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Case Report

A Rare Case of Gastroschisis in a Neonatal Jersey Crossbred Calf

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Abstract

Gastroschisis is a rare congenital defect characterized by a full-thickness abdominal wall opening, resulting in evisceration of abdominal organs without a protective membrane. A day-old Jersey calf weighing 29 kg presented with an exposed intestine lateral to the umbilicus, dehydration, and hypothermia. Initial management included fluid resuscitation with warmed Ringer's lactate and maintenance of visceral hydration and body warmth. Premedication was performed using atropine and butorphanol, followed by induction and maintenance of anesthesia with diazepam and ketamine. Surgical correction involved thorough lavage of exposed viscera, careful assessment of tissue viability, and replacement into the abdominal cavity, followed by layered closure of the abdominal wall and skin, with placement of a stent bandage to prevent seroma formation. Postoperative care included intravenous fluids, analgesia, broad-spectrum antibiotics, and gastroprotective therapy. The calf recovered uneventfully, passed normal urine and feces, and tolerated feeding of colostrum and milk replacer. This case highlights that with prompt surgical intervention, appropriate anesthetic management, and diligent perioperative care, successful correction of gastroschisis in neonatal calves is achievable. Awareness of potential congenital associations and accurate differentiation from omphalocele and umbilical hernia is essential for optimal clinical outcomes.

Keywords: Abdominal, Calf, Congenital, Gastroschisis, Surgery.

Introduction

Gastroschisis is a rare congenital malformation of the abdominal wall characterized by a full-thickness defect, typically located right lateral to the umbilicus, allowing the evisceration of abdominal contents without a protective membrane (Rousseaux, 1994; Martin-Alguacil and Avedillo, 2020). Unlike an omphalocele, gastroschisis does not involve the umbilical cord and lacks a covering sac, exposing the intestines directly to the external environment, which can lead to desiccation, inflammation, and functional impairment (Moore and Persaud, 2004). While gastroschisis is well-documented in human neonates, reports in veterinary literature, particularly in cattle, are rare (López Valdéz *et al.,* 2011; Bhat *et al.,* 2020). The precise etiology remains uncertain, though several hypotheses suggest vascular disruption, failure of lateral fold closure, or increased intra-abdominal pressure during fetal development (Vermeij-Keers *et al.,* 1996). Dystocia in heifers has also been associated with congenital abdominal wall defects, including gastroschisis, where herniation severity varies from umbilical hernias to complete eventration of abdominal organs (Rousseaux, 1994). Surgical correction remains the treatment of choice, with options including immediate or staged closure of the abdominal defect, sometimes utilizing a temporary prosthetic bag for gradual visceral repositioning (Marven and Owen, 2008). Given the limited literature on gastroschisis in neonatal calves, this case report describes the diagnosis, surgical intervention, and outcome of a rare presentation of gastroschisis in a bovine neonate.

Fig. 1: One-day-old calf exhibiting intestinal evisceration resulting from gastroschisis.

Fig. 2: Calf on the eleventh day following surgical correction of gastroschisis. PIC NOT PROPRRLY PLACED

Case History and Observations

A one-day-old Jersey crossbred calf from Handwara, Jammu and Kashmir, was presented to the Government Veterinary Hospital with a history of abdominal organ evisceration and umbilical edema. The calf was delivered unassisted, showed no congenital anomalies at birth, and was separated from its dam after receiving colostrum. Within 2–3 hours postnatally, the owner observed an umbilical bulge with externalized viscera and applied a makeshift bandage before seeking veterinary care. On admission, the calf was recumbent, lethargic, and exhibited intermittent muscle fasciculations, with clinical signs of severe dehydration (10–12%), emaciation, and hypothermia (98.5°F). A systemic evaluation revealed sluggish neuro-ophthalmic reflexes, pale pink oral mucous membranes with a capillary refill time of 1–2 seconds, tachycardia (180 bpm) without murmurs or arrhythmias, and an elevated respiratory rate, though difficult to quantify due to fasciculations and vocalization, with no adventitious lung sounds. The calf weighed 29.0 Kg and lacked a suckling reflex. The examination of the ventral abdomen revealed a 3–4 cm defect slightly right of the midline, with exposed small intestine contaminated with straw, mud, and fibrin, appearing red-purple and sticky. The calf vocalized in distress upon palpation, and perineal examination showed a perforated anus devoid of meconium stains. The anal opening was anatomically patent and no evidence of atresia ani or anorectal obstruction was detected on clinical examination. The absence of meconium staining was considered to be due to delayed passage of meconium, which was subsequently confirmed by the passage of normal meconium and feces approximately five hours after surgical correction. Hematological analysis revealed leukocytosis, indicative of an inflammatory response or early-stage infection, while other parameters remained within normal

limits. Based on clinical findings, the case was diagnosed as "gastroschisis" secondary to a ventral abdominal wall defect.

Treatment

Initial assessment revealed dehydration, necessitating fluid resuscitation. A total of 400 ml of warm Ringer's lactate was administered intravenously through the jugular vein over one hour. The exposed viscera were gently covered with warm sterile saline-soaked gauze to prevent desiccation, and warmth was maintained using hot-water bottles wrapped in towels to avoid direct heat exposure. Once the calf was hemodynamically stable, premedication for anesthesia was carried out with atropine sulphate (1.9 ml, IM) and butorphanol tartrate (0.15 ml, IM). Induction was achieved using diazepam (1.2 ml, slow IV) followed by ketamine hydrochloride (0.7 ml, slow IV) after satisfactory muscle relaxation. Anesthesia was maintained with intermittent ketamine doses (0.29 ml IV every 15–20 minutes) throughout the procedure.

A ventral midline celiotomy was performed with the calf in dorsal recumbency. The exteriorized viscera were thoroughly lavaged with warm physiologic saline until all debris was cleared, and the serosal surfaces were evaluated for tissue viability, adhesions, and rupture before replacement. Sterile surgical lubricant was applied to facilitate reduction of the viscera into the abdominal cavity. As initial manipulation was difficult, the abdominal rent was extended by approximately 3 cm cranially and caudally through sharp dissection, which allowed easier repositioning. After complete reduction, the defect was temporarily closed using nonabsorbable suture material in a simple continuous pattern, and a second surgical preparation was performed. Before definitive abdominal closure, the cavity was lavaged with copious warm saline and residual fluid was removed with suction. The abdominal wall was closed in three layers, muscular layers in a simple continuous pattern, subcuticular tissue in a simple continuous pattern to minimize dead space, and skin closure with non-absorbable suture (Polyamide) in a simple continuous pattern. Prior to suturing, the incision site was thoroughly irrigated with a 0.1% iodine solution, and a latex drain was positioned on either side of the wound. A stent bandage was placed over the incision site to provide pressure and reduce seroma formation.

The total anesthetic period lasted approximately three hours, and the calf exhibited mild hypothermia during recovery. Postoperative management included warming air therapy, intravenous isotonic crystalloids at 80 ml/h, and bottle-feeding of calf milk replacer four times daily at 10% of body weight, with daily weight monitoring. The calf passed normal urine, meconium, and feces after 5 hours of surgery. Antibiotic therapy consisted of broad-spectrum coverage using ceftriaxone (600 mg IV once daily), along with anti-inflammatory treatment using meloxicam (6 mg IM once daily). Gastroprotective management included administration of sucralfate slurry (1 g orally, four times daily) and pantoprazole (1 mg/kg IV once daily) to minimize the risk of abomasal ulceration associated with surgical stress and concurrent medication. These therapies were continued for six days, during which the calf showed progressive recovery without postoperative complications. The incision site was examined daily, and the drains were gradually removed over a period of four days. Each day, the wound was cleansed and flushed with a diluted chlorhexidine solution as part of local wound management. Over the next two weeks, progressive healing was observed, with a marked reduction in the wound size and noticeable improvement in the abdominal contour.

Discussion

Congenital body wall defects in calves represent a critical group of embryonic developmental failures where the ventral body wall fails to close completely during early gestation (Koebel *et al.*, 2023). This structural failure typically leads to herniation or eventration, allowing abdominal and sometimes thoracic viscera to develop outside the protective body cavity. These defects range in severity from relatively benign, surgically correctable conditions like simple umbilical hernias to severe, fatal anomalies such as gastroschisis and *Schistosomus reflexus* (Park *et al.*, 2023). These malformations often originate from an early-pregnancy interplay of autosomal recessive genes, infectious diseases, or environmental toxins. Because they frequently result in severe dystocia, stillbirth, or immediate neonatal death, they pose a significant economic and reproductive challenge that necessitates careful genetic management and prompt veterinary intervention (Mir *et al.*, 2024). In human medicine, the standard treatment for gastroschisis involves postnatal abdominal wall closure, either immediately or gradually, often using a temporary prosthetic bag to allow progressive reduction of the exposed intestines (Marven and Owen, 2008). Complications such as intestinal atresia, stenosis, necrosis, or serosal fibrous inflammation ("peel") can significantly

influence postnatal management (Sadler and Feldkamp, 2008). Severe cases may result in abdominal compartment syndrome or require long-term parenteral nutrition, increasing the risk of liver failure. Experimental and clinical observations suggest that gastroschisis may also occur alongside other congenital anomalies. Mastroiacovo *et al.* (2007) reported that among 52 cases analyzed, 26% were associated with anencephaly, hydrocephaly, or limb shortening. Similarly, Alberto *et al.* (2008) observed a consistent association between meroanencephaly and gastroschisis in embryos, likely reflecting the impact of teratogenic agents disrupting normal organogenesis.

Studies in animal models provide insights into the possible embryologic and environmental causes of gastroschisis. Alberto *et al.* (2008) described embryos with gastroschisis exhibiting hepatomegaly and liver extrusion without involvement of other abdominal organs. Microscopic evaluation revealed irregularities in the formation of the abdominal epidermis, although no cellular changes were observed in the ectoderm lining. Moore and Persaud (2004), Brewer and Williams (2004) and Padmanabhan (2006) have suggested that maternal exposure to drugs and environmental chemicals may promote protrusion of viscera into the amniotic cavity, interfering with the normal closure of the lateral body folds. Singh (2003) further demonstrated in a rat model that exposure to carbon monoxide combined with zinc deficiency increased fetal mortality and malformations, including gastroschisis, highlighting the importance of maternal nutrition and toxin-free gestational environments for proper embryonic development.

Veterinary reports of gastroschisis indicate similar anatomical and clinical considerations. A piglet aborted at 90 days of gestation weighing 0.420 kg exhibited a lateral abdominal wall defect just left of the ventral midline with externalization of intestinal loops, a normal umbilical cord, and absence of a surrounding membrane (Martin-Alguacil and Avedillo, 2020). While this defect is often isolated, additional anomalies such as liver malformations or limb deformities may occur (Torres *et al.*, 2015). Although the absence of meconium staining initially raised suspicion of a concurrent anorectal abnormality such as atresia ani, clinical examination confirmed a patent anal opening. The subsequent passage of normal meconium and feces following surgery indicated that gastrointestinal continuity and anorectal patency were preserved. Therefore, this finding was considered transient rather than representing an associated congenital malformation. Careful evaluation for concurrent congenital anomalies remains essential in neonatal calves with gastroschisis, as the presence of additional defects may influence prognosis and therapeutic planning. In the present case, surgical management required careful preoperative stabilization with warmed Ringer's lactate to correct dehydration and shock. Anesthesia was induced using atropine sulphate to reduce salivation and prevent bradycardia, butorphanol to provide analgesia and mild sedation, diazepam for muscle relaxation, and ketamine to induce anesthesia. Postoperative care focused on maintenance of body temperature, analgesia, and gastroprotective therapy, emphasizing the need for meticulous supportive management. Accurate differentiation of gastroschisis from omphalocele or congenital umbilical hernia remains critical, as these conditions differ in chronology, pathogenesis and surgical approach.

Conclusion

Gastroschisis in neonatal calves is a rare congenital defect requiring prompt recognition and careful surgical management. Successful outcomes depend on preoperative stabilization, appropriate anesthesia, meticulous handling of exposed viscera, and layered abdominal closure combined with postoperative supportive care including fluid therapy, analgesia, antibiotics, and gastroprotective treatment. This case demonstrates that with timely intervention and comprehensive perioperative management, even severe congenital abdominal wall defects in neonates can be corrected successfully, minimizing complications and ensuring recovery. Awareness of potential associations with other congenital anomalies and differentiation from omphalocele or umbilical hernia remains essential for accurate diagnosis and effective treatment planning.

Contribution by Authors

All the authors contributed equally to writing the manuscript. The final manuscript was read by all authors and consented to publication.

Conflict of Interests

There is no conflict of interest.

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