The Evolution of Infantile Pyloric Stenosis

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We present the case of a 14 day old baby in whom we observed the evolution of idiopathic hypertrophic pyloric stenosis. We discuss the importance of follow-up ultrasound in an infant who is clinically suspected for IHPS and present an update on treatments for this condition.

Patient Description

A 14 day old firstborn male baby was admitted to our pediatric department with a 2 day history of vomiting, which became projectile on the day of admission. He was born at term after an uneventful pregnancy and delivery. Birth weight was 3.06 kg. A family history of pyloric stenosis was noted in an uncle. Physical examination revealed a jaundiced baby. The abdomen was soft without evidence of a pyloric tumor. The rest of the physical examination was normal. An indirect hyperbilirubinemia of 12.1 mg/dl was the only abnormality found in the laboratory results, which included a complete blood count, blood gases, glucose, electrolytes, transaminases, and thyroid-stimulating hormone. An ultrasound done on admission was negative for a pyloric tumor [Figure A]. Due to the continuation of projectile vomiting, an upper gastrointestinal study was performed 2 days later and showed a narrow pyloric canal (not shown), with normal transit to the small bowel. A repeat ultrasound showed a prolonged pyloric canal of 20.4 mm and a thickened pyloric muscle of 3.4 mm [Figure B] (abnormal values: thickness > 4 mm, length > 16 mm).

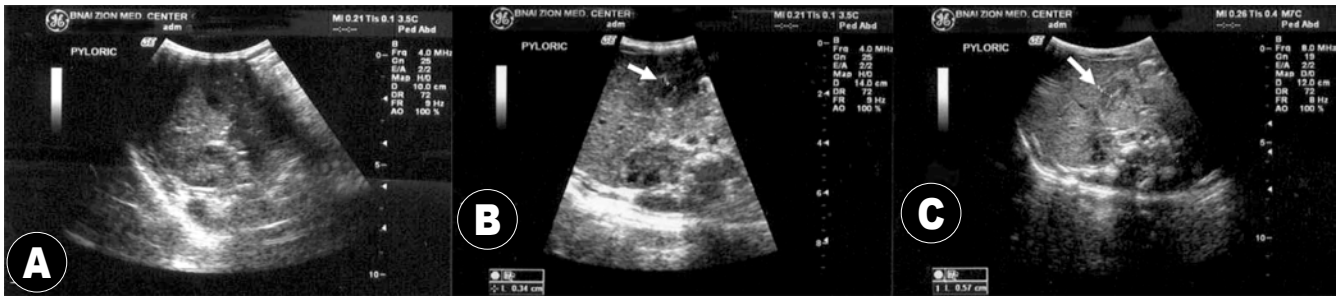
Due to the uncertainty of the diagnosis, the child was followed conservatively with small feedings and intravenous fluids. A repeat ultrasound on the fourth day of admission showed a prolonged pyloric canal of 21.4 mm and a thickened pyloric muscle of 5.7 mm [Figure C]. A pyloromyotomy was performed without complications and the child was discharged home after 2 days.

Comment

Although the precise etiology of idiopathic hypertrophic pyloric stenosis remains unknown, there is evidence that it may be an acquired condition rather than a congenital disorder, as previously thought. A detailed update on the etiology of IHPS can be found in an editorial by Udassin published in *IMAJ* [1].

In this condition, gastric outlet obstruction results from hypertrophy of the pyloric muscle, edema of the pyloric canal, and spasm of the antropyloric muscle, which leads to vomiting, dehydration, and metabolic alkalosis. Clinical manifestations of pyloric stenosis begin at a mean age of 3 weeks after birth, but they 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 nonbilious vomiting, which is often projectile [2]. Physical examination may reveal an olive-like pyloric mass palpated in the upper abdomen in up to 90% of patients. When palpated, a pyloric mass is considered pathognomonic [2]. Hypochloremic metabolic alkalosis is a typical laboratory finding. Nevertheless, in a study reported in *IMAJ*, we compared the clinical presentation of 70 infants with a surgically confirmed diagnosis of IHPS and found that a third of the infants lacked projectile vomiting by history, a pyloric tumor was not palpated in half, and only a quarter had metabolic alkalosis [3]. This was in

IHPS = idiopathic hypertrophic pyloric stenosis



[A]. Abdominal ultrasound at presentation showing a normal pyloric canal

[B]. Repeat ultrasound on the second day of admission, showing a prolonged pyloric canal of 20.4 mm and a thickened pyloric muscle of 3.4 mm

[C]. Repeat ultrasound on the fourth day of admission, showing a prolonged pyloric canal of 21.4 mm and a thickened pyloric muscle of 5.7 mm

accordance with a paper by Hulka et al. who compared the data of 901 infants with IHPS during five periods from 1969 through 1994.

Since the advent of ultrasonography in 1977 by Teele and Smith as a non-invasive efficient diagnostic modality for IHPS, the confirmation of the diagnosis has become more efficient and is usually reached at an earlier stage of the evolving clinical picture. However, since it is an evolving disease, as this case presents, a negative ultrasound should not rule out pyloric stenosis. Futhermore, in any case with typical clinical symptoms ultrasound rather than upper gastrointestinal tract series should be repeated every few days to ascertain the diagnosis.

Vomiting is a common symptom of many disease states. The differential diagnosis of the infant with vomiting is broad and includes congenital anatomic, gastrointestinal, infectious, neurologic, genetic, and metabolic disorders. The physician must remain alert to the huge differential and not assume that all infants who vomit have gastroesophageal reflux. Special attention should be given to the infant with bile-stained emesis, which suggests intestinal obstruction beyond the duodenum. It is an ominous sign that mandates immediate evaluation. Abdominal X-rays should be performed in neonates with persistent emesis and in all infants with bile-stained emesis to detect air-fluid levels, distended bowel loops, characteristic patterns of obstruction (double bubble: duodenal atresia), and pneumoperitoneum (intestinal perforation). A barium swallow X-ray with small bowel follow-through is indicated in the presence of bilious emesis.

Non-operative therapy for IHPS consists of the use of atropine and atropine-like medications and paste-consistency feedings until resolution [4]. Because of the high failure rate, the prolonged recovery period, and the low risk of pyloromyotomy, the non-operative approach is rarely used. Recently, an endoscopic pyloromyotomy has been suggested as an alternative to surgery [5].

In conclusion: this case presents the evolution of IHPS as visualized by ultrasound. This case should increase the awareness of pediatricians and pediatric surgeons to the fact that a negative ultrasound in an infant suspected for IHPS should not be considered the final word. In any case with typical clinical symptoms ultrasound should be repeated to ascertain the diagnosis.

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