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Case Report

Gastroesophageal reflux disease with Thal fundoplication

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Gastroesophageal reflux (GER) is a common phenomenon among healthy infants, with approximately 50% of infants aged 0 to 3 months and 67% of infants aged 4 months experiencing at least one episode of vomiting per day. GER defined as regurgitation of gastric contents into the esophagus or mouth. GER typically improves through the first postnatal year, with only 5% of healthy 12 month old infants experiencing vomiting.[1,2] Complicated GER or gastroesophageal reflux disease (GERD) has been reported to affect up to 8% of infants and children with GER.[1,3] Antireflux procedures (ARPs) are increasingly offered to control GERD symptoms. We report a case of a baby with GERD and treated with Thal fundoplication procedures. Fundoplication procedure is rarely performed in management of GERD, and this is the first fundoplication procedure in children with GERD at M. Djamil Hospital.

The Case

A 6 month old baby boy, was referred to M. Djamil Hospital, Padang on April 3, 2007 with four days history of abdominal distention. He had vomited 2-3 times per day since born and pallor appearance. On physical examination, he looked moderately ill. Body weight (BW) was 4.5 kg (< percentile 3 CDC 2000), body length (BL) was 64 cm (percentile 10-25 CDC 2000), with head circumference was 43 cm (normal for Nelhaus standard), BW/Age was 62%, BL/Age was 94% and BW/BL was 71%. Sunken anterior fontanel and eye with anemic conjungtiva were noted. There was purulent discharge from right ear, distended and tender abdomen, normal bowel sound, no enlargement of the liver and spleen, slow return of skin turgor, and pitting edema on pretibial region.

Laboratory results disclosed hemoglobin 7.3 g/dl, erythrocyte count 2.93 million/μL, hematocrit 23%, reticulocyte 7%, MCH 24.91%, MCV 78.50%, and MCHC 31.74%. From blood smear, erythrocyte was microcytic hypochrome, leukocyte and platelet morphology was normal. Blood glucose was 72 mg/dl, cholesterol 67 mg/dl, sodium 130 mg/dl, potassium 2.5 mg/dl, chloride 90 mg/dl, serum iron 38.5 μg/dl, total iron binding concentration 176.6 μg/dl, albumin 2.03 g/dl, and globulin 1.87 g/dl. No proteinuria was found on urinalysis. Barium meal examination suggested a hypertrophy pyloric stenosis (HPS), but further investigation with abdominal ultrasound did not proof the diagnosis.

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Based on history, clinical manifestation and laboratory examination, the patient was diagnosed as hypertrophy pyloric stenosis (HPS), kwashiorkor, iron deficiency anemia, and perforative otitis media. Patient was treated with WHO protocol for malnutrition for 4 weeks, upright position 30 minute after meal and small frequent feeding. After 4 weeks BW was increased to 4.9 kg (< P3 CDC 2000). Laboratory results revealed hemoglobin 11.3 g/dl, leukocyte count 10.000/μL , platelet count 585.000/μL, differential leukocyte count 0/2/2/2/53/37/6, reticulocyte 15%, sodium level was 148 mg/dl, potassium 4.3 mg/dl, and chloride 122 mg/dl. If formula volume was raised above 40 cc, he would vomit.

Pilorotomy (pylorectomy???) was performed on this patient, but hypertrophic pylorus was not found. During the surgery, surgeon found dullness in "angle of His" area so that, they performed Thal fundoplication procedure (**Figure 1**) to eliminate GERD in this patient. After hospitalized for 8 weeks, the patient can consume 100 cc of milk per times without vomiting. Body weight increased to 5.3 kg (<P3 CDC 2000), BW/BL 78%. No pallor or fever experienced. On June 9, 2007, he was sent home. His mother had been taught how to take daily care of the baby.

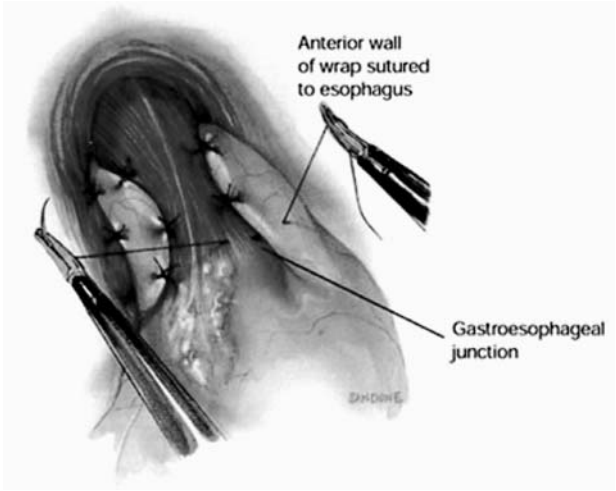

Figure 1. Thal Fundoplication
From Renee C. Minjarez and Blair A. Jobe. GI Motility online (2006) doi:10.1038/gimo56

On follow up, 6 months after the patient was sent home, there was no feeding problem reported. Body weight was 9.6 kg (P10-25 CDC 2000), BL 75 cm (P1-25 CDC 2000), BW/A was 88%, BL/A was 96% and BW/BL was 94%. He can sit without support, walks with 2 hands held, and speaks 3-5 word.

Discussion

On admission, the working diagnosis was acute diarrhea with moderate dehydration, suspect hypertrophy pyloric stenosis (HPS), observation of meteorismus *et causa* suspect unbalance serum electrolyte, microcytic hypochrome anemic, and under nutrition.

There are several diagnosis often provide in non bilious vomiting infants include milk intolerance, HPS, duodenal atresia, and GER. In this patient, the diagnosis of milk intolerance can be eliminated because the baby is breastfed. Meanwhile, patient with duodenal atresia if not given a parenteral diet are unlikely to survive until 6 months, because the food intake cannot reach intestine, then being absorbed.[4] Approximately 85% of infants vomit during the first week of life, and 60-70% manifest as clinical gastroesophageal reflux at age 3-4 months. Resolution of symptoms occurs in approximately 90% of infants by age 8-10 months.[5]

Typical presentations of infant with HPS begin at average 3 weeks after birth, but may occur at any time between birth and 5 months of age. The onset of clinical symptoms is heralded by regurgitation of feeds and progresses to the classic (often) projectile, non bilious vomiting. Physical examination may reveal a dehydrated or wasted infant who is an avid sucker. An olive-like pyloric mass may be palpated in the upper abdomen in up to 90% of patients. Imaging procedures are reserved for instances when pyloric stenosis is highly suspected, but the infant does not have a palpable pyloric mass. Upper gastrointestinal radiography with contrast and ultrasound was used.[6]

This patient began non bilious vomiting from birth, not at 3rd week of life; this may happen if the HPS occur with GER. There was no olive-like pyloric mass in this patient so we decide to perform barium meal and abdominal ultrasound, but the result was contradictive, the patient was treated with conservative management for GER such as upright position for 30 minute after meal, small frequent feeding and food thickening. However, the patient was vomit if formula volume was raised above 40 cc.

Prokinetic agent should not be used for regurgitation or vomiting without prior upper gastrointestinal radiography to assure normal anatomy, including the absence of malrotation with its potential for intermittent volvulus as the cause of the vomiting.[7] We suspected HPS as gastrointestinal abnormality, so we did not give prokinetic to the patient.

Iron is actively transported across the placenta at a gradient that favors the fetus. Consequently, newborn iron stores are altered only when maternal iron deficiency is severe. Approximately 85% of iron stores in the newborn are in RBC hemoglobin. Infants have a dietary requirement for iron because they are growing rapidly and expanding their RBC mass. The peak incidence of dietary iron deficiency in infancy occurs between the ages of 6 months and 2 years. It rarely is encountered in term infants younger than 4 months of age unless the baby has lost iron through blood loss.[8] This patient was 6 month old and had feeding problem, thus had high risk to suffer iron deficiency anemia. Iron deficiency anemia usually treated with oral ferrous sulfate (3 to 6 mg/kg per day of elemental iron). The iron is absorbed better if given between meals and along with a vitamin C containing beverages . It is appropriate to have the patient take iron for at least 1 to 2 months after the hemoglobin level has been corrected to fill iron storage sites.[9] After 5 week therapy, hemoglobin had increased to 11.3 g/dl and reticulocyte to 15%.

Kwashiorkor is a form of protein energy malnutrition (PEM) characterized by insufficient protein intake and reasonable carbohydrate intake. It is marked with hypoalbuminemia resulting in edema, dermatosis, and growth retardation. Clinical features can include irritability, mild growth failure, developmental delay, and edema of the extremities, which is the hallmark of kwashiorkor. Hypoalbuminemia is universal. Interestingly, affected children often have normal or near normal weight and height for age.[24] On admission, patient had no clinical signs of kwashiorkor, such as edema, mental changes, changes in hair color or skin disorders, and BW/A was 62%, BL/A was 94%. and BW/BL was 71% that still can be grouped in under nutrition. On 2nd week after admission, there was tibial edema, decreased serum albumin (2.03 g/dl), and cholesterol level was 67mg/dl so we diagnosed that patient as kwashiorkor. The reasons beyond this phenomenon maybe because at admission patient was dehydrate in which edema rarely seen, while serum albumin level was not markedly decrease, Politzer and Wayburne[5] stated that edema was seen if the serum albumin of about 1,3-1,9 mg/dl.

Ear, nose, and throat complications of GERD include hoarseness, laryngitis, sinusitis, dental erosions, and recurrent otitis media.[2] In this patient there was acute otitis media which suspected as complication of GERD.

Consultation with the Pediatric Surgery Department established the diagnosis of hypertrophy pyloric stenosis and planned to perform pylorotomi. During the surgery, HPS was not found thus the surgeon perform Thal fundoplication procedure to correct GER in this patient. The principles of surgical therapy for GERD include lengthening of the intra-abdominal esophagus, accentuation of the angle of His, increasing the pressure barrier at the esophago-gastric junction, and approximation of the crura.[1] After the procedure, patient had experienced no vomiting and able to feed more amount of formula.

In conclusion, the finale diagnosis was GERD because GER occurs in this patient followed by kwashiorkor type of malnutrition. GERD should be treated for eliminating kwashiorkor. Fundoplication procedure is rarely performed in Indonesia, and this case was the first fundoplication procedure in children with GERD at M. Djamil Hospital.

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