e-ISSN: 2807-6257

Open Access Indonesian Journal of Medical Reviews

[OAIJMR]

https://hmpublisher.com/index.php/oaijmr

Early Intervention in Neonatal Gastric Volvulus: A Case Report on Prompt Diagnosis and Surgical Success

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ARTICLE INFO

Keywords:
Early diagnosis
Gastric volvulus
Gastropexy
Neonatal
Pediatric surgery

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/oaijmr.v5i4.750

A B S T R A C T

Neonatal gastric volvulus (NGV) is an uncommon but potentially life-threatening surgical emergency characterized by the abnormal rotation of the stomach around one of its axes. This condition can lead to gastric outlet obstruction, ischemia, necrosis, and perforation if not promptly diagnosed and managed. The clinical presentation is often nonspecific, posing significant diagnostic challenges. Early recognition and immediate surgical intervention are paramount for favorable outcomes. We report the case of a 7-day-old female neonate who presented with recurrent postprandial, non-bilious vomiting and significant orogastric tube residuals since the first few days of life. Antenatal and perinatal histories were largely unremarkable. Initial physical examination revealed a generally stable neonate with mild distress during vomiting episodes, but the abdomen was soft, non-distended, and without palpable masses or signs of peritonitis. Laboratory investigations were within normal neonatal limits. A plain abdominal radiograph was initially inconclusive but suggested some degree of gastric distension. Subsequent upper gastrointestinal (UGI) contrast study revealed features consistent with organoaxial gastric volvulus, demonstrating abnormal positioning of the greater curvature of the stomach and obstruction to contrast passage. Exploratory laparotomy confirmed organoaxial gastric volvulus secondary to marked laxity of the gastrocolic and gastrosplenic ligaments. Detorsion of the stomach and an anterior gastropexy were performed. Postoperatively, the patient had an uneventful recovery, with gradual introduction and tolerance of oral feeds. Follow-up at 6 months showed normal growth and no recurrence of symptoms. Neonatal gastric volvulus remains a diagnostic challenge due to its rarity and nonspecific symptoms. A high index of suspicion, particularly in neonates with persistent, non-bilious vomiting, is crucial. Prompt radiological evaluation, typically with an UGI series, can confirm the diagnosis. Early surgical intervention involving detorsion and gastropexy is the mainstay of treatment and is associated with excellent outcomes, preventing potentially lethal complications. This case underscores the importance of early diagnosis and timely surgical management in achieving successful outcomes in neonatal gastric volvulus.

1. Introduction

Gastric volvulus (GV) in the neonatal period, defined as an abnormal rotation of the stomach of more than 180 degrees around one of its axes, is a rare yet critical condition that demands prompt recognition and surgical intervention to prevent life-threatening complications such as ischemia, necrosis, and perforation. While GV can occur at any age, neonatal gastric volvulus (NGV) presents unique diagnostic and management challenges due to the subtlety and nonspecificity of its early clinical signs, often mimicking more common neonatal conditions like gastroesophageal reflux or sepsis. The true incidence of NGV is difficult to ascertain precisely due to its rarity, with estimates in the pediatric population being low, and neonatal cases representing a small fraction of these. Some studies suggest an incidence of approximately 0.1–0.5 per 1,000 live births for

congenital gastrointestinal anomalies that might predispose to volvulus, but NGV itself is less common than other forms of intestinal volvulus.[1,2]

The stomach is normally anchored by four primary ligaments: the gastrohepatic, gastrophrenic, gastrosplenic, and gastrocolic ligaments. These ligaments, along with the fixed points at the gastroesophageal junction proximally and the pylorus distally, provide stability while allowing for normal gastric motility and distension. Embryologically, the stomach undergoes a complex developmental process involving rotation and fixation. During the fifth week of gestation, the primitive stomach appears as a fusiform dilation of the foregut. It then undergoes a 90-degree clockwise rotation around its longitudinal axis, bringing the original dorsal border (future greater curvature) to the left and the ventral border (future lesser curvature) to the right. Differential growth rates establish the curvatures. Subsequently, a rotation around an anteroposterior axis occurs, causing the caudal (pyloric) end to move rightward and upward, and the cephalic (cardiac) end to move leftward and slightly downward. Failure or incompleteness of these rotational processes or, more commonly, abnormalities in the development and subsequent laxity or absence of the supporting gastric ligaments are believed to be the primary etiological factors in primary NGV. This inherent ligamentous laxity, particularly of the gastrocolic and gastrosplenic ligaments, allows for abnormal gastric mobility and predisposes to volvulus.[3,4]

Gastric volvulus is broadly classified based on the axis of rotation: Organoaxial Volvulus: Rotation occurs along the long axis of the stomach, connecting the cardia and the pylorus. This is the more common type in both children and adults (accounting for approximately two-thirds of cases) and is often associated with diaphragmatic defects such as congenital diaphragmatic hernia or eventration of the diaphragm, or paraesophageal hernias. In this type, the antrum rotates in the opposite direction to the fundus. Strangulation and necrosis are more commonly associated with this type. Mesenteroaxial Volvulus: Rotation occurs around the short axis, which bisects the lesser and greater curvatures, perpendicular to the longitudinal axis. This type is less common and often results from laxity of the gastrosplenic and gastrocolic ligaments. It tends to be primary (idiopathic) and may present with more chronic or intermittent symptoms. Combined or Mixed Volvulus**:** This involves rotation around both axes and is the rarest type.

Furthermore, GV can be classified by etiology: Type 1 (Primary/Idiopathic): Occurs in the absence of any other underlying abnormality, attributed primarily to congenital laxity, elongation, or absence of the gastric ligaments. Approximately one-third of pediatric cases are primary. Type 2 (Secondary/Acquired): Occurs due to an underlying congenital or acquired anatomical defect or condition that predisposes the stomach to twist. Common associated conditions include congenital diaphragmatic hernia, eventration of the diaphragm, paraesophageal hernia, wandering spleen, asplenia or polysplenia syndromes, gastric tumors or cysts, adhesions, pyloric stenosis, or other intestinal malrotations.

The pathophysiology of NGV involves not only mechanical obstruction but also potential vascular compromise. As the stomach twists, it can create a closed-loop obstruction, leading to the accumulation of gas and fluid, gastric distension, and subsequent vomiting. More critically, the torsion can compromise the blood supply to the stomach, which is derived from the celiac axis (left and right gastric arteries, gastroepiploic arteries, and short gastric arteries). While the stomach possesses a rich collateral circulation, significant rotation (typically >180 degrees) can strangulate these vessels, leading to ischemia, edema, mucosal sloughing, full-thickness necrosis, and eventual perforation. If untreated, this can rapidly progress to peritonitis, sepsis, shock, and death, with historical mortality rates for acute, complicated GV being as high as 30-50%.[5,6]

Clinical presentation of NGV is notoriously variable and often nonspecific, making diagnosis challenging. Neonates may present acutely with the sudden onset

of non-bilious vomiting (a key feature, as the obstruction is usually proximal to the ampulla of Vater), epigastric distension, feeding intolerance, and signs of distress or pain (manifesting as inconsolable crying). Hematemesis can occur due to mucosal ischemia or ulceration. In severe cases with strangulation, the neonate may rapidly develop signs of shock, respiratory distress (due to upward displacement of the diaphragm by the distended stomach), and peritonitis. Borchardt's triad, consisting of severe epigastric pain, retching without vomiting, and inability to pass a nasogastric tube, is classic in adults but rarely seen in neonates. A chronic or intermittent form of NGV can also occur, presenting with failure to thrive, recurrent episodes of vomiting, abdominal discomfort, or early satiety, often leading to significant diagnostic delay.[7,8]

Given the potential for rapid deterioration, a high index of suspicion is paramount for early diagnosis. While plain abdominal radiography may show a distended stomach, an abnormal gastric air-fluid level, a "double bubble" sign (if the volvulus creates two separate air pockets), or paucity of distal gas, these findings are often nonspecific. The gold standard for diagnosis is an upper gastrointestinal (UGI) contrast study, which can delineate the abnormal anatomy, showing the twisted stomach, the point of obstruction (often a "beaking" or "corkscrew" appearance of the contrast column), and delayed gastric emptying. Abdominal ultrasound, particularly in skilled hands, may demonstrate the "whirlpool sign" of twisted mesenteric vessels or abnormal gastric positioning. Computed tomography (CT) can also confirm the diagnosis and identify complications, but is less frequently used as a primary diagnostic tool in neonates due to radiation exposure and the often sufficient information provided by UGI studies.

Once NGV is diagnosed or strongly suspected, prompt management is critical. Initial measures include gastric decompression with an orogastric or nasogastric tube (if it can be passed), intravenous fluid resuscitation, and correction of electrolyte imbalances. Broad-spectrum antibiotics may be indicated if ischemia or perforation is suspected. Definitive treatment is surgical. The goals of surgery are to decompress the stomach, assess its viability, reduce the volvulus (derotation), resect any necrotic tissue, and perform a gastropexy to prevent recurrence. Various gastropexy techniques have been described, including anterior gastropexy (fixing the anterior gastric wall to the anterior abdominal wall), fundoplication (though less common as a primary treatment for volvulus alone), or suture fixation of specific gastric ligaments if identifiable and robust enough. Laparoscopic approaches to gastropexy are increasingly utilized in older children and adults and have been reported in infants, offering the potential benefits of minimally invasive surgery. The prognosis for NGV, when diagnosed and treated promptly before the onset of irreversible ischemia, is generally excellent. However, delays in diagnosis can lead to significant morbidity and mortality.[9,10] This case report describes a 7-day-old neonate who presented with recurrent vomiting and was subsequently diagnosed with organoaxial gastric volvulus secondary to ligamentous laxity. The report aims to highlight the clinical presentation, diagnostic workup, surgical management, and successful outcome, thereby emphasizing the critical role of early intervention in this rare neonatal emergency.

2. Case presentation

A 7-day-old female neonate was referred from a peripheral hospital to the pediatric surgical unit at Dr. Moewardi General Hospital, Surakarta, Indonesia, due to persistent, recurrent postprandial vomiting and significant gastric tube residuals since the first 2-3 days of life. The vomiting was described as non-bilious, occurring shortly after feeds, and forceful at times. The infant was born via spontaneous vaginal delivery to a G1P0A0 mother. The gestational age at birth was aterm; birth weight was 2740 grams, and other neonatal characteristics were within normal limits. APGAR scores were reported as 6 at one minute, 8 at five minutes, and 8 at ten minutes, suggesting some initial need for resuscitation or adaptation. The mother

reported an uneventful pregnancy with no known exposure to teratogens or history of maternal illness. There was no family history of significant congenital anomalies.

Postnatally, the baby was initially started on breast milk. However, she began vomiting after feeds of even small volumes, such as 2 ml of breast milk. This led to the placement of an orogastric tube (OGT) for gastric decompression and to assist with feeding management. Despite OGT placement, significant residuals were frequently aspirated, and vomiting persisted. On admission to our unit at 7 days of life, the neonate weighed 2700 grams. Her vital signs were: temperature 37.0°C, heart rate 140 beats/minute, respiratory rate 45 breaths/minute, and oxygen saturation 98% in room air. She appeared comfortable at rest but became irritable with episodes of vomiting. There were no dysmorphic features. Cardiovascular examination revealed normal heart sounds with no murmurs. Respiratory examination showed clear breath sounds bilaterally, although a concurrent radiological finding of right unilateral pneumonia was noted (Table 1).

The abdomen was soft, not distended, and symmetrical. There were no visible peristaltic waves, skin discoloration, or signs of abdominal wall defects. Bowel sounds were present and seemed normoactive. There was no tenderness on palpation, no palpable masses, and no organomegaly. Digital rectal examination was not performed initially as there was no history of delayed meconium passage. An OGT was in situ, draining small amounts of clear to milky fluid. The external genitalia were normal female. Neurological examination was grossly normal for a neonate.

Initial laboratory results showed a hemoglobin (Hb) level of 17.2 g/dL, hematocrit (Hct) of 51%, white blood cell count (WBC) of 7.7 x 10^3/µL (with a normal differential), and platelet count of 190 x 10^3/µL. Serum electrolytes (sodium, potassium, chloride, bicarbonate), blood urea nitrogen, and creatinine were within normal limits. C-reactive protein was mildly elevated at 8 mg/L (normal <5 mg/L), possibly related to the pneumonia or early stress response. Blood gas analysis was unremarkable. A plain abdominal X-ray (babygram) was performed. It revealed findings consistent with right unilateral pneumonia. The cardiac silhouette was normal. The gastric bubble appeared somewhat distended, but its position was difficult to definitively assess as abnormal on the plain film alone. Prominent fecal material was noted in the colon. The OGT was seen projecting over the stomach. Due to the persistent vomiting and suspicion of a proximal obstruction, an Oesophago-Gastro-Duodenal (OMD) contrast study (upper gastrointestinal series) was performed using non-ionic water-soluble contrast (Figure 1).

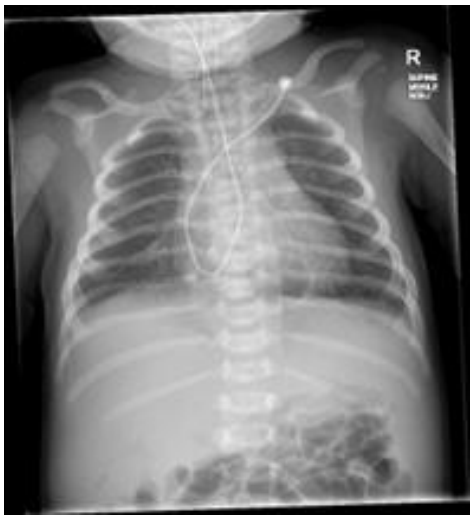

Figure 1. Oesophagus-Maag-Duodenum (OMD) contrast imaging.

The UGI study demonstrated a significantly dilated esophagus and stomach. The contrast revealed an abnormal rotation of the stomach: the greater curvature was positioned superiorly and to the right, while the lesser curvature was inferior and to the left, consistent with an organoaxial gastric volvulus. There was a near-complete obstruction at the distal stomach/pyloric region, with only a trickle of contrast entering the duodenum after a prolonged period. The duodenal loop appeared normally positioned, and there was no evidence of intestinal malrotation seen on the limited views of the duodenum. The findings were diagnostic of gastric volvulus. The initial report from the barium study also raised a suspicion of associated duodenal stenosis, which prompted the surgical decision, although this was later ruled out intraoperatively.

Based on the clinical presentation and conclusive UGI findings of gastric volvulus, a decision was made for urgent surgical intervention. The patient was kept nil per os, an intravenous line was secured, and maintenance intravenous fluids (10% Dextrose with 0.225% NaCl + 20 mEq/L KCl at an appropriate neonatal rate) were administered. The OGT was placed on continuous low-pressure suction for gastric decompression. Intravenous antibiotics (Ampicillin and Gentamicin) were commenced preoperatively, considering the risk of aspiration pneumonia and potential gastric compromise. Informed consent was obtained from the parents after a detailed explanation of the condition, the proposed surgical procedure, potential risks, and benefits. The patient underwent an exploratory laparotomy under general anesthesia in a supine position. A transverse supraumbilical skin incision, approximately 5 cm in length, was made. The abdominal cavity was entered layer by layer. Upon entering the peritoneum, the stomach was immediately identified to be significantly dilated and rotated along its organoaxis in a clockwise direction by approximately 270 degrees. The greater curvature was found superiorly and anteriorly, and the pylorus was displaced. There were no signs of frank ischemia or necrosis of the gastric wall; the color was predominantly pink, with good serosal sheen, although some areas appeared mildly congested. A small amount of clear peritoneal fluid was noted. The stomach was carefully detorted by gentle counter-clockwise rotation. After detorsion, the stomach immediately regained a more normal pink coloration throughout, and its anatomical orientation was restored. Examination of the gastric ligaments revealed significant laxity and attenuation of the gastrocolic and gastrosplenic ligaments. The gastrohepatic and gastrophrenic ligaments appeared relatively intact but perhaps more pliable than typical. The duodenum was carefully inspected, and no evidence of the suspected duodenal stenosis or any other intrinsic or extrinsic duodenal obstruction was found; the initial suspicion from the barium study was likely an artifact of the gastric volvulus itself or contrast hold-up. There was no associated intestinal malrotation; the ligament of Treitz was in its normal position, and the cecum was in the right lower quadrant. No diaphragmatic hernia or eventration was identified (Table 2).

To prevent recurrence, an anterior gastropexy was performed. The anterior wall of the gastric antrum and body was sutured to the anterior abdominal wall (peritoneum and posterior rectus sheath) using four interrupted stitches of a non-absorbable multifilament suture (such as 3-0 Silk or Ethibond). Care was taken to ensure the sutures did not compromise gastric motility or the lumen. The OGT was repositioned to ensure it was draining freely. The abdominal wound was closed in layers: peritoneum with continuous absorbable suture, rectus fascia with interrupted absorbable suture, subcutaneous tissue with absorbable suture, and skin with subcuticular absorbable sutures.

Postoperatively, the patient was managed in the Neonatal Intensive Care Unit (NICU) for close monitoring. She remained hemodynamically stable. The OGT was kept on free drainage and aspirated minimal amounts postoperatively. Intravenous fluids and antibiotics were continued. The surgical wound remained clean, dry, and without signs of infection or

dehiscence. Parenteral nutrition was not required. On the second postoperative day, as bowel sounds returned and OGT drainage was minimal, cautious introduction of oral feeding with expressed breast milk (EBM) was initiated via the OGT, starting with small volumes (5 ml every 3 hours). Feeds were gradually increased as tolerated, monitoring for vomiting or increased OGT residuals. The patient tolerated the graded feeds well. By the fifth postoperative day, she was on full oral feeds (20-30 ml EBM every 3 hours) without vomiting or significant residuals, and the OGT was removed. Her abdominal examination remained benign, with no distension or tenderness. The right unilateral pneumonia was treated concurrently with intravenous antibiotics, and her respiratory status improved gradually. She was discharged home on the tenth postoperative day, tolerating full breastfeeding, gaining weight, and with a well-healed surgical wound. Follow-up in the pediatric surgical outpatient clinic at 1 month, 3 months, and 6 months post-discharge showed continued good progress. She was thriving, meeting developmental milestones, and had no recurrence of vomiting or other gastrointestinal symptoms. At 6 months, her weight was appropriate for her age, and the parents reported no concerns. Further long-term follow-up was planned.

Table 1. Summary of patient's clinical, laboratory, and imaging findings.

Category	Finding	Details & significance
Demographics	Age at Presentation	7 days old
	Gender	Female
	Birth Weight	2740 grams
	Gestational Age	Aterm
	Mode of Delivery	Spontaneous Vaginal Delivery
	APGAR Scores	6 (1 min), 8 (5 min), 8 (10 min)
Anamnesis (History)	Chief Complaint	Recurrent postprandial vomiting, significant orogastric tube (OGT) residuals.
	Onset of Symptoms	Since 2-3 days of life.
	Characteristics of Vomiting	Non-bilious, forceful at times, occurring shortly after feeds (even small volumes, for example 2 ml breast milk).
	Feeding History	Initially breast milk, leading to vomiting; OGT placed for feeding assistance and decompression.
	OGT Findings	Significant residuals frequently aspirated despite OGT placement.
	Maternal & Family History	G1P0A0 mother, uneventful pregnancy, no known teratogen exposure or maternal illness. No family history of significant congenital anomalies.
Physical examination (on Admission)	Weight	2700 grams
	Vital Signs	Temperature: 37.0°C; Heart Rate: 140 beats/minute; Respiratory Rate: 45 breaths/minute; Oxygen Saturation: 98% in room air
	General Appearance	Comfortable at rest, but became irritable with episodes of vomiting. No dysmorphic features.
	Abdominal Examination	Soft, non-distended, symmetrical. No visible peristaltic waves, skin discoloration, or signs of abdominal wall defects. Bowel sounds present and normoactive. No tenderness on palpation, no palpable masses, no organomegaly. OGT in situ, draining small amounts of clear to milky fluid.
	Cardiovascular System	Normal heart sounds, no murmurs.
	Respiratory System	Clear breath sounds bilaterally on auscultation.
	Other Systems	External genitalia: Normal female. Neurological examination: Grossly normal for a neonate.
Laboratory findings	Complete Blood Count (CBC)	Hemoglobin (Hb): 17.2 g/dL; Hematocrit (Hct): 51%; White Blood Cell (WBC): 7.7 x $10^3/\mu L$ (Normal differential); Platelet Count: 190 x $10^3/\mu L$
	Serum Biochemistry	Electrolytes (Sodium, Potassium, Chloride, Bicarbonate), Blood Urea Nitrogen (BUN), Creatinine: All within normal limits.
	Inflammatory Markers	C-reactive Protein (CRP): 8 mg/L (Normal <5 mg/L)
Imaging findings	Plain Abdominal X-ray (Babygram)	- Right unilateral pneumonia identified. - Cardiac silhouette normal. - Gastric bubble appeared somewhat distended; position difficult to definitively assess as abnormal. - Prominent fecal material in the colon. - OGT tip projected over the stomach.
	Upper Gastrointestinal (UGI) Contrast Study / OMD	- Esophagus: Significantly dilated. - Stomach: Significantly dilated, abnormal organoaxial rotation (greater curvature superiorly & right, lesser curvature inferiorly & left). - Obstruction: Near-complete at the distal stomach/pyloric region; only a trickle of contrast entered duodenum after prolonged period. - Duodenum: Loop appeared normally positioned; no evidence of intestinal malrotation.
Clinical diagnosis (Preoperative)	Primary Diagnosis	Neonatal Gastric Volvulus (Organoaxial Type) with Gastric Outlet Obstruction.
	Secondary/Associated Diagnosis	Right Unilateral Pneumonia.

Table 2. Summary of treatment procedures and postoperative follow-up.

Category	Phase / Aspect	Details
Preoperative management	Decision for Surgery	Made urgently following UGI contrast study confirmation of neonatal gastric volvulus.
	Patient Preparation	- Nil per os (NPO). - Intravenous (IV) access secured. - Orogastric tube (OGT) placed on continuous low-pressure suction for gastric decompression.
	Fluid Management	Maintenance IV fluids: 10% Dextrose with 0.225% NaCl + 20mEq/L KCl, administered at neonatal maintenance rate.
	Antibiotic Prophylaxis	IV Ampicillin and IV Gentamicin commenced preoperatively (considering risk of aspiration pneumonia and potential gastric compromise).
	Informed Consent	Obtained from parents after detailed explanation of the diagnosis, proposed surgical procedure (exploratory laparotomy, detorsion, gastropexy), potential risks (infection, bleeding, anesthesia risks, recurrence, need for further surgery), and benefits (relief of obstruction, prevention of necrosis).
Surgical intervention: Exploratory laparotomy	Anesthesia & Positioning	General endotracheal anesthesia; Patient in supine position.
	Surgical Incision	Transverse supraumbilical skin incision, approximately 5 cm in length. Abdominal cavity entered layer by layer.
	Intraoperative Findings	- **Gastric Volvulus:** Stomach significantly dilated, rotated organoaxially (clockwise ~270 degrees). Greater curvature superior/anterior, pylorus displaced. - **Gastric Viability:** No frank ischemia or necrosis; stomach predominantly pink with good serosal sheen, some areas mildly congested. A small amount of clear peritoneal fluid noted. - **Ligamentous Anatomy:** Significant laxity and attenuation of gastrocolic and gastrosplenic ligaments. Gastrohepatic and gastrophrenic ligaments relatively intact but pliable. - **Associated Anomalies:** Duodenum normal (no stenosis). No intestinal malrotation (Ligament of Treitz normally positioned, cecum in RLQ). No diaphragmatic hernia or eventration.
	Operative Procedure Steps	**Detorsion:** Gentle manual counter-clockwise rotation of the stomach to its anatomical position. **Viability Reassessment:** Stomach color improved to uniform pink post-detorsion. **Gastric Decompression:** Ensured via OGT. **Anterior Gastropexy:** Four interrupted sutures of 3-0 non-absorbable multifilament (Silk or Ethibond) used to fix the anterior wall of the gastric antrum and body to the anterior abdominal wall (peritoneum and posterior rectus sheath). Sutures placed to avoid luminal compromise. **OGT Repositioning:** Confirmed for free drainage.
	Wound Closure	Layered closure: Peritoneum (continuous absorbable suture), rectus fascia (interrupted absorbable suture), subcutaneous tissue (absorbable suture), skin (subcuticular absorbable sutures).
Postoperative management	Immediate Postoperative Care	Managed in Neonatal Intensive Care Unit (NICU) for close monitoring of vital signs, respiratory status, and surgical site.
	Hemodynamic & Respiratory Status	Remained hemodynamically stable. Respiratory status gradually improved with ongoing treatment for pneumonia.
	OGT Management	Kept on free drainage; aspirated minimal amounts postoperatively.
	Fluid & Nutrition Management	- Continued IV fluids initially. - **Day 2 Post-op:** Commenced cautious enteral feeding with expressed breast milk (EBM) 5 ml every 3 hours via OGT, following return of bowel sounds and minimal OGT residuals. - Feeds gradually increased as tolerated. - **Day 5 Post-op:** Tolerating full oral feeds (20-30 ml EBM every 3 hours); OGT removed.
	Antibiotic Therapy	Continued IV Ampicillin and Gentamicin for 7 days postoperatively (for pneumonia and surgical prophylaxis).
	Pain Management	Neonatal appropriate analgesia (paracetamol) administered as needed.
	Wound Care	Surgical wound kept clean and dry; monitored daily for signs of infection or dehiscence. No complications noted.
	Abdominal Status	Abdomen remained soft, non-distended, with no tenderness or signs of peritonitis. Normal bowel function restored.
	Hospital Discharge	Day 10 postoperatively. Patient tolerating full breastfeeding, gaining weight, afebrile, with a well-healed surgical wound.
Follow-up	Schedule & Location	Pediatric surgical outpatient clinic visits scheduled at: 1 month, 3 months, and 6 months post-discharge.
	1-Month Follow-up	- **Clinical Status:** Good weight gain, no vomiting, tolerating feeds well. Parents reported satisfaction. - **Wound:** Well healed, no concerns. - **Plan:** Continue routine neonatal care, encourage breastfeeding.
	3-Month Follow-up	- **Clinical Status:** Continued thriving, meeting developmental milestones appropriately. No gastrointestinal symptoms. - **Examination:** Abdomen soft, non-tender, no palpable masses. - **Plan:** Reinforce normal feeding practices.
	6-Month Follow-up	- **Clinical Status:** Excellent overall health, normal growth parameters for age. No recurrence of vomiting or any adverse symptoms. Parents reported no concerns. - **Examination:** Unremarkable. - **Outcome:** Considered surgically successful with no evidence of recurrence.
	Long-term Plan	Longer-term follow-up planned annually through early childhood to monitor for any late complications (though rare) and overall development.

3. Discussion

Neonatal gastric volvulus (NGV) is a rare but critical surgical emergency that, if not diagnosed and treated promptly, can lead to devastating consequences, including gastric ischemia, necrosis, perforation, and mortality. This case report highlights the successful management of a 7-day-old neonate with organoaxial gastric volvulus attributed to laxity of the gastric ligaments, emphasizing the importance of a high index of suspicion, timely diagnostic imaging, and immediate surgical intervention.[11,12]

The etiology of NGV is multifactorial and can be primary (idiopathic) or secondary to other congenital or acquired conditions. Primary NGV, as suspected in our patient due to the intraoperative finding of significantly lax gastrocolic and gastrosplenic ligaments without other associated anomalies, is thought to result from congenital defects in the gastric suspensory apparatus. These ligaments, derived from the embryonic dorsal and ventral mesogastrium, are crucial for maintaining the stomach's normal anatomical position and preventing excessive mobility. Deficiencies in their length, strength, or points of attachment can render the stomach abnormally mobile and prone to twisting. A previous study reported a case of congenital gastric volvulus emphasizing the role of ligamentous laxity. Organoaxial volvulus, as seen in this case, is the more common type and involves rotation around the longitudinal axis extending from the cardia to the pylorus. This type of rotation can lead to obstruction of both the gastric inlet and outlet, as well as compromise of the blood supply running along the curvatures.[13,14]

Secondary NGV is associated with a range of conditions. Congenital diaphragmatic hernia (CDH) is a well-recognized cause, where herniation of the stomach into the thoracic cavity facilitates its abnormal rotation. Eventration of the diaphragm, phrenic nerve palsy leading to diaphragmatic paralysis, and paraesophageal hernias can similarly predispose to NGV. Wandering spleen, a condition characterized by absent or underdeveloped splenic suspensory ligaments (including the gastrosplenic ligament), often coexists with NGV because the stomach and spleen share common embryological origins and ligamentous attachments. Some studies highlighted that patients with gastric volvulus recurrence have a high incidence of wandering spleen, often requiring concurrent gastropexy and splenopexy. Other reported associations include asplenia/polysplenia syndromes, gastric duplication cysts, tumors, Ladd's bands in the context of malrotation (though malrotation primarily causes midgut volvulus, it can occasionally influence gastric position), and post-surgical adhesions. In our case, a thorough intraoperative examination ruled out these common secondary causes. Another study described an unusual presentation of CDH with gastric volvulus and splenic herniation, underscoring the complexity of associated anomalies.[15,16]

The pathophysiology involves a vicious cycle. Gastric rotation leads to obstruction, causing fluid and gas accumulation. This distension can further exacerbate the degree of volvulus. If the rotation exceeds 180 degrees, it can occlude the arterial supply and venous drainage of the stomach. The rich collateral blood supply of the stomach offers some protection, but prolonged or severe torsion can lead to ischemia, infarction, and perforation. The mortality rate for acute GV with complications like gangrene can be as high as 30-50% if intervention is delayed.

The clinical presentation of NGV is often nonspecific, making it a diagnostic challenge, particularly in neonates, where conditions like severe gastroesophageal reflux, pyloric stenosis (though typically later onset), sepsis, or other causes of intestinal obstruction are more common. Non-bilious vomiting is a cardinal symptom, as the obstruction is usually proximal to the ampulla of Vater. Our patient presented with this key feature. Other symptoms include epigastric distension, feeding intolerance, and irritability. Hematemesis may occur if there is mucosal ischemia. Borchardt's triad (severe epigastric pain, retching without emesis, and inability to pass a nasogastric tube) is classic in adults but rarely

observed in neonates. The presence of concurrent pneumonia in our patient, likely aspiration-related due to recurrent vomiting, further complicated the initial clinical picture, a common scenario in such cases.[17,18]

A high index of suspicion is crucial. While plain abdominal radiographs may show suggestive signs like a massively distended stomach, an abnormal position of the gastric bubble, a "single bubble" with paucity of distal gas, or sometimes a "double bubble" (if the volvulus creates two distinct air-filled compartments within the stomach), they are often not diagnostic. In our case, the plain film was not definitively diagnostic but did show gastric distension. The gold standard diagnostic investigation is an upper gastrointestinal (UGI) contrast study. The UGI series can demonstrate the abnormal rotation of the stomach (for instance, inverted greater and lesser curvatures in organoaxial volvulus), the point of obstruction (often appearing as a "beak" or "bird's beak" sign), a "corkscrew" appearance of the twisted esophagus or duodenum, and delayed or absent gastric emptying. This was pivotal in confirming the diagnosis in our patient. Ultrasound can be a useful adjunct, particularly in experienced hands, and may show the "whirlpool sign" of twisted gastric mesentery and vessels or abnormal gastric positioning, but its utility is operator-dependent and may not always be conclusive. CT scans can provide detailed anatomical information, but are generally reserved for equivocal cases or when complications are suspected, given the radiation burden in neonates.[19,20]

Once NGV is diagnosed, the management involves initial resuscitation and urgent surgical intervention. Gastric decompression via an OGT (if it can be passed beyond the twisted cardia) is essential to reduce distension and prevent further aspiration. Intravenous fluids are administered to correct dehydration and electrolyte imbalances. Broad-spectrum antibiotics are typically given, especially if there's suspicion of ischemia or associated pneumonia, as in our patient. Surgical treatment aims to: Decompress the stomach: This is often achieved preoperatively with an OGT and intraoperatively if needed. Assess gastric viability: After derotation, the stomach must be carefully inspected for any signs of ischemia or necrosis. Areas of compromised viability may require resection, though extensive gastric resection in neonates carries high morbidity. Fortunately, in our case, the stomach was viable. Derotate the stomach: Gentle manual reduction of the volvulus is performed. Perform gastropexy: This is the crucial step to prevent recurrence. Various techniques have been described: Anterior gastropexy: The anterior wall of the stomach (usually antrum and body) is sutured to the anterior abdominal wall (peritoneum and posterior rectus sheath). This was the technique employed in our patient and is a commonly performed and effective method. Non-absorbable sutures are typically used to ensure permanent fixation. Fundoplication (such as Nissen or Toupet): While primarily an anti-reflux procedure, it also helps to anchor the fundus and gastroesophageal junction. However, it is generally not considered necessary for NGV unless significant GERD is also present. Stamm or Janeway gastrostomy: Creating a gastrostomy tube site inherently pexies the stomach to the anterior abdominal wall. This can be considered if long-term enteral access is also required. Repair of associated defects: If NGV is secondary to a CDH, eventration, or paraesophageal hernia, these must be repaired. Laparoscopic gastropexy: This minimally invasive approach is gaining popularity and has been successfully used in children, including infants. It involves intracorporeal suturing to achieve anterior gastropexy. Iacona et al. (2022) described laparoscopic-assisted percutaneous anterior gastropexy for acute and chronic GV in infants, highlighting less invasive options.

The choice of gastropexy technique often depends on the surgeon's preference, the patient's condition, and whether the volvulus is primary or secondary. In primary NGV due to ligamentous laxity, anterior gastropexy is a widely accepted and effective method, as demonstrated by the successful outcome in our

case. The use of non-absorbable multifilament sutures aims to create lasting adhesions.

The prognosis for NGV largely depends on the timeliness of diagnosis and intervention and the presence or absence of gastric necrosis. If surgery is performed before irreversible ischemia occurs, the outcomes are generally excellent, with low rates of recurrence after adequate gastropexy. Our patient had an uneventful postoperative course and remained symptom-free at 6-month follow-up, consistent with favorable outcomes reported in the literature for timely managed cases. Delays leading to gastric gangrene significantly increase morbidity and mortality, potentially requiring extensive gastric resection, which can result in long-term nutritional challenges and dumping syndrome. Recurrence after gastropexy is rare but can occur if the fixation is inadequate or if there are strong predisposing factors not fully addressed.

The presentation of our case with non-bilious vomiting, diagnosis via UGI series, finding of ligamentous laxity, and successful treatment with anterior gastropexy aligns well with many reports in the literature. The initial suspicion of duodenal stenosis on the UGI study, which was later ruled out intraoperatively, highlights how severe gastric distension and abnormal positioning in NGV can sometimes distort the appearance of adjacent structures on imaging, leading to potential diagnostic pitfalls. The concurrent pneumonia is a common comorbidity, often secondary to aspiration from persistent vomiting, and underscores the need for comprehensive neonatal care.

The significant laxity of both gastrocolic and gastrosplenic ligaments observed intraoperatively strongly points to a primary defect in gastric fixation as the etiology in this neonate. The successful outcome in our patient, with prompt feeding advancement and no recurrence, reinforces the efficacy of early surgical intervention with detorsion and anterior gastropexy for primary NGV.

4. Conclusion

This case of neonatal gastric volvulus underscores the necessity of considering NGV in neonates with non-bilious vomiting, even with confounding factors like pneumonia. It confirms that primary ligamentous laxity is a key etiology and that open detorsion with anterior gastropexy remains a highly effective and safe treatment, leading to excellent short-term outcomes when gastric viability is preserved. Early surgical intervention, consisting of manual detorsion of the volvulus and subsequent anterior gastropexy, proved to be a definitive and successful treatment, leading to an uneventful recovery and no recurrence of symptoms at follow-up. The favorable outcome in this patient reiterates that the prognosis for NGV is excellent when the condition is addressed before the onset of vascular compromise and gastric necrosis. Pediatricians, neonatologists, and pediatric surgeons must maintain awareness of NGV to facilitate early referral and intervention.

5. References

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