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GANGRENOUS MASTITIS IN DAIRY CATTLE: ETIOLOGY, PATHOPHYSIOLOGY, AND COMPREHENSIVE MANAGEMENT

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Abstract

Gangrenous mastitis represents one of the most severe, life-threatening forms of clinical mastitis in dairy cattle, characterized by an acute inflammatory response that rapidly escalates to tissue ischemia, necrosis, and eventual sloughing of the affected mammary parenchyma. Driven primarily by opportunistic bacterial pathogens and their potent exotoxins or endotoxins, the condition carries a guarded prognosis, posing a significant threat to animal welfare, herd longevity, and dairy economics. This article reviews the etiology,

pathophysiology, clinical presentation, and evidence-based medical and surgical management strategies required to optimize clinical outcomes.

1. Introduction

Mastitis remains a predominant health and economic challenge within the global dairy industry. Among its various clinical manifestations, gangrenous mastitis is universally recognized as a peracute, devastating emergency (Islam et al., 2008).

Unlike milder forms of mastitis characterized by localized inflammation and reversible tissue changes, gangrenous mastitis involves a catastrophic disruption of the local microvasculature. The resulting thrombosis and ischemia lead to rapid cell death, leaving affected quarters cold, cyanotic, and necrotic. Prompt recognition and aggressive intervention are vital to prevent systemic collapse, septic shock, and mortality.

2. Etiology and Pathophysiology

The etiopathogenesis of gangrenous mastitis is typically multifactorial, involving the synergistic interaction of bacterial virulence factors and host immune status. The critical risk window is concentrated around the periparturient period, spanning three weeks prepartum to three weeks postpartum, due to physiological immunosuppression, negative energy balance, and heightened oxidative stress (Cheng & Han, 2020).

Primary microbial agents implicated in the disease process include:

- **Staphylococcus aureus**: A frequent primary culprit, particularly virulent strains producing alpha toxins and other dermonecrotic enzymes. These toxins provoke intense local vasoconstriction, endothelial damage, platelet aggregation, and subsequent thrombosis of the mammary vessels (Hussain et al., 2014).
- **Escherichia coli and Coliforms**: Associated with peracute toxic mastitis, releasing systemic lipopolysaccharide (LPS) endotoxins that trigger profound systemic inflammatory response syndrome (SIRS) and disseminated intravascular coagulation (DIC).
- **Clostridium perfringens (Type A)**: Frequently acts as a primary or secondary invader, proliferating rapidly in the hypoxic, anaerobic environment of the damaged gland and producing histolytic toxins and gas, culminating in emphysematous and moist gangrene (Schoonderwoerd et al., 1990).

Following bacterial invasion via the teat canal, rapid colonization within the milk cistern triggers an overwhelming host immune response. Neutrophil influx and inflammatory mediator release induce severe edema, increasing intramammary pressure and ultimately collapsing the microvasculature. Deprived of oxygen and nutrients, the affected teat and udder tissue undergoes coagulative or liquefactive necrosis.

3. Clinical Presentation and Diagnosis

Affected cows typically present with a dramatic and rapid deterioration in overall health within 24 to 48 hours of infection onset.

Clinical Manifestations

- **Local Findings:** The affected quarter transitions swiftly from being hot, hyperemic, and exquisitely painful to cold, insensate, cyanotic (dark red to bluish-black), and leather-like. A distinctive line of demarcation frequently develops between healthy and necrotic tissue.
- **Secretory Changes:** Milk production ceases entirely in the affected quarter. The secretion changes to a foul smelling, serosanguineous, or brownish watery fluid containing tissue debris.
- **Systemic Signs:** Patients exhibit severe endotoxemia or septicemia characterized by high fever (or subnormal temperature in terminal shock), profound depression, complete anorexia, rumen stasis, tachycardia, tachypnea, severe dehydration, and sternal or lateral recumbency.

Diagnosis

Diagnosis is primarily established via physical examination and patient history. Confirmation of the primary etiologic agent involves microbiological culture and antimicrobial sensitivity testing of milk or exudate samples, accompanied by gram staining (Islam et al., 2008; Hussain et al., 2014).

4. Therapeutic and Surgical Management

Because thrombosis of the local vasculature prevents systemic antibiotics from effectively reaching the core of the infection, conservative medical management alone is frequently insufficient once necrosis is established. A multi-modal therapeutic protocol is mandatory:

1. **Fluid Resuscitation and Supportive Care:** Aggressive intravenous fluid therapy (isotonic saline or lactated Ringer's solution) combined with hypertonic saline and buffer solutions is essential to correct severe dehydration, electrolyte derangements, and metabolic acidosis. Non-steroidal anti-inflammatory drugs (NSAIDs) are critical to combat endotoxic shock and control pain.
2. **Antimicrobial Therapy:** Broad-spectrum systemic antimicrobial therapy should be initiated immediately, adjusted subsequently based on culture and sensitivity profiles to control secondary bacteremia and check the spread of infection.
3. **Surgical Intervention:** Radical partial or total mastectomy (amputation of the gangrenous quarter) is often the definitive treatment of choice. Surgical removal of the necrotic, toxin-producing tissue halts systemic absorption of toxins, relieves pain, and significantly improves the patient's chances of survival (Islam et al., 2008; Lalzawmliana et al., 2019).



Conclusion

Gangrenous mastitis represents a severe clinical crisis in dairy herd management. While modern antimicrobial therapy and intensive supportive care provide foundational support, the peracute destruction of mammary tissue often necessitates prompt surgical amputation. Early identification of high-risk periparturient cows, stringent milking hygiene, and rapid therapeutic intervention remain the cornerstones of minimizing mortality and optimizing animal welfare.

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