



Hypoxic-Ischemic Colitis at Griffiths' Point in a Term Infant with Hypoxic-Ischemic Encephalopathy and Acute Kidney Injury Requiring Emergency Subtotal Colectomy

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Abstract

A term male neonate with moderate Hypoxic-Ischaemic Encephalopathy (HIE) developed persistent rectal bleeding and rising inflammatory markers during therapeutic hypothermia. Despite conservative management, he deteriorated with signs of colonic ischaemia. Imaging suggested segmental hypoperfusion centred at Griffiths' point at the splenic flexure, a recognised colonic watershed zone. Emergency laparotomy revealed extensive non-viable colon requiring subtotal colectomy with end ileostomy. Histopathology confirmed transmural ischaemic necrosis. This case highlights the vulnerability of neonatal colonic watershed regions to systemic hypoperfusion and underscores the need for early recognition of gastrointestinal complications in infants with HIE.

Keywords: Hypoxic-Ischaemic Colitis; Griffiths' Point; Splenic Flexure Ischaemia; Neonatal Watershed Injury; Hypoxic-Ischaemic Encephalopathy (HIE); Acute Kidney Injury (AKI); Therapeutic Hypothermia; Rectal Bleeding in Neonates; Subtotal Colectomy; Neonatal Gastrointestinal Ischaemia; Transmural Colonic Necrosis; Mesenteric Hypoperfusion

Introduction

Hypoxic-ischaemic encephalopathy is a multisystem condition in which gastrointestinal hypoperfusion is common but often under-recognised [1]. The colon contains two classical watershed zones - Sudeck's point and Griffiths' point - where collateral circulation between mesenteric territories is limited [2]. In adults, ischaemic colitis frequently localises to these regions [3]; however, neonatal cases centred on Griffiths' point are rarely reported [4]. Persistent rectal bleeding in infants with HIE warrants careful evaluation for ischaemic injury, particularly when accompanied by systemic inflammatory responses or multi-organ dysfunction [5].

We hope that this report will provide valuable insights for clinicians involved in neonatal, surgical, and intensive care, and will support earlier recognition of gastrointestinal ischaemia in infants with multisystem hypoxic injury.

Case Presentation

A term male infant (birthweight 3.4 kg) was delivered via emergency caesarean section for prolonged fetal bradycardia. Apgar scores were 2, 4, and 6 at 1, 5, and 10 minutes. Cord pH was 6.92 with a base deficit of -18 mmol/L. He met criteria for moderate HIE and commenced therapeutic hypothermia within 2 hours of birth.

By day 2 of life, he developed persistent fresh rectal bleeding, abdominal distension, and increased gastric aspirates. He remained hemodynamically borderline, requiring fluid boluses. Urine output declined to <0.5 mL/kg/h, and serum creatinine rose to 140 μ mol/L, consistent with Acute Kidney Injury (AKI).

Investigations

Blood tests showed CRP peaked at 78 mg/L, Neutrophilia with left shift, worsening metabolic

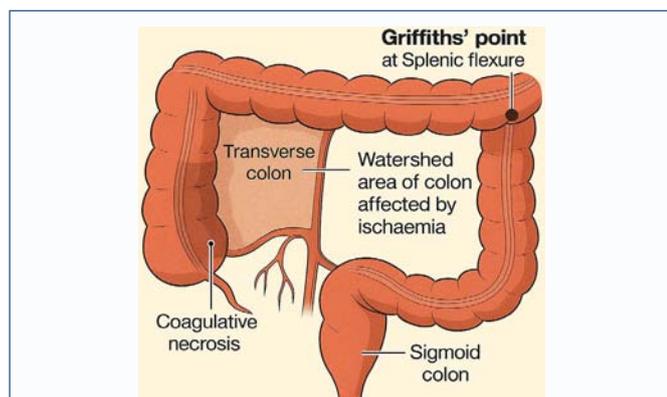


Figure 1: Anatomical schematic of the neonatal colon highlighting Griffiths' point at the splenic flexure (black dot), a classical watershed zone between the Superior Mesenteric Artery (SMA) and Inferior Mesenteric Artery (IMA) territories. The shaded region from mid-transverse colon to descending colon represents the watershed area affected by ischaemia in this case. The marginal artery of Drummond is shown supplying the colon, with limited collateral flow at Griffiths' point. This vascular vulnerability contributed to segmental hypoxic-ischaemic colitis requiring subtotal colectomy.

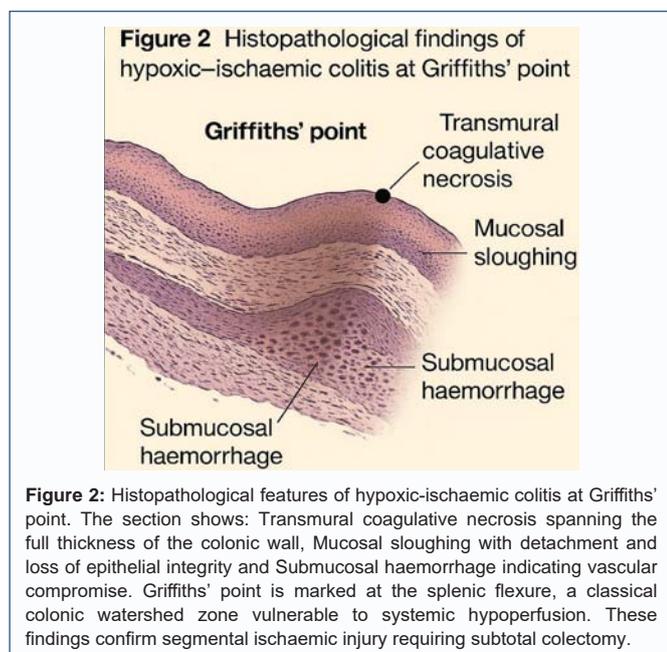


Figure 2: Histopathological features of hypoxic-ischaemic colitis at Griffiths' point. The section shows: Transmural coagulative necrosis spanning the full thickness of the colonic wall, Mucosal sloughing with detachment and loss of epithelial integrity and Submucosal haemorrhage indicating vascular compromise. Griffiths' point is marked at the splenic flexure, a classical colonic watershed zone vulnerable to systemic hypoperfusion. These findings confirm segmental ischaemic injury requiring subtotal colectomy.

acidosis, Coagulation: Mild prolongation of PT and APTT, insufficient to explain bleeding. Blood and stool cultures were negative.

Abdominal radiography demonstrated progressive dilation of the transverse and descending colon, no pneumatosis or free air. Ultrasound with Doppler revealed markedly reduced perfusion centred at the splenic flexure, no portal venous gas.

The findings suggested segmental colitis centred at Griffiths' point, consistent with hypoxic-ischaemic injury.

Clinical Deterioration and Surgical Intervention

Over the next 24 hours, rectal bleeding intensified and abdominal tenderness increased. Repeat imaging showed worsening colonic dilation. By day 3, the infant developed signs of peritonism and haemodynamic instability.

Emergency laparotomy revealed a sharply demarcated,

non-viable colonic segment from the distal transverse colon across the splenic flexure into the proximal descending colon, patchy serosal hemorrhage in remaining colon, no small-bowel involvement and no perforation, but high risk of progression.

A subtotal colectomy with end ileostomy was performed.

Histopathology

Microscopy demonstrated a transmural coagulative necrosis, mucosal sloughing, submucosal hemorrhage, thrombotic microvascular changes. Findings were consistent with hypoxic-ischaemic colitis.

Treatment

Postoperative management included ventilatory support, parenteral nutrition, broad-spectrum antibiotics, careful fluid and electrolyte management for AKI, ongoing neuroprotective care for HIE and serial abdominal examinations.

Outcome and Follow-Up

Rectal bleeding resolved immediately after surgery. Inflammatory markers normalised over the following week. Renal function improved, with creatinine returning to baseline by day 14. Enteral feeds were reintroduced gradually enterally when the ileostomy started functioning fully and were well tolerated.

At 3-month follow-up, the infant demonstrated appropriate growth and stable ileostomy function. Plans for future stoma reversal were under discussion. Neurodevelopmental follow-up for HIE was ongoing.

Discussion

We believe this case is of particular interest because:

- It highlights an under-recognised gastrointestinal complication of HIE.
- It emphasises the anatomical vulnerability of Griffiths' point in neonates.
- It demonstrates the importance of early recognition of rectal bleeding in infants undergoing therapeutic hypothermia.
- It contributes to the limited literature on neonatal colonic watershed ischaemia and its surgical implications.

This case illustrates the potential severity of hypoxic-ischaemic colitis in neonates with systemic hypoperfusion [1]. The localisation of injury at Griffiths' point reflects the anatomical vulnerability of this watershed region, where collateral flow between the superior and inferior mesenteric arteries may be tenuous [2]. In neonates with HIE, blood flow redistribution prioritises vital organs, leaving the gastrointestinal tract susceptible to ischaemia [3].

Unlike Necrotising Enterocolitis (NEC), hypoxic-ischaemic colitis may present early, often within the first 48 hours of life, and without pneumatosis or portal venous gas [4]. The coexistence of AKI in this infant supports a global hypoxic insult affecting multiple organs [5].

Progression to transmural necrosis requiring subtotal colectomy is rare but underscores the need for early surgical involvement when deterioration occurs [6]. This case adds to the limited neonatal literature describing colonic ischaemia centred on Griffiths' point and highlights the importance of recognising gastrointestinal

manifestations in infants undergoing therapeutic hypothermia [7, 8].

This case describes a rare and clinically significant presentation of segmental neonatal colonic ischaemia localised to Griffiths' point, a watershed region seldom reported as the primary site of injury in term infants. The infant's course was further complicated by Hypoxic-Ischaemic Encephalopathy (HIE) and acute kidney injury, illustrating the multisystem nature of perinatal hypoxic insults. Despite initial conservative management, the infant progressed to transmural necrosis requiring emergency subtotal colectomy, with histopathology confirming hypoxic-ischaemic colitis.

Learning Points

- Griffiths' point at the splenic flexure is a key colonic watershed zone vulnerable to ischaemia in neonates with systemic hypoperfusion.
- Hypoxic-ischaemic colitis can present early in infants with HIE and may progress rapidly.
- Persistent rectal bleeding with rising inflammatory markers should prompt evaluation for ischaemic colitis, even in the absence of classical NEC features.
- Emergency subtotal colectomy may be lifesaving when conservative management fails or necrosis is suspected.
- Multi-organ involvement, such as AKI, supports a systemic hypoxic-ischaemic mechanism.

Conclusion

This case highlights the critical vulnerability of colonic watershed zones - particularly Griffiths' point - in neonates with systemic hypoperfusion. In the context of hypoxic-ischaemic encephalopathy and acute kidney injury, early gastrointestinal symptoms such as rectal bleeding may signal evolving ischaemic colitis. Recognition

of this anatomical and pathophysiological pattern is essential, as progression to transmural necrosis may necessitate emergency surgical intervention. Awareness of watershed anatomy and its clinical implications can guide timely diagnosis and management, potentially improving outcomes in affected neonates.

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