Hypertrophic pyloric stenosis in a Premature Infant: A Case Report

Chan-Yao Wu, Kai-Sheng Hsieh, Shu-Ming Lin, Ying-Yao Chen

Hypertrophic pyloric stenosis is uncommon in premature babies. In term infants, the typical presentations include a palpable mass over the epigastric area, exaggerated gastric peristalsis after feeding and projectile non-bilious vomiting. However these symptoms are not commonly seen in premature infants. The onset of symptoms in pyloric stenosis in premature infants is later than that in term infants. The pyloric index (including pyloric wall thickness, and the length and the width of pyloric canal) measured by sonography gives a definite diagnosis in term infants. But the sonographic diagnostic criteria are unclear in premature infants. We report a case of a premature baby (gestational age of 34 weeks, birth body weight of 2,000 g, twin B) with pyloric stenosis who developed non-bilious projectile vomiting in the 3rd week of life. Pyloromyotomy was performed at 5 weeks of age with a smooth post-operative course. Feeding was started on the 3rd postoperative day and he was discharged on the 10th postoperative day with a body weight of 2,200 g.

Key words: hypertrophic pyloric stenosis, prematurity

Hypertrophic pyloric stenosis in term infants presenting with typical symptoms include projectile vomiting, exaggerated gastric peristaltic wave after feeding and epigastric mass typical of the pyloric " olive ". These symptoms are uncommon in premature infants. A delayed onset of these classic symptoms until the 5th week of life in premature infants has been reported compared with an onset in the 3rd week of life in a term baby [1]. The etiology of pyloric stenosis in term infants is unknown. In premature infants, some authors reported a relationship between pyloric stenosis and transpyloric feeding [2]. Hypertrophic pyloric stenosis in premature infants constitutes a small subgroup of patients with the disease. The incidence of pyloric stenosis in premature infants is unusual [3]. A definite diagnosis in premature infants is impaired by the absence of objective imaging diagnostic criteria of the kind that exists for term infants. The standard therapy for pyloric stenosis is Ramstedt pyloromyotomy. Use of the umbilical route for intra-abdominal pyloromyotomy is a recent technical improvement [4].

Case Report

A premature male infant boy with a birth body weight of 2,000 gm and gestational age of 34 weeks, was born to a 30-year-old gravida one, para two mother. No family history of this problem was elucidated. His brother, twin A, appeared healthy, but the patient, twin B, had frequent projectile non-bilious vomiting starting in the third week of life. His body weight had reached 2,500 gm at that time, but decreased gradually after the development of vomiting. The body weight decreased to 2,000 gm at the fifth week of age. He was transferred to our hospital for further management under the impression of bowel obstruction. Physical examination showed a weak, small infant with poor skin turgor. Abdominal examination showed a flat abdomen with an epigastric mass typical of the pyloric " olive ". No gastric peristalsis wave was noted. Arterial blood gas revealed metabolic alkalosis (pH: 7.577, PaCO₂: 35.2 mmHg, HCO₃⁻: 32 mmol/L, BE: 10.1 meq/L). Complete blood count showed white blood cell count: 17,000/mm³, Hgb: 14 mg/dl and platelet count: 380,000/mm³. Blood chemistry revealed sodium: 121 mmol/L, potassium: 3.5 mmol/L, chloride: 87 mmol/L, BUN: 19 mg/dL, creatine: 1.1 mg/dL and anion gap: 11.5 mmol/L. Initially, KUB showed the stomach distended with gas, without evidence of bowel gas (Fig. 1). Ultrasonic examination of the abdomen (Fig. 2) demonstrated hypertrophy of the pyloric wall, measuring about 5.5 mm in thickness, and a target appearance of the pylorus which was 17 mm in length and 14 mm in external diameter (normal values of pyloric measurement for term infants in this hospital are less than 3 mm, 17 mm and 13 mm respectively). Intravenous fluid supplements were given...
and dehydration and electrolytes imbalances were corrected. A pyloromyotomy through the umbilical route was performed the following day. A white glistening pyloric mass about 4.5 x 2.5 cm was found. The patient was fed via a nasogastric tube starting on the 3rd postoperative day. No further projectile vomiting occurred. Feeding amounts increased gradually and he was discharged in stable condition on the 10th postoperative day with a body weight of 2,200 gm. At the age of 4 months, he was thriving with a body weight of 4,600 gm.

Discussion

The first case of pyloric stenosis of a full term baby was reported in 1907 [5]. It is uncommon in premature babies. The incidence of pyloric stenosis in premature babies ranges from 1% [6] to 16% [7,8] of all cases. In Benson and Lloyd's review of 1,120 cases, 3.1% were described as premature [3].

![Fig. 1. The KUB picture revealed a distended stomach and no bowel gas.](image1)

![Fig. 2. Sonography of the pylorus showed the hypertrophy of the pyloric wall with 5.5 mm in thickness, and a target appearance of the pylorus which was 17 mm in length and 14 mm in external diameter.](image2)

The etiology of pyloric stenosis in premature infants remains unclear. Transpyloric feedings as a causative agent in the premature infant have been reported. Cosman et al reviewed 13 premature infants with pyloric stenosis related to transpyloric feedings [2]. Transpyloric intubation for enteral feeding might delay the diagnosis of this disorder and has been thought to play a role in its development. Our patient had no transpyloric feeding except for a short period postoperatively.

The typical presentation in full term infants including projectile vomiting, exaggerated gastric peristalsis after feeding and epigastric mass typical of the pyloric "olive" are often partially expressed in premature babies. Henderson et al had attempted to define the clinical features in premature infants with pyloric stenosis [9]. These included absence of the voracious appetite that characterizes the full term infant with pyloric stenosis, absence of projectile vomiting, and absence of visibly exaggerated gastric peristalsis. The onset of vomiting in premature infants is later than in term infants. Kehl and Lange reported a delayed onset of vomiting in the 5th week of life [1], which is later than in full term infants in whom vomiting usually appears at 3 weeks [10].

In our case, the onset of projectile non-bilious vomiting was at the age of 3 weeks. The symptom of voracious appetite was not obviously expressed and gastric peristalsis after feeding was not found. So, it is easy to misdiagnose the pyloric stenosis in premature infants. People involved in caring for premature infants, especially family health practitioners and general pediatricians need to be aware of its existence and the possibility of an atypical presentation in premature infants.

Our patient had typical hypochloremic metabolic alkalosis. In spite of loss of large amounts of fluid, the sodium concentration is usually normal, because loss of water and sodium usually occur in proportion to that in the extracellular fluid. On the other hand, because preferential loss of gastric hydrochloric acid occurs, the serum chloride is low and the serum bicarbonate is elevated, leading to metabolic alkalosis. As a consequence of the alkalosis, hypoventilation occurs.

Radiologists use the muscle length, diameter, and wall thickness — either individually or in combination — as a "pyloric muscle index" to establish whether the pylorus is hypertrophic [11,12]. The normal range for the pyloric muscle index in term infants had been established. But, the untrasoundographic diagnostic criteria is controversial in premature infants. Previous studies [2,13] reported that sonography is nondiagnostic for hypertrophic pyloric stenosis because the normal range of pyloric measurements is not easy to establish. Upper gastrointestinal series examination with barium has been suggested as one of the definitive diagnostic tools by some authors [14]. Most pediatric surgeons rely on physical findings for
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The standard therapy for pyloric stenosis is Ramstedt pyloromyotomy. Laparoscopic pyloromyotomy has been reported to be a safer alternative technique [15]. Performing a pyloromyotomy through a supraumbilical skin fold incision will leave an almost invisible scar and therefore has definitive cosmetic advantages [4]. Surgical findings in premature infants show that the mass is a softer, more pliable, less gritty and thinner than that found in full-term infants [14]. In general, in full term infants with hypertrophic pyloric stenosis, milk feeding begins 12-24 hrs after surgery. In our case, nasogastric feeding was not begun until the third postoperative day due to series of KUB that revealed a single bubble sign over the stomach area. We think the cause was that severe swelling over the incision area over the pylorus delayed the passage of gas. We concluded that routine imaging follow up is necessary after surgery.

We recommend that normal pyloric measurements correlated with corrected gestational age, birth body weight and sex should be established as early as possible.

References