



Subacute Infantile Hypoxic-Ischaemic Colitis Secondary to Congenital Absence of the Vascular Arcade at Griffith's Point: A Multidisciplinary Case Report

Rakesh Mamilal H Patel

Department of Mathematics, Government Science College, Sector - 15, Gandhinagar, Gujarat State, India.

DOI: <https://doi.org/10.68050/JAMS.2026.835>

Received: 14 October 2026; Accepted: 20 October 2026; Published: 27 October 2026

ABSTRACT

Background: Subacute infantile hypoxic-ischaemic colitis is an uncommon form of intestinal injury that differs from classical necrotising enterocolitis.

Case Presentation: A term infant weighing 2,100 g developed persistent hematochezia and transfusion-dependent anaemia following severe birth asphyxia complicated by hypoxic-ischaemic encephalopathy, acute kidney injury, and patent ductus arteriosus. Despite broad-spectrum antimicrobial therapy and successful enteral feeding, gastrointestinal bleeding persisted. Diagnostic laparoscopy followed by laparotomy demonstrated localized hypoxic-ischaemic colitis extending from the left transverse colon to the sigmoid colon associated with congenital absence of the marginal vascular arcade at Griffith's point. Left colectomy and primary colocolic anastomosis were successfully performed.

Results: Histopathological examination confirmed transmural hypoxic-ischaemic colitis. Enteral feeding resumed on postoperative day three. At one-year follow-up the infant remained asymptomatic with normal growth and development.

Conclusion: Congenital absence of collateral circulation at Griffith's point may predispose term infants to localized colonic ischaemia following severe perinatal hypoxia. Early multidisciplinary evaluation and timely surgical intervention can achieve excellent outcomes.

Keywords: Hypoxic-ischaemic colitis, Griffith's point, Watershed area, Congenital vascular anomaly, Infantile colectomy, Multidisciplinary care.

Running Title: Infantile Hypoxic-Ischaemic Colitis at Griffith's Point

1. INTRODUCTION

Hypoxic-ischaemic injury of the gastrointestinal tract is most commonly recognized as **necrotising enterocolitis (NEC)**, a devastating condition that predominantly affects premature infants and is associated with intestinal immaturity, dysregulated inflammation, and impaired mesenteric perfusion [1]. However, ischaemic colonic injury may also occur in term infants, particularly following severe perinatal hypoxia, congenital cardiovascular abnormalities, or regional vascular insufficiency [2].

The colon contains several anatomically vulnerable watershed regions where perfusion depends on collateral circulation between major mesenteric vessels. **Griffith's point**, located at the splenic flexure, represents the junction between the territories supplied by the middle colic artery of the superior mesenteric artery and the left colic artery of the inferior mesenteric artery [3]. Although the marginal artery of Drummond usually provides an adequate collateral network, anatomical variations including hypoplasia, discontinuity, or congenital absence may significantly compromise blood flow during systemic low-flow states [4].



Perinatal asphyxia causes redistribution of cardiac output away from the gastrointestinal tract in favour of cerebral and myocardial perfusion. Prolonged mesenteric vasoconstriction may produce intestinal hypoperfusion, mucosal injury, and eventual ischaemic necrosis [2,5]. While diffuse intestinal involvement is well recognised in NEC, isolated left-sided colonic ischaemia due to congenital deficiency of the vascular arcade at Griffith's point has rarely been described in infancy [6].

The clinical presentation frequently overlaps with infectious colitis, food protein-induced enterocolitis, allergic proctocolitis, or resolving NEC, often delaying diagnosis [1,5]. Furthermore, endoscopic evaluation of a severely compromised infant colon carries a substantial risk of perforation and may not be feasible in critically ill neonates [5]. We report a unique case of subacute infantile hypoxic-ischaemic colitis secondary to congenital absence of the vascular arcade at Griffith's point in a term infant who survived severe birth asphyxia. The case highlights the importance of multidisciplinary decision-making, recognition of vascular anatomical variants, and timely surgical intervention in achieving an excellent long-term outcome [6,7].

2. CASE PRESENTATION

2.1 Patient Demographics and Resuscitation at Birth

A term infant was born via an emergent Category 1 Caesarean section due to acute fetal distress. The birth weight was **2,100 grams**, indicative of intrauterine growth restriction (IUGR). At delivery, the infant required extensive resuscitation in the delivery room due to profound birth asphyxia. Initial cord blood gas evaluation revealed a severe metabolic acidosis driven by a highly elevated serum lactate level.

The infant was admitted to the Neonatal Intensive Care Unit (NICU) with a clinical diagnosis of **Hypoxic-Ischaemic Encephalopathy (HIE)**, concurrent **Acute Kidney Injury (AKI)**, and a hemodynamically significant **Patent Ductus Arteriosus (PDA)**. Under close multidisciplinary monitoring, the HIE and AKI resolved with supportive care, and the PDA was managed conservatively. Crucially, during this critical stabilizing period, the patient exhibited no clinical or radiographic signs of classic necrotising enterocolitis (NEC).

2.2 Progress and Clinical Course

Following stabilization, enteral feeds were gradually introduced. The infant tolerated breast milk well, successfully reaching full enteral volumes. However, the clinical course was complicated by the onset of persistent bloody stools (hematochezia) and chronic, recurrent anaemia. The anaemia was severe enough to require the transfusion of two separate units of packed red blood cells (pRBC) to maintain hemodynamic stability.

In response to the bloody stools, the clinical team initiated an empiric course of triple-antibiotic therapy comprising **Ampicillin, Gentamicin, and Metronidazole**. Despite prolonged antimicrobial coverage and full breast milk tolerance, the hematochezia persisted, prompting a multidisciplinary team (MDT) consultation including pediatric neonatologists, gastroenterologists, and surgeons. A diagnostic colonoscopy and biopsies was proposed but ultimately rejected by the MDT, as it was deemed a highly unsafe and unacceptably risky procedure given the fragility and suspected ischaemic changes of the infant loop architectures.

2.3 Surgical Exploration and Findings

Based on the MDT consensus, the patient was taken to the operating theatre for a diagnostic laparoscopy. Initial laparoscopic visualization demonstrated localized, chronic ischaemic changes isolated to the left side of the colon, prompting immediate conversion to an open exploratory laparotomy.

[Vascular Arcade Interrogation]

SMA (Middle Colic) -----X [Griffith's Point] X----- IMA (Left Colic)

(Primitive / Absent)

|
[Ischaemic Watershed Zone]
(Left Transverse Colon ---> Sigmoid Colon)

Surgical exploration revealed severe, subacute hypoxic-ischaemic colitis accurately demarcated from the **left transverse colon to the sigmoid colon**. Detailed dissection and vascular interrogation of the mesentery revealed that the marginal vascular arcade at Griffith's point was remarkably primitive and almost non-existent. This congenital anatomical absence resulted in a total lack of a functional collateral circulation anastomosis across the colonic watershed.

Because the rest of the bowel was clean and well-prepared, the surgeons performed a targeted **left colectomy**, resecting the ischaemic portion from the left transverse colon down to the sigmoid colon. Intestinal continuity was successfully restored via a primary transverse colon to sigmoid colon end-to-end anastomosis.

2.4 Histopathological Evaluation

Gross and microscopic evaluation of the excised left colonic specimen confirmed the clinical suspicion. Histological sectioning demonstrated definitive patterns of classic **hypoxic-ischaemic colitis** associated with patchy, transmural necrosis of the left colonic wall, marked mucosal ulceration, and submucosal granulation, consistent with chronic low-flow vascular deprivation.

3. OUTCOMES AND FOLLOW-UP

The patient's postoperative course in the pediatric intensive care unit was entirely uneventful. Due to the pristine intraoperative preparation of the colonic borders and a secure primary anastomosis, enteral feeds were safely reinitiated on the **third postoperative day**. The infant tolerated the feeds without any signs of distension, emesis, or recurring bloody stools, and was discharged home in stable condition.

CLINICAL TIMELINE

Birth: Term, 2100g, Cat 1 CS -> High Lactate -> NICU Resuscitation
(Diagnoses: HIE, AKI, PDA; No signs of NEC)

|
Weeks 1–4: Gradual breast milk tolerance achieved

|
Weeks 5–8: Persistent bloody stools & recurrent anaemia
(Required 2 units pRBC; Unresponsive to Triple Antibiotics)

|
MDT Choice: Colonoscopy rejected as unsafe -> Diagnostic Laparoscopy

|
Surgery: Conversion to Laparotomy -> Left Colectomy with End-to-End
Transverse-Sigmoid Anastomosis (Congenital absence of arcade)

|
Post-Op Day 3: Reintroduction of enteral feeds; uneventful recovery

|
1-Year Follow-Up: Completely asymptomatic; thriving normally

At the **1-year clinical follow-up**, the patient was thoroughly evaluated in the outpatient pediatric clinic. The infant is completely asymptomatic, demonstrates perfect bowel regulation, has no further episodes of hematochezia, and is thriving well with physical growth and developmental milestones plotting consistently along normal percentiles for age.



4. DISCUSSION

This case highlights a rare cause of localized infantile colonic ischaemia resulting from the combined effects of severe perinatal hypoxia and a congenital vascular deficiency at Griffith's point. Although NEC remains the most common form of neonatal intestinal ischaemic injury, its pathogenesis and distribution differ significantly from the lesion observed in the present patient [1]. Unlike classic NEC, which typically affects premature neonates and frequently involves the terminal ileum and proximal colon, this infant developed isolated left-sided colonic disease despite being born at term and demonstrating satisfactory tolerance of enteral feeding during recovery.

The clinical circumstances strongly suggest that perinatal hypoxia played a central role in triggering the ischaemic insult. The infant experienced profound birth asphyxia complicated by hypoxic-ischaemic encephalopathy, acute kidney injury, patent ductus arteriosus, and severe lactic acidosis. During systemic hypoperfusion, compensatory mesenteric vasoconstriction preserves blood flow to vital organs at the expense of the gastrointestinal tract [2,5]. In most infants, collateral circulation from the marginal artery limits tissue injury; however, the operative finding of a primitive or virtually absent arterial arcade at Griffith's point created a uniquely vulnerable watershed zone that lacked adequate collateral perfusion [3,4].

Anatomical variations affecting the marginal artery of Drummond are well described and have been implicated in watershed ischaemia of the colon [3,4]. Similar vascular vulnerability has been reported in congenital gastrointestinal disorders involving arterial insufficiency, including certain forms of intestinal atresia and other developmental abnormalities thought to arise from intrauterine vascular accidents [6]. The present case extends this concept by demonstrating how a congenital deficiency of collateral mesenteric circulation may predispose to localized postnatal ischaemic injury following a major hypoxic event.

Another important lesson from this case concerns diagnosis. Persistent hematochezia and transfusion-dependent anaemia despite full enteral feeding and broad-spectrum antibiotic therapy argued against simple infectious colitis or resolving NEC. The persistence of symptoms suggested an ongoing structural or vascular pathology [5]. Colonoscopic assessment was considered but rejected because of the risk of perforation in a potentially necrotic bowel segment. This decision proved appropriate, as direct laparoscopic evaluation safely identified the underlying pathological process and guided definitive treatment.

The operative findings demonstrated sharply demarcated disease extending from the left transverse colon to the sigmoid colon. Histopathology confirmed transmural ischaemic injury with necrosis, mucosal ulceration, and reparative changes consistent with chronic hypoxic-ischaemic colitis. Importantly, the proximal and distal bowel segments remained healthy, allowing a limited segmental colectomy with primary anastomosis rather than a more extensive resection. Early return of enteral feeding and sustained recovery support the effectiveness of this tailored surgical approach.

Recent reports have highlighted the importance of recognising regional vascular vulnerability and mesenteric watershed anatomy in neonatal intestinal disease [6-8]. Awareness of mesenteric anatomical variations may help clinicians identify infants at risk for atypical ischaemic presentations when persistent gastrointestinal bleeding occurs despite apparently successful neonatal recovery. Early multidisciplinary collaboration between neonatologists, radiologists, gastroenterologists, pathologists, and paediatric surgeons remains essential for timely diagnosis and optimal management.

5. CONCLUSIONS

1. Isolated subacute infantile hypoxic-ischaemic colitis can manifest in full-term infants who suffer significant perinatal asphyxia, even when classic signs of NEC are absent.
2. Congenital absence or underdevelopment of the mesenteric vascular arcade at Griffith's point eliminates collateral compensation, making the left colon highly susceptible to localized tissue necrosis during low-flow states.

3. In infants presenting with persistent hematochezia and transfusion-dependent anaemia unresponsive to medical therapy, diagnostic colonoscopy should be avoided if ischaemia is suspected. Early surgical exploration via diagnostic laparoscopy provides a safer path to direct anatomical evaluation and definitive management.

Learning Points

1. Hypoxic-ischaemic colitis may occur in term infants even in the absence of classical necrotising enterocolitis.
2. Griffith's point is a vascular watershed zone that becomes highly vulnerable when collateral circulation is congenitally deficient.
3. Persistent hematochezia and transfusion-dependent anaemia despite appropriate medical therapy should raise suspicion of underlying structural or vascular pathology.
4. Diagnostic laparoscopy may provide a safer alternative to colonoscopy when bowel ischaemia is suspected.
5. Early multidisciplinary evaluation allows timely definitive treatment and excellent long-term outcomes.

What This Case Adds to Existing Literature

- Describes a rare case of subacute infantile hypoxic-ischaemic colitis associated with congenital absence of the vascular arcade at Griffith's point.
- Demonstrates the interaction between severe perinatal hypoxia and developmental mesenteric vascular deficiency.
- Highlights the diagnostic value of surgical exploration when endoscopic assessment is unsafe.
- Supports limited segmental colectomy with primary anastomosis as an effective treatment in selected infants with localized disease.

REFERENCES

1. Neu J, Walker WA. Necrotizing enterocolitis. *N Engl J Med*. 2011;364(3):255-264.
2. Brandt LJ, Feuerstadt P, Longstreth GF, et al. ACG Clinical Guideline: Colon ischemia. *Am J Gastroenterol*. 2015;110:18-44.
3. Griffiths JD. Surgical anatomy of the blood supply of the distal colon. *Ann R Coll Surg Engl*. 1956;19(4):241-256.
4. Longo WE, Ballantyne GH, Gusberg RJ. Ischemic colitis: patterns and prognosis. *Dis Colon Rectum*. 1992;35(8):726-730.
5. Patel RV, Trambadia RA, Anthony FM, Chhaniara RA, Govani DR, Patel RR, Correia RC. Congenital neonatal colonic atresias arising in watershed areas of the colonic blood supply: a case series. *J Indian Med Assoc*. 2024;122(2):55-60.
6. Khatri LK, More AB, Govani PS, Zapparackaite I, More BB, Singh SJ, Swamy KB, Mehta AR, Midha PK, Patel RV. Fulminating enterocolitis in term neonates with major cardiac defects: a review of hypoxic mucosal shedding and postoperative risk. *WebLog J Cardiol*. 2026.
7. Zapparackaite I, Govani ND, Singh H, Singh SJ, Mehta AR, Midha PK, Patel R, Patel RV. Applied clinical anatomy of gut vascular supply: a narrative review of segmental perfusion, watershed vulnerability, and surgical implications. *WebLog J Anat*. 2026.
8. Govani DR, Swamy KB, Mehta AR, Midha PK, Govani ND, Panchasara NG, Patel RR, Patel RV. Hypoxic-ischaemic colitis at Griffith's point in a term infant with hypoxic-ischaemic encephalopathy

and acute kidney injury requiring emergency subtotal colectomy. WebLog J Cerebrovasc Dis Stroke. 2026.

Figure Captions for Manuscript Submission

Figure 1. Serial Plain Abdominal Radiographs.

A–C. Chronological anteroposterior supine radiographs demonstrating asymmetric, moderate distension of bowel loops with a progressive diminishing of gas within the left colonic segment and a localized ground-glass appearance, indicating evolving mural thickening, edema, and fluid accumulation without evidence of pneumatosis intestinalis or free intraperitoneal air.

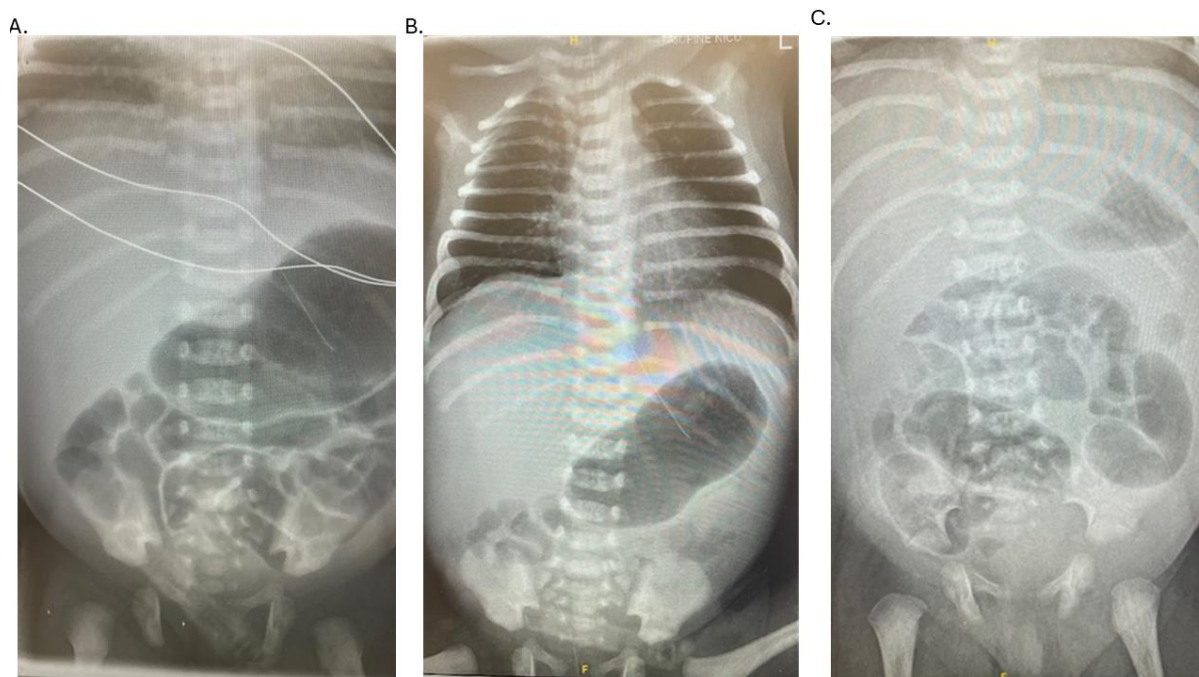


Figure 2. High-Resolution Grey-Scale and Colour Doppler Ultrasonography.

Serial transabdominal sonograms of the left flank demonstrating pronounced circumfused thickening, stratification anomalies, and secondary structural inflammatory changes localized to the left colonic wall. Parallel colour Doppler interrogation reveals significant, confluent transmural hyperaemia ("hyperaemic gut signature") meticulously centred over the anatomical watershed territory of Griffith's point and the adjacent left colonic segments, confirming tissue viability and a severe, subacute reactive hypoperfusion injury.

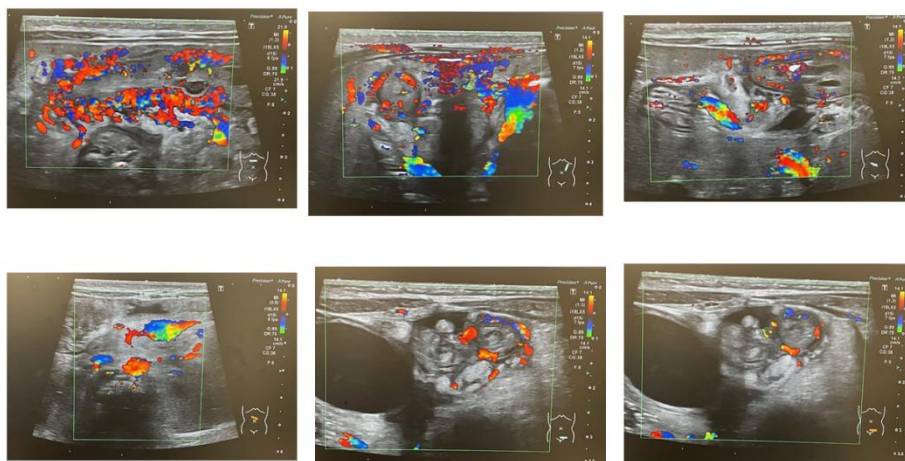




Table 1. Key Differential Diagnoses in Persistent Infantile Hematochezia

Diagnosis	Key Features	Reasons Excluded
NEC	Prematurity, pneumatosis	Absent
Infectious colitis	Positive cultures	Negative response to treatment
Food protein-induced colitis	Feeding related	Persistent severe anaemia
Vascular/ischaemic colitis	Localized disease	Confirmed histologically

Submission Transparency & Sign-off:

- **Patient Consent:** Signed institutional informed consent was obtained from the parents for the academic publication of this anonymized clinical course and surgical summary.
- **Conflict of Interest:** The authors declare no competing financial or institutional conflicts of interest.