Conclusions

The incidence rate of hydrocephalus was 1.05% amongst 4502 patients attended the Paediatric Neurology Subdivision Department of Child Health, while the highest incidence was under 1 year old (75.5%). Congenital hydrocephalus were found in 33 cases (73.9%), and conservative treatment performed to almost all of the cases and only 4 cases had ventriculo-peritoneal shunt.

REFERENCES

5. Hadirekno, Daulay EM, Lily E, Saing B. Hydrocephalus from Paediatric Neurology Subdivision of Child Health Department, Medical Faculty University of North Sumatera, Pirngadi Hospital, Paediatr Indone 1988; 28 : 255-8.

CASE REPORT

Congenital Hypertrophic Pyloric Stenosis
A Case at the Gunung Wenang Hospital Manado - North Sulawesi Indonesia

by

SM SALENDU W and ABDULLAH B

(From the Department of Child Health School of Medicine Sam Ratulangi University, Manado - North Sulawesi)

Abstract

On a baby girl of 4 weeks old with the diagnosis of congenital hypertrophic pyloric stenosis, an extremely rare case in our Hospital, surgical operation was done using the Fredet Ramsted method.

The main complaint was frequent vomiting. The diagnosis was based on projectile vomiting, retarded growth, constipation, moderate dehydration and was confirmed by barium meal study.

Ten days after the operation, she was discharged in a good condition.

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Introduction

Congenital hypertrophic pyloric stenosis is a condition due to hypertrophy of the longitudinal and circular muscles of the pylorus [1]. The cause of this condition is not clear yet [2]. Male infants are four times more affected by this condition than female infants [3].

This abnormality was found in preterm more, in still birth preterm infants. The symptoms occur 2 - 5 weeks after birth [4], but in 10% of cases they may begin at birth [5]. The incidence of congenital hypertrophic pyloric stenosis is 6,2 per 1000 person - per year [6].

The treatment is surgery, using the Fredet Ramstedt surgery method, which give satisfactory results [7]. A few patients could spontaneously recover, but this is extremely rare [8].

The following is a report of a case of congenital hypertrophic pyloric stenosis.

Case Report

A-four-week-old Indonesian girl from Kotamobagu (North Sulawesi, Indonesia), was admitted to the Pediatric Department Gunung Wenang General Hospital, Manado North Sulawesi - Indonesia, with the main complaint of vomiting.

Seven days before admission she suffered from projectile vomiting occurring 2 - 3 times a day after every feeding. The vomit contained of the previous feeding and there was no bile or blood. Shortly after vomiting the patient looked hungry. Because of the persistent vomiting which became more frequently, the patient was then transferred to Gunung Wenang General Hospital, Manado.

She never had passed stools since 5 days before hospitalization. Micturition was normal.

She was the second child of 2 siblings. Her parents and the other sibling were healthy. There was family history of this similar illness or any congenital abnormality. The patient was born spontaneously, with the body weight of 2800 gram.

Physical examination on admission revealed an alert child, looking ill, with a respiratory rate of 28 X/minute, a pulse rate 132 X/minute, body temperature 38,3°C, body weight 2,800 gram and body length 51 cm.

Discussion

The diagnosis of hypertrophic pyloric stenosis was based on: three clinical features. The dominant symptom was typically projectile and free of bile vomiting, the onset being between 2 - 3 weeks after birth, though in 10% of cases, symptoms began at birth, and rarely symptoms develop as late as 3 1/2 years of age. Initially the vomiting can be intermittent but it increases in severity and frequency until it occurs with every feed. Constipation usually develops, but early before there may be mild diarrhea, so that gastroenteritis is occasionally the initial mistaken diagnosis. The patient's nutritional status deteriorates; poor weight gain progresses to weight loss, dehydration and electrolyte imbalance.

In our case we found projectile and frequent vomiting, constipation and retarded growth/weight loss.

The diagnostic physical findings are a palpable pyloric mass and visible peristaltic waves across the epigastrium. Peristalsis proceeding from left to right: towards the pylorus is particularly prominent after feeding, just before vomiting. The hypertrophic pyloric muscles are felt as a mobile, non tender, firm, olive shaped mass in the epigastrium or right hypochondrium. The mass may be missed early in the course of the disease. Once a pyloric mass is felt, the diagnosis is made needing no further investigations.

In our case we did not find any visible and palpable mass, therefore we made the barium meal X-rays. In approximately 10% of infants with pyloric stenosis, the tumor is not palpable, thus needing radiological examination to reveal the elongated, narrowed pyloric canal [2]. Conservative therapy is not recommended, because it takes quite a long time and the complication could be fatal for the infant [2].

Recently some studies suggested that the best treatment is Fredet Ramstedt pylorotomy, and the results are satisfactory.

In our case, the pyloric stenosis was corrected by using Fredet Ramstedt Pylorotomy and was successful.

REFERENCES


Preprandial plasma and urine amino acid concentrations were measured in 28 growing, very low birth weight, appropriate-for-gestational-age infants randomly assigned to either protein-unenriched (n = 14) or human milk protein-enriched (n = 14) human milk. The two groups of infants had similar birth weights (900 to 1500 g) and gestational ages (26 to 32 weeks). The study was initiated at a mean age of 19 days when the infants tolerated full feeding volumes and lasted for a mean time of 28 days. Mean protein intake values were 2.1 ± 0.3 and 3.6 ± 0.3 g/kg per day (mean ± SD) and weight gain values were 26.6 ± 7.4 and 35.1 ± 3.6 g/day in the protein-unenriched and the protein-enriched groups of infants, respectively. Human milk protein enrichment resulted in significantly increased concentrations of all plasma amino acids except serine, taurine, and histidine. Most urine amino acid concentrations correlated with protein intake and with the plasma concentrations, suggesting that the effects of protein quality and quantity can be evaluated by measuring urinary amino acid concentrations alone, thereby making such studies less invasive. Infants fed protein-unenriched human milk had growth rates below the estimated intrauterine rate as well as low plasma and urine amino acid concentrations, indicating suboptimal protein intake levels. When the plasma concentrations of the essential amino acids in the protein-enriched infants from the present study were compared with concentrations found in the literature in fetal and umbilical cord plasma essential amino acid concentrations in the well-growing, protein supplemented infants from the present study corresponded best to plasma concentrations found in breast-fed, growing, term infants at 1 to 3 months of age. It is suggested that preprandial plasma amino acid concentrations found in healthy, growing, breast-fed, term infants can be used as reference standard values when evaluating preprandial plasma amino acid concentrations in appropriate-for-gestational-age, very low birth weight infants.


Concentrations of 11 plasma proteins were measured in 28 healthy, growing, very low birth weight, appropriate for gestational-age infants fed varying levels of human milk protein intake (range 1.7 to 3.9 g/kg per day). Significant positive correlations were found between mean protein intake and concentrations of 7 of the plasma proteins studied (transferrin, retinol-binding protein, and transferrin: P < .001; vitamin D-binding protein and apolipoprotein B: P < .01; albumin and apolipoprotein A I; P < .05). A weak negative correlation with mean protein intake was seen for the plasma level of orosomucoid, whereas no significant correlations were found for the