Scientific Research Publishing

Open Journal of Obstetrics and Gynecology, 2026, 16(1), 11-16
https://www.scirp.org/journal/ojog
ISSN Online: 2160-8806
ISSN Print: 2160-8792

Complicated Gastroschisis with Progressive Fetal Bowel Dilatation: A Case Report

Basma Alsayegh*, Omar Taso, Alaa Dreidi, Ahmed Alzubi, Suhib Alzwaherh

Department of Obstetrics and Gynecology, King Hussein Medical Center, Royal Medical Services, Amman, Jordan
Email: *dr.basooom89@gmail.com

How to cite this paper: Alsayegh, B., Taso, O., Dreidi, A., Alzubi, A. and Alzwaherh, S. (2026) Complicated Gastroschisis with Progressive Fetal Bowel Dilatation: A Case Report. *Open Journal of Obstetrics and Gynecology*, **16**, 11-16.
https://doi.org/10.4236/ojog.2026.161002

Received: December 8, 2025
Accepted: December 28, 2025
Published: December 31, 2025

Abstract

Background: Gastroschisis is a congenital abdominal wall defect associated with significant morbidity and mortality, particularly when complicated by severe intestinal inflammation or ischemia. **Case Presentation**: We report the case of a 27-year-old primigravida with fetal gastroschisis diagnosed at 18 weeks' gestation. Serial prenatal ultrasonography demonstrated progressive bowel wall thickening and marked dilatation of the eviscerated bowel, increasing from 1.9 cm at 33 weeks to 2.53 cm at 35 weeks, suggestive of severe intrauterine intestinal injury. Following antenatal corticosteroid administration, a male neonate was delivered at term. At birth, the bowel appeared markedly edematous, congested, and ischemic. Surgical management on day of life one consisted of bowel reduction and primary fascial closure. The postoperative course was complicated by rapid hemodynamic deterioration consistent with systemic inflammatory response syndrome and abdominal compartment syndrome, refractory to maximal supportive therapy. The neonate died on day three of life due to multi-organ failure. **Conclusion**: This case underscores the prognostic value of serial prenatal ultrasound in identifying severe intestinal compromise in gastroschisis. In fetuses demonstrating marked bowel dilatation and wall thickening, the findings support consideration of staged reduction strategies over primary closure to mitigate the risk of postoperative systemic inflammatory response syndrome and abdominal compartment syndrome.

Keywords

Gastroschisis, Complicated Gastroschisis, Fetal Bowel Dilatation, Prenatal Ultrasound, Neonatal Surgery, Systemic Inflammatory Response Syndrome, Abdominal Compartment Syndrome

1. Introduction

Gastroschisis is a neonatal surgical emergency characterized by herniation of ab-

dominal viscera through a paraumbilical abdominal wall defect. Its global incidence has increased over recent decades and is currently estimated at 2 - 5 per 10,000 live births [1]. Despite advances in neonatal intensive care and surgical techniques, gastroschisis remains associated with substantial morbidity, particularly in its complicated form [2].

The pathophysiology of in utero intestinal injury in gastroschisis is attributed to two interdependent mechanisms. Chronic chemical peritonitis results from prolonged exposure of the bowel serosa to amniotic fluid, leading to inflammation, edema, and bowel wall thickening [3]. In addition, vascular compromise plays a critical role and is thought to arise from constriction and intermittent compression of the mesenteric vessels at the narrow abdominal wall defect, resulting in reduced perfusion, ischemia, and progressive inflammatory injury to the bowel [4].

Complicated gastroschisis—defined by the presence of intestinal ischemia, necrosis, atresia, or perforation—accounts for a disproportionate share of adverse outcomes and mortality [1] [4]. Prenatal identification of severe intestinal compromise is therefore essential for prognostication and postnatal management planning. This report describes a case of extreme antenatal bowel deterioration culminating in rapid postoperative systemic collapse.

2. Case Presentation

2.1. Antenatal Course and Ultrasound Findings

A 27-year-old healthy primigravida was referred to the Fetal Medicine Unit following detection of fetal gastroschisis at 18 weeks' gestation. Serial ultrasound examinations were performed to monitor fetal growth and bowel characteristics.

At 30 weeks' gestation, ultrasound demonstrated bowel wall thickening with mild dilatation of the eviscerated bowel measuring 1.2 cm. By 33 weeks and 5 days, bowel dilatation had progressed to 1.9 cm (**Figure 1**), a measurement associated with increased risk of intestinal ischemia and complicated gastroschisis. Given the concerning progression, antenatal corticosteroids were administered at 34 weeks' gestation.

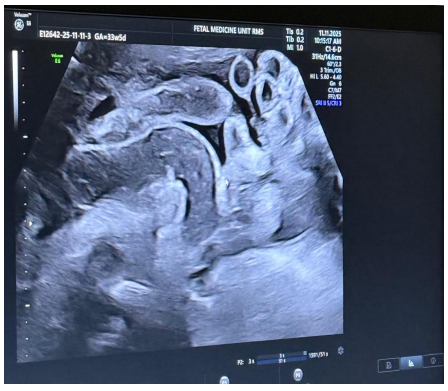

Figure 1. Fetal ultrasound at 33 weeks + 5 days showing the gastroschisis defect. A caliper measurement confirms the maximal diameter of the eviscerated bowel is 1.9 cm.

At 35 weeks and 5 days, ultrasound revealed severe dilatation of the eviscerated bowel with a maximal diameter of 2.53 cm, far exceeding established high-risk thresholds. Marked bowel wall thickening was also noted (Figure 2 and Figure 3), indicating advanced intrauterine intestinal injury.

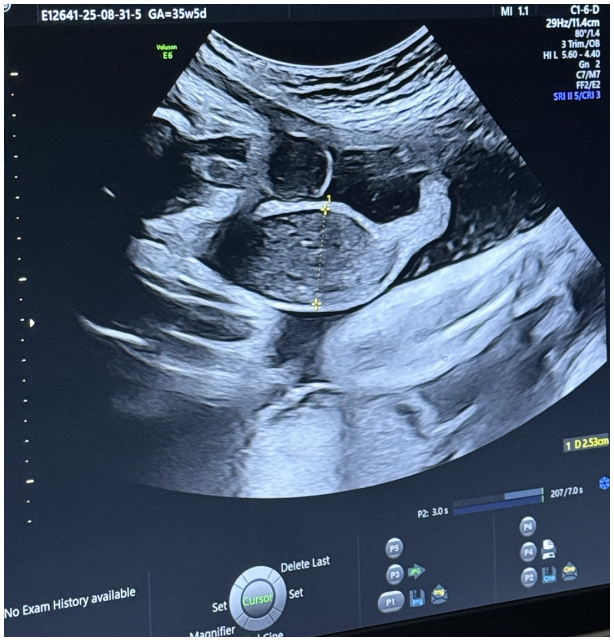

Figure 2. Fetal ultrasound at 35 weeks + 5 days confirming the severe eviscerated bowel dilatation with a caliper measurement of 2.53 cm.

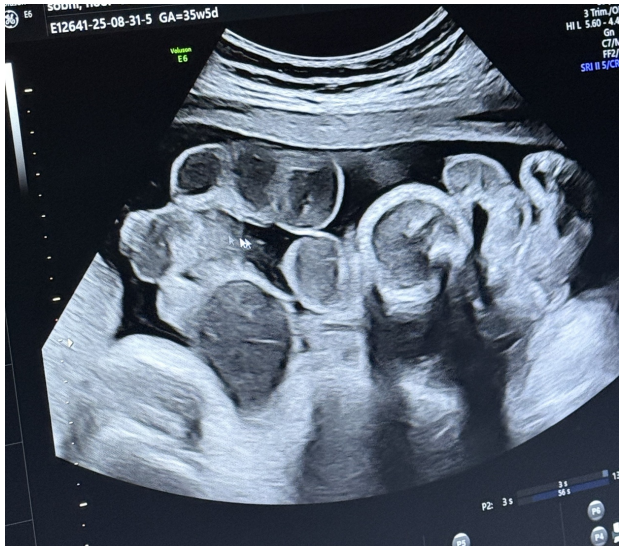

Figure 3. Thickened bowel wall, marked bowel wall thickening.

2.2. Delivery and Initial Neonatal Status

The neonate was delivered at term via elective cesarean section. Apgar scores were 8 at one minute and 9 at five minutes. At birth, the eviscerated bowel loops appeared markedly edematous, congested, and dusky, consistent with severe is-

chemic enteritis (Figure 4). The neonate was transferred to the neonatal intensive care unit, where thermal stabilization was initiated. Initial arterial blood gas analysis revealed metabolic acidosis, reflecting early systemic hypoperfusion.

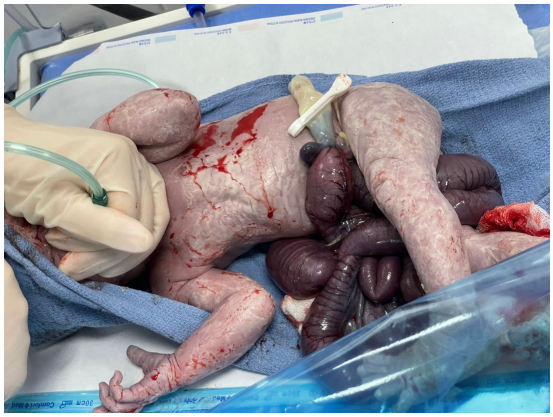

Figure 4. Immediate post-delivery photograph of the neonate showing the abdominal defect and the severely congested and inflamed bowel loops.

2.3. Surgical Intervention and Postoperative Course

Neonate underwent surgical intervention on day one due to hypothermia. Intraoperatively, the small bowel and colon demonstrated diffuse inflammatory changes and ischemic discoloration. No intestinal atresia, perforation, or overt necrosis was identified [5].

Despite the concerning prenatal imaging and gross bowel appearance, intraoperative assessment suggested that the abdominal cavity could accommodate the reduced viscera without excessive tension. The bowel was considered viable, and abdominal wall compliance was judged acceptable. Based on these findings, the surgical team elected to proceed with bowel reduction and primary fascial closure rather than staged reduction [2].

Within 12 hours postoperatively, the infant's condition deteriorated rapidly, requiring escalating ventilatory and hemodynamic support. The clinical picture was dominated by severe systemic inflammatory response syndrome with refractory hypotension [3]. Concurrent signs of abdominal compartment syndrome—including oliguria, increasing ventilatory pressures, and worsening hypotension—were observed [2]. Despite maximal supportive therapy, the neonate developed progressive multi-organ failure and died on day of life three.

3. Discussion

This case illustrates well-established prenatal predictors of complicated gastroschisis. Progressive bowel dilatation exceeding 1.8 cm and increasing bowel wall thickness have consistently been associated with intestinal ischemia, atresia, necrosis, and adverse neonatal outcomes [6] [7]. In the present case, dilatation progressed to more than 2.5 cm, representing extreme intrauterine intestinal compromise [6].

The rapid postoperative deterioration can be explained by the synergistic interaction between reperfusion-induced systemic inflammation and elevated intra-abdominal pressure. Primary closure of severely ischemic bowel results in sudden reperfusion, triggering a massive cytokine release that precipitates systemic inflammatory response syndrome [3] [4]. Simultaneously, primary closure in the setting of marked bowel edema acutely increases intra-abdominal pressure, resulting in abdominal compartment syndrome [2]. The mechanical effects of abdominal compartment syndrome further impair venous return, ventilation, and renal perfusion, compounding hemodynamic instability.

Previous cohort studies and case series report mortality rates of up to 20% in complicated gastroschisis, particularly in cases associated with significant antenatal bowel dilatation and wall thickening [1] [4]. Measurements exceeding 2.5 cm correlate strongly with postoperative septic morbidity and mortality [6] [8]. While primary fascial closure is appropriate for simple gastroschisis, staged reduction using a silo is recommended in high-risk cases to permit gradual decompression and reduce systemic complications [2] [5].

4. Conclusion

Progressive antenatal bowel dilatation and wall thickening are strong predictors of complicated gastroschisis and adverse neonatal outcomes [6]. In this case, an eviscerated bowel diameter exceeding 2.5 cm represented an indicator of extreme intrauterine intestinal injury and profound physiological risk. Such findings should prompt early consideration of staged reduction strategies to reduce the risk of systemic inflammatory response syndrome and abdominal compartment syndrome and to prioritize neonatal physiological stability [2] [4].

Patient Consent

Written informed consent for publication of this case report and images was obtained from the infant's legal guardian.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Bradnock, T.J., Marven, S., Owen, A., Johnson, P., Kurinczuk, J.J., Spark, P., *et al.* (2011) Gastroschisis: One Year Outcomes from National Cohort Study. *BMJ*, **343**, d6749. https://doi.org/10.1136/bmj.d6749

[2] Molik, K.A., Gingalewski, C.A., West, K.W., Rescorla, F.J., Scherer, L.R., Engum, S.A., *et al.* (2001) Gastroschisis: A Plea for Risk Categorization. *Journal of Pediatric Surgery*, **36**, 51-55. https://doi.org/10.1053/jpsu.2001.20004

[3] Carnaghan, H., *et al.* (2014) Predicting Complicated Gastroschisis: Prenatal Ultrasound Markers. *Ultrasound in Obstetrics & Gynecology*, **43**, 406-412.

[4] Emam, D.M. (2024) A Case Report of Midgut Atresia Presenting with Gastroschisis. *International Journal of Health Sciences*, **8**, 1654-1656.

https://doi.org/10.53730/ijhs.v8ns1.15354

[5] Balasundaram, P., Lautz, T.B., Gale, R. and Remedios-Smith, K.G. (2024) Case Report of a Neonate with Complex Gastroschisis: A Multidisciplinary Approach. *Pediatric Reports*, **16**, 779-785. https://doi.org/10.3390/pediatric16030065

[6] Đorđević, I., Slavković, A., Marjanović, Z., Živanović, D. and Petrović, M. (2018) Gastroschisis Associated with Colonic Atresia—CASE Report. *Facta Universitatis*, *Series: Medicine and Biology*, **19**, 81-83. https://doi.org/10.22190/fumb170217017d

[7] Ishii, D., Miyagi, H., Hirasawa, M. and Miyamoto, K. (2020) Congenital Multiple Colonic Atresias with Intestinal Malrotation: A Case Report. *Surgical Case Reports*, **6**, Article No. 45. https://doi.org/10.1186/s40792-020-00822-z

[8] Skarsgard, E.D., *et al.* (2012) Outcomes in Complicated Gastroschisis. *Journal of Pediatric Surgery*, **47**, 959-963.