Gastroschisis

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Introduction:

Gastroschisis is one of the spectrum of congenital abdominal wall defects.[1] Gastroschisis is usually less than 4cm in diameter, with no covering membrane and the contents float within the amniotic fluid. The contents are usually the midgut with stomach and sometimes a gonad. It is almost always to the right side of the umbilical cord and does not involve the umbilicus.[1, 2] The other anomalies of the spectrum of congenital abdominal wall defects include umbilical defects with intact rectus abdominis muscles like Omphalocele, pentalogy of Cantrell, ectopia cordis thoracis, cloacal exstrophy, hernia of umbilical cord and umbilical hernia.[1] The absence of a sac and the typical right-sided location in gastroschisis differentiates it from the other abdominal wall defects.

At birth, the protruding bowel may appear normal, but with passage of time, usually after 20 minutes of birth, the extruded intestine will get thickened and covered with fibrinous exudate and get matted together and the individual loops cannot be then distinguished from each other.[1] Gastroschisis has been reported to be associated with gastrointestinal conditions like atresia, perforation, necrosis or volvulus. Such association has been termed as complicated gastroschisis and has been associated with poor prognosis.[1]

Epidemiology:

1) Global Incidence and Temporal Trends:

The global incidence of gastroschisis ranges from 2 to 5 per 10,000 live births, with marked geographic variation.[3, 4, 5] Since the late 20th century, many high-income nations of North America, Europe, and Australasia have reported a significant increase in incidence, from the 1980s through the early 2010s, particularly among infants born to young mothers (<20 years).[3, 4, 5] More recent registry data from high-income countries suggest plateauing or modest declines in incidence since approximately 2016, potentially reflecting changes in maternal risk-factor exposure, including reduced smoking prevalence.[6, 7] Despite these temporal shifts, young maternal age remains the strongest and most consistent epidemiologic risk factor worldwide.[3, 4, 5]

2) Epidemiology in India:

In contrast to countries with established congenital anomaly surveillance systems, reliable population-based epidemiologic data on gastroschisis in India are lacking. Published Indian literature consists predominantly of hospital-based case series from tertiary neonatal surgical

centres, which provide valuable clinical insights but are inherently limited by referral bias and incomplete case ascertainment.[8, 9] Recent published reports suggest incidence of approximately 2–5 per 10,000 live births.[10] The recent increase might be attributed to improvements in antenatal diagnosis, referral networks, increase in premature deliveries and better survival of preterm babies.[10, 11, 12]

Associated Risk Factors[1, 10]:

Epidemiologic studies consistently identify several risk factors as follow[1, 10]:

i) Demographic Risk Factors:

- Young maternal age
- Low socioeconomic status
- Poor maternal prenatal care
- Primigravida status

ii) Nutritional factors:

- Low levels of glutathione and a-carotene
- High levels of nitrosamines
- Absence of supplemental vitamin intake during pregnancy
- Absence of folic acid fortification
- Poor nutrition (Maternal obesity has actually been found to be protective)

iii) Most studies show **no familial or genetic risk factors**

iv) Environmental and other risk factors:

- Living near chemical plants, farms, landfills
- Use of vasoconstrictive agents and drugs like cocaine, methamphetamine, and marijuana during pregnancy
- Cigarette smoking
- Alcohol use

***Maternal Age Distribution*:** Amongst the above stated risk factors, a consistent and striking epidemiologic feature seen in gastroschisis is the strong association with young maternal age. Mothers younger than 20 years have a 5–10-fold higher risk compared with women aged 25–29 years. The risk decreases progressively with increasing maternal age, suggesting an environmental or behavioural contributors.[3, 4, 12]

Sex Distribution: Most studies report a slight male predominance, although the difference is small and not always statistically significant.[4, 10, 13]These associations support a multifactorial etiology, likely involving vascular disruption during early embryogenesis.[1, 3, 10, 14, 15]

Associated Anomalies and their impact on outcomes: Unlike omphalocele, gastroschisis is usually an isolated anomaly, with chromosomal abnormalities being rare (<2%). However, intestinal complications such as atresia can occur in 30% patients.[10] Other systems are rarely involved. Advances in neonatal intensive care and surgical management have improved survival to >90% in high-income countries, though outcomes remain poorer in low-resource settings.[1, 10, 13, 15]

Clinical Features of Gastroschisis:

The defect in gastroschisis is small (usually less then 4cm) and is almost always to the right side of umbilicus (Figure 1). Sometimes, a skin bridge is present between the defect and the umbilicus. The small bowel protrudes through the defect with no covering and is directly exposed to the environment. Sometimes, stomach, large bowel and gonads may be seen protruding. On the other hand, omphalocele is the defect of the umbilicus and the herniating bowel is covered with sac (Figure 2).

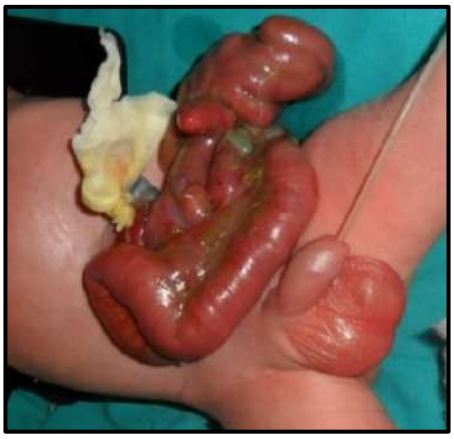

Figure 1: Gastroschisis – Clinical Picture

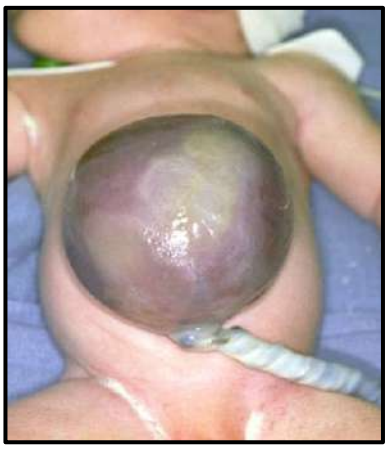

Figure 2: Omphalocele: Clinical Picture

At birth, the protruding bowel may appear normal, but with passage of time, usually after 20 minutes of birth, the extruded intestine will get thickened and covered with fibrinous exudate and get matted together and the individual loops cannot be then distinguished from each other.[1] Gastroschisis has been reported to be associated with gastrointestinal conditions like atresia, perforation, necrosis or volvulus. Such association has been termed as complicated gastroschisis and has been associated with poor prognosis.[1]

The difference between Gastroschisis and omphalocele is tabulated in Table 1.

S.No.	Features	Gastroschisis	Omphalocele
1	Embryologic defect	Failure of closure of the abdominal wall (para-umbilical defect)	Failure of midgut return into abdominal cavity
2	Location of defect	Mostly to the right of umbilicus	Midline at the umbilical ring

3	Covering sac	Absent – bowel is directly exposed to the environment	Present (composed of amnion + Wharton's jelly + peritoneum)
4	Contents	Usually small intestine ± stomach/colon; sometimes gonads	Bowel ± liver and other viscera
5	Umbilical cord insertion	Normal, separate from defect	Inserts onto the sac
6	Bowel appearance	Thickened, edematous, inflamed with "peel"	Usually normal in appearance
7	Associated anomalies	Rare (intestinal atresia might be present)	Common (cardiac, neural tube, chromosomal)
8	Chromosomal association	Uncommon	Frequent (Trisomy 13, 18, 21; Beckwith–Wiedemann)
9	Gestational age / birth weight	Often preterm, low birth weight	Usually term or near-term
10	Antenatal maternal AFP	Markedly elevated	Mild to moderate elevation
11	Risk of infection and sepsis	High due to exposed bowel	Lower (intact sac unless ruptured)
12	Immediate neonatal issue	Fluid, heat loss; bowel injury	Risk of sac rupture
13	Management at birth	Bowel protection + primary or staged closure (silo)	Sac protection; staged closure if giant omphalocele
14	Prognosis	Generally good if isolated	Depends on associated anomalies

Embryogenesis of Gastroschisis:

Multiple theories for embryogenesis of gastroschisis have been proposed, but none of them are well-accepted. To understand the embryogenesis of gastroschisis, it is essential to understand the embryology during the organogenesis period. At 3 weeks' gestation, the four folds develop into the embryonic disk which enclose the body cavities - two lateral folds, one cephalic fold and the caudal fold. Simultaneously, the gut tube is formed along the length of embryo and its elongation and development take place within the umbilical coelom till about10 weeks, after which the gut returns from the space within the umbilical stalk to the peritoneal cavity and undergoes rotation and fixation.[1]

With the above background comes the first theory of its embryogenesis, i.e., failure of development of umbilical coelom. The elongating intestine then has no space in which to expand and the gut then ruptures out of the body wall to the right of umbilicus, which is relatively unsupported because of resorption of the right umbilical vein at about 4 weeks' gestation.

Inadequate mesoderm formation has also been proposed which leads to absence or weakness of the muscular and connective tissue layers, producing a paraumbilical wall defect through which abdominal contents herniate.[16]

An alternate explanation is that the yolk sac and associated vitelline structures fail to incorporate into the umbilical cord, hereby allowing the midgut to exit the abdomen at the point they exit. This theory also seems reasonable as gastroschisis has no covering membrane.[1]
Another theory of embryogenesis of gastroschisis is abnormal or premature regression of the right umbilical vein, leading to localised ischemia and weakening of the right periumbilical wall and causing gastroschisis.[17, 18]
It has also been proposed that gastroschisis results from defective lateral body folding, specifically failure of the right lateral fold to advance and fuse properly.[16, 19] The resulting incomplete closure of the abdominal wall leaves a persistent paraumbilical opening (Figure 3). This theory aligns with the anatomic timing of abdominal wall formation during weeks 4–5 of gestation.

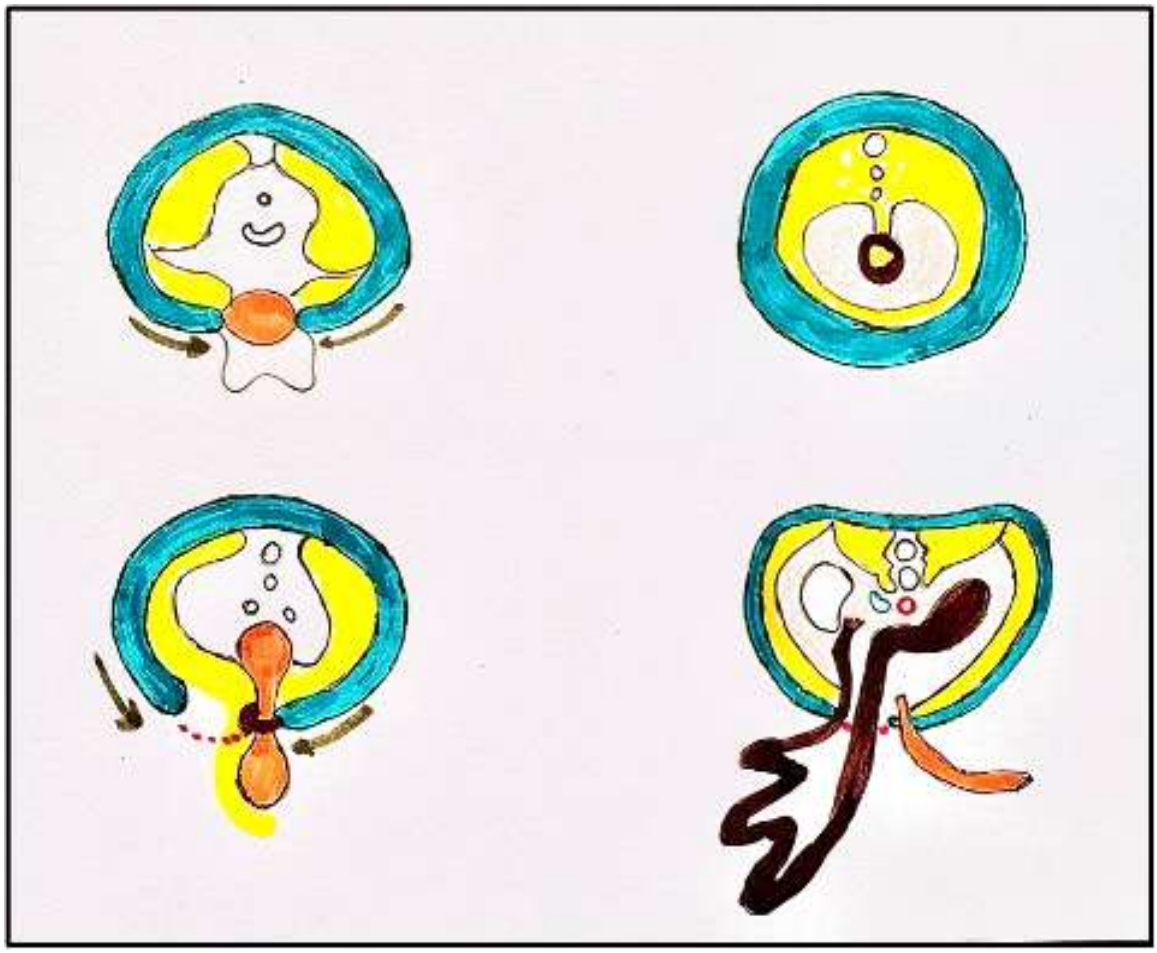

Figure 3: A. Normal lateral body folding: Medial migration of right and left lateral body folds (yellow) during the 4th week of gestation; **B. Normal anterior abdominal wall formation:** Fusion of lateral body folds resulting in a closed ventral abdominal wall and normal umbilical ring; **C. Failure of right lateral body fold:** Incomplete closure of the right lateral body fold producing a persistent ventral abdominal wall defect (red dots); **D. Gastroschisis:** Right-sided para-umbilical defect with uncovered herniation of midgut loops into the amniotic cavity.

The most widely accepted and leading hypothesis is that gastroschisis results from a vascular accident, typically involving the right omphalomesenteric artery or right umbilical vein, leading to ischemic injury and subsequent full-thickness disruption of the abdominal wall.[16, 18] This also explains the defect's consistent right-sided location and is supported by the frequent association of bowel injury and atresia, which are compatible with an ischemic mechanism.
Recent studies suggest that the above stated embryologic pathways might be modified or triggered by external teratogenic factors—especially vasoactive exposures such as smoking, young maternal age, NSAIDs, and certain recreational drugs.[20, 21] These factors might increase the susceptibility to vascular disruption or interfere with mesodermal or umbilical ring development. In a comprehensive review of the literature gastroschisis was reported to be induced by 22 teratogens.[1]
The appearance of the bowel is most often a postnatal event. At the moment of birth, the bowel in gastroschisis is usually quite normal. Twenty minutes later it begins to acquire the characteristic changes. These changes may be due to exposure to air, but more likely they are related to mesenteric venous occlusion at the level of the abdominal wall defect with resultant edema and transudation of proteinaceous fluid.[1]

Recent systematic evidence (2023–2025) on etiopathogenesis of gastroschisis:

Several recent systematic reviews reported a multifactorial model vascular susceptibility along with environmental triggers, possibly modified by genetic susceptibility in the etiopathogenesis of gastroschisis, as follows[14, 22, 23, 24, 25]:

- **Genetics/genomics:** A recent systematic review by Marquart JP et al concluded that though the current human genetic studies are limited, they point toward gene–environment interactions for gastroschisis etiology; more modern omics/epigenetic studies are recommended.[22]
- **Experimental (animal) models:** Another systematic review of fetal lamb experiments by Arai T et al summarized the different surgical/experimental methods which induce simple versus complex gastroschisis. They tried to clarify prenatal manipulations which produce ischemic/structural bowel changes.[23]
- **Infectious exposures (GUTI):** The systematic review and meta-analysis by Vinogradov R et al concluded that periconceptional genitourinary infections (UTI and STI) might be associated with increased gastroschisis risk, supporting environmental/vascular-mediated mechanisms.[24]
- **Medication exposures/teratogens:** Baldacci S et al, published a recent meta-analysis on medication use in pregnancy and reported associations between first-trimester exposure to several over the counter drugs like aspirin, ibuprofen, pseudoephedrine/phenylpropanolamine and increased risk of gastroschisis, again consistent with environmental/vascular or teratogenic contributors to embryogenesis.[14]

Antenatal Diagnosis, Monitoring, and Management:

Accurate prenatal diagnosis is important so that planning of the management and counselling of to be parents could be done well.

Ultrasonography: Antenatal ultrasonography, especially the second-trimester anomaly scan (18–22 weeks), is the commonest diagnostic modality used. Although first-trimester diagnosis is now been increasingly reported with advances in imaging technology. The characteristic sonographic features include free-floating bowel loops herniating through a right-sided paraumbilical abdominal wall defect, absence of a covering membrane, and a normal insertion of umbilical cord.[13, 16, 20, 26]

Other markers: Alpha-fetoprotein in both maternal serum and amniotic fluid and Acetylcholine esterase in amniotic fluid are elevated in abdominal wall defects; provided open neural tube defects have been ruled out.

Gastroschisis can be differentiated from omphalocele at ultrasound by its paraumbilical location and lack of a covering membrane. Presence of liver and large size of the defect would help to differentiate ruptured omphalocele from gastroschisis. Sensitivity of ultrasound and maternal serum Alpha-fetoprotein screening is close to 80% in diagnosing abdominal wall defects[10]

Antenatal Counselling and Monitoring: Counselling of the to be parents about the nature of primary defect, associated anomalies and genetic syndromes. After the associated lethal defects have been ruled out, the pregnancy is to be carried till term with close monitoring and surveillance as follows:

a) ***Serial Ultrasound Surveillance:*** After diagnosis, serial ultrasound surveillance is recommended every 2–4 weeks, increasing to weekly or biweekly in the third trimester. Monitoring of the fetus is to be done for fetal growth, amniotic fluid volume, and bowel characteristics, including dilatation, wall thickness, and echogenicity.
b) Fetuses with gastroschisis are at increased risk of fetal growth restriction and stillbirth, antenatal surveillance should include umbilical artery Doppler studies, as well as non-stress testing or biophysical profiles from 32–34 weeks' gestation [10, 27, 28, 29]
c) Parents should be encouraged to follow-up and get delivery done at a tertiary care centre with well-equipped NICU, paediatric surgery and neonatal anaesthesia availability

A recent systematic review and meta-analysis (2023) demonstrated that intra-abdominal bowel dilatation is significantly associated with complex gastroschisis, including bowel atresia, necrosis, and perforation, whereas isolated extra-abdominal bowel dilatation has limited predictive value.[27]

***Timing and mode of Delivery*:** Current evidence supports expectant antenatal management with close surveillance rather than routine early preterm delivery. A systematic review and meta-analysis by Morris et al[30] showed that elective delivery before 36 weeks does not reduce mortality and increases neonatal respiratory morbidity. [30]

The optimal timing of delivery remains controversial. Recent systematic reviews and individual participant data meta-analyses suggest that delivery at 37–38 weeks' gestation provides the best balance between the risks of stillbirth and prematurity in uncomplicated gastroschisis.[30, 31] Planned early preterm delivery has not demonstrated a survival benefit.[11]

Multiple systematic reviews and meta-analyses consistently demonstrate no advantage of elective caesarean section over vaginal delivery in terms of neonatal survival, bowel outcomes, or length of hospital stay[32, 33] Accordingly, vaginal delivery is recommended[32]; however, the mode of delivery should be best decided by the treating Obstetrician and should be as per the obstetric indications

Antenatal Interventions: Antenatal corticosteroids can be administered according to routine obstetric indications for threatened preterm birth; there is no evidence of a gastroschisis-specific benefit.[30] Experimental approaches such as amnio-exchange or in-utero bowel covering remain investigational and have not recommended outside clinical trials.[34]

Summary of evidences as per the recent Randomized Controlled Trials:

- RCTs done were small and underpowered, limiting definitive conclusions.[35-38]
- Elective early preterm delivery (34–36 weeks) has been tested but does not improve outcomes and may increase complications (e.g., sepsis). Early elective delivery hence is not recommended solely to improve gastroschisis outcomes.
- There is no established prenatal intervention from randomized trials that alters the course of gastroschisis. Amnio-exchange has not been shown to have benefit in reducing bowel-related morbidity.
- A large ongoing RCT (GOOD Study) has been designed to address the gaps in evidence on delivery timing, but the results are still pending[38]

Postnatal Management:

As stated earlier, delivery of such babies is recommended at a tertiary care centre with well-equipped NICU, paediatric surgery and neonatal anaesthesia availability. Rapid transfer of the neonate from delivery room to the neonatal operating room increases the chances of primary closure of the defect.[1]

The main principles of management are:

- Stabilization of neonate
- Reduction of viscera

- Closure of the defect
- Management of the associated anomalies (if any)

Immediate Postnatal Management:

Stabilization of the neonate: It begins in the delivery room itself.

Protection of the exposed bowel: The exposed bowel should be immediately protected using a sterile warm saline-soaked gauze or sterile urobag, to minimize evaporative heat loss and bowel dilatation. It should be ensured that the defect in gastroschisis is not very narrow to compromise the bowel vascularity of the exposed bowel. The neonate should be positioned with support to the bowel to prevent traction on the mesentery.[10, 13] Orogastric tube placement, catheterization and per rectal wash are recommended to decompress the gut and reduce bowel distension and aspiration risk.[10]

Thermoregulation and Fluid Management: Infants with gastroschisis are at high risk of hypothermia and third-space fluid losses. Aggressive fluid resuscitation (150 – 175ml/kg/day) is required during the first 24 – 48 hours, guided by urine output, serum electrolytes, and acid–base status.[1, 10, 13, 39]

Respiratory Support and Antibiotics: Respiratory support should be provided as needed. Broad-spectrum intravenous antibiotics should be initiated early to reduce the risk of sepsis related to bowel exposure, typically continued until definitive abdominal closure and clinical stabilization.

Surgical Management:

1) **Reduction and Primary Closure:**

    - ***Without mesh:*** The surgical goal is safe reduction of herniated viscera and abdominal wall closure without causing abdominal compartment syndrome. This can be accomplished either without prosthetic mesh in selected neonates with minimal bowel edema and adequate abdominal domain.
    - ***With mesh:*** Closure with mesh may result in complications and require removal later

Steps of primary closure:

- The bowel contents are reduced gradually; this can be aided by decompressing the bowel by gastric aspiration, rectal washes and milking.
- The abdominal wall may be gently stretched by finger posteriorly to anteriorly to prevent closure under tension
- If still the closure feels tight, then relaxing incisions can be given in the abdominal wall fascia
- The closure should be done using interrupted sutures.

- It is important to ensure that the primary closure is not under tension which can cause abdominal compartment syndrome. The following parameters, if present, should prompt the surgeon to apply silo instead of primary closure[10]:

    - Mean airway pressure more than 25cm of water
    - Intravesical pressure more than 20cm of water
    - Saphenous intravenous line unable to flow by gravity

Even after primary closure, the neonate should be monitored post-operatively for early signs of abdominal compartment syndrome like persistent tachycardia, oliguria, increased ventilator setting requirements and tensed abdomen. Such patients might need decompression with silo placement.

Figure 4: Intra-operative image of Gastroschisis

Figure 5: Post-operative image of primary repair of gastroschisis

2) **Gradual/Staged Reduction:** This is done using a silo and is preferred when bowel edema is significant or primary closure would compromise ventilation or perfusion.[40, 41, 42] This can be applied bed side and kept tied to keep the bowel in place and tightened gradually to cause gradual reduction without compromising the ventilation. When the complete eviscerated bowel can be reduced, then primary closure can be done.

Figure 6: Neonate with silo-bag applied over the gastroschisis

3) ***Creation of ventral hernia with delayed repair:*** In this technique, only the skin is closed after reduction of the bowel at neonatal period and the resulting ventral hernia is repaired later when the child grows. This might be difficult and may require prosthesis or tissue expanders as the bowel grows into the defect and ventral hernia becomes larger and difficult to reduce.

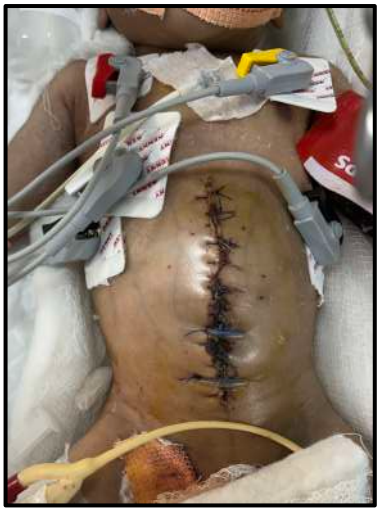

Figure 7: Immediate post-operative image with creation of ventral hernia

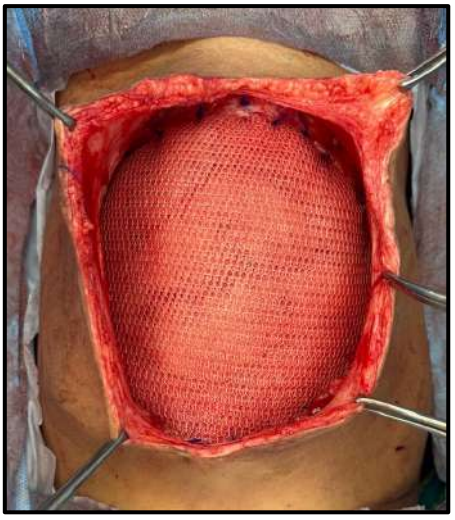

Figure 8: Mesh repair of the ventral hernia at the end of one year of age

Multiple systematic reviews have demonstrated no difference in survival between primary closure and silo placement, although silo use may reduce ventilatory compromise and improve physiologic stability.[11, 41]

Management of Complex Gastroschisis: Approximately 10–20% of infants have complex gastroschisis, characterized by intestinal atresia, necrosis, perforation, or volvulus.43 These infants often require:

- Delayed definitive surgery
- Resection of nonviable bowel
- Prolonged parenteral nutrition
- Increased risk of short bowel syndrome and mortality[43, 44]

Postoperative and Nutritional Management:

1. ***Parenteral and Enteral Nutrition:*** Total parenteral nutrition (TPN) is initiated early and continued until bowel function recovers. Enteral feeds are introduced gradually, usually after resolution of ileus and reduction of gastric aspirates 40 Early trophic feeding, when feasible, should be encouraged as it is associated with shorter hospital stay and earlier achievement of full enteral nutrition.[45]
2. ***Infection and Complication*** Surveillance
3. ***Management of post-operative bowel dysmotility:*** This poses a difficult challenge as bowel motility may take long to recover. Early gradual trophic feeds should be encouraged as tolerated. Use of prokinetic drugs like domperidone, erythromycin and metoclopramide have been tried with variable success.
4. Infants require close monitoring for:

    - Central line–associated bloodstream infections
    - Necrotizing enterocolitis-like bowel injury
    - TPN-associated cholestasis
    - Abdominal compartment syndrome[40]

Management of Cryptorchidism[10]:

This condition is associated with gastroschisis with an incidence of 15-30%. The herniated testis should be replaced in the abdominal cavity at the time of first surgery as this results in normal descent of the testis into the scrotum in majority of the cases.

Hospital Outcomes:

Survival rates for simple gastroschisis exceed 90–95% in high-resource settings. However, morbidity remains substantial, particularly in complex cases, with prolonged hospitalization and feeding difficulties being common.[11, 43, 44] Major causes of death are prematurity, sepsis and ischemia of the bowel.

Long-Term Follow-up:

1. ***Growth and Nutrition:*** Long-term follow-up focuses on growth, nutritional adequacy, and gastrointestinal function. While most survivors achieve normal growth by early childhood, infants with complex gastroschisis are at increased risk of:

    - Growth failure
    - Feeding intolerance
    - Intestinal dysmotility[44, 45]

2. ***Neurodevelopmental Outcomes:*** Most children with uncomplicated gastroschisis have normal neurodevelopmental outcomes, although prematurity and prolonged hospitalization may contribute to subtle delays.46, 47 Routine developmental surveillance is recommended during infancy and early childhood.

3. ***Gastrointestinal and Surgical Sequelae:*** Late complications may include:

    - Adhesive small bowel obstruction
    - Incisional hernia
    - Chronic abdominal pain or constipation[48, 49]

Lifelong follow-up is usually not required for simple gastroschisis unless for children with complex disease or short bowel syndrome.

Conclusion/Summary:

Prognosis of antenatally detected gastroschisis remains good in institutes with well-equipped and co-ordinated multidisciplinary neonatal, surgical, and nutritional care. Early stabilization, appropriate surgical strategy, meticulous postoperative management, and long-term follow-up are essential to optimize survival and functional outcomes.

Emerging Concepts and Research Gaps:

Despite extensive observational data, randomized evidence for antenatal interventions remains limited. Systematic reviews conclude that procedures such as amnio-exchange and fetal bowel covering have no proven benefit and should remain experimental.30, 34 Ongoing multicentre randomized trials evaluating optimal timing of delivery are anticipated to address key evidence gaps.

Future Prospects in Gastroschisis:

1. ***Improved Antenatal Risk Stratification:*** One of the major future goals in gastroschisis care is the development of reliable antenatal predictors of complex disease and adverse outcomes. Future research needs to focus upon:

- Composite prediction models integrating ultrasound parameters (intra-abdominal bowel dilatation, growth restriction), Doppler findings, and gestational age[3]
- Machine-learning–based risk models using longitudinal imaging and clinical data to better predict complex gastroschisis and neonatal morbidity[50]

Such tools may be beneficial for individualized counselling, optimized timing of delivery, and targeted neonatal preparedness.

2. ***Biomarkers and Amniotic Fluid Research:*** Experimental and translational research suggests that inflammatory mediators in amniotic fluid contribute to bowel damage in gastroschisis.27 Although investigational, the future prospects might focus on:

- Identification of biochemical biomarkers (cytokines, bile acids, oxidative stress markers) to predict bowel injury severity[28, 30]
- Development of non-invasive maternal serum or amniotic fluid markers to supplement ultrasound findings[28, 30]

3. ***Refinement of Antenatal Management Strategies***: There are no high-quality randomized evidence guiding antenatal management available yet. Future advances are expected from:

- Large multi-center randomized controlled trials evaluating optimal timing of delivery (e.g., early-term vs expectant management)[51]
- Standardization of antenatal surveillance protocols based on risk stratification rather than uniform intensive monitoring

4. ***Innovations in Fetal Therapy:*** At present, fetal surgical intervention for gastroschisis remains experimental and is limited to research settings.[32, 33] Although previous trials of amnio-exchange and fetal bowel covering have not shown benefit,[32] future fetal therapies might focus on:

- Targeted modulation of the intra-amniotic inflammatory environment
- Advanced biomaterials or minimally invasive fetal interventions

5. ***Advances in Neonatal and Surgical Care:*** Future neonatal management aims to reduce morbidity rather than mortality, which is already low in simple gastroschisis. Anticipated advances include:

- Standardized feeding protocols and enhanced recovery pathways to reduce time to full enteral feeds[11]
- Improved strategies to prevent intestinal failure–associated liver disease

- Refinement of minimally invasive and bedside closure techniques to reduce ventilator dependence and hospital stay[40]

6. Long-Term Outcomes and Quality of Life: Future research should also focus on long-term functional outcomes, including:

- Growth, gastrointestinal function, and nutrition
- Neurodevelopmental and psychosocial outcomes
- Health-related quality of life in adolescence and adulthood[44, 48]

This might be done by establishment of long-term follow-up registries to understand lifelong outcomes.

7. Global Surveillance and Prevention: Future prospects also include strengthening population-based congenital anomaly surveillance, particularly in low- and middle-income countries.[16] Improved data quality may:

- Clarify true global incidence trends
- Enable identification of environmental and behavioural risk factors
- Inform preventive public health strategies

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