ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 9| Issue: 12| December 2023|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2023: 8.224 || ISI Value: 1.188

ABDOMINAL WALL DEFECTS: GASTROSCHISIS, DEFINITION, EPIDEMIOLOGY, PATHOGENESIS, TYPES, DIAGNOSIS AND MANAGEMENT

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Article DOI: https://doi.org/10.36713/epra15076
DOI No: 10.36713/epra15076

SUMMARY

Introduction: *Abdominal wall defects are common congenital anomalies, both gastroschisis and omphalocele conform to fetal developmental anomalies that favor multidisciplinary support before and after birth. Outcomes for newborns with gastroschisis usually depend on the distinctive features of the abdominal wall defect and underlying intestinal viability, whereas omphalocele outcomes are determined by the size of the defect and the presence of other related anomalies.*

Objective: *to detail current information related to the two fetal abdominal wall defects, especially gastroschisis definition, epidemiology, pathogenesis, types, diagnosis and management.*

Methodology: *a total of 48 articles were analyzed in this review, including review and original articles, as well as clinical cases, of which 36 bibliographies were used because the other articles were not relevant to this study. The sources of information were PubMed, Google Scholar and Cochrane; the terms used to search for information in Spanish, Portuguese and English were: fetal abdominal wall defects, gastroschisis, prenatal anomalies, surgery and prosthetic silo.*

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Results: *The incidence of gastroschisis ranges from 0.4 to 3 per 10,000 births and shows an upward pattern, the incidence of omphalocele ranges from 1.5 to 3 per 10,000 births. About 10% of infants with gastroschisis have intestinal stenosis or atresia because of vascular insufficiency due to volvulus or compression of the vascular pedicle by a narrowing abdominal ring. Periconceptional genitourinary tract infections were significantly associated with gastroschisis (OR, 1.5; 95 % CI, 1.3 to 1.9). Other significant associations presented in various studies with gastroschisis pregnancies include smoking (odds ratio (OR), 2.86; 95 % CI, 2.22-3.66), history of pregestational or gestational diabetes (OR, 2.81; 95 % CI, 1.42-5.5), and use of antidepressant medications (OR, 4.4; 95 % CI, 1.38-11.8).*

Conclusions: *Abdominal wall defects are common congenital anomalies, both gastroschisis and omphalocele represent different embryological outcomes, which depend on accurate prenatal diagnosis and referral to a multidisciplinary fetal center. Gastroschisis is a congenital anomaly of the rudimentary umbilical ring, in which there is herniation of a variable portion of viscera through the defect, with no membrane or covering sac, and its prevalence has been steadily increasing. Gastroschisis is classified into simple and complex types. Prenatal ultrasound scans can diagnose gastroschisis as early as 12 weeks of gestation. Routine preterm delivery or elective cesarean section has not been shown to improve outcome. Surgical management after birth is aimed at reduction of the herniated viscera and closure of the abdominal wall. Survival rates are usually high; however, the prognosis depends on the state of the bowel at birth. Those affected with significant intestinal alteration at birth are at higher risk of premature death or later complications. Future fetal surgery could improve the outlook and management of the condition.*

KEY WORDS: *gastroschisis, abdominal wall defect, congenital.*

INTRODUCTION

Abdominal wall defects are common congenital anomalies, where gastroschisis and omphalocele are most commonly encountered. These two are the result of errors in the embryological development of the fetal abdominal wall, being unique disorders with different clinical sequelae. Gastroschisis usually appears alone with postnatal outcomes linked to the underlying integrity of the prolapsed bowel. In contrast, omphalocele is associated with other structural anomalies or genetic syndromes that contribute more to postnatal outcomes than the omphalocele disorder. These 2 pathologies present embryologic differences, however, both gastroschisis and omphalocele are fetal developmental anomalies that benefit from multidisciplinary support before and after birth.Gastroschisis and omphalocele have reported incidences approaching 1 in 4000 live births; however, the incidence of omphalocele presented on second trimester ultrasound is as high as 1 in 1100, which highlights the significant rate of intrauterine fetal death related to diagnosis and prenatal therapies that may someday change. In counterpoint, the incidence of gastroschisis has increased in recent years. Both omphalocele and gastroschisis are usually diagnosed prenatally, and each has a related spectrum of postnatal outcomes. Outcomes for infants with gastroschisis usually depend on the distinctive features of the abdominal wall defect and underlying intestinal viability, whereas omphalocele outcomes are determined by the size of the defect and the presence of other related anomalies. Both gastroschisis and omphalocele require multidisciplinary and translational approaches. Prenatal ultrasounds also predict the high probability of intestinal atresia. The timing and mode of delivery in mothers with gastroschisis fetuses is controversial(1-9).

METHODOLOGY

A total of 48 articles were analyzed in this review, including review and original articles, as well as cases and clinical trials, of which 36 bibliographies were used because the information collected was not important enough to be included in this study. The sources of information were Cochrane, PubMed and Google Scholar; the terms used to search for information in Spanish, Portuguese and English were: fetal abdominal wall defects, gastroschisis, prenatal anomalies, surgery and prosthetic silo.

The choice of bibliography exposes elements related to the two fetal abdominal wall defects, especially gastroschisis definition, epidemiology, pathogenesis, types, diagnosis and management.

DEVELOPMENT

Definition

It is a congenital malformation characterized by a visceral hernia usually through a right-sided abdominal wall defect to an intact umbilical cord not covered by a membrane. About 10% of affected individuals with gastroschisis present intestinal stenosis or atresia resulting from vascular insufficiency caused by a volvulus or compression of the vascular pedicle by a narrowing abdominal ring(6,10,11).

Gastroschisis is thought to be caused by an interruption in the migration of the ventral lateral body folds in the early stages of embryonic development, which forms a paramedian line defect. Normally, the elongating intestine herniates out of the abdominal cavity around the sixth week of gestation and in the next four subsequent weeks, undergoes a process of midgut rotation with the return of the intestines to the abdomen. However, if the abdominal wall is not fully created, the intestine may herniate into the amniotic cavity. Gastroschisis is usually not associated with other anomalies unlike omphalocele. Postnatal outcomes are linked to the level of intestinal disruption and subsequent gastrointestinal complications. Complex gastroschisis is any related intestinal atresia, necrosis, perforation or volvulus; simple gastroschisis has no intestinal complications. Complex gastroschisis has a high mortality rate, prolonged hospital stays, increased infectious complications and a higher risk of intestinal failure compared to simple gastroschisis(1).

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Epidemiology

There are regional variations in the incidence of abdominal wall disorders and the relative proportions of gastroschisis and omphalocele; however, the incidence of gastroschisis is between 0.4 and 3 per 10,000 births and the incidence of omphalocele ranges between 1.5 and 3 per 10,000 births(11).

Gastroschisis disproportionately affects younger mothers, mostly in mothers under 20 years of age approximately 15.7 per 10,000 live births. Prevalence rates are higher in white and Hispanic compared to black mothers. Clinical studies show that genitourinary tract infections during the periconceptional period are significantly associated with gastroschisis (OR, 1.5; 95% CI, 1.3 to 1.9). Other literature points to significant associations of pregnancies with gastroschisis with smoking (odds ratio (OR), 2.86; 95 % CI, 2.22-3.66), history of pregestational or gestational diabetes (OR, 2.81; 95 % CI, 1.42-5.5) and using antidepressant medications (OR, 4.4; 95 % CI, 1.38-11.8)(12-15).

Pathogenesis

There are multiple embryological hypotheses to clarify the pathogenesis of gastroschisis, such as altered differentiation of the embryonic mesenchyme due to teratogenic exposure, rupture of the amniotic membrane at the base of the umbilical cord, abnormal involution of the right umbilical vein leading to a defect in the viability of the surrounding mesenchyme, disruption of the omphalomesenteric artery leading to necrosis located in the abdominal wall at the base of the cord, abnormal folding of the embryo leading to a ventral body wall defect, failure of the yolk sac and associated yolk structures to attach to the umbilical stalk, resulting in a perforation in the abdominal wall separate from the umbilicus, and amniotic rupture in the flaccid zone of the umbilical cord secondary to genetic predisposition or exogenous factors, such as: drugs, viruses, toxins or radiation(16-19).

In recent times, Lubinsky devised a binary vascular-thrombotic model for gastroschisis, in which normal involution of the umbilical vein makes a potential site for thrombosis adjacent to the umbilical ring. Subsequent thrombosis, related to factors that increase maternal estrogen, decreases the strength of the umbilical ring, forming a site for a probable hernia. This would explain the morphological findings and epidemiological risk factors. However, there is insufficient evidence that this is happening in humans, human evidence currently supports the theory that gastroschisis is not a defect of the abdominal wall, but an anomaly of the rudimentary umbilical ring, presenting in a separation of the fetal ectoderm from the epithelium of the amnion on the right side(6,20).

Types

Gastroschisis is subdivided into simple and complex according to the condition of the intestine.

Simple gastroschisis: intestine is in good condition without intestinal complications.

Complex gastroschisis: related to congenital intestinal complications such as atresia, perforation, ischemia, necrosis or volvulus. Closed or closure gastroschisis belongs to a subset of complex gastroschisis, in which the abdominal wall defect closes around the prolapsed intestine, generating intestinal stenosis of exit and/or entrance, atresia, ischemia, necrosis or resorption. These intestinal complications are due to a mixture of exposure to digestive compounds in the amniotic fluid and ischemia due to mesenteric constriction in the defect(6,21).

Figure 1. Gastroschisis.

Source: The Authors.

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Diagnosis

It is performed prenatally more than 90% of the time. Recognition of free-floating intestines outside the abdominal cavity to the right of a normally inserted umbilical cord on prenatal ultrasound is diagnostic. Prenatal diagnosis is usually made in the second trimester, however it has been reported in the twelfth week of gestation. A diagnosis of gastroschisis made in the first trimester should be judged with caution and confirmed later in gestation, because normal visceral rotation may not end until the conclusion of the first trimester(22,23).

It is possible to diagnose gastroschisis through prenatal ultrasound at 12 weeks of gestation. When gastroschisis is suspected, the corresponding ultrasound features should be observed such as:

- Appearance of the eviscerated bowel, e.g., dilatation and/or thickening.
- Identification of the cord insertion site in relation to the defect.
- Identification of eviscerated organs.
- Absence of a covering membrane or sac.
- Identification of associated malformations(24,25).

Prenatal Management

Following the diagnosis of gastroschisis, the multidisciplinary team consisting of a pediatric surgeon, a neonatologist, an obstetrician and a social worker should provide proper counseling and care. Prenatal ultrasound can serve to show reliable predictors of postnatal outcome and better prenatal counseling. There is literature that favors delivery at less than 37 weeks gestation for fetuses with gastroschisis to reduce fetal death and neonatal morbidity, however, preterm delivery is not without probable risks, primarily those related to physiologic immaturity and preterm delivery. Among the potential risks are respiratory morbidity, neurocognitive deficits, increased mortality, cholestasis and sepsis(1,6,26).

About 30 to 40% of pregnancies with gastroschisis show spontaneous preterm labor and delivery, compared to 6% of controls. Higher rates of preterm labor were related to elevated levels of proinflammatory cytokines in the amniotic fluid. Spontaneous preterm labor was associated with markedly affected bowel loops, intestinal occlusion and stained amniotic fluid, probably connected with repeated fetal vomiting of gastrointestinal contents into the amniotic fluid, increasing the number of inflammatory mediator mediators. The incidence of intrauterine fetal death (IUFD) in pregnancies complicated by gastroschisis is close to 5%(27-31).

Postnatal Management

The herniated bowel should be protected by covering it with a warm gauze soaked in saline solution, placing it in a central location of the abdominal wall and covering it with a plastic lining or a plastic bag to reduce heat and fluid evaporation. It is advisable to position the patient in the right lateral decubitus position so as not to cause vascular damage due to torsion of the mesenteric vascular pedicle. Systematic aggressive fluid resuscitation or excessive maintenance fluids should not be used. Fluid boluses are recommended when there is clinical evidence of metabolic acidosis and hypovolemia(6).

Surgical Management

Among the objectives of surgical treatment of gastroschisis are:

- The closure of the abdominal wall defect.
- The reduction of the herniated viscera into the peritoneal cavity while avoiding direct trauma to the intestine and excessive intra-abdominal pressure.

While the condition of the exposed bowel and the level of abdominovisceral disproportion predict the type and timing of surgical intervention, it is prudent to consider other factors such as gestational maturity, weight and comorbidities of the infant. The surgical alternatives for closure are:

1. Primary reduction, either with immediate closure with suture or without suture.
2. Prosthetic silo placement, gradual visceral reduction followed by delayed closure with or without suture.

After birth, the unprotected intestine is exposed to mechanical injury, dehydration, infection and pressure necrosis. To avoid these complications, 2 postnatal preventive techniques have been used as standard for the management of gastroschisis. These are either by repositioning the prolapsed bowel and subsequent closure of the abdominal wall, or by temporarily covering the prolapsed bowel with a preformed silo bag that is fixed under the abdominal wall of the newborn, leading to a gradual repositioning of the prolapsed bowel and subsequent delayed closure; secondary closure is used only in cases where primary closure is not possible. Modifications of primary closure, such as sutureless repair of the abdominal defect, using the umbilical cord and adhesive drapes, have had similar results with better benefits, but have a higher risk of umbilical hernia(32).

There is much controversy about the ideal surgical technique to practice and also about the best gestational age at delivery(33,34). There are some techniques for the treatment of gastroschisis such as:

- Open fetal coverage.
- Fetoscopic coverage.
- Open fetal repair.
- Open fetal ligation of the esophagus.
- Fetoscopic repair.
- EXIT procedures.
- Amnioinfusion and amnioexchange.

The most promising techniques for prenatal treatment of complex gastroschisis at the moment are amnioexchange and fetoscopic lining of the protruding bowel(32).

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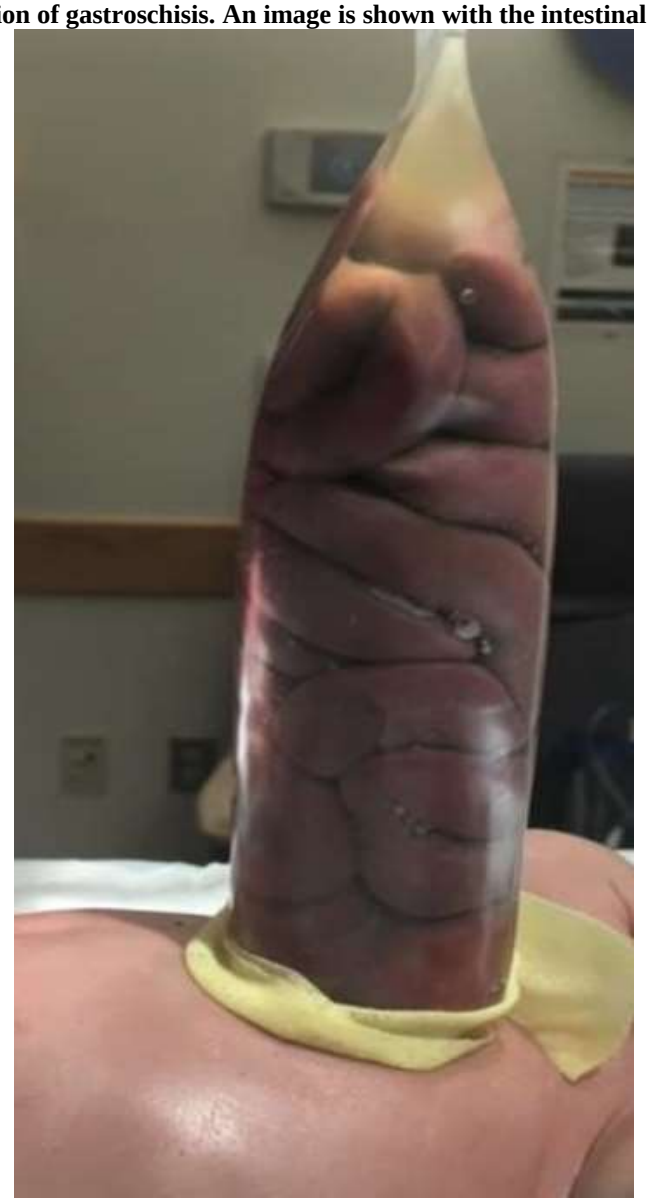
Figure 2. Step reduction of gastroschisis. An image is shown with the intestinal loops placed in a silo.



Source: Bhat V, Moront M, Bhandari V. Gastroschisis: A State-of-the-Art Review(6).

Post Surgical Management

Usually infants present with some measure of adynamic ileus, intestinal dysmotility and nutrient malabsorption, requiring nasogastric decompression through a nasogastric tube and parenteral nutrition through a secure central venous catheter, which may be a peripherally inserted central catheter (PICC) or a tunneled catheter, Broviac Catheter. Feeding begins upon recovery of bowel function. The period for the start of feeding differs from one to two weeks, or even longer, according to the state of the bowel. Early initiation of trophic feeding can promote peristalsis, prevent villous atrophy and decrease excessive bacterial growth. Feeding progresses slowly because feeding intolerance is common due to altered intestinal motility. Feeding expressed breast milk may help protect the infant from the development of necrotizing enterocolitis. Early oral stimulation is excellent for maintaining sucking and swallowing reflexes and preventing oral aversion. Infants with gastroschisis have abnormal esophageal motor function leading to delayed acquisition of oral feeding milestones and the possible requirement for chronic tube feeding(35,36).

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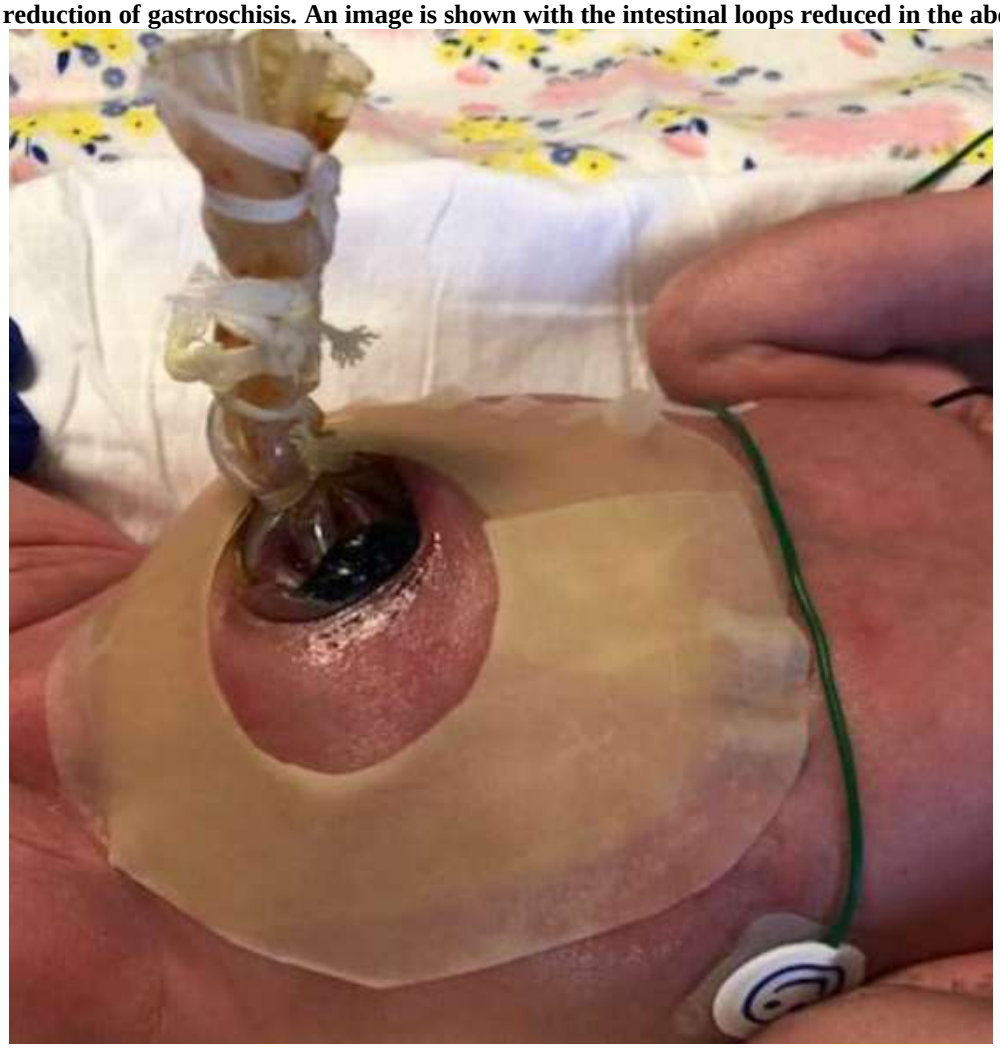
Figure 3. Stepwise reduction of gastroschisis. An image is shown with the intestinal loops reduced in the abdomen using a silo.



Source: Bhat V, Moront M, Bhandari V. Gastroschisis: A State-of-the-Art Review(6).

Prognosis

The prognosis of infants with gastroschisis varies as it is directly related to the state of the bowel at birth. About 25% of infants with simple gastroschisis and more than 70% of infants with complex gastroschisis will form posterior intestinal obstruction due to adhesions, anastomotic stricture or volvulus, theoretically necessitating several surgical interventions(6).

CONCLUSIONS

Abdominal wall defects are common congenital anomalies, both gastroschisis and omphalocele represent different embryological outcomes, which depend on accurate prenatal diagnosis and referral to a multidisciplinary fetal center. Gastroschisis is a congenital anomaly in the rudimentary umbilical ring, in which there is herniation of a variable portion of viscera through the defect, with no covering membrane or sac, and its prevalence has been steadily increasing. Gastroschisis is classified into simple and complex types. Prenatal ultrasound scans can diagnose gastroschisis as early as 12 weeks of gestation. Routine preterm delivery or elective cesarean section has not been shown to improve outcome. Surgical management after birth is aimed at reduction of the herniated viscera and closure of the abdominal wall. Survival rates are usually high; however, the prognosis depends on the state of the bowel at birth. Those affected with significant intestinal alteration at birth are at higher risk of premature death or later complications. Fetal surgery may improve the outlook and management of the condition in the future.

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Conflict of Interest Statement

The authors report no conflicts of interest.

Funding

The authors report no funding by any organization or company.