Educational Gap
A better understanding of the prenatal, postnatal, and surgical management of gastrochisis and omphalocele is needed to further improve care of affected infants.

Abstract
Omphalocele and gastrochisis are the two most common congenital abdominal wall defects requiring neonatal intensive care. Historically treated as a single entity, they represent two distinct pathologies with different clinical management algorithms and associated outcomes. With improvements in prenatal diagnosis, neonatal intensive care, and pediatric surgical practices, good long-term outcomes are possible in the absence of catastrophic bowel injury or debilitating associated anomalies.

Learning Objectives
After completing this article, readers should be able to:

1. Recognize that omphalocele and gastrochisis represent two distinct pathologies with different genetic and embryologic origins.
2. Be aware that initial management requires cardiopulmonary stabilization and protection of the eviscerated bowel or omphalocele sac.
3. Understand that small defects are often closed primarily, but definitive management of larger defects often requires serial reduction with silo placement (gastrochisis) or epithelialization with delayed closure (omphalocele).
4. Acknowledge that good long-term outcomes are common for both defects in the absence of catastrophic bowel injury or significant associated anomalies.

Introduction
Omphalocele and gastrochisis are the most common congenital abdominal wall defects requiring neonatal intensive care. Omphalocele is a midline defect characterized by eviscerated abdominal contents covered by a protective sac (Fig 1). It is associated with advanced maternal age and karyotype abnormalities. Gastrochisis, in comparison, is a defect in the abdominal wall located just lateral to the umbilicus that exposes the eviscerated abdominal contents to the amniotic fluid (Fig 2). As a consequence, omphalocele is rarely associated with intestinal injury, whereas gastrochisis may be complicated by inflamed, volvulized, atretic, and/or perforated intestine. Gastrochisis is associated with young maternal age, maternal smoking, alcohol use, illicit substance abuse, and use of over-the-counter vasoactive medications and salicylates. Historically, omphalocele had an incidence twice that of gastrochisis; however, the incidence of gastrochisis has more recently increased for reasons that are unclear. Both defects now occur at a rate of ~1 to 3 per 10,000 live births.

Pathogenesis
Omphalocele
Omphalocele is a congenital malformation resulting from incomplete body wall folding during embryogenesis. The defect is most commonly located at the base of the umbilical
stalk in the midline, with less common variants occurring in the epigastric and infraumbilical regions. The eviscerated contents, which include small bowel and sometimes colon and liver, are contained within a protective sac comprising peritoneum, Wharton’s jelly, and amnion. Other solid viscera are occasionally involved. Unlike gastroschisis, omphalocele is associated with other anomalies 50% to 70% of the time, with up to 50% of affected infants having significant congenital cardiac disease.

The intact sac of the omphalocele that partitions the viscera from the amniotic fluid usually protects it from the prolonged ileus characteristic of gastroschisis. Because of the broad base of the defect, strangulation is unlikely. However, a ruptured omphalocele is often complicated by ileus, the severity of which seems to be related to the length of exposure of the viscera to the amniotic fluid in utero. Ileocolonic volvulus is also a possibility that may not be initially apparent.

The immediate and long-term outcomes of children who have omphalocele are directly related to the severity of the associated anomalies. In addition to congenital cardiac disease, omphalocele is frequently associated with genetic anomalies and syndromic presentations. Approximately 10% of affected infants will have Beckwith-Wiedemann syndrome (gigantism, macroglossia, omphalocele, and hypoglycemia secondary to pancreatic hyperplasia). Other associations of multiple anomalies include CHARGE (coloboma, heart defects, choanal atresia, mental retardation, and genitourinary and ear anomalies) and VACTERL (vertebral, anal, cardiac, tracheoesophageal, renal, and limb deformities).

GENETICS. Associated karyotype abnormalities are found in ~30% of infants who have omphalocele, the most common being trisomy 13, 18, and 21. Pituitary homeobox 2, insulin-like growth factor 2, cyclin-dependent kinase inhibitor 1C, and a polymorphism in the methylentetrahydrofolate reductase gene (677C-T) have all been associated with omphalocele, but the exact extent to which these genes are involved in its pathogenesis remains uncertain.

EMBRYOLOGY. The anatomic pathology (and subsequent clinical management) of an omphalocele is intimately related to the events of embryogenesis and fetal development. The third week of gestation is characterized by the formation of the trilaminar germ disc composed of endoderm, mesoderm, and ectoderm. The rapid growth of the ectoderm and mesoderm (the outer two layers) leads to ventral folding of the embryo around the endoderm (the main precursor of the visceral organs). This process involves four body wall folds (cranial, caudal, and two lateral folds), which in normal development approximate in the midline to form the umbilical ring. The sixth week of gestation is characterized by physiologic herniation of the midgut through the umbilical ring, with subsequent intestinal rotation (270 degrees,
converging counterclockwise) and spontaneous reduction of the herniated bowel by week 10 of gestation. An omphalocele arises from incomplete folding of the lateral body wall folds and incomplete reduction of the physiologic intestinal herniation. The umbilical stalk persists as a broad-based protective sac composed of peritoneum and amnion separated by a layer of Wharton’s jelly (mucopolysaccharides).

Variants of omphalocele occur in the epigastrium and the infraumbilical region and are the result of incomplete folding of the cranial and caudal body wall folds, respectively. Epigastric omphalocele in combination with sternal cleft, ectopia cordis/cardiac defects, pericardial defects, and diaphragmatic hernia is known as the Pentalogy of Cantrell. Infraumbilical omphalocele, often associated with bladder or cloacal exstrophy, occurs in conjunction with imperforate anus and spinal defects to form the OEIS association (omphalocele, exstrophy of the bladder, imperforate anus, and spinal defects).

**Gastroschisis**

Gastroschisis is an abdominal wall defect that almost always occurs 1 to 2 cm to the right of the umbilicus. Unlike omphalocele, the eviscerated structures do not have a protective sac and are therefore in direct contact with the amniotic fluid in utero. This contact leads to the characteristic edematous, foreshortened, fluid-filled bowel. Gastroschisis typically occurs as an isolated malformation, and morbidity in the neonatal period is almost always directly related to the extent of gastrointestinal disease.

Approximately 10% of infants born with gastroschisis will have a concurrent intestinal atresia or stenosis, a process likely related to in utero vascular compromise from volvulus or compression of the bowel at the abdominal wall. As with omphalocele, intestinal nonrotation is frequently present in affected individuals. Prolonged ileus that delays enteral feeding is encountered to some extent in virtually all infants who have gastroschisis. This outcome is thought to be secondary to the effects of utero exposure of the bowel to amniotic fluid. Animal studies have shown that complete amniotic fluid replacement prevents the development of characteristic bowel inflammation; this finding has not, however, been translated into clinically useful therapy.

Gastroschisis is frequently associated with intrauterine growth restriction, with ~30% of affected infants born below the 10th percentile for birthweight. This causes the phenomenon is not well understood but is potentially related to both placental anomalies and direct nutritional wasting secondary to the exposed viscera. Conversely, omphalocele is not associated with intrauterine growth restriction but rather is found in neonates who are often large for gestational age.

Gastroschisis is also associated with prematurity in ~30% of affected individuals. This population is not immune to the complications of prematurity and low birthweight. Necrotizing enterocolitis is frequently encountered both before and after correction of the abdominal wall defect and can be a catastrophic complication. Similar to premature infants who do not have gastroschisis, necrotizing enterocolitis tends to occur after the initiation of enteral feeds and is more common in infants weighing less than 2500 g at birth.

**GENETICS.** The genetic basis for gastroschisis, as with omphalocele, is not well defined. Gastroschisis is most often sporadic, but there are reports of familial cases, including concordance for the defect in monozygotic twins, dizygotic twins, and distant relatives. Multiple genes have been implicated in the development of gastroschisis, including those encoding endothelial nitric oxide synthase, intracellular adhesion molecule 1, and atrial natriuretic peptide. Polymorphisms within these genes are associated with a nearly twofold increase in the odds of developing gastroschisis; this figure increases to greater than fivefold when combined with maternal smoking.

**EMBRYOLOGY.** Unlike omphalocele, the embryologic etiology of gastroschisis is a debated topic for which a number of theories have emerged over the last 50 years. Importantly, the association with endothelial nitric oxide synthase and intracellular adhesion molecule 1, along with the significant influence of maternal smoking, lends support to theories of vasculogenic pathogenesis. In 1980, deVries suggested that untimely involution of the right umbilical vein leads to apoptosis in the surrounding mesenchyme and resorption of the body wall. This theory is favored because it explains the right-sided preponderance of the defect and fits with the genetic associations described earlier. Interestingly, there are rare cases of left-sided gastroschisis found in the setting of left umbilical vein resorption. Others have suggested that early involution of the vitelline artery causes body wall ischemia and necrosis. Counterarguments to this second theory, however, note that segmental vessels from the aorta, not the vitelline vessels, typically perfuse the abdominal wall. Other theories of pathogenesis include rupture of the umbilical membrane before reduction of the physiologic visceral herniation (essentially a ruptured omphalocele), teratogenic interference with mesenchymal differentiation, and failed fusion of the yolk sac with the umbilical stalk, leaving the yolk sac as a lead point for herniation.
sensitivity of diagnosis of an abdominal wall defect. Ultrasound has a sensitivity of routine prenatal ultrasound often provides the initial diagnosis of an abdominal wall defect. Ultrasound has a sensitivity of routine prenatal ultrasound often provides the initial diagnosis of an abdominal wall defect. Ultrasound has a sensitivity of routine prenatal ultrasound often provides the initial diagnosis of an abdominal wall defect. Ultrasound has a sensitivity of 0% to 75% and a specificity of 95% for both omphalocele and gastroschisis. As is the case with ultrasound in many settings, utility is affected by the timing of the study and operator experience. After the in utero diagnosis of an abdominal wall defect, comprehensive fetal imaging, including echocardiography and magnetic resonance imaging, should be performed to identify any associated anomalies. Laboratory studies have little diagnostic or prognostic value with the exception of maternal serum α-fetoprotein (AFP) levels. Maternal serum AFP is commonly used to screen for chromosomal abnormalities and neural tube defects, and is also helpful in screening for abdominal wall defects. Gastroschisis is associated with high levels of maternal serum AFP (approximately seven to nine multiples of the mean), whereas omphalocele typically has a more modest elevation (approximately four multiples of the mean).

Elevated AFP levels or positive ultrasound findings should lead to consideration of amniocentesis or chorionic villus sampling. High-risk pregnancies are identified by the presence of significant karyotype abnormalities and allow for parental counseling and mobilization of health care resources. After prenatal diagnosis, referral for delivery at a center with access to both a neonatologist and a pediatric surgeon is an important next step in management.

The mode and timing of delivery for both omphalocele and gastroschisis are controversial. Sonographic evidence of fetal bowel dilation and thickened bowel wall has been suggested as an indication for induced preterm delivery, and some authors suggest that cesarean delivery is preferred in the setting of omphalocele to avoid sac rupture. There is currently no definitive evidence, however, to recommend induced preterm delivery or elective cesarean delivery for neonates who have abdominal wall defects.

Postnatal Care
Both omphalocele and gastroschisis present with varying degrees of severity, and the involvement of both a neonatologist and a pediatric surgeon is mandatory to achieve the best possible outcomes. The basic principles of initial management include cardiopulmonary stabilization followed by protection of the exposed viscera or omphalocele. An important initial consideration for neonates who have either gastroschisis or omphalocele is thermoregulation because the exposed viscera predispose to hypothermia, particularly with gastroschisis. Immediate measures include protection with saline-soaked dressings and placement of a “bowel bag” to limit insensible fluid and heat losses. The surgeon must then assess the viability of the exposed intestine and consider the severity of any associated anomalies while formulating a comprehensive care plan with the neonatologists. Both the embryologic and pathophysiologic distinctions between omphalocele and gastroschisis lead to differences in the options for definitive treatments (discussed in the following sections).

OMPHALOCELE. Immediate management considerations for omphalocele include airway protection with intubation and mechanical ventilation if necessary, fluid resuscitation (omphalocele predisposes to increased insensitive fluid loss, although not as dramatically as gastroschisis), gastric decompression, and protection of the sac with saline-soaked dressings. After initial stabilization, efforts are directed toward identifying any associated evidence is lacking in each case, and this remains an area of both active debate and investigation.

**Differential Diagnosis**
Prune-belly (Eagle-Barrett) syndrome is defined as the congenital absence of abdominal wall musculature, bilateral cryptorchidism, and ureteral, bladder, and urethral anomalies. It is a rare condition, with an incidence of ~1 per 50,000 live births with a strong male preponderance. The characteristic flaccidity and wrinkling of the abdominal skin is a direct result of the absent musculature but rarely causes functional impairment. The outcomes associated with prune-belly syndrome are directly related to the severity of the genitourinary anomalies. Prune-belly syndrome is differentiated from omphalocele by containment of the abdominal viscera within a continuous layer of skin and the presence of a normal umbilicus.

Umbilical hernias occur in roughly 80% of neonates with birthweights <1200 g and in 20% of neonates with birthweights <2500 g, and they represent incomplete closure of the umbilical ring after separation of the umbilical stalk. They are differentiated from other congenital abdominal wall defects by the presence of epithelialized skin overlying the defect and the presence of an intact, but enlarged, umbilical ring. The majority of umbilical hernias are directly related to the severity of the genitourinary anomalies. It is a rare condition, with an incidence of 60% to 75% and a specific karyotype abnormalities and allow for parental counseling and mobilization of health care resources. After prenatal diagnosis, referral for delivery at a center with access to both a neonatologist and a pediatric surgeon is an important next step in management.

**Management**

**Prenatal Care**
Routine prenatal ultrasound often provides the initial diagnosis of an abdominal wall defect. Ultrasound has a sensitivity of ~60% to 75% and a specificity of ~95% for both omphalocele and gastroschisis. As is the case with ultrasound in many settings, utility is affected by the timing of the study and operator experience. After the in utero diagnosis of an abdominal wall defect, comprehensive fetal imaging, including echocardiography and magnetic resonance imaging, should be performed to identify any associated anomalies. Laboratory studies have little diagnostic or prognostic value with the exception of maternal serum α-fetoprotein (AFP) levels. Maternal serum AFP is commonly used to screen for chromosomal abnormalities and neural tube defects, and is also helpful in screening for abdominal wall defects. Gastroschisis is associated with high levels of maternal serum AFP (approximately seven to nine multiples of the mean), whereas omphalocele typically has a more modest elevation (approximately four multiples of the mean).
anomalies. This evaluation includes a detailed physical examination, karyotype analysis, echocardiography, and renal ultrasonography.

The definitive management of omphalocele depends on both the size of the defect and the integrity of the sac (intact or perforated). A “giant omphalocele” (base diameter >4 cm) with an intact sac is treated with daily application of silver sulfadiazine to promote progressive epithelialization (Figs 3A and 3B). A body wrap is placed and progressively tightened to facilitate reduction of the abdominal contents and to reestablish the peritoneal domain. Elective repair of the fascial defect is performed after a minimum of 6 months but typically after 2 years of age. If the sac is ruptured, a loose silo fashioned from silastic mesh is secured to the edges of the defect and used to cover the intestine. Sequential cinching of the silo in the midline allows gradual reduction of the bowel and reestablishment of abdominal domain, after which the silo is removed and a second interposition mesh is placed to achieve abdominal wall closure. It is rarely possible to achieve primary facial closure.

Small omphalocles, which are more frequently associated with karyotype abnormalities, are either closed primarily or epithelialized with delayed closure. If primary closure is considered, the eviscerated structures must be reducible without inducing an abdominal compartment syndrome. This is typically defined as bladder pressure >20 mm Hg or central venous pressure >4 mm Hg. The presence of associated anomalies requiring more urgent intervention also precludes primary closure.

GASTROSCHISIS. As with omphalocele, the initial management of gastroschisis begins with airway protection, fluid resuscitation, placement of an orogastric tube, and protection of the eviscerated structures with moist dressings and a protective covering (“bowel bag”). If segments of necrosis or perforation are present, immediate surgical resection is required. No attempt is made at primary anastomosis in the presence of ongoing inflammation, and any identified atretic segments are often left untouched. If the peritoneal cavity will accommodate, the eviscerated structures are reduced, stomas are created if necessary, and the abdomen is closed. If there are bowel anomalies, reexploration and reestablishment of intestinal continuity are planned only after resolution of bowel inflammation in 6 weeks’ time.

If resection is needed, it is imperative to preserve as much bowel length as possible to avoid long-term parenteral nutrition dependence. This process typically requires at least 30 cm of small bowel if the ileocecal valve is left intact or >50 cm if the ileocecal valve is removed. The development of short bowel syndrome is dependent on a number of factors other than bowel length, however, including gestational age, quality of remaining bowel, and the presence of the ileum for absorption of vitamin B₁₂ and fat-soluble vitamins.

![Figure 3. A giant omphalocele A. before and B. after full epithelialization.](http://neoreviews.aappublications.org/)

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If the bowel is viable at birth, the options for definitive management include reduction with primary closure or serial reduction with delayed closure. When the eviscerated structures are reducible without causing an abdominal compartment syndrome or respiratory compromise, primary closure is a reasonable course of action. Delayed closure involves placement of a preformed silo with a spring-loaded base over the eviscerated structures, with anchoring of the atraumatic spring deep to the fascial defect (Fig 4A). This procedure can be done either at the bedside or in the operating room based on the stability of the infant and surgeon preference. Serial reduction is then accomplished by once- or twice-daily ligation of the silo until the eviscerated contents are entirely reduced (Figs 4B and 4C).

After reduction (either primary or delayed), the defect is typically repaired by suturing the fascial defect and attempting a cosmetic reconstruction of the skin defect and umbilicus. Sutureless closure has been proposed as an alternative to the traditional sutured closure and is gaining popularity, with emerging reports of decreased length of mechanical ventilation and improved cosmesis at the umbilicus (Figs 5A–5D). This technique involves primary or staged reduction of the intestine followed by placement of a plastic-occlusive dressing over the defect (without any attempt at suture closure of the defect). If primary reduction is achieved and a sufficient length of umbilical cord is intact, the cord can be used as a biological bowel cover under the plastic dressing. With silo reduction, the cord is typically already removed, in which case a nonabrasive absorptive sponge such as Mepitel® (Mölnlycke Health Care US, LLC, Norcross, GA) is placed between the plastic dressing and the bowel. The wound is then allowed to heal by secondary intention. The umbilical ring closes by contraction, and full epithelialization is achieved by 2 weeks. The child is typically left with a sometimes sizable umbilical hernia, which in most cases closes by 1 year of age.

**Postreduction Management**

Antibiotics are continued for 48 hours after reduction or silo placement. In the absence of other major physiologic or anatomic anomalies, attention is focused on providing adequate caloric intake and electrolyte homeostasis. Because prolonged ileus is almost always encountered, parenteral nutrition is initiated early in all cases of

![Figure 4. Delayed closure is performed when the abdominal domain is insufficient for primary reduction. A. A silo with a spring-loaded base is placed, and; B. the silo is tied down over several days until; C. complete reduction is achieved.](http://neoreviews.aappublications.org/)
gastrostomias. Selective use of parenteral nutrition is appropriate for infants who have an omphalocele. Enteral feeding is initiated as early in the postnatal period as possible and can be started even while waiting for definitive closure of the abdominal wall defect. We initiate trophic enteral feeding as soon as gastric tube output decreases and titrate to meet caloric requirements as tolerated. A variation of this method, the so-called minimal enteral feeding, has been shown to decrease the length of dependence on parenteral nutrition, rates of nosocomial infections, and length of hospitalization. Given the frequency of prolonged ileus in the setting of gastroschisis, the evaluation for mechanical obstruction (ie, small bowel contrast study) is not undertaken until the sixth week of life followed by elective repair of any identified intestinal stricture or atresia.

Outcomes
The outcomes associated with omphalocele are directly related to the presence of associated anomalies. Isolated omphalocele has a mortality rate of ~10%, but this figure increases to nearly 60% when other anomalies are present. The most common complications related to the omphalocele include ileus, wound infection, and sepsis, all of which occur in ~15% of affected individuals. Concomitant cardiopulmonary disease and chromosomal
anomalies are the leading causes of death in the acute setting.

In comparison, the outcomes associated with gastrochisis are directly related to the severity of the gastrointestinal disease. The overall mortality rate is ~4% to 7%, and the majority of those deaths are in the setting of gastrointestinal catastrophe (eg, extensive necrosis, multiple atresias, short bowel syndrome). The most common survivable complications of gastrochisis are prolonged ileus, catheter-related infections, and sepsis, all of which occur in ~15% to 30% of affected individuals.

If survival is achieved in the neonatal period, excellent long-term outcomes are now relatively common for both isolated omphalocele and uncomplicated gastrochisis. Childhood studies cite higher rates of learning disabilities in survivors of abdominal wall defects; however, adult survivors attain levels of education and positions of employment similar to the general population. Furthermore, roughly nine of 10 adult survivors of omphalocele or gastrochisis consider themselves to be in generally good health. The most common complaints in this population include abdominal scarring and intermittent abdominal discomfort and/or symptoms of gasoressophageal reflux.

Summary

Omphalocele and gastrochisis represent two distinct pathologies with different genetic, embryologic, and environmental components. Aggressive resuscitative measures along with patience in reduction and closure of the defect allow for preservation of gastrointestinal function in the majority of cases. Long-term survival with excellent functional outcomes is possible in the absence of catastrophic bowel injury or significant associated anomalies.

Suggested Reading


American Board of Pediatrics Neonatal–Perinatal Content Specifications

- Know the pathogenesis, clinical manifestations, and associated abnormalities of gastrochisis.
- Know the approach to management, the complications, and the difficulties in providing enteral nutrition to neonates with gastrochisis.
- Know the pathogenesis and associated anomalies associated with omphalocele.
- Know the approach to management, clinical manifestations, the differential diagnosis of, and the complications of treatment of omphalocele in neonates.
- Realize the association of major congenital anomalies involving the gastrointestinal tract and abdominal wall with those involving other organs.
NeoReviews Quiz

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1. A prenatal diagnosis of gastroschisis is made for a 20-year-old mother who will deliver at your institution. Which of the following is true for the mother/fetus regarding this diagnosis?
   A. Compared with omphalocele, the diagnosis of gastroschisis is much more likely to be associated with other anomalies, with 50% having congenital heart disease.
   B. The most common abdominal wall defect in gastroschisis occurs 1 to 2 cm to the left of the umbilicus.
   C. Gastroschisis is associated with prematurity in 30% of affected patients.
   D. Although there is a lack of a continuous protective sac, the intestines are usually covered by a thick coating of Wharton’s jelly.
   E. Approximately one-half of infants who have gastroschisis have pseudo-gigantism and will be large for gestational age.

2. A 30-year-old mother has a previous child who had gastroschisis. She is now pregnant again and asks about the possibility of prenatal diagnosis of gastroschisis. Which of the following is true?
   A. Because it is a gross abdominal wall defect, ultrasound at 18 to 20 weeks should have 100% sensitivity, although specificity for gastroschisis may be 80% to 90%.
   B. Gastroschisis is associated with decreased levels of maternal serum α-fetoprotein on the prenatal screen.
   C. If gastroschisis is detected, the mother should opt for a cesarean delivery between 30 and 32 weeks' gestational age.
   D. Maternal smoking is associated with increased risk of developing gastroschisis, particularly in the context of polymorphisms within certain genes such as those encoding endothelial nitric oxide synthase.
   E. The standard of care has moved to fetal surgery at 24 weeks to repair the defect in utero to reduce inflammation of the intestines.

3. A mother who has a known prenatal diagnosis of omphalocele in the fetus presents in active labor at 37 weeks' gestational age. Which of the following is an appropriate aspect of initial management for this mother and infant?
   A. Tocolysis should be initiated to avert delivery until 39 weeks.
   B. There is clear evidence that cesarean delivery will improve prognosis for both the mother and infant.
   C. After birth, the omphalocele should be protected with saline-soaked dressings and placement of a “bowel bag.”
   D. Intubation of the infant in the delivery room should be avoided to prevent bowel distension, and blow-by oxygen can be given if the patient is cyanotic.
   E. In almost all circumstances, if the patient is stable from a respiratory standpoint, the patient should be taken to the operating room directly after delivery.

4. A male infant who has gastroschisis is born at 33 weeks' gestational age. On initial examination, the bowel generally looks healthy and viable. Which of the following is an appropriate aspect of clinical management for this infant?
   A. The patient should be stabilized in the NICU for at least 1 week of antibiotics before any surgery or procedure.
   B. The patient should be taken to the operating room on the first day, have any suspicious necrotic segments removed, with primary anastomoses at those segments and primary closure of the abdomen.
C. The patient should be started on feedings first, and if not tolerated within 1 week, started on parenteral nutrition at that time.

D. Because the amniotic fluid can cause inflammation to the exposed bowel, the patient should have the bowel soaked in an antibiotic-infused solution within the “bowel bag” or silo for at least 2 days.

E. If the eviscerated structures are reducible without causing abdominal compartment syndrome or respiratory compromise, primary closure may be a reasonable option.

5. A 35-weeks'-gestational-age female infant who has gastroschisis is now 2 weeks old and has had definitive closure of her defect. Which of the following is appropriate in terms of nutrition management for this infant?

A. Because the patient is now 2 weeks old and 37 weeks’ corrected age, parenteral nutrition can be discontinued if her weight is back up to birthweight, with the goal of preventing liver disease.

B. Enteral feedings should be started at 40 weeks' gestation or later; feedings will not be tolerated until that time, and parenteral nutrition will supply adequate calories.

C. Trophic enteral feedings can be started when gastric output via the orogastric tube decreases.

D. Before any feedings, a small-bowel contrast study should be performed to assess for patency of the intestines.

E. In most cases of gastroschisis, definitive closure leads to active bowel function, and an infant at this age should be able to feed ad libitum without other nutrition supplementation in the next 2 to 3 days.
Ventral Abdominal Wall Defects
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