malnutrition nexus in Diabetes Mellitus type 2:

The pathophysiologic changes with implications on nutrition/malnutrition in type 2 diabetes are complex. The degree of glucose wastage by excretion in the urine is the primary cause of the metabolic symptomatology. The classic symptoms of diabetes polyuria [frequent urination], polydipsia [increased thirst], polyphagia [increased hunger], fatigue and weight loss, all have nutritional implications. The energy deficit due to the glycosuria gives rise to hunger and polyphagia. This and the water loss lead to thirst, polydypsia and weight loss. Protein ingested is largely made into sugar and therefore used inefficiently. Unlike type 1 diabetes, while there is very little tendency toward ketoacidosis, it is not unheard of, as loss of both exogenous and endogenous carbohydrate results in rapid mobilization of fat leading eventually in severe cases to ketosis and coma.

Hyperglycemia adversely affects fluid balance and immune function. As the filtered load of glucose increases, it eventually exceeds tubular reabsorptive capacity. As a result, glucose remains in the tubular lumen and acts as an osmotic diuretic, increasing the urinary loss of electrolytes and water. Hyperglycemia related impaired immune function is associated with abnormalities in granulocyte adhesion, chemotaxis, phagocytosis, and intracellular killing. Phatocytosis can be corrected or substantially improved with control of blood glucose.

Unanticipated discontinuation of nutrition support, is the potential cause of hypoglycemia. Other non nutritional causes are: resolution of severe stress, discontinuation or decreased doses of glucocorticoids or sympathomimetic agents, renal dysfunction, severe hepatitis, sepsis, and diabetic gastroparesis.

Irrespective of whether the individual is symptomatic or asymptomatic, administration of sugar packets/cubes/tablets/gels or fruit juice orally or administering of glucose/dextrose by IV route is mandatory to resolve hypoglycemia.

The nutritional implications of managing Diabetes Mellitus only through drug therapy would be an increase in insulin levels which could, in some situations, result in hypokalemia, hypophophatemia, and hypomagnesemia, all of which have a detrimental effect on nutritional status. Hyperinsulinemia shifts potassium and magnesium into skeletal muscle and hepatic cells. Glucose-

and insulin-stimulated glycolysis stimulate the cellular uptake and utilization of phosphorus for the phosphorylation of glucose and fructose and for the synthesis of adenosine triphosphate.

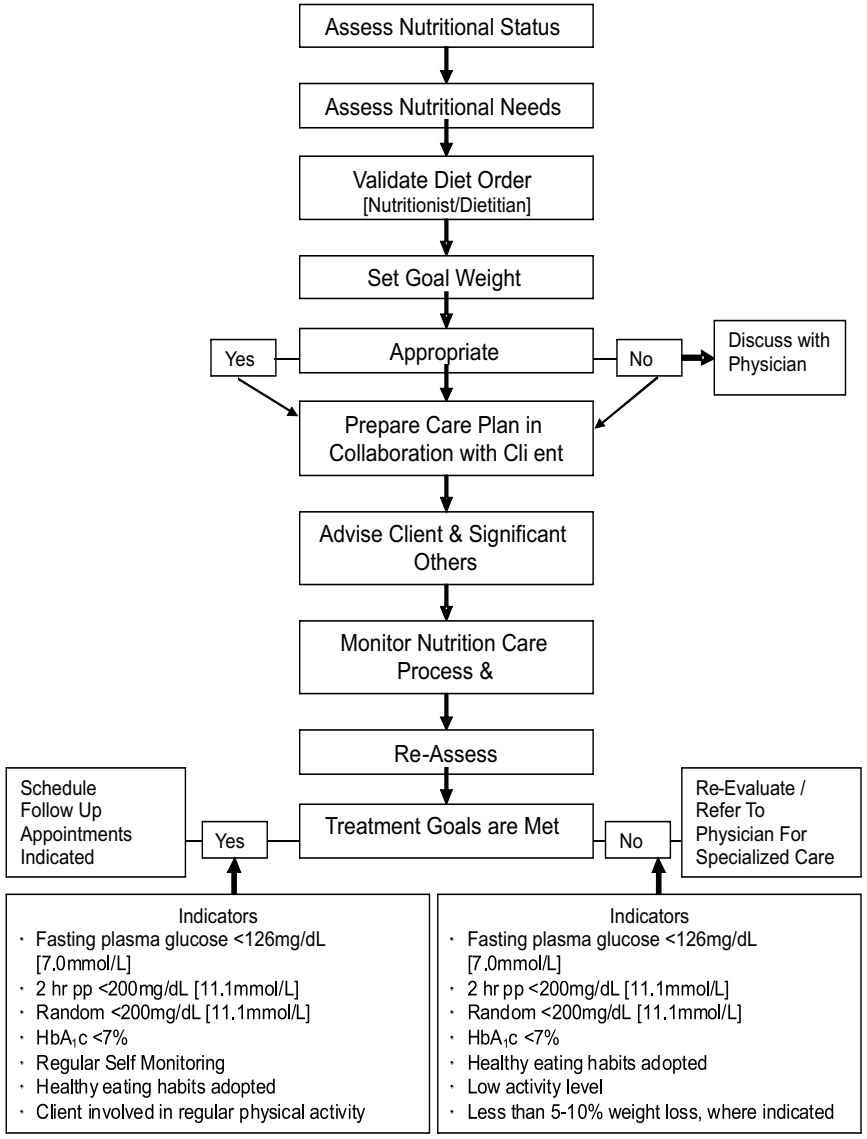
Under the classic etiology classification [1. too little in; 2. failure to absorb; 3. too much out; 4. failure to utilize; 5. increased requirements] of nutritional deficiency, diabetes mellitus appears to subscribe to all of the 5 categories in varying degrees. One defect is that people lose their sense of satiety the feeling that they are full and should stop eating. Also the stomach empties too quickly in type 2 diabetes at least initially. There are other gastrointestinal flaws such as inappropriate levels of incretin mimetics the substances that respond to a meal and these incretin mimetics are metabolized too quickly. The alpha cells of the pancreas do not 'read' the blood glucose level correctly. Abnormal function of intestinal mucosa results in Diabetes Mellitus with steatorrhoea.

Nutritional Management for Diabetes:

Nutrition therapy is an integral component of the successful management of Diabetes Mellitus and has remained one of the most challenging aspects of care due to the complexity of nutrition issues. Inputs in developing appropriate nutrition intervention strategies is extremely important in ensuring that the plan is appropriate to the individual's lifestyle and cultural practices. All nutrients play an important role in diabetes management, moderation in intake is usually the key.

To document the impact of diabetes mellitus on nutritional status specifically, the critical path would require an intensive interventional nutritional care [Figure 1]. The major goals of therapy are to achieve metabolic control and to prevent or delay the macrovascular and microvascular complications of Diabetes. Overall therapy for Diabetes includes: education; nutrition therapy; physical activity and exercise; blood glucose monitoring; behavior modification and self-care and management of medication [if required]. The components of intensive interventional nutrition care are: physical examination, weight, blood pressure, other concurrent abnormalities, laboratory, fasting and/or 2 hour post prandial blood glucose, fasting lipids and micro albuminuria at the initial visit. The frequency of follow-up is: physical examination annually; weight monthly or every regular visit; blood pressure every regular visit; other concurrent

Figure 1: CRITICAL PATH FOR THE NUTIRTIONAL MANAGEMENT OF DIABETES MELLITUS [12]



abnormalities every regular visit; laboratory quarterly more often if there are complications; fasting and/or 2 hour post prandial blood glucose 1. quarterly if treatment changes or if client is not meeting goals 2. twice per year if stable; fasting lipids annually, unless abnormal; and micro albuminuria semi annually.

Indications for referral to Nutritionist/Dietitian are: poor appetite or client not eating; clients who express difficulty following nutrition treatment regimen that must be handled with competence or expertise and for development of meal plan; clients who have difficulty maintaining treatment targets; e.g. acceptable blood pressure, blood sugar or weight goals; change of treatment, e.g. from diet only to oral hypoglycemic agent or a shift to insulin.

The expected outcomes of nutritional care are: [1] improved nutritional status; [2] improved food and nutrient intake; [3] improved knowledge; [4] positive behavior change; [5] improved laboratory values, weight, blood pressure; [6] risk factor reduction; [7] prevention or delay of complications; [8] ability to identify and access

Available community resources; [9] reduced hospital admissions; [10] improved self management; [11] improved quality of life.

In view of the above facts and the conclusion one is forced to drawn thereon that *healthy dietary choices* be promoted, the objective of this article was to foster clear understanding and interpretation of the nutrition/malnutrition nexus. The express aim was not just to highlight the significance of the effect of Diabetes mellitus disease since its advent/manifestation and the duration of its manifestation on the nutritional status of the afflicted individual, but to undertake evidence based documentation exercise in this direction.

Hence, assessment of nutritional status in 2122 type 2 diabetics, using anthropometry [height, weight, BMI], biochemical parameters [hemoglobin, fasting blood sugar, post prandial blood sugar and HbA1c diabetes status, serum albumin and lipid profile] and manifestation of diabetic cardiopathy, diabetic neuropathy, diabetic nephropathy, and diabetic retinopathy in relation to the duration of Diabetes Mellitus has been undertaken at MV Hospital for Diabetes, WHO Regional Center for Diabetes.

Preliminary data analysis reveals that within 5 years, duration of type 2 diabetes excursions in the fasting and postprandial blood sugars are erratic and unsuccessful attempts to control blood glucose levels through polypharmacy are also increasing, with an increased need for inclusion of insulin as a treatment modality. In general, intake compliance to prescribed oral

hypoglycemic agents and/or insulin is adhered to but there is a great mismatch with respect to dietary intake resulting in malnutrition as well as diabetes related complications nephropathy, retinopathy, cardiopathy and neuropathy. With advent of diabetes related complications the tendency is to further restrict dietary intake. Several foods/food groups, especially pulse, legumes, lentils, vegetables and fruits are excluded, meal schedule indicates long intervals between each meal, and there is increased consumption of black tea/coffee.

The study protocol also attempts to document the medication modality and its impact on the nutritional status. This is also imperative as 6 types of oral medicines (and 11 individual drugs) are now available to help people with type 2 Diabetes control their blood sugar when modifications in diet and lifestyle are not sufficient, but they are not without side- effects that may impact nutritional status.

An in-depth analysis is ongoing as it would be imperative to use this data to not only foster identification of the key factors that result in the manifestation of the disease and advance its progression but also contribute to a greater understanding of their interplay with respect to the Indian diabetic populace and proceed with formulating nutrition support prescriptions and management strategies and evaluating their efficacy.

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Use of home based naso-jejunal feeding for long term nutritional support in a chronically ill patient A case report

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This is an overview of the nutritional interventions in acutely ill patients.

INTRODUCTION:

A 78 year old male patient was admitted to the hospital with sudden loss of consciousness at home and laboured breathing. He was continually being provided assisted ventilation through an Ambu bag and mask on way to the hospital . The patient was a known case of diabetes, hypertension, hypothyroidism and surgically drained subdural hematoma who developed right hemiparesis post-operatively. On admission, the patient was diagnosed to have pulmonary embolism (D-Dimer test positive) leading to cardio-respiratory arrest.

On examination, at the time of arrival, there was no spontaneous breathing, no palpable pulse and no recordable blood pressure. After extensive cardio-pulmonary resuscitation carried out for 45 minutes, the patient was resuscitated. The patient was kept on ventilatory support for few days because of the impaired neurological status of the patient and poor ventilatory efforts and impaired deglutition reflexes.

Biochemical profile showed major derangements in fasting blood glucose (400 gm/ dl); serum transaminases (SGOT/ SGPT- 834/ 740 I.U., respectively); total leukocyte count (23,600/ cu.mm); total serum protein (5.9 gm/ dl); serum albumin (2.0 gm/ dl) and hemoglobin (9.5 gm/ dl). However, the serum electrolytes (Na 148; K 4.0 mEq/ day); blood urea (39 mg/ dl) and serum creatinine (1.0 mg/ dl) were normal.

During the course of hospital stay, serum creatinine started to increase gradually indicating pre-renal and ischemic renal injury due to prolonged hypotension as a

result of cardiac arrest.

Course of treatment:

During the course of admission, the patient's neurological condition did not improve much and he had sustained hypoxic brain damage.

Blood sugar monitoring was performed every 2 hourly initially and then appropriately monitored in response to the patient's status. Albumin was infused intravenously 6 times during the total hospital stay. A total of 5 units of blood were transfused during hospital stay. On the 11th day of hospital stay, the endotracheal tube was removed and a tracheostomy was performed as the the patient was unable to swallow (owing to an impaired neurological status).

Feeding protocol:

Feeding was accomplished using a nasogastric feeding tube and initiated on the 3rd day in hospital with a minimum daily volume of 200 ml in the form of 50 ml every 4 hours to assess feed tolerance. The feeds were well tolerated by the patient, with no significant nasogastric aspirates and the volume of feed was gradually increased from approximately 300 ml/ day (50 ml X 3 hourly) on the fourth day in hospital to 450 ml/day on the fifth day and then to 800 ml/day on the sixth day in hospital. On the sixth day, the patient was being administered a total of 850 kcal energy and 15 gm protein. The enteral feed was milk-based. There was no overnight feeding.

The total energy requirements (in the form of Resting Energy Expenditure REE) were calculated by the Harris Benedict's equation (given below).

REE = 66.47 + 13.75W + 5.00H-6.76A
Where, W weight of the patient; H Height of the patient and A age of the patient.

A stress factor of 1.2 was multiplied and the final REE was calculated to be 2150 Kcals/ day. The protein requirement was calculated as 1.2 gm protein/ Kg Ideal body Weight/ day and was approximately 80-85 gm/ day. The feed composition was planned in such a way as to include all food groups (milk, cereals, pulses, vegetables and fruits).Oil was added to increase the energy density and viscosity of the feed. Commercially available immunonutrients (glutamine) and a commercially available protein supplement were also added to the prepared

enteral feed.

The feed volume was gradually increased to a maximum of 2500 ml daily (250 ml every 2 hourly) which provided a daily energy intake of 2185 kcal and a daily protein intake of 85 gm on the 20th day of hospital stay. However, on day 22, there was documented enteral feed intolerance in the form of increasing nasogastric aspirates. The feed intolerance persisted over the next 4 days despite changing the composition of the feed to a semi-elemental lactose-free formula. At this stage, the treating medical unit, in consultation with the nutrition team, decided to change the route of enteral feeding from nasogastric to nasojejunal through a nasojejunal tube inserted fluoroscopically. The patient and his family were also informed about the alternative option of feeding through a surgically placed feeding jejunostomy, which the patient refused. The patient was subsequently discharged from the hospital on nasojejunal feeding which has been continued effectively till date (7 months following discharge from hospital). At the time of discharge, the patient's blood investigations which were deranged at admission had become normal.

There is evidence that nasojejunal feeding is more effective as compared to nasogastric feeding in specific clinical situations necessitating prolonged enteral feeding using a semi-elemental feed preparation (1). It is also useful in decreasing both the cost of and need for parenteral nutrition when used for the correct indication(s) and judiciously.

However, there are certain precautions that have to be considered when using NJ feeds:

- Placing of the tube is difficult and has to be placed and monitored radiologically from time to time (1);
- The tube can easily become blocked so requires frequent flushing (2);
- Longer periods of feeding may reduce mobility of the patient (3);
- Type of feed given may require review (4).

Take Home Message :

Nasojejunal feeding can be an effective modality for prolonged enteral feeding, even in the home setting, in clinical situations where it is clearly indicated. However, the feeding protocol requires a patient-tailored approach dictated by the specific clinical situation and patient's response.

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Understanding Practical Issues Involving Feeding In Infants And Young Children Through Case Scenarios

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Q.1.

A 2 month old exclusively breast-fed baby boy is brought to you with complaints of vomiting milk 3-4 times daily and passing light yellow semi-liquid stools 12-15 times daily for the past 4-6 weeks. His birth weight was 3.1 kg, he was born by normal (vaginal) delivery, he is the first child of his parents. His current weight is 5.2 kg. He expresses willingness to feed, is active and alert. There is no perianal redness or excoriation. What is his problem? How will you manage him?

EXPLANATION:

The practical approach would be to leave the baby alone, there is nothing wrong with him! What must be done, however, is reassuring the parents that their baby boy is normal and growing well. Frequent, small volume, light yellow coloured stools are commonly observed in exclusively breast-fed infants and there is wide variation in stool frequency. There is absolutely no cause for alarm if the exclusively breast-fed child is feeding and growing well.

Q.2.

A 18 day old baby girl has been brought to you with complaints of passing frank blood in stool (approx. 3-5 ml) on at least 4 separate occasions since birth. She was delivered normally (vaginally), birth weight was 2.7 kg and she is the first and only child of her parents. She has been actively feeding, alert and has no frequent vomiting. She has been on both breast feeds (predominantly) and occasional top feeds since birth. How will you manage this baby?

EXPLANATION:

An important, although not very common, cause of frank bleeding in an alert and active infant is CMPA (cow's milk protein allergy). The term is a misnomer

because it refers to allergy not only to cow's milk but also buffalo milk and many other animal milk and milk products like curd, buttermilk and cheese. The diagnosis is clinical (demonstrating disappearance of bleeding on withdrawing animal milk protein from the feed), although, there is recent evidence that this can be supported by a rectal mucosal biopsy showing more than 6 eosinophils/high power field. The practical approach would be to first ensure that the infant is exclusively breast fed and stop feeding any animal milk to the infant. If at the end of 2 weeks, the blood is still present, the mother should be strictly instructed to completely avoid all milk and milk products in her own diet because, in severe cases of CMPA, the animal milk protein antigen in the mother's body is excreted into her breast milk which is then passed on to the baby during feeding. If the bleeding still persists, the baby should be investigated for other causes of gastrointestinal bleeding such as Meckel's diverticulum and duplication cyst of small intestine.

Q.3.

A 45 day old infant girl, birth weight 2.3 kg has been brought to the pediatric emergency with the complaints of increasing frequency of vomiting since Day 12 of life, the baby has not responded to modifying the feeding strategy (small frequent feeds) initially and subsequently to pharmacotherapy ranitidine for the past 2 weeks the problem has actually worsened. The baby has gained weight very poorly (current weight 2.5 kg, only 200 gm weight gain in 6 weeks)! What would you like to do how will you investigate and manage this baby?

EXPLANATION:

Failure of the vomiting to respond to both modification in the approach to feeding as well as pharmacotherapy for suspected gastroesophageal reflux(which ideally should have been documented with a milk scan before starting treatment) raises the most likely possibility of congenital hypertrophic pyloric stenosis. An ultrasound abdomen would be helpful but is not a substitute to a thorough clinical evaluation for the disorder. The management is surgical. The differential diagnosis should include various inborn errors of metabolism when the diagnosis of pyloric stenosis cannot be documented.

Q.4.

A 7 month old baby girl weighing 6.8 kg was exclusively breast-fed till 5 months of age, then started on a formula feed in addition to weaning foods. She

developed loose stools which lasted 6-7 days, stools frequency was 18-20 per day, small volume stools, no mucus or blood, some stools were passed explosively. The baby has had perianal redness followed by excoriation. Stool pH is 5.5, reducing substances +++. She has never had increased frequency of loose stools before. What is the likely explanation? How will you manage?

EXPLANATION:

A very common cause of watery diarrhea in the latter half of infancy is due to the rotavirus this is the case here. The investigations also indicate that she has lactose intolerance, which, in this part of the world, is usually a secondary (post-infective) lactose intolerance, is transient, lasting a few days to weeks. This is unlike primary lactose intolerance which is common in Scandinavia and is due to congenital lactase deficiency (milk is avoided throughout life). In this child, a practical approach would be to decrease the total daily intake of milk by the infant (but not discontinue milk altogether, unless the stool frequency and/or volume increases) and provide the required quantity of milk subsequently after the infant recovers from the diarrheal episode.

Q.7.

A 4 year old boy weighing 12 kg and height 92 cms is brought to the pediatric outpatient department by his parents with the complaint that he has been growing poorly since the age of 2 years. He was 3.2 kg at birth. He is active, alert, feeds well and there is no vomiting, abdominal pain. He passes intermittent bulky stools. What could he be having? What would you do?

EXPLANATION:

The most likely explanation is that this boy is suffering from Celiac Disease (gluten allergy), a genetic disorder characterized by intolerance to dietary wheat and wheat products, about a third of children with Celiac Disease present with inadequate weight and height gain and absent gastrointestinal symptoms. A serological test (anti - tissue transglutaminase antibody) initially and if positive, should have the diagnosis confirmed by a duodenal mucosal biopsy obtained by an upper gastrointestinal endoscopy. If the diagnosis of Celiac Disease is confirmed histopathologically, the child should be started on a gluten-free diet which should be adhered to very meticulously for the rest of his life.

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