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Review article

Post-Parturition Umbilical Cord Bacterial Infections (Omphalitis): Etiology, Management, and Global Health

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Abstract

Omphalitis, a bacterial infection of the umbilical stump and surrounding periumbilical tissues, represents a significant cause of preventable neonatal morbidity and mortality, particularly in resource-limited settings. While its incidence is low in high-income countries due to standardized hygienic practices, it remains a formidable clinical challenge in vulnerable populations globally. This infection typically presents within the first two weeks of life, with a spectrum ranging from mild cellulitis to a life-threatening systemic illness. The pathophysiology involves bacterial invasion through the necrotic cord stump, with the unique patency of the umbilical vessels facilitating rapid progression to severe complications such as necrotizing fasciitis, umbilical vein thrombophlebitis, portal vein thrombosis, and sepsis. The bacteriology is often polymicrobial, predominantly featuring *Staphylococcus aureus* (including MRSA), *Streptococcus pyogenes*, and Gram-negative organisms like *Escherichia coli* and *Klebsiella pneumoniae*. Risk factors are multifactorial, including low birth weight, unhygienic cord care practices, and application of harmful traditional substances. Diagnosis is primarily clinical, supported by laboratory investigations (inflammatory markers, blood cultures) and imaging, notably ultrasonography, to detect deep tissue involvement. Management necessitates a prompt, aggressive approach with broad-spectrum intravenous antibiotics, with surgical intervention required for complications like abscess formation or necrotizing fasciitis. Crucially, prevention is the cornerstone of reducing the disease burden, with "dry cord care" recommended in hygienic settings and topical application of chlorhexidine to the umbilical stump proven to significantly reduce incidence and mortality in high-risk environments. This comprehensive review synthesizes current evidence on the etiology, pathogenesis, clinical presentation, management, and preventative strategies for omphalitis, highlighting the critical intersection of clinical vigilance and public health intervention.

Keywords:
Omphalitis, Neonatal Infection, Umbilical Cord Care, Neonatal Sepsis, Chlorhexidine, Necrotizing Fasciitis, Umbilical Vein Thrombophlebitis, Neonatal Mortality, Postpartum Complications, Public Health.

I. Introduction

The umbilical cord serves as the critical lifeline *in utero*, facilitating the exchange of gases, nutrients, and waste between the fetus and the placenta. Following parturition, this structure is clamped and severed, leaving a residual stump that undergoes a natural, aseptic process of desiccation, necrosis, and eventual separation, typically within 5 to 15 days postpartum. This transitional period, however, renders the umbilical stump a unique and vulnerable site. As an avascular piece of necrotic tissue, it provides a fertile medium for bacterial colonization and proliferation. Omphalitis is formally defined as a bacterial infection of the umbilical stump and the surrounding periumbilical soft tissues (the subcutaneous fat and skin), which can range from a mild, localized cellulitis to a fulminant, life-threatening systemic illness (Painter, 2022).

The global incidence of omphalitis exhibits a stark disparity, with rates as low as 0.2-0.7% in high-income nations but soaring to over 20% in regions with limited access to clean birth practices and postnatal care (Mullany *et al*., 2007). While its occurrence in developed countries is a relative rarity, the profound

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severity of its potential complications underscores its continued clinical significance. The prompt recognition and aggressive, multifaceted management of omphalitis are paramount, as the infection can rapidly progress via the patent umbilical vessels to cause sepsis, necrotizing fasciitis, and portal vein thrombosis (Fraser *et al*., 2006). This comprehensive review synthesizes contemporary literature to elaborate on the etiology, risk factors, clinical spectrum, diagnostic modalities, therapeutic interventions, and preventative strategies for neonatal omphalitis, serving as an updated resource for clinicians and public health practitioners.

II. Etiology and Pathogenesis

The pathogenesis of omphalitis begins with the inevitable bacterial colonization of the umbilical stump. Within hours of birth, the umbilicus is colonized by a diverse microbiome, often derived from the maternal genital tract, the environment, and caregiver handling. Infection ensues when these commensal organisms breach the skin barrier and invade the deeper, previously sterile tissues of the cord and surrounding structures.

2.1 Common Causative Agents

The bacteriology of omphalitis is typically polymicrobial, reflecting the mixed flora of the colonization site. The predominant pathogens can be categorized as follows:

Clinical Significance	Common Pathogens	Bacterial Classification
Most common isolates globally; S. aureus is frequently the primary pathogen, with Methicillin-Resistant S. aureus (MRSA) posing a significant therapeutic challenge. S. pyogenes (GAS) is notorious for its association with rapid, invasive disease and toxic shock syndrome (AAP, 2022).	*Staphylococcus aureus* (including MRSA), *Streptococcus pyogenes* (Group A Streptococcus)	**Gram-Positive**
Often implicated in more severe or systemic infections; these organisms are common in healthcare-associated infections and in settings with poor sanitation. They are associated with a higher risk of gram-negative sepsis and endotoxin-mediated shock (Medscape, 2025).	*Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Pseudomonas aeruginosa*	**Gram-Negative**
A devastating, albeit rare, cause; C. tetani spores, introduced via contaminated instruments or substances applied to the cord, germinate in the anaerobic environment of the necrotic stump, producing a potent neurotoxin (tetanospasmin).	*Clostridium tetani*	**Anaerobic**
Often part of a polymicrobial infection; their role is increasingly recognized, particularly in foul-smelling discharge and in conjunction with other pathogens.	*Bacteroides* spp., *Peptostreptococcus* spp.	**Anaerobes**

The precise epidemiological profile is dynamic, influenced by geographic location, community hygiene standards, and local antibiotic resistance patterns (Turyasiima *et al*., 2020).

2.2 Pathogenesis and Pathways of Spread

The initial infection manifests as a localized cellulitis. The bacteria gain entry through the moist, granulating tissue at the base of the cord stump. The unique anatomical vulnerability of this site lies in the direct vascular connections of the umbilical vessels. The umbilical vein provides a direct conduit to the portal

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circulation of the liver, while the two umbilical arteries connect to the internal iliac arteries. This vascular architecture allows for the rapid hematogenous dissemination of infection.

A critical complication is umbilical vein thrombophlebitis, where infection leads to the formation of an infected thrombus within the vein. This thrombus can propagate into the portal venous system, causing pylephlebitis (portal vein thrombophlebitis), which can subsequently seed liver abscesses and lead to pre-hepatic portal hypertension (Fraser *et al*., 2006). Furthermore, local spread can lead to myonecrosis of the abdominal wall, peritonitis via a patent urachus, or the devastating soft tissue infection known as necrotizing fasciitis.

III. Risk Factors

A multifactorial set of conditions predisposes a neonate to developing omphalitis. These can be systematically categorized:

Source	Specific Risk Factors	Category
Celik *et al*. (2021)	Younger maternal age, primiparity, lower maternal education level, and poor maternal handwashing habits.	Maternal
Medscape (2025)	Low birth weight (<2500g), prematurity, congenital immune deficiencies (e.g., leukocyte adhesion deficiency), prolonged rupture of membranes (>24 hours), septic delivery, and iatrogenic trauma from umbilical catheterization.	Neonatal
Mullany *et al*. (2007)	Unhygienic cord-cutting practices (using unsterilized instruments), application of harmful traditional substances (e.g., animal dung, ash, mustard oil), home birth without a skilled attendant, prolonged hospitalization (nosocomial exposure), and being born during hot, humid seasons.	Environmental/Care-Related

The application of contaminated traditional substances is a particularly potent risk factor, as it directly inoculates pathogens while creating a moist, occlusive environment that impedes natural drying and mummification of the cord (Mullany *et al*., 2007).

IV. Clinical Presentation, Diagnosis, and Grading

The onset of omphalitis typically occurs between the 3rd and 9th day of life, though presentations outside this window are possible (Cleveland Clinic, 2024).

4.1 Clinical Signs

- Local Signs: The diagnosis is suspected with findings that extend beyond mild, transient periumbilical erythema. Key signs include:
    - Erythema and Induration: Redness and firm, palpable hardening of the skin extending >2 cm from the cord base.
    - Purulent Discharge: Frank pus or cloudy, serosanguinous fluid exuding from the stump.

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- Foul Odor: A distinct, putrid smell.
- Tenderness: Evidenced by infant crying upon palpation of the area.
- Lymphangitis: The presence of red streaks radiating onto the abdominal wall is an ominous sign of progressive infection.

- Systemic Signs: Indicate severe disease and possible sepsis:
  - Thermoregulatory instability (fever >38°C or hypothermia <36.5°C).
  - Lethargy, irritability, or a high-pitched cry.
  - Feed intolerance, vomiting, or abdominal distension.
  - Tachycardia, tachypnea, or signs of poor perfusion (mottling, delayed capillary refill).

4.2 Diagnostic Workup and Severity Grading

Diagnosis is primarily clinical, but a thorough workup is essential to guide management. A common grading system (Medscape, 2025) is outlined below:

Management Implication	Clinical Presentation	Grade
Requires hospitalization and initiation of intravenous broad-spectrum antibiotics due to the high risk of progression. Close monitoring is essential.	Purulent, malodorous discharge from the cord stump (funisitis). Erythema is minimal and confined to the cord. No systemic signs.	Grade 1 (Mild)
Requires immediate hospitalization and IV antibiotics. Imaging (ultrasound) is indicated to rule out deep tissue involvement.	Erythema and induration extending to the periumbilical skin (cellulitis). No systemic signs of toxicity.	Grade 2 (Moderate)
A medical and potential surgical emergency. Requires aggressive IV antibiotics, intensive supportive care, and immediate surgical consultation for possible debridement.	Grade 2 signs PLUS systemic toxicity (e.g., fever, lethargy, hemodynamic instability) OR any signs of necrotizing fasciitis (skin bullae, crepitus, skin necrosis).	Grade 3 (Severe)

- Laboratory Investigations:
  - Complete Blood Count (CBC) with Differential: Leukocytosis or, more ominously, leukopenia. Neutropenia can be a sign of overwhelming sepsis.
  - Inflammatory Markers: Elevated C-reactive Protein (CRP) and Procalcitonin are sensitive markers for bacterial infection and are useful for monitoring response to therapy.

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- Blood Cultures: Essential for all cases of Grade 2 and 3 omphalitis to identify bacteremia and guide targeted antibiotic therapy (AAP, 2022).

- Microbiological Studies:
  - Umbilical Swab Culture: A deep swab of purulent material should be sent for Gram stain, aerobic, and anaerobic culture with antibiotic susceptibility testing (Turyasiima *et al*., 2020).

- Imaging:
  - Abdominal Ultrasonography: The cornerstone of imaging. It is non-invasive and can detect umbilical vein thrombophlebitis, portal vein thrombosis, intra-abdominal abscesses, and subcutaneous gas indicative of necrotizing fasciitis (Fraser *et al*., 2006).
  - Computed Tomography (CT): Reserved for complex cases where US is inconclusive or when there is a high clinical suspicion for deep intra-abdominal abscess or extensive necrotizing fasciitis.

4.3 Differential Diagnosis

Clinicians must distinguish omphalitis from benign umbilical conditions:

- Umbilical Granuloma: A persistent, moist, friable, pinkish-red nodule of granulation tissue without surrounding cellulitis.
- Umbilical Hernia: A soft, reducible bulge that is not tender or erythematous.
- Patent Urachus: Presents with clear, serous drainage that may increase with crying; infection can occur but initial presentation lacks cellulitis.
- Allergic Contact Dermatitis: Often from antiseptics or soaps; presents with erythema and vesicles but lacks induration and purulent discharge.

V. Management and Treatment

Management is dictated by the severity grade, but a low threshold for aggressive treatment is warranted in neonates.

5.1 Medical Management

- Empirical Antibiotic Therapy: For Grade 2 and 3 omphalitis, immediate IV broad-spectrum antibiotics are mandatory. A common regimen includes:
  - An anti-staphylococcal penicillin (e.g., Oxacillin, Nafcillin) or a glycopeptide (e.g., Vancomycin) in areas with high MRSA prevalence.
  - PLUS an aminoglycoside (e.g., Gentamicin) or a third-generation cephalosporin (e.g., Cefotaxime) to provide robust Gram-negative coverage (Medscape, 2025).
  - Metronidazole should be added if there is foul-smelling discharge or suspicion of anaerobic involvement.

- Tailored Therapy: The regimen should be de-escalated based on culture and sensitivity results. A typical course lasts 10-14 days, extended if complications like septic thrombosis are present.

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5.2 Adjunctive and Surgical Management

- Supportive Care: Includes thermoregulation, fluid resuscitation, hemodynamic support, and correction of electrolyte imbalances.
- Surgical Intervention: Indicated for:
  - Necrotizing Fasciitis: Requires immediate, radical, and repeated surgical debridement until viable, bleeding tissue is reached.
  - Abscess Formation: Requires incision and drainage.
  - Non-responsive Infection: Surgical exploration may be necessary if the patient fails to improve despite appropriate antibiotics, to drain undetected collections.

VI. Complications

The morbidity and mortality of omphalitis are directly related to its complications:

- Local: Progression to abscess formation, necrotizing fasciitis (with mortality rates exceeding 50%), and myonecrosis.
- Vascular: Umbilical vein thrombophlebitis, portal vein thrombosis (pylephlebitis) leading to portal hypertension and extrahepatic portal vein obstruction, and septic embolization.
- Systemic: Sepsis, septic shock, disseminated intravascular coagulation (DIC), meningitis, and end-organ damage.

VII. Prevention and Cord Care Practices

Prevention is the cornerstone of reducing the global burden of omphalitis.

7.1 Dry Cord Care

In high-resource, hygienic settings, the WHO and AAP recommend "dry cord care" (Stewart *et al*., 2016). This involves:

- Washing hands before and after handling the cord.
- Keeping the cord clean and dry, exposed to air.
- Folding the diaper down to prevent contamination.
- Avoiding submersion in water until the cord has separated.

7.2 Topical Antiseptics

In community settings with high neonatal mortality rates (>30 per 1000 live births) or where harmful traditional practices are common, the application of a topical antiseptic to the cord stump is a life-saving intervention. 7.4% Chlorhexidine Digluconate is the agent of choice, with robust evidence demonstrating its efficacy in reducing omphalitis incidence by 50-75% and all-cause neonatal mortality by 20-25% (Stewart *et al*., 2016). Single-use chlorhexidine delivery systems are now widely promoted in public health campaigns across South Asia and sub-Saharan Africa.

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VIII. Conclusion

Omphalitis represents a critical nexus of neonatal medicine and global public health. While its incidence in the developed world is low, its potential for catastrophic sequelae demands vigilant clinical awareness and a low threshold for aggressive intervention. The persistent high burden in low-resource settings highlights profound health inequities. The path forward requires a dual approach: first, the continued global scale-up of evidence-based preventative strategies, primarily chlorhexidine cord care and clean birth practices; and second, within clinical practice, the unwavering principles of prompt diagnosis, aggressive empirical antibiotic therapy, and early surgical consultation for severe cases. Future research must focus on the evolving antimicrobial resistance landscape, cost-effective delivery models for chlorhexidine, and improved point-of-care diagnostics to guide therapy in remote settings.

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