



Fulminating Enterocolitis in Term Neonates with Major Cardiac Defects: A Review of Hypoxic Mucosal Shedding and Postoperative Risk

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Abstract

Objectives: To review the pathophysiology and management of fulminating Enterocolitis (EC) in term neonates with major cardiac defects.

Background: Unlike classic NEC in preterm infants, cardiac EC is a "vascular catastrophe" driven by mesenteric low-flow states and anatomical vulnerabilities.

Design: Narrative review of published literature, clinical guidelines, and surgical series across pediatric cardiology, neonatal and pediatric surgery, and neonatal/perinatal medicine.

Data Sources: MEDLINE, Embase, Cochrane Library, and major pediatric surgical and cardiology and cardiothoracic surgery society guidelines.

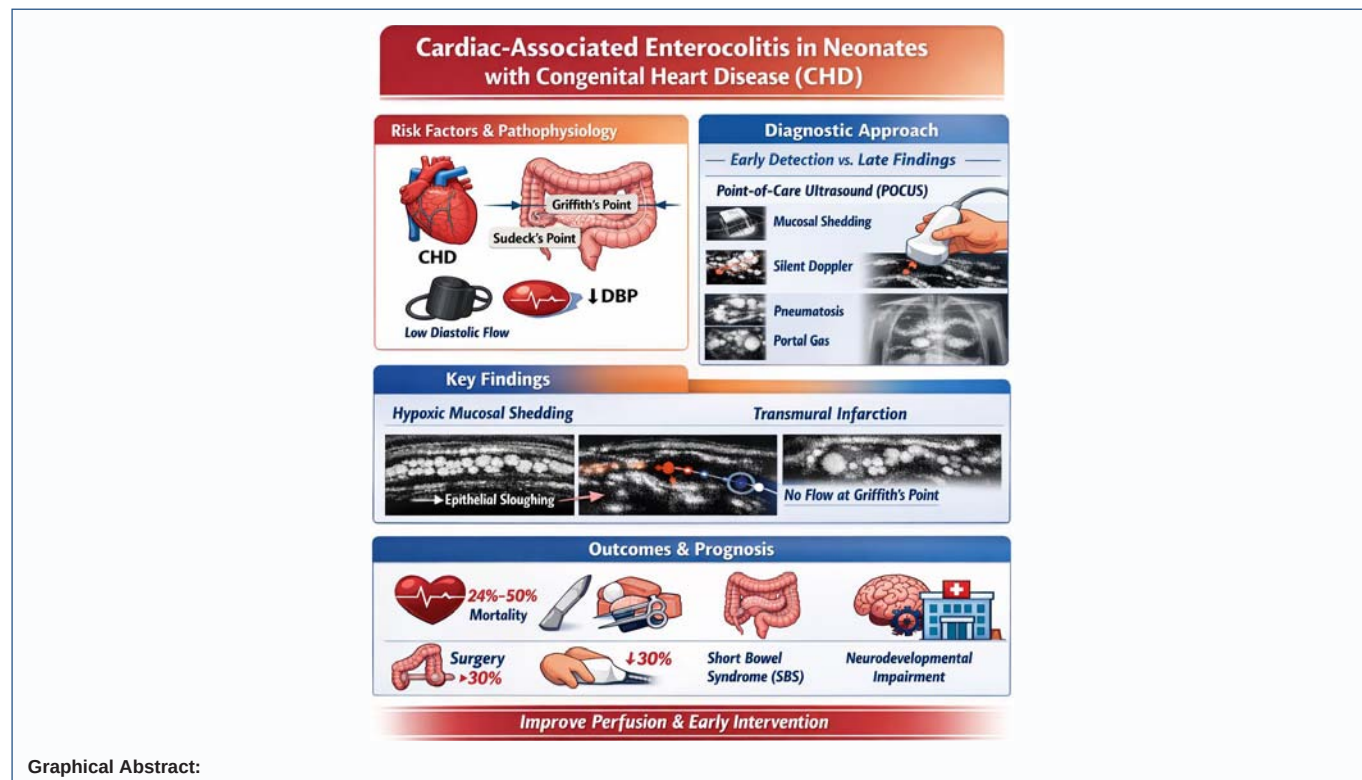
Eligibility Criteria: Studies describing structural, functional, or inflammatory and infective lesions of the fulminating necrotising enterocolitis in neonatal term cardiac patients from birth to 1 month, with emphasis on conditions requiring surgical assessment or intervention.

Results: The clinical course of Enterocolitis (EC) in neonates with Congenital Heart Disease (CHD) is characterized by a significantly higher burden of disease compared to classical neonatal NEC. High-risk lesions include HLHS and Coarctation. Key risks include the watershed areas of marginal colonic supply (Griffith's, Sudeck's, and ileocecal points). Furthermore, the terminal reach of vasa recta makes the antimesocolic area a zone of relative ischaemia. Postoperative ileus exacerbates this by compressing microvascular flow, leading to rapid hypoxic mucosal shedding.

Conclusions: Fulminating Enterocolitis (EC) in term neonates with congenital heart disease (CHD) represents a distinct vascular phenotype, diverging from the dysbiosis-driven Necrotising Enterocolitis (NEC) of prematurity. This condition is characterised by segmental ischaemia at anatomical watershed zones and rapid progression to transmural infarction. Mortality ranges from 24% to 50%, with single ventricle physiology reaching up to 97%. Early Point-of-Care Ultrasound (POCUS) detection of mucosal shedding and mural avascularity offers a proactive diagnostic alternative to late-stage radiographic signs. Surgical management prioritises perfusion-preserving strategies and anatomical sparing. This manuscript synthesises clinical outcomes, imaging findings, and anatomical risk factors, advocating for a paradigm shift in diagnostic and operative management. Early recognition of these anatomical triggers via Point-of-Care Ultrasound (POCUS) is critical to reducing the high morbidity and mortality in this population.

Keywords: Enterocolitis; Necrotizing; Heart Defects; Congenital; Watershed Areas; Fulminating

Enterocolitis; Congenital Heart Disease; Hypoxic Mucosal Shedding; Anatomical Watershed Zones; Antimesocolic Ischaemia; Postoperative Ileus; Bacterial Translocation; Point-of-Care Ultrasound (POCUS); Low Cardiac Output Syndrome (LCOS)



Summary Box

What is already known on this topic

- Necrotising Enterocolitis (NEC) in term neonates is strongly linked to congenital heart disease (CHD) and low cardiac output.
- "Diastolic steal" in ductal-dependent lesions reduces mesenteric perfusion, predisposing the gut to ischaemia.

What this study adds?

- Identifies three critical watershed anatomical zones - Griffith's point (splenic flexure), Sudeck's point (rectosigmoid), and the ileocecal point - as primary sites of failure.
- Highlights the antimesocolic border as a zone of relative ischaemia due to the circumferential nature of the long and short terminal arteries.
- Explains how postoperative ileus acts as a mechanical catalyst, where bowel distension compresses terminal vessels to trigger rapid mucosal shedding.

How this might change clinical practice?

- Advocates for a "POCUS-First" approach to detect the loss of the "gut signature" at the antimesocolic border before X-ray markers appear.
- Suggests that managing postoperative distension is a critical vascular intervention to prevent microvascular bowel infarction.

Strengths and Limitations of This Study

Strengths

- Specific Clinical Phenotype: This review provides a dedicated

analysis of the "cardiac gut," distinguishing it from the traditional prematurity-based model of NEC, which is often conflated in general neonatal literature.

- Anatomical Precision: By identifying the three specific watershed points (Griffith's, Sudeck's, and ileocecal) and the antimesocolic microvascular risk, this study provides a concrete anatomical framework for surgical and radiological suspicion.

- Diagnostic Innovation: The integration of Point-of-Care Ultrasound (POCUS) markers - specifically the "loss of gut signature" - offers a proactive alternative to the late-stage findings of traditional radiography.

- Interdisciplinary Scope: The review bridges the gap between paediatric cardiac surgery, neonatal intensive care, and gastrointestinal pathology, making it highly relevant for the IICR readership.

Limitations

- Heterogeneity of Cardiac Lesions: The "cardiac-NEC" label covers a wide spectrum of CHD (e.g., HLHS vs. Truncus Arteriosus), and the exact haemodynamic trigger (low flow vs. diastolic steal) may vary between these lesions.

- Retrospective Evidence Base: Much of the existing data on neonatal cardiac enterocolitis stems from retrospective registry reviews or single-centre case series, which may introduce selection bias.

- Lack of Standardised POCUS Protocols: While ultrasound shows superior sensitivity for mucosal shedding, there is currently a lack of globally standardised training and reporting protocols for

"cardiac gut" POCUS in the CVICU.

• **Confounding Factors:** Postoperative variables, such as the timing of enteral feeds and the specific choice of inotropic agents, vary significantly between institutions, making it difficult to isolate a single causative factor for mucosal shedding.

Introduction

Necrotising Enterocolitis (NEC) in term neonates with major cardiac defects follows a "fulminating" course distinct from the prematurity-associated variant. In these patients, the primary insult is acute or chronic mesenteric ischaemia resulting from ductal steal or postoperative Low Cardiac Output Syndrome (LCOS). This review explores the progression from hypoxic mucosal shedding to transmural necrosis, focusing on the unique anatomical and microvascular risks inherent to the cardiac neonate.

This manuscript provides a comprehensive analysis of the unique pathophysiology of enterocolitis in the cardiac neonatal population. Specifically, we highlight the critical role of anatomical watershed areas - including Griffith's point, Sudeck's point, and the ileocecal point - and the microvascular vulnerability of the antimesocolic border as primary risk factors for fulminant necrosis. We further discuss how postoperative ileus-induced distension exacerbates these risks, facilitating rapid mucosal shedding and bacterial translocation.

Necrotising Enterocolitis (NEC) is traditionally defined as a disease of prematurity and gut immaturity. However, a distinct and "fulminating" clinical phenotype occurs in term neonates with major Congenital Heart Disease (CHD), representing approximately 10-15% of all NEC cases [1, 2]. In this population, the primary insult is not developmental but haemodynamic and anatomical, driven by acute or chronic mesenteric "low-flow" states.

The pathophysiology of enterocolitis in the cardiac neonate is often a "two-hit" phenomenon. Preoperatively, lesions with large left-to-right shunts or ductal-dependent systemic flow (such as Hypoplastic Left Heart Syndrome or Coarctation of the Aorta) create a "diastolic steal," diverting essential blood flow away from the mesenteric circulation [3]. Postoperatively, the gut is subjected to a secondary insult from Cardiopulmonary Bypass (CPB), which triggers a systemic inflammatory response, increases capillary leak, and necessitates high-dose vasoactive support that further induces mesenteric vasoconstriction [4].

Central to this catastrophic progression is the concept of hypoxic mucosal shedding. Unlike the more gradual onset seen in preterm infants, cardiac associated enterocolitis often presents as a rapid, transmural infarction. This vulnerability is dictated by the colonic watershed areas - specifically Griffith's point at the splenic flexure, Sudeck's point at the rectosigmoid junction, and the ileocecal point - where marginal blood supply is most tenuous [5, 6]. Furthermore, the circumferential nature of the long and short terminal arteries (*vasa recta*) renders the antimesocolic border a zone of relative ischaemia. During postoperative ileus, intraluminal distension compresses these microvascular vessels, facilitating a rapid breach of the mucosal barrier and subsequent bacterial translocation [7].

Given the high morbidity and mortality associated with this condition, early recognition of these anatomical and microvascular triggers is paramount. This review examines the current understanding of fulminating enterocolitis in the cardiac neonate, advocating for a transition from traditional radiography to advanced

Table 1: Integrated Risk factors for cardiac enterocolitis.

Risk Factor	Mechanism	Clinical Marker
Watershed Zones	Absent/poor marginal anastomosis	Segmental necrosis at Splenic Flexure.
Terminal Arteries	Antimesocolic relative ischaemia	Antimesocolic wall thinning on US.
Post-Op Ileus	Increased intramural pressure	Rapid bacterial translocation.
Vasoactive Burden	Mesenteric vasospasm	Sudden metabolic acidosis.

Point-of-Care Ultrasound (POCUS) to detect early mucosal shedding before irreversible necrosis occurs.

Enterocolitis in neonates with CHD is increasingly recognised as a distinct clinical entity. Unlike classical NEC, which primarily affects preterm infants due to mucosal immaturity and dysbiosis, cardiac associated EC is a vascular catastrophe driven by haemodynamic instability and anatomical vulnerability. This manuscript explores the pathophysiology, diagnostic challenges, prognostic benchmarks, and surgical principles underpinning this high-risk condition (Table 1).

Pathophysiology: Three anatomical landmarks - Griffith's point (splenic flexure), Sudeck's point (rectosigmoid junction), and the ileocecal point - are identified as primary sites of ischaemic failure. These watershed zones are particularly susceptible to hypoperfusion during postoperative low cardiac output states. The antimesocolic border, supplied by terminal circumferential arteries, exists in a state of relative ischaemia even under normal conditions. Postoperative ileus-induced distension further compresses these vessels, triggering hypoxic mucosal shedding and bacterial translocation.

The "Two-Hit" Model

1. The Diastolic Steal: Lesions like Hypoplastic Left Heart Syndrome (HLHS) or large PDAs divert blood from the mesenteric arteries.
2. Postoperative Inflammation: Cardiopulmonary Bypass (CPB) triggers a systemic inflammatory response, increasing capillary leak and gut oedema.
3. Mucosal Shedding: Hypoxia triggers the sloughing of the intestinal epithelium, a precursor to rapid transmural necrosis.

Anatomical and microvascular determinants of ischaemia

The neonatal colon possesses three high-risk "watershed" zones where collateral circulation is tenuous:

1. Griffith's Point (Splenic Flexure): The junction between the SMA and IMA.
2. Sudeck's Point (Rectosigmoid Junction): The area between the last sigmoid artery and the superior rectal artery.
3. Ileocecal Point: The terminal flow zone between ileal and colic branches.

The Antimesocolic Risk Zone: The vasa recta wrap around the bowel wall to meet at the antimesocolic border. During postoperative ileus, increased intraluminal pressure stretches the bowel wall, compressing these terminal arteries. This localized hypoxia at the antimesocolic area is the primary site for mucosal shedding and bacterial translocation.

Diagnostic paradigm

Traditional reliance on abdominal radiography (AXR) often results in reactive management. By the time pneumatosis or portal

venous gas is visible, the window for bowel salvage may have closed. POCUS enables direct visualisation of the gut signature and its loss, particularly at the antimesocolic border, serving as a high-fidelity biomarker for imminent necrosis. Doppler imaging can detect mural avascularity before radiographic signs emerge.

Diagnostic modalities: AXR vs. POCUS in cardiac enterocolitis

In the "fulminating" cardiac phenotype, the diagnostic window is exceptionally narrow. Relying solely on clinical signs often leads to delayed intervention.

1. Plain Abdominal Radiography (AXR)

AXR remains the first-line screening tool, but its limitations in the cardiac neonate must be acknowledged:

- The "Fixed Loop" Sign: A single loop of bowel that remains unchanged in position or gas configuration on serial films (6-12 hours apart). This is a hallmark of transmural necrosis, often occurring at the splenic flexure (Griffith's point).
- Pneumatosis Intestinalis: Submucosal or subserosal gas. In cardiac patients, this may appear "bubbly" or "linear" and progress rapidly to the portal venous system.
- Pneumoperitoneum: The "Football Sign" or "Rigler Sign" (gas on both sides of the bowel wall). While definitive for perforation, it is often a late finding in fulminant cases.

2. Point-of-Care Ultrasound (POCUS): The new gold standard?

POCUS offers dynamic, real-time assessment of the antimesocolic border and watershed zones, often showing changes 4-8 hours before AXR.

- Loss of "Gut Signature": A healthy bowel wall shows five distinct layers. Loss of these layers (especially the hyperechoic mucosa) indicates mucosal shedding.
 - Mural Thickness & Echogenicity: In the early "post-bypass" phase, POCUS reveals bowel wall thickening (>2.5mm) due to SIRS-induced oedema. As ischemia progresses to necrosis, the wall becomes dangerously thin (<1mm) and "paper-like."
 - Color doppler assessment: Hyperaemia: Suggests early inflammatory "second hit."
- Avascularity: A "silent" Doppler signal at the antimesocolic border is a precursor to perforation.
- Free Fluid with Debris: Unlike the simple serous ascites common after bypass, POCUS can identify "complex" echogenic fluid, suggesting a "weeping" necrotic bowel or micro-perforation (Table 2).

Methods

Search strategy

Narrative review of published literature, clinical guidelines, and surgical series across pediatric cardiology, neonatal and pediatric surgery, and neonatal/perinatal medicine. MEDLINE, Embase, and Cochrane Library were searched using terms related to topic.

Eligibility and inclusion criteria for the cardiac-NEC phenotype

For the purposes of this review and clinical identification, patients are identified based on the following criteria:

Table 2: Comparison of imaging features in cardiac-NEC.

Feature	Plain Film (AXR)	Point-of-Care US (POCUS)
Early Detection	Low (needs gas-wall interface)	High (detects wall oedema/flow)
Mucosal Integrity	Inferred via pneumatosis	Directly visualised (Gut signature)
Watershed Zones	Non-specific gas patterns	Site-specific Doppler flow
Perforation	High sensitivity (Free Air)	High sensitivity (Complex Fluid)

1. Inclusion criteria (the target cohort)

Gestational Age: Term or near-term neonates (≥36 weeks gestation) to exclude the primary confounder of gut immaturity.

Cardiac anatomy: Presence of major Congenital Heart Disease (CHD), specifically:

Ductal-dependent systemic flow: Hypoplastic Left Heart Syndrome (HLHS), critical aortic stenosis, or interrupted aortic arch.

Cyanotic lesions with "steal": Truncus arteriosus or large Patent Ductus Arteriosus (PDA) causing diastolic flow reversal in the descending aorta.

Clinical Status: Neonates in the pre-operative phase (ischaemic priming) or within the first 10 days post-cardiac surgery (bypass related SIRS and Low Cardiac Output Syndrome).

2. Clinical "Fulminant" markers

To be categorised as "fulminating," the case must exhibit at least two of the following:

Rapid Progression: Transition from first clinical sign (e.g., gastric residuals) to radiographic pneumatosis or perforation in <12 hours.

Hypoxic Mucosal Shedding: Clinical observation of "tissue-like" mucosal sloughing in stools or POCUS-confirmed loss of the "gut signature."

Refractory Metabolic Acidosis: Sudden, unexplained increase in serum lactate (>4 mmol/L) despite stable cardiac filling pressures.

3. Exclusion criteria

Preterm neonates (<34 weeks) where immaturity-related NEC is the primary differential.

Neonates with primary gastrointestinal anomalies (e.g., Gastroschisis, Hirschsprung's disease).

Cases of "Spontaneous Intestinal Perforation" (SIP) which typically lack the systemic inflammatory and ischaemic markers of the cardiac-NEC phenotype (Summary Table 1 and Table 3).

Results: Morbidity and mortality in cardiac associated enterocolitis

The clinical course of Enterocolitis (EC) in neonates with Congenital Heart Disease (CHD) is characterized by a significantly higher burden of disease compared to classical neonatal NEC.

1. Mortality outcomes

- Overall Fatality Rates: Mortality in neonates with both CHD and EC is consistently higher than in those with either condition alone, with rates typically reported between 24% and 50%.

- Risk Amplification: The presence of CHD in an infant with EC is associated with a seven-fold increased risk of mortality compared

Summary Table 1: Eligibility checklist.

Category	Requirement
Primary Cohort Term	Neonates with major CHD.
Anatomical Risk	Documented "Diastolic Steal" or Post-Bypass LCOS.
Diagnostic Focus	Evidence of Watershed Ischaemia (Griffith's/Sudeck's).
Exclusion	Prematurity-related NEC (<34 weeks). Data Extraction

Table 3: Surgical decision-making.

Finding	Surgical Action	Rationale
Antimesocolic Shedding	Proximal Stoma	Reduces tension; improves micro-perfusion.
Watershed Infarction	Segmental Resection	Removes necrotic focus; prevents SIRS.
Post-Bypass LCOS	Second-Look Laparotomy	Allows for "flow-dependent" recovery of bowel.
Transmural Necrosis	Resection + Stoma	Avoids primary anastomosis in unstable neonates.

to infants without CHD (OR 7.0).

- Critical Lesions: Mortality is highest in infants with single ventricle physiology (e.g., HLHS), where the condition can be fatal in up to 97% of surgical cases.
 - 30-Day vs. Long-Term: While 30-day mortality may hover around 20%, long-term mortality for term infants with Bell's stage 2-3 disease can reach 40%.
2. Morbidity and clinical trajectory
- Surgical Intervention: Approximately 21% to 38% of cardiac-EC cases require surgical management (laparotomy or drain placement).
 - Segmental Necrosis: Due to the watershed anatomy (Griffith's and Sudeck's points), survivors frequently suffer from:
 - Intestinal Strictures: Occurring in up to 30% of survivors.
 - Short Bowel Syndrome (SBS): Resulting from radical resection of necrotic watershed zones.
 - Hospital Burden: The occurrence of EC in CHD neonates significantly prolongs the hospital stay - often doubling it - and

Summary Table 2: Prognostic benchmarks.

Metric	Cardiac-EC Cohort	Clinical Significance
Prevalence in CHD	2%-5%	Common in high-complexity lesions.
Mortality (Unadjusted)	24%-50%	Seven-fold risk vs. non-CHD EC.
Surgical Necessity	~30%	Higher in Trisomy 21/ Stage III disease.
Median Time to Onset	6.5-14 Days	Postoperative peak at ~1 week.

Table 4: Prognostic indicators.

Indicator	Impact on Outcome	Clinical Correlation
Portal Venous Gas	Increased Mortality	Indicates advanced mucosal shedding and sepsis.
LCOS (Post-Op)	High Morbidity	Synergistic hit from low flow and inflammation.
Watershed Necrosis	Risk of SBS	Segmental loss at Splenic Flexure/Rectosigmoid.
Antimesocolic Leak	Micro-perforation	Ominous sign on POCUS; leads to peritonitis.

increases total hospital charges.

- Neurodevelopmental Outcomes: Survivors often exhibit cognitive and motor composite scores in the low-normal range, with EC being a cumulative risk factor for neurodevelopmental delay.
3. Independent risk factors
- Haemodynamic Triggers: A minimum diastolic blood pressure DBP of 30 mmHg is an independent predictor for the development of EC in duct-dependent lesions (OR 8.7).
 - Genetics: Trisomy 21 is a notable co-morbidity, with these infants being more likely to require surgical management for EC compared to those without the genetic diagnosis (Summary Table 2 and Table 4).

Discussion

Cardiac associated EC demands a shift in diagnostic and surgical strategy. The condition's rapid progression is dictated by macro-anatomical watershed zones and microvascular vulnerabilities. Early detection via POCUS and perfusion-preserving surgical techniques

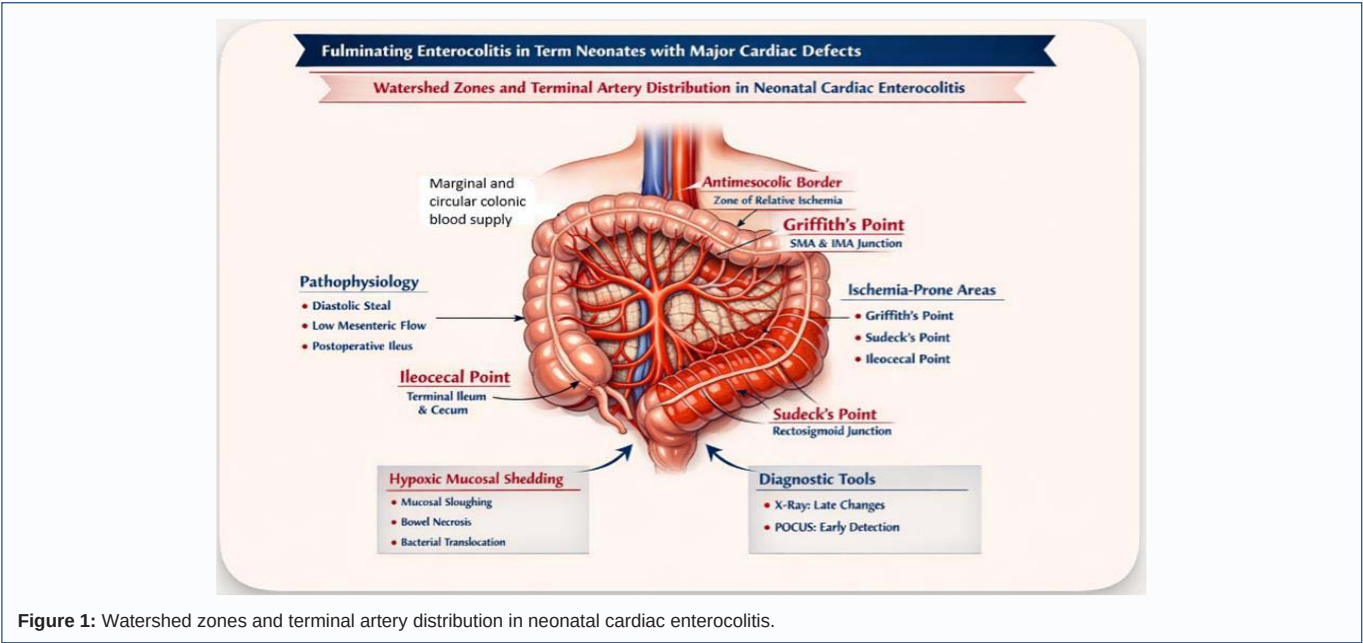


Figure 1: Watershed zones and terminal artery distribution in neonatal cardiac enterocolitis.

are essential. Vasoactive stewardship should favour lusitropy (e.g., milrinone) over pure vasoconstriction to protect mesenteric flow.

The recognition of fulminating enterocolitis as a distinct "cardiac gut" phenotype is essential for improving neonatal survival. While classic NEC in preterm infants is primarily a disorder of mucosal immaturity and dysbiosis, the condition in term neonates with major cardiac defects is a vascular catastrophe. This discussion highlights the critical intersection of surgical anatomy, hemodynamics, and the limitations of current diagnostic paradigms.

The vulnerability of watershed zones

Our review identifies three specific anatomical landmarks - Griffith's point, Sudeck's point, and the ileocecal point - as the primary "islands of failure." In the setting of a "diastolic steal" (common in HLHS or large PDAs), the marginal artery of Drummond and the arc of Rioloan may provide insufficient collateral flow to these distal territories [6, 8-11]. When systemic oxygen delivery drops postoperatively, these areas are the first to undergo hypoxic mucosal shedding.

The antimesocolic "Microvascular Trap"

A key insight presented here is the role of the circumferential terminal arteries. Because the vasa recta reach their terminal limit at the antimesocolic border, this zone exists in a state of relative ischaemia even under normal physiological conditions [9, 12, 13]. During postoperative ileus, the resulting bowel distension increases intramural tension, which mechanically compresses these terminal vessels. This "microvascular trap" explains why the antimesocolic border is the most frequent site of initial mucosal breach and subsequent bacterial translocation in the cardiac patient.

Rethinking the diagnostic approach

The traditional reliance on Plain Abdominal Radiography (AXR) often results in a "reactive" rather than "proactive" management strategy. By the time pneumatosis intestinalis or portal venous gas is visible on AXR, the window for bowel salvage has often closed [7]. We propose that Point-of-Care Ultrasound (POCUS) should become the standard of care in the CVICU. POCUS allows for the

Summary Table 3: Surgical decision-making.

Finding	Surgical Action	Rationale
Antimesocolic Shedding	Proximal Stoma	Reduces tension; improves micro-perfusion.
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Post-Bypass LCOS	Second-Look Laparotomy	Allows for "flow-dependent" recovery of bowel.
Transmural Necrosis	Resection + Stoma	Avoids primary anastomosis in unstable neonates.

direct visualisation of the "gut signature"; its loss, specifically at the antimesocolic border, serves as a high-fidelity biomarker for imminent necrosis [8, 14, 15].

Clinical implications for the cardiologist

For the cardiac intensivist, these findings mandate a shift in vasoactive stewardship. While adrenaline and noradrenaline are vital for maintaining systemic pressures, their potent-adrenergic effects can exacerbate mesenteric vasospasm at watershed points. In high-risk neonates, favouring lusitropy (e.g., milrinone) and early aggressive management of postoperative distension may preserve the microvascular flow required to prevent mucosal shedding.

Key surgical principles in cardiac associated enterocolitis

The surgical management of fulminating enterocolitis in the cardiac neonate is governed by the "Perfusion-First" principle. Unlike preterm NEC, where maturity is the issue, here the issue is flow.

1. The "Window of Opportunity"- Perfusion-First Strategy

- Early Diversion over Resection: In cases of suspected mucosal shedding at watershed zones (Griffith's or Sudeck's points), early proximal diversion (stoma) may be preferred over radical resection. Relieving intraluminal pressure (from postoperative ileus) can sometimes salvage a marginally ischaemic antimesocolic border.
- Peritoneal Drainage (GPD): While often used in unstable preterm infants, GPD is frequently insufficient in cardiac neonates due to the segmental and transmural nature of the "cardiac gut" infarction. Formal laparotomy is usually required to assess the extent of watershed necrosis.

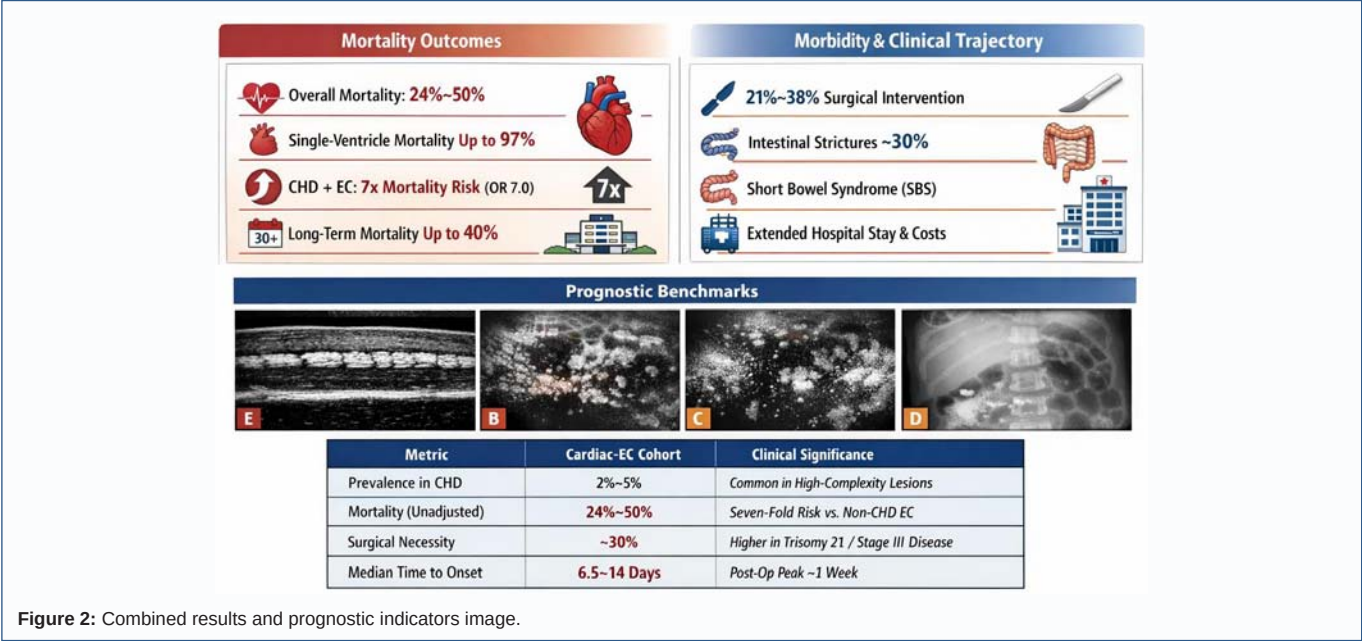
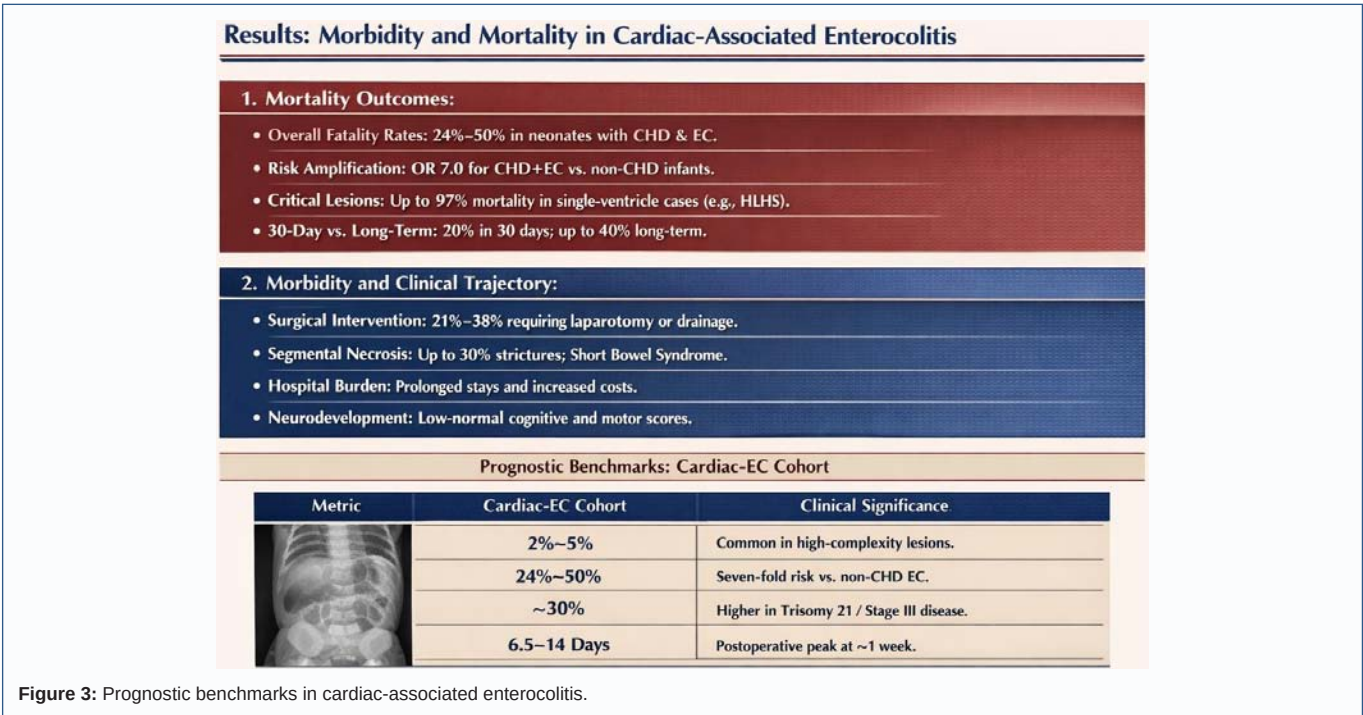


Figure 2: Combined results and prognostic indicators image.



2. Tactical resection: "Wait and See"

- Limit Initial Resection: Given the high risk of Short Bowel Syndrome (SBS), surgeons should resect only frankly necrotic/perforated segments.
- Planned Second-Look: Because the mesenteric flow is dynamic (dependent on cardiac output/inotropes), a second-look laparotomy in 24-48 hours is mandatory. Segments that appear "dusky" during LCOS may recover once cardiac stability is achieved and "diastolic steal" is corrected.

3. Anatomical sparing

- Preserving the Ileocecal Valve: Every effort must be made to preserve the ileocecal point. If the necrosis is limited to the antimesocolic border, tapering enteroplasty or patch repairs are generally avoided in favour of a temporary stoma to ensure complete bowel rest.
- Watershed Awareness: Surgeons must specifically inspect the Splenic Flexure (Griffith's Point) and Rectosigmoid Junction (Sudeck's Point), as these are the most common sites for occult, skip lesion necrosis.

4. Intraoperative flow assessment

- Doppler and Fluorescence: Use of intraoperative Doppler or Indocyanine Green (ICG) fluorescence can help delineate the microvascular "no-flow" zones at the antimesocolic border, guiding the precise margins of resection (Summary Table 3).

Research Highlights

- Distinct Phenotype: Fulminating enterocolitis in term neonates with major cardiac defects is a vascular catastrophe distinct from the necrotising enterocolitis of prematurity.
- Anatomical Vulnerability: Identifies three critical colonic watershed zones - Griffith's point (splenic flexure), Sudeck's point

(rectosigmoid), and the ileocecal point - as primary sites of ischaemic failure.

- Microvascular Risk: Highlights the antimesocolic border as a zone of relative ischaemia due to the circumferential nature of the long and short terminal arteries.
- Mechanical Catalyst: Explains how postoperative ileus-induced distension compresses these terminal vessels, triggering rapid hypoxic mucosal shedding and bacterial translocation.
- Diagnostic Paradigm Shift: Advocates for Point-of-Care Ultrasound (POCUS) over plain radiography to detect the loss of the "gut signature" and mural avascularity in the immediate post-bypass period.

Future Perspectives

The phenomenon of fulminating enterocolitis in cardiac neonates is a distinct clinical entity. Hypoxic mucosal shedding serves as the critical transition point between a reversible low-flow state and irreversible necrosis. Future strategies must focus on Near-Infrared Spectroscopy (NIRS) for continuous mesenteric monitoring and vasoactive stewardship—favouring lusitropy over pure vasoconstriction - to protect the "cardiac gut."

Conclusion

Fulminating enterocolitis in term cardiac neonates is a vascular catastrophe distinct from classical NEC. Its management requires early ultrasound-based detection, anatomical awareness, and flow-preserving surgical principles. Future strategies should incorporate continuous mesenteric monitoring and tailored haemodynamic support to protect the "cardiac gut." Fulminating enterocolitis in the term cardiac neonate represents a specific vascular catastrophe that differs significantly from the classic necrotising enterocolitis of prematurity. Its rapid progression is dictated by a unique intersection of macro-anatomical watershed zones and microvascular vulnerabilities.

The presence of critical perfusion points - specifically Griffith's point at the splenic flexure, Sudeck's point at the rectosigmoid junction, and the ileocecal point - creates "islands" of ischaemic risk. This is further compounded by the circumferential distribution of terminal arteries, leaving the antimesocolic border as a zone of relative ischaemia. In the high-stakes postoperative period, the mechanical stress of ileus-induced distension acts as the final trigger, compressing these delicate vessels and leading to rapid hypoxic mucosal shedding and bacterial translocation.

To improve survival in this fragile population, the clinical paradigm must shift:

1. Early Detection: Move beyond the late signs of plain radiography to the proactive use of Point-of-Care Ultrasound (POCUS) to monitor "gut signature" and mural flow.

2. Vascular Stewardship: Prioritise mesenteric perfusion by balancing vasoactive support and minimising factors that exacerbate bowel distension.

3. Anatomical Suspicion: Maintain a low threshold for intervention when clinical or metabolic instability aligns with ischaemia at known watershed locations.

By recognising these anatomical and physiological drivers, clinicians can transition from reactive management to a preventative strategy that protects the "cardiac gut" from irreversible necrosis.

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Competing Interests

The authors declare that they have no competing interests.

Ethics Approval

Not applicable. This study is a narrative review of published literature and did not involve human participants or animal subjects.

Patient and Public Involvement

No patients or members of the public were directly involved in the design, conduct, reporting, or dissemination of this review.

Data Availability Statement

All data relevant to the study are included in the article or uploaded as supplementary information.

Author Contributions

All authors conceived the study, conducted the literature search and drafted the manuscript. All authors contributed to revisions, approved the final version, and agree to be accountable for all aspects of the work.

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