CASE REPORT

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Gastroschisis- A Case Report

ANATOMY

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ABSTRACT

Gastroschisis is a birth defect in the anterior abdomen wall and is often referred to as "ventral wall defect". Babies with gastroschisis are born with portions of their small and large intestines exposed outside the body through a small opening generally found to the right of the belly button. Gastroschisis is seen in approximately 1 in 4000 live births, and is more common in mothers under 20 years of age.

A fetus with an abdominal wall defect is a high risk pregnancy. For gastroschisis, there is an increased risk of intra uterine growth retardation (IUGR), fetal death, and premature delivery. So careful obstetric follow-up with serial ultrasounds and other tests of fetal well being are indicated. To emphasise its importance this case is presented.

***Key words:** Gastroschisis, Ventral wall defect, Fetus, Intra uterine growth retardation*

Introduction

The word gastroschisis, freely translated from the Greek, means "belly cleft." The principal features of this rare condition are: a defect in the abdominal wall which is extra-umbilical in location with no membranous sac covering the eviscerated mass of intestines, and a normal umbilical cord insertion into the abdominal wall that is not involved in the evisceration [1].

Gastroschisis is a full-thickness defect in the abdominal wall usually just to the right of a normal insertion of the umbilical cord into the body wall [2]. Gastroschisis occurs in 1 in 4000 live births [3]. The majority of pregnancies complicated by gastroschisis are diagnosed prenatally [4]. Subsequent sonographic visualization of freely floating loops of bowel within the amniotic fluid with an abdominal wall defect to the right of the insertion of the umbilical cord at any point after the normal embryonic return of the intestine to the abdominal cavity at 10 weeks of gestation confirms the diagnosis [5-8]. Preterm delivery is more frequent in infants with gastroschisis, with an incidence of 28% compared with 6% of normal deliveries [9]. Bowel atresia is the most common associated anomaly in patients with gastroschisis. Recent studies report concomitant atresia of bowel and gastroschisis in 6.9-28% of patients [10,11].

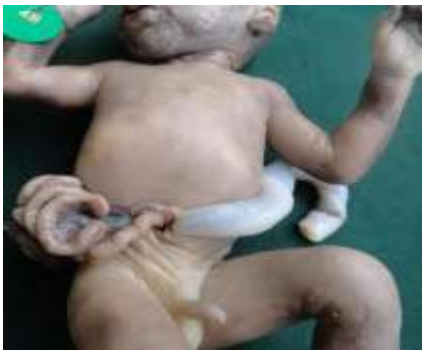
Case Report

Aborted human male fetues of 24 weeks of gestation with anterior abdominal wall defect was received in the Department of Anatomy from Department of Obstetrics and

Gynecology, NRI Medical College & General Hospital, Chinakakani for study with proper consent of parents .The foetus was fixed immediately with 10% formalin.

After recording the parameters for age determination, foetus was examined for external and internal defects. Except herniation of gut loops through anterior abdominal wall to the right of umbilicus, no other external defects were detected. Herniated gut contains jejunum, ileum, caecum with appendix, segment of colon and the mesenteric vessels. All these structures were entangled and twisted around each other (Fig.1).

Figure 1: Herination of gut loops through the umbilical opening



After opening the abdomen a part of colon is seen extending down below the pancreas running in front of left kidney and into left iliac fossa (Fig.2). This part of colon was assumed as descending colon because initial part of colon was seen in herniated mass of intestines in continuation with caecum. This descending colon abruptly turned towards right side, posterior to the herniating loop and continued to the right iliac fossa forming anterior and inferior relation to the right kidney (Fig.3). Again the loop has turned down wards and to the left upto the mid line and continued in to the pelvis. Below the pancreas it formed a double 'S' loop.

Figure 2: Relation of Descending colon to Antero inferior part of left kidney

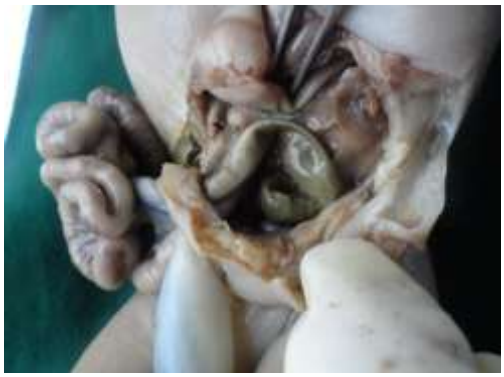


Figure 3: Relation of Descending colon to Antero inferior part of right kidney

Discussion

Gastroschisis is a protrusion of abdominal contents through the anterior body wall directly into the amniotic cavity. It occurs lateral to the umbilicus usually on the right and the defect is most likely due to abnormal closure of the body wall around the connecting stalk. Viscera are not covered by peritoneum or amnion [12].

Exact cause of gastroschisis is uncertain, but various causes have been proposed including ischemic injury to the anterior abdominal wall (absence of the right omphalomesentric artery), rupture of the anterior abdominal wall, weakness of the wall caused by involution of right umbilical vein [13]. Gastroschisis has a very strong

association with young maternal age, with most of these mothers being age of 20 years or younger. In addition, gastroschisis has been linked to maternal exposure to cigarette smoking, illicit drugs, vasoactive over-the-counter drugs (such as pseudoephedrine), and environmental toxins [14]. These associations are consistent with the vascular insufficiency of the abdominal wall for the etiology of gastroschisis [14]. Oligohydramnios is also common in gastroschisis, being present up to 25% of cases. The cause is unknown and it is usually of moderate severity and associated with IUGR, fetal distress, and birth asphyxia [15].

Gastroschisis can be differentiated from omphalocele as it is herination of abdominal viscera through an enlarged umbilical ring. The origin of defect is failure of the bowel to return to the body cavity from its physiological herniation during 6^{th} week to 10^{th} week. The viscera are covered by amnion. Omphalocele occurs in 2.5/10,000 births, where as gastroschisis occurs in 1/10,000 births. Unlike gastroschisis, omphalocele is associated with high rate of mortality (25%) and severe malformations, such as cardiac anomalies (50%) and neural tube defects (40%) [12].

Gastroschisis can be differentiated from umbilical hernia. The intestines return to the abdominal cavity during the 10^{th} week but the mass again herniate through an imperfectly closed umbilicus, thus forming umbilical hernia. It differs from gastroschisis being covered with skin and subcutaneous tissue. The herniating mass usually contains greater omentum and small intestine. The hernia usually does not reach its maximum size until the end of the first month after birth. The defect through which the hernia occurs is in the linea alba. The hernia protrudes during straining and can be easily reduced through the fibrous ring at the umbilicus [13].

Abdominal wall defects are often diagnosed by prenatal ultrasound done for routine screening or for obstetric indications such as evaluating an elevated maternal alpha fetoprotein of serum (AFP). AFP is also usually elevated with abdominal wall defects. The magnitude and of AFP elevation varies between gastroschisis and omphalocele [2]. In gastroschisis, maternal serum AFP is markedly abnormal, with an average elevation of more than nine multiples of the mean (MoM). In contrast, in omphalocele, AFP is elevated by an average of only four MoM, with a much wider range [16]. Prenatal ultrasound could potentially identify the overwhelming majority of abdominal wall defects and accurately distinguish omphalocele from gastroschisis.

Conclusion

Improved understanding of gastroschisis, its early diagnosis by prenatal ultrasound, safe delivery of the foetus with a ventral wall defect, advanced surgical techniques for its correction and intensive care management of neonates reduces the morbidity and mortality.

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References

1. Goodhead. B Gastroschisis, Brit.med.J.,1965,1,771-772.(PMCID:PMC:2166158).

2. Led better.D J.Gastroschisis and Omphalocele.Surg Clin N A.2006;86.249-260.(PMID:16580922).

3. Baird PA, MacDonald EC. An epidemiologic study of congenital malformations of the anterior abdominal wall in more than half a million consecutive live births. Am J Hum Genet 1981;33:470-8.(PMID:6454342).

4. Barisic I, Clementi M, Husler M, et al. Evaluation of prenatal ultrasound diagnosis of fetal abdominal wall defects by 19 European registries. Ultrasound Obstet Gynecol 2001;18:309-16.(PMID:11778988).

5. Rasmussen SA, Frias JL. Non-genetic risk factors for gastroschisis. Am J Med Genet 2008;148C:199-212. (PMID:18655102).

6. Castilla EE, Mastroiacovo P, Orioli IM. Gastroschisis: international epidemiology and public health perspective. Am J Med Genet 2008;148C:162-79.(PMID:18655097)

7. Loane M, Dolk H, Bradbury I. Increasing prevalence of gastroschisis in Europe 1980-2002: a phenomenon restricted to younger mothers? Paediatr Perinat Epidemiol 2007;21:363-9.(PMID:17564594).

8. Alvarez SM, Burd RS. Increasing prevalence of gastroschisis repairs in the United States: 1996-2003. J Pediatr Surg 2007;42:943-6.(PMID:17560199).

9. Lausman AY, Langer JC, Tai M, et al. Gastroschisis: what is the average gestational age of spontaneous delivery? J Pediatr Surg 2007;42:1816-21.(PMID:18022429).

10. Kronfli R, Bradnock TJ, Sabharwal A. Intestinal atresia in association with gastroschisis: a 26 year review. J Pediatr Surg 2010;26:891-4.(doi:10.1007/s00383-010-2676-4).

11.Arnold MA, Chang DC, Nabaweesi R, et al. Risk stratification of 4344 patients with gastroschisis into simple and complex categories. J Pediatr Surg 2007;42:1520-5.(doi:10.1016/j.jped surg.2007.04.032)

12.Sadler T W.Langman's Medical Embrology.12th ed.New Delhi:Wolters Kluwer India Pvt.Ltd;2013.Chapter 15,Digestive system:225.

13.Moore K.L and Persaud T.V.N The Developing Human Clincially Oriented Embrology,8th ed.New Delhi.Elsevier;2008:229-231.

14.Curry JI, McKinney P, Thornton JG, et al. The aetiology of gastroschisis. Br J Obstet Gynaecol 2000; 107(11):1339–46.(doi:10.1111/j.1471-0528.2000.tb11645.x).

15.Tan KH, Kilby MD, Whittle MJ, et al. Congenital anterior abdominal wall defects in England and Wales
1987–93: retrospective analysis of OPCS data. BMJ 1996;313(7062):903–6.(PMCID:PMC 2352268).

16. Saller DN Jr, Canick JA, Palomaki GE, et al. Second-trimester maternal serum alpha-fetoprotein,
unconjugated estriol, andhCGlevels in pregnancies with ventral wall defects. Obstet Gynecol 1994;84(5):852–5. (PMCID:PMC 1446119).

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