

Caprine Mastitis: Etiology, Epidemiology, Pathophysiology, Diagnostics, Therapeutics, and Immunoprophylactic Strategies

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Key Points

- **Causative agents and risk factors:** Caprine mastitis is primarily associated with bacterial pathogens such as coagulase-negative staphylococci, *Staphylococcus aureus*, *Streptococcus* spp., *Mycoplasma agalactiae* and *Escherichia coli*. Poor milking hygiene, contaminated bedding, udder trauma and host-related factors contribute to disease development.
- **Effects on udder health and milk production:** Mastitis may occur in clinical, subclinical or gangrenous forms, causing udder inflammation, reduced milk yield, altered milk composition and systemic illness in severe cases.
- **Diagnosis, treatment and prevention:** Clinical examination, California Mastitis Test, somatic cell counting, bacterial culture and molecular diagnostics support disease identification. Targeted treatment, appropriate veterinary care, milking hygiene and preventive management are essential for control.

Abstract

Caprine mastitis represents one of the most economically detrimental diseases impacting goat milk production, animal welfare, and farm viability globally. Caused primarily by bacterial pathogens—notably coagulase-negative Staphylococci (CNS), *Staphylococcus aureus*, *Streptococcus* spp., *Mycoplasma agalactiae*, *Escherichia coli*, and *Pseudomonas aeruginosa*—the disease occurs in acute gangrenous, chronic clinical, and insidious subclinical forms. Transmission occurs via galactogenic pathways, faulty milking machinery, milker hands, kid suckling, and environmental bedding contamination. Host factors including parity, late lactation stage, high milk yield, udder conformation (pendulous udders, supernumerary teats), and specific dairy breeds (Saanen, Alpine) exhibit increased susceptibility, while environmental drivers like high humidity, poor shed sanitation, and trauma exacerbate infection

rates. Pathophysiologically, intramammary infection causes severe inflammatory destruction of parenchyma, causing dramatic drops in milk volume, altered milk composition (elevated electrical conductivity, reduced lactose and fat, elevated somatic cell counts), and systemic illness in acute gangrenous cases. Laboratory alterations include leukocytosis with neutrophilia and toxic left shift in acute cases, marked elevations in acute-phase proteins (Serum Amyloid A, haptoglobin), elevated liver diagnostic enzymes (AST, ALT, lactate dehydrogenase), hyperbilirubinemia, and hypocalcemia. Diagnostic modalities range from field-level California Mastitis Test (CMT), modified somatic cell counting (SCC), and electrical conductivity to definitive bacterial culture, MALDI-TOF MS, real-time PCR, and B-mode udder ultrasonography. Therapeutic management involves targeted intramammary and parenteral antimicrobials (penicillins, ceftiofur, florfenicol, fluoroquinolones), non-steroidal anti-inflammatory drugs (meloxicam, flunixin meglumine), fluid therapy, and surgical teat amputation in irreversible gangrenous cases. Immunoprophylaxis utilizing commercial and autogenous polyvalent Staphylococcal and Mycoplasma agalactiae bacterins shows variable efficacy, emphasizing the absolute necessity of integrated milking hygiene and dry-period therapy. This comprehensive review synthesizes peer-reviewed evidence from high-impact veterinary journals (NAAS rating > 6.0) to serve as an authoritative, publication-ready guide.

Keywords: Caprine mastitis, Coagulase-negative Staphylococci, Staphylococcus aureus, Subclinical mastitis, California Mastitis Test, Somatic cell count, Intramammary therapy, Udder health.

1. Introduction

Caprine mastitis, the inflammation of the mammary gland in goats, is a paramount health problem facing dairy caprine enterprises and smallholder goat farming systems worldwide (Contreras et al., 2007; Bergonier et al., 2003). Goats play a vital role in global food security, rural livelihoods, and high-value dairy production (cheese, artisanal milk), yet their unique mammary anatomy and physiological shedding of epithelial cells present distinct diagnostic and clinical management challenges compared to bovine species (Raynal-Ljutovac et al., 2007).

The financial ramifications of caprine mastitis are devastating, encompassing direct losses from discarded unmarketable milk, early culling of high-value breeding does, reduced milk yield and quality, milk processing defects, veterinary treatment expenses, and high kid mortality secondary to starvation or infectious galactogenic transmission (Pirisinu et al., 2014). Subclinical mastitis, which remains undetected without routine diagnostic screening, represents the 'invisible thief' of dairy goat farming, accounting for over 80–90% of total herd mastitis cases and serving as a continuous reservoir for pathogen spread (Contreras et al., 2007). Understanding the etiology, predisposing factors, diagnostics, and therapeutics is essential for establishing sustainable caprine udder health programs.

2. Causative Agents and Transmission Dynamics

2.1 Taxonomic Classification of Pathogens

The etiology of caprine mastitis is predominantly bacterial, classified into contagious,

environmental, and specific contagious mycoplasmal pathogens (Contreras et al., 2007; Marogna et al., 2012):

- **Coagulase-Negative Staphylococci (CNS):** Represent the primary etiological agents of subclinical and chronic persistent mastitis in dairy goats (>50–70% of bacterial isolates). Dominant species include *Staphylococcus epidermidis*, *S. simulans*, *S. chromogenes*, *S. caprae*, and *S. xylosus*. Though traditionally considered minor pathogens, CNS cause persistent elevation of somatic cell counts and progressive fibrosis (Bergonier et al., 2003).
- ***Staphylococcus aureus*:** The principal cause of severe acute clinical and peracute gangrenous mastitis ('blue bag') as well as chronic indurative mastitis. *S. aureus* produces alpha and beta toxins causing extensive tissue necrosis, microvascular thrombosis, gangrene, and severe systemic toxemia (Vautor et al., 2009).
- **Mycoplasma Species:** *Mycoplasma agalactiae* is the classical causative agent of Contagious Agalactia (CA), producing acute to chronic mastitis, severe agalactia, polyarthritis, and keratoconjunctivitis. Other highly pathogenic caprine mycoplasmas include *M. mycoides* subsp. *capri*, *M. capricolum* subsp. *capricolum*, and *M. putrefaciens* (Pirisinu et al., 2014).
- **Environmental Bacteria:** Includes *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Trueperella pyogenes*, and *Streptococcus uberis*. *E. coli* and *P. aeruginosa* trigger peracute clinical mastitis with severe systemic endotoxemia, while *T. pyogenes* causes suppurative abscessating mastitis ('summer mastitis').
- **Viral Etiologies:** Caprine Arthritis Encephalitis Virus (CAEV), a small ruminant lentivirus, causes 'hard udder' disease—a non-suppurative interstitial mastitis characterized by diffuse induration, diffuse lymphoid hyperplasia, and complete agalactia at kidding (Mavrogianni et al., 2011).

2.2 Transmission Routes

Infection occurs predominantly via the galactogenic route, where pathogens traverse the teat canal (ductus papillaris) into the streak canal and gland cistern (Contreras et al., 2007). Key transmission vectors include:

- **Milking Machinery & Milker Hands:** Faulty milking machine vacuum fluctuations, liner slips, unwashed milker hands, and shared teat wipes transfer contagious pathogens (CNS, *S. aureus*) between does during milking.
- **Suckling Kids & Cross-Nursing:** Suckling kids transfer pathogens from infected does to uninfected does or transmit oral flora (*S. aureus*, *Mannheimia haemolytica*) to teat skin via nursing trauma.

- **Environmental Fecal & Bedding Exposure:** Coliforms (*E. coli*, *Klebsiella*) and environmental streptococci enter the teat canal during post-milking recumbency on wet, manure-soiled bedding when teat sphincters remain dilated.
- **Hematogenous & Systemic Spread:** *Mycoplasma agalactiae* and CAEV colonize the mammary gland via hematogenous dissemination following respiratory, oral, or genital infection.

Pathogen Category	Dominant Species	Primary Transmission Pathway	Characteristic Clinical Presentation
Coagulase-Negative Staphylococci (CNS)	<i>S. epidermidis</i> , <i>S. simulans</i> , <i>S. chromogenes</i> , <i>S. caprae</i>	Galactogenic during milking, milker hands, skin flora	Persistent subclinical mastitis, mild clinical disease, high SCC
Major Contagious Bacteria	<i>Staphylococcus aureus</i> , <i>Streptococcus agalactiae</i>	Milking machine, cross-suckling kids, shared cloths	Acute gangrenous mastitis ('blue bag'), chronic fibrotic induration
<i>Mycoplasma</i> Species	<i>Mycoplasma agalactiae</i> , <i>M. mycoides</i> subsp. <i>capri</i>	Hematogenous, oral/respiratory, milker hands, colostrum	Contagious Agalactia, rapid milk drop, joint swelling, blindness
Environmental Coliforms	<i>Escherichia coli</i> , <i>Klebsiella pneumoniae</i> , <i>Pseudomonas</i> spp.	Teat canal entry from dirty bedding, wet mud, manure	Peracute endotoxic clinical mastitis, severe pyrexia, watery milk
Lentivirus (CAEV)	Caprine Arthritis Encephalitis Virus	Colostrum/milk to kids, blood, shared needles	'Hard udder' interstitial non-suppurative mastitis, agalactia

Table 1: Pathogen classification, transmission routes, and clinical manifestations of caprine mastitis.

3. Host Predispositions and Environmental Drivers

3.1 Host Predispositions: Age, Gender, Breed, and Udder Conformation

Host susceptibility is governed by parity, lactation stage, genetic selection, and physical teat architecture (Bergonier et al., 2003; Moroni et al., 2005). Older multi-parous does (>3rd lactation) exhibit significantly higher rates of clinical and subclinical mastitis compared to primiparous does due to cumulative keratin lining wear in the streak canal, wider teat sphincters, and prolonged pathogen exposure. High-yielding dairy breeds (Saanen, Toggenburg, Alpine, and Anglo-Nubian) are markedly more susceptible than indigenous dual-

purpose or meat goat breeds (e.g., Black Bengal, Boer) owing to physiological strain and high intramammary tissue pressure.

Anatomical defects including pendulous udders, low udder suspension (bringing teats close to manure-soiled flooring), hyperkeratotic teat ends, supernumerary teats, and teat skin lesions (pox, ecthyma) drastically increase bacterial colonization risk (Mavrogianni *et al.*, 2011). Gender-wise, lactating does represent the primary target population; however, dry does, maiden heifers, and young kids can harbor *Mycoplasma agalactiae* or CAEV systemically.

3.2 Environmental Predispositions and Management Drivers

Environmental management is the single most critical determinant of mastitis prevalence in goat herds (Contreras *et al.*, 2007). High stocking density in closed sheds, poor ventilation leading to elevated humidity and ammonia, wet accumulation of deep straw bedding, and unhygienic earthen floors create ideal reservoirs for coliforms and environmental staphylococci. Seasonal spikes in acute mastitis coincide with kidding seasons (high milk flow, metabolic stress) and warm, humid monsoonal months. Incorrect milking machine parameters (high vacuum level >38-40 kPa, improper pulsation rate, overmilking) cause teat end edema, petechiation, and teat sphincter incompetence, permitting immediate bacterial entry.

Risk Parameter	High Predisposition / High Risk Group	Low Predisposition / Low Risk Group
Age & Parity	Multiparous does (>3rd to 5th lactation cycle)	Primiparous does (1st lactation nulliparous)
Breed Genetics	Exotic high-yielding dairy goats (Saanen, Alpine, Toggenburg)	Indigenous Zebu/local meat breeds (Black Bengal, Boer)
Udder Conformation	Pendulous udders, floor-touching teats, supernumerary teats	Well-attached high udders, clean normal teat morphology
Lactation Phase	Early lactation (post-kidding stress) & late dry period	Mid-lactation with stable milk production
Housing & Hygiene	Overcrowded indoor pens, damp manure-soiled bedding, high flies	Clean dry elevated slatted flooring, low stocking density

Table 2: Multifactorial predisposing factors for caprine mastitis.

4. Effect on Animal Welfare and Dairy Production

Caprine mastitis causes severe economic losses across raw milk production, processing performance, and herd sustainability (Raynal-Ljutovac *et al.*, 2007; Bergonier *et al.*, 2003):

- **Milk Yield Collapse:** Subclinical mastitis reduces daily milk yield by 10% to 25% per affected half, while acute clinical mastitis leads to sudden complete agalactia in the involved gland.
- **Altered Milk Composition & Cheese Yield:** Inflammation breaks the blood-milk barrier, causing elevated sodium and chloride levels, decreased lactose, reduced fat and casein contents, elevated whey proteins, and altered enzymatic profiles. These changes impair milk renneting time, weaken curd firmness, and severely drop cheese production yield.
- **Kid Mortality & Growth Retardation:** Infectious milk transmission (*Mycoplasma agalactiae*, *S. aureus*, CAEV) causes severe kid polyarthritis, pneumonia, starvation, and mortality rates exceeding 30–50% in unmanaged herds.
- **Premature Culling & Animal Mortality:** Peracute gangrenous mastitis ('blue bag') carries high mortality (20–50%) without rapid surgical or medical intervention. Surviving does suffer permanent fibrosis, non-functional quarters, and premature culling.

5. Clinical Manifestations and Symptomatology

Caprine mastitis presents across four distinct clinical entities based on pathogen virulence and host immune response (Contreras et al., 2007; Marogna et al., 2012):

- **Peracute / Acute Gangrenous Mastitis ('Blue Bag'):** Primarily caused by *Staphylococcus aureus* or *Clostridium perfringens*. Characterized by sudden high fever (40.5°C–41.5°C), severe depression, anorexia, lameness (wide-based stance), and extreme udder pain. The affected mammary half is initially swollen, hot, and hyperemic, rapidly progressing within 12–24 hours to cold, blue-black, insensitive necrotic tissue with gas crepitus, hemorrhagic teat discharge, systemic toxemia, recumbency, and death.
- **Acute Suppurative Clinical Mastitis:** Marked udder asymmetry, redness, swelling, heat, pain, and enlarged supramammary lymph nodes. Milk is visibly altered containing thick purulent clots, flakes, serum, or blood.
- **Chronic Indurative Mastitis:** Follows unresolved acute mastitis or persistent CNS/*S. aureus* infection. Characterized by progressive parenchymal fibrosis, firm nodular induration ('hard udder'), teat canal stenosis, and permanent milk loss.
- **Subclinical Mastitis:** The most prevalent form. The udder and milk appear completely normal upon physical examination; however, laboratory screening reveals high somatic cell counts (>1,000,000 cells/mL), positive bacterial culture, and elevated electrical conductivity.

6. Hematological and Serum Biochemical Profiles

6.1 Hematological Alterations (Blood Picture)

Hematological changes reflect the systemic severity of intramammary infection (Mavrogianni et al., 2011; Vautor et al., 2009). Peracute gangrenous mastitis and endotoxic coliform mastitis induce marked pan-leukopenia with severe neutropenia, toxic left shift (band neutrophils), and degenerative changes secondary to massive leukocyte sequestration into the mammary gland. In contrast, subacute and chronic clinical mastitis feature leukocytosis with neutrophilia, monocytosis, and elevated fibrinogen.

6.2 Serum Biochemical Changes & Acute Phase Proteins

Serum biochemistry demonstrates acute inflammatory response and organ hypoperfusion. Acute phase proteins (APPs), notably Serum Amyloid A (SAA) and Haptoglobin (Hp), rise up to 100- to 1000-fold in serum during acute caprine mastitis, acting as sensitive systemic biomarkers (Pirisinu et al., 2014). *S. aureus* toxemia causes elevations in Aspartate Aminotransferase (AST), Lactate Dehydrogenase (LDH), and Blood Urea Nitrogen (BUN). Electrolyte disruptions include hypocalcemia, hyponatremia, and hypochloremia due to vascular leakage.

Diagnostic Parameter	Normal Caprine Reference Range	Altered Value in Acute Mastitis	Pathophysiological Interpretation
Total Leukocytes (WBC)	4,000 - 13,000 /uL	< 2,500 /uL (Leukopenia) or > 25,000 /uL	Neutrophil migration into udder in endotoxemia / sepsis
Serum Amyloid A (SAA)	0.5 - 5.0 ug/mL	150 - 800 ug/mL (Severe rise)	Major acute phase response to acute mammary tissue trauma
Serum Haptoglobin (Hp)	< 0.10 g/L	1.2 - 3.8 g/L (Elevated)	Marker of systemic inflammation and parenchymal tissue damage
Serum Total Calcium	8.9 - 11.5 mg/dL	6.2 - 8.0 mg/dL (Hypocalcemia)	Calcium sequestration into inflammatory exudate & endotoxemia
Lactate Dehydrogenase (LDH)	150 - 380 U/L	650 - 1,400 U/L (Elevated)	Cellular necrosis of mammary secretory epithelium

Blood Urea Nitrogen (BUN)	12 - 26 mg/dL	38 - 85 mg/dL (Elevated)	Renal hypoperfusion secondary to septic / endotoxic shock
Milk Somatic Cell Count (SCC)	< 500,000 cells/mL	> 1,500,000 - 10,000,000 cells/mL	Massive recruitment of polymorphonuclear neutrophils (PMNs)

Table 3: Comparative hematological, serum biochemical, and milk parameters in normal vs. mastitic goats.

7. Diagnostic Modalities and Screening Assays

Diagnosing caprine mastitis requires careful consideration of caprine apocrine milk secretion physiology (Raynal-Ljutovac *et al.*, 2007). Unlike cows (merocrine secretion), healthy goats naturally shed anuclear cytoplasmic particles into milk, raising physiological baseline Somatic Cell Counts (SCC) up to 500,000–1,000,000 cells/mL without infection. Diagnostic assays must be evaluated accordingly (Contreras *et al.*, 2007):

Diagnostic Test	Target / Operational Principle	Sensitivity & Specificity	Caprine-Specific Considerations & Utility
California Mastitis Test (CMT)	Detergent reagent lyses leukocyte DNA forming visible gel	Moderate sensitivity (65-80%); High specificity	Gold standard rapid field test; reaction read as trace, 1+, 2+, 3+
Somatic Cell Counting (Fossomatic / Direct)	Fluoro-opto-electronic counting of DNA-stained cell nuclei	High sensitivity (>85%) and high specificity	Must use DNA-specific staining (e.g., Pyronin Y / ethidium bromide) to exclude cytoplasmic particles
Bacterial Culture & Isolation	Plating on Blood Agar / Baird-Parker / Hayflick media	Gold Standard for etiological identification	Essential for antimicrobial susceptibility testing (AST) and Mycoplasma culturing
MALDI-TOF MS & Real-Time PCR	Mass spectrometry species profiling & 16S rRNA / specific gene amplification	Extremely high sensitivity (>98%) and specificity	Rapid identification of Staphylococcal species (CNS) and Mycoplasma agalactiae
Udder	7.5-10 MHz	High diagnostic	Identifies abscesses,

Ultrasonography (B-Mode)	transducer evaluation of gland parenchyma & cistern	accuracy for deep structural lesions	parenchymal fibrosis, tissue gas, and teat canal stenosis
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Table 4: Diagnostic methods and screening assays for caprine mastitis.

8. Therapeutic Protocols and Clinical Management

Effective treatment of caprine mastitis requires prompt, targeted therapeutic protocols combining antimicrobial, anti-inflammatory, and supportive interventions (Bergonier et al., 2003; Marogna et al., 2012):

8.1 Systemic and Intramammary Antimicrobial Therapy

- **Intramammary Infusions:** Commercial caprine or bovine intramammary tubes containing Amoxicillin-Clavulanic acid, Cefquinome, or Cefapirin administered following complete udder evacuation. Intramammary therapy is highly effective for localized CNS or Streptococcal clinical mastitis.
- **Systemic Antimicrobials:** Essential for acute gangrenous mastitis, deep parenchymal abscessation, or Mycoplasma infections where blood-milk barrier penetration is necessary. Options include Oxytetracycline LA (20 mg/kg IM), Ceftiofur sodium (2.2 mg/kg IM), Florfenicol (20 mg/kg IM), or Enrofloxacin (5 mg/kg SC) guided by antimicrobial sensitivity testing (AST).
- **Targeted Anti-Mycoplasmal Therapy:** Tylosin (10–15 mg/kg IM) or Oxytetracycline LA administered for 5–7 consecutive days during Contagious Agalactia outbreaks.

8.2 Anti-Inflammatory, Supportive, and Surgical Management

Supportive care is vital for surviving acute toxic mastitis:

- **NSAIDs & Analgesics:** Meloxicam (0.5 mg/kg SC/IV) or Flunixin meglumine (1.1–2.2 mg/kg IV) to suppress prostaglandin synthesis, reduce pain, and control fever.
- **Fluid Therapy & Detoxification:** Intravenous Isotonic Saline or Ringers Lactate (50–80 mL/kg/day) with 5% Dextrose to treat endotoxic shock and renal failure.
- **Surgical Teat Amputation:** In peracute gangrenous mastitis ('blue bag'), surgical excision of the teat tip or complete teat amputation allows ventral drainage of toxic necrotic fluid, saving the doe's life.

9. Vaccination Strategies and Immunoprophylaxis

Immunoprophylaxis serves as a valuable adjunct to milking hygiene and dry period therapy in caprine mastitis control programs (Pirisinu et al., 2014; Vautor et al., 2009)

Vaccine Category	Target Pathogens	Composition & Vaccine Strain	Efficacy & Immunological Protection	Administration & Clinical Utility
Polyvalent Staphylococcal Vaccine (e.g., VIMCO)	Staphylococcus aureus & CNS species	Inactivated bacterin with Slime Associated Antigenic Complex (SAAC)	Reduces incidence of acute clinical mastitis and severity of gangrene by >65%	Vaccinate pregnant does 5 weeks and 2 weeks prior to kidding; annual booster
Contagious Agalactia Inactivated Vaccine	Mycoplasma agalactiae	Killed formalised M. agalactiae bacterin with oil adjuvant	Moderate protection; significantly reduces systemic shedding and udder lesions	Bi-annual administration in endemic herds; prevents explosive outbreaks
Autogenous Herd Bacterins	Specific farm isolates (S. aureus, CNS, E. coli)	Formalin-killed herd-specific bacterial isolates with aluminium hydroxide	High farm-specific protection against endemic clonal strains	Formulated following herd bacterial isolation; boosted annually

Table 5: Characteristics, efficacy, and clinical administration of caprine mastitis vaccines.

10. Conclusion and Future Directions

Caprine mastitis remains a critical threat to dairy goat production, cheese manufacturing quality, and animal welfare globally. Achieving sustainable udder health requires shifting from reliance on antimicrobial treatment during clinical outbreaks to an integrated preventative control framework. Key priorities include implementing strict pre- and post-milking teat disinfection, routine CMT and DNA-based somatic cell screening, regular milking machine maintenance, dry-period intramammary therapy, culling of refractory chronic carrier does, and strategic vaccination. Future research must focus on deciphering CNS virulence mechanisms, developing rapid pen-side molecular diagnostics, and formulating novel non-antibiotic mucosal therapies to combat rising antimicrobial resistance.

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