Antenatal Ultrasound Findings from a Fetus that was Ultimately Diagnosed with Pyloric Stenosis in the Mid Neonatal Period: A Case Report

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Infantile hypertrophic pyloric stenosis is the most common surgical cause of vomiting in infants. The pyloric muscle is hypertrophied and the pyloric channel becomes narrow and elongated, causing gastric outlet obstruction. This is a case report of antenatal ultrasound findings from a fetus that was ultimately diagnosed with pyloric stenosis in the mid neonatal period.

Key words: Hypertrophic pyloric stenosis, Non-bilious vomiting, Prenatal ultrasonic diagnosis

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Introduction

Pyloric stenosis, also known as infantile hypertrophic pyloric stenosis (IHPS), is the most common cause of intestinal obstruction in infancy. IHPS occurs secondary to hypertrophy and hyperplasia of the muscular layers of the pylorus, causing a functional gastric outlet obstruction. The incidence of IHPS is 2-4 per 1000 live births with a male-to-female predominance of 4:1. The usual age of presentation is approximately the 3rd week of life with progressively worsening of vomiting.[1]

Pyloric stenosis is mainly a postnatal event and the postnatal US pattern of pyloric stenosis is widely recognized but antenatal presentation is quite unknown with only a few case reports so far and presence of such case reports suggests that the process probably starts antenatally.

Herein we report an infant who presented with a constant cystic structure near the stomach at 20 weeks of gestation. The fetus was followed up at 25th and 37th weeks of gestation with no change in the shape and place of the cystic structure. The newborn was ultimately diagnosed with pyloric stenosis 3 weeks after the birth.

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Case Report

A 35-year-old gravida 2, para 1 pregnant female attended to the radiology clinic at 20 weeks of gestation for an antenatal ultrasound examination. The patient had an amniocentesis before admission because of elevated risk at triple test [risk for trisomy 21:1/150 with maternal serum human chorionic gonadotrophin 2.5 multiples of median (MoM)]. The results of the amniocentesis were normal.

Fetal US examination revealed a 5.5 mm, cystic, nodular structure with smooth thin borders adjacent to the left lobe of the liver, abutting the medial border of the stomach (Figure 1). No additional pathologies were noted on the US exam.

The cystic structure was assumed to be due to a duplication cyst or diverticula because of its close proximity with the stomach. A simple liver cyst and mesenteric cyst were also included in the differential diagnosis as well. Duodenal atresia was also considered but ruled out because fluid in the intestines and colon were detectable during the initial exam and the follow-up. The patient presented with no additional anomalies and the size and shape of stomach, gallbladder were normal and amniotic fluid was within normal limits. Also amniocentesis ruled out the possible genetic abnormalities. Therefore the patient was followed up with serial US examinations. The cyst remained stable in shape and location but mild increase in size was noted which was 7.5 mm in 26th week (Figure 2) and 10 mm in 32nd weeks. The fetus was delivered via vaginal route and no complications ensued. The neonate was discharged from the hospital and the family was advised to return for an US exam 2 days later but did not show-up for the exam. The infant was readmitted to the hospital on the third week due to regurgitation and vomiting upon feeding. Pyloric steno-

sis was confirmed and surgically treated. There was no other distortion of anatomy at the preoperative imaging work-up and at surgery.

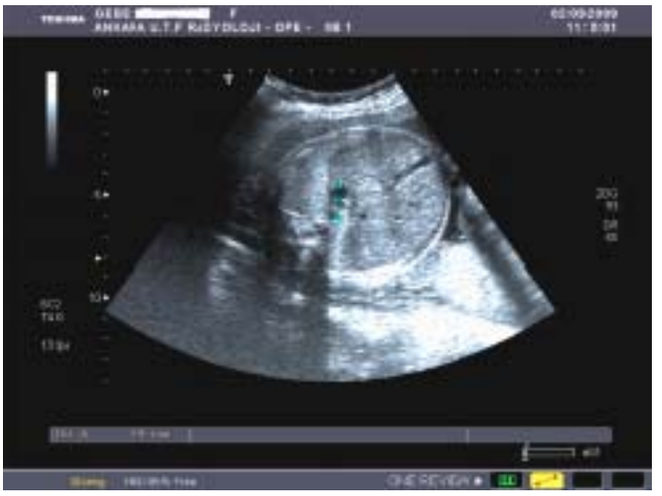

Figure 1: A smooth contoured cystic structure abutting the medial border of the stomach within close vicinity of the left lobe of the liver is seen on the US image (calipers) in the 20th week of gestation

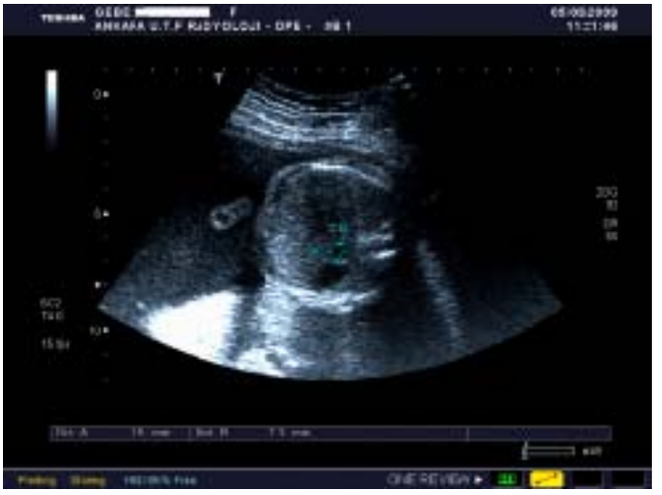

Figure 2: The size of the cystic structure increases mildly but the location and shape remains the same. The proximity of the structure (calipers) with the stomach is also clearly visualized

Discussion

The causes of IHPS are multifactorial.[2] Both environmental factors and hereditary factors are believed to be contributory. Possible etiologic factors include deficiency of nitric oxide synthase containing neurons, abnormal myenteric plexus innervation, infantile hypergastrinemia, and exposure to macrolide antibiotics. Although IHPS has a strong genetic predisposition, the characteristics pyloric hypertrophy is a postnatal event. Barium studies performed in the newborn period showed no difference between the infants who later developed IHPS and the others.[3] Similarly Rollins et al., stated that congenital preformed muscular hypertrophy do not appear to be present in babies who later develop pyloric stenosis by ultrasonography.[4] In contrast there exist well-documented cases of IHPS with persistent vomiting dating from birth,[5-7] and there has been one report of in-utero gastric dilatation in a neonate who later developed IHPS.[8] In addition polyhydramnios has been reported complicating the pregnancy leading to the birth of a baby with IHPS, indicating pyloric obstruction in utero.[6-9]

Singh et al., reported a case where the diagnosis of IHPS was entertained in the antenatal period and the neonate was followed up until the IHPS became manifest.[10] On their antenatal US scan polyhydramnios was noted and there was unusually active peristalsis in the pyloric antral area with normal thickness of the circular muscle. Tashjian et al., reported an infant with IHPS seen on prenatal ultrasound.[11] US scan which was performed one day prior to delivery revealed a grossly distended fetal stomach with an identifiable duodenal cap. In our case we did not observe polyhydramnios, gastric dilatation or increased gastric peristaltism. The cystic structure noted in the current prenatal US exam could be compared to the "duodenal cap" described in prenatal exam by Tashjian et al.,[11] but the images for this case were not too convincing for the cystic structure to actually be the duodenal cap that was described in the pediatric GI, surgery and neonatal imaging literature because of the circular shape and the location of the cyst which seemed more posterior to represent the duodenum at the distal side of the pyloric juncture.

It is possible that the evolution of IHPS may begin in the antenatal period and as the process progresses postnatally, the whole pyloric musculature becomes increasingly hypertrophied; the final outcome is gastric outlet obstruction. However our case showed absence of polyhydramnios, gastric dilatation and gastric peristaltism also suggests that in the antenatal period symptomatic pylorospasm and pyloric thickening do not exist.

If a "duodenal cap" is noted in the antenatal US exam, duodenal atresia and stenosis are the initial and primary considerations in the differential diagnosis of such a cystic structure within close proximity of the stomach. The most typical sonographic finding is the characteristic "double bubble" sign caused by the simultaneous dilatation of the stomach and the proximal duodenum. Up to half of the duodenal atresia cases are complicated by polyhydramnios. In addition differential diagnosis includes choledocal cyst, hepatic cyst, dilated gallbladder, biliary atresia, mesenteric and omental cyst. Mesenteric and omental cysts appear as a thin-walled, unilocular or multilocular cystic mass sonographically.

In conclusion while pyloric stenosis is relatively common,

the ability to diagnose it prenatally has been elusive. IHPS should be included in the differential diagnosis among others if a duodenal cap like cystic structure was detected within close proximity of the stomach. Moreover, this allows maternal referral to a center with appropriate neonatal and surgical expertise, which may improve the outcome. Limitation of this case report is that there are no images immediately after delivery to follow-up the reported findings. It seems essential to prove that the cystic structures seen before birth have not just disappeared, but have transformed into a more typical picture of pyloric stenosis.

İlk Orta Neonatal Dönemde Gözlenip Devamında Pilor Stenozu Teşhisi Alan Bir Fetüsteki Antenatal Ultrason Bulguları: Bir Olgu Sunumu

İnfantil hipertrofik pilor stenozu infantlarda kusmanın en sık cerrahi nedenidir. Pilor kası hipertrofiye olur, pilor kanalı darlaşır ve gastrik çıkış obstrüksiyonuna neden olarak uzar. Burada orta neonatal dönemde pilor stenozu tanısı almış olan bir fetusdaki antenatal ultrasonografi bulguları sunulmuştur.

Anahtar Kelimeler: Hipertrofik pilor stenozu, Safrasız kusma, Prenatal ultrasonografik tanı

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