ScienceDirect

Animal Reproduction Science

Volume 256, September 2023, 107318

Review article

A systematic review of mammary gland inflammations in queens (*Felis catus*)

Iosif Vasiu [a], Roman Dąbrowski [b], Marco Wochnik [b], Anna Płusa [c], Asta Tvarijonaviciute [d]

Show more

Outline | Share Cite

https://doi.org/10.1016/j.anireprosci.2023.107318

Get rights and content

Highlights

- Queen mastitis affects both females and kittens.
- It is encounterd during the periparturient period and secondarily to FEH.
- In cats, mastitis cases are classified into nonseptic and septic.

Abstract

Mastitis is a rare condition that can be diagnosed in female cats of all breeds and ages. It is usually caused by bacterial agents and is frequently encountered secondarily in feline mammary fibroepithelial hyperplasia (FEH). If left untreated, it can peril both the queen and her kittens. Therefore, a reliable and quick diagnosis is the primary key to successful treatment. Conventional diagnosis consists of a clinical and ultrasound evaluation of the queen's mammary glands. However, there is a lack of literature specifically focused on

mastitis in queens. Consequently, the present review brings forth the existing knowledge regarding mammary gland inflammations in cats, with the goal of improving current clinical evaluation and treatment plans.

Previous Next

Keywords

Felis catus; Mammary gland; Inflammation; Mastitis

1. Introduction

Mastitis is a rarely reported condition in this particular species. The existing literature does not provide sufficient information on the prevalence of mastitis. However, it is worth noting that mastitis may commonly occur as a secondary condition to feline mammary fibroepithelial hyperplasia (FEH) (Chisholm, 1993, Payan-Carreira, 2013). Furthermore, mastitis can affect all actively lactating, nursing, puerperal, or pseudopregnant queens (Johnston et al., 2001, Root Kustritz, 2010).

In cats, there are recognized cases of mammary gland inflammation categorized as nonseptic (i.e., galactostasis) and septic (i.e., acute mastitis, gangrenous mastitis, and chronic mastitis) (Akgül and Kaya, 2016, Al-salihi et al., 2018, Burstyn, 2010, Demirel and Ergin, 2014, Park et al., 2007, Roudebush and Wheeler, 1979, Tawfik et al., 2020, Wilson, 2013).

However, in bitches and cats, mastitis is considered an emergency (Biddle and Macintire, 2000, Jutkowitz, 2005, Vasiu et al., 2020). The presence of acute mastitis, and especially the development of gangrenous mastitis, poses a similar risk to both queens and their litters (Al-salihi et al., 2018, Demirel and Ergin, 2014, Roudebush and Wheeler, 1979, Wilson, 2013).

This systematic review aims to consolidate all relevant information on feline nonseptic and septic mastitis while assessing the specific characteristics of this species. Furthermore, the review will cover the treatment and prophylactic measures for this condition.

2. Materials and methods

A comprehensive systematic review was conducted to explore the available literature on mastitis in queens. To gather relevant data, searches were performed using Google Scholar and PubMed. The search terms employed included “queen,” “cat,” “feline,” “mastitis,” “mammary gland,” “inflammation,” “infection,” and “pathology.” Various definitions and combinations of these terms were utilized to find the necessary

information.

The search strategy was restricted to include only papers published in English, resulting in the exclusion of papers edited in different languages, such as Indonesian and Russian. The database search encompassed the earliest year for which data was available. Additionally, the reference lists of all articles and reports were examined to identify any relevant published articles that might not have been indexed in the selected database. In case further information was necessary, the corresponding authors of the selected papers were contacted.

Hard copies of any articles or other reports not available in the University of Agricultural Sciences and Veterinary Medicine Library, Cluj-Napoca, Romania, were obtained from other European libraries through the University's interlibrary loan services.

The authors chose articles from the search results by assessing titles and abstracts, excluding those irrelevant to the topic at hand (such as mastitis in women, mastitis in bitches, mastitis in ruminants, mastitis in rabbits does, or mastitis in other small companion animals). Subsequently, all the chosen articles underwent a comprehensive review of their full texts, conducted independently by the authors (IV, RD). In cases of disagreement, resolutions were reached through mutual consent. The study includes all the relevant data obtained from the literature, including case reports, research papers, book chapters, reviews, and symposium abstracts.

3. Results

A total of 23 research papers were identified as eligible for inclusion in this review. Among these, 11 (47.83%) were categorized as case reports (see Table 1, Table 5 (21.74%) were book chapters, 3 (13.04%) were reviews, and 4 (17.39%) were symposium abstracts. However, it should be noted that one case report was unavailable for online consultation and therefore excluded from the analysis.

Table 1. The case reports included in the systemic review.

	References	Mammary diagnostic	Analyses	Main conclusions/ findings
1	Chisholm (1993)	Acute mastitis	-	Local hot packing and general antibiotics are recommended in mastitis or ulcerations secondary to FEH. Thus, mastectomy should be reserved for cases that do not respond to a conservative approach
2	MacDougall (2003)	Acute mastitis	Histopathology	Secondary mammary glands ulcerations and mastitis can complicate the underlying mammary hyperplasia
3	Akgul and Kaya (2016)	Acute mastitis	Milk microbiology;	*E. coli* and *S. simulans* co-infection can cause acute mastitis in queens

			PCR	
4	Burstyn (2010)	Acute mastitis	X-rays; CBC	Acute mastitis can be secondary to mammary glands' FEH. Treated accordingly, it can hasten the recovery of the mammary glands, avoiding an unnecessary surgical outcome
5	Tawfik et al. (2020)	Gangrenous mastitis	Milk microbiology	Skin disorders are more frequent in dogs than in cats
6	Wilson (2013)	Gangrenous mastitis	Milk microbiology	The treatment of the mammary glands gangrene as an open wound using oral antibiotics and wound honey dressings represents an optimal treatment plan
7	Demirel and Ergin (2014)	Gangrenous mastitis	Milk microbiology; CBC	Lymphatic drainage of the mammary glands can be responsible for bacterial translocations
8	Al-Salihi et al. (2018)	Gangrenous mastitis	Milk microbiology; PCR	*E. coli* and *S. aureus* co-infection can cause gangrenous mastitis in queens
9	Roudebush and Wheeler (1979)	Gangrenous mastitis	Milk microbiology; CBC	Queens can withstand severe necrotic processes and have remarkable local healing resources. Moreover, monoinfections with *E. coli* can be responsible for gangrenous mastitis in queens.
10	Park et al. (2007)	Mammary lump	Histology; ELISA, PCR	*T. gondi* should be included in the differential diagnostic of nodular mammary lesions in queens

Abbreviations: CBC, complete blood count; E., Escherichia; ELISA, enzyme-linked immunosorbent assay; PCR, polymerase chain reaction; S., Staphylococcus; T., Toxoplasma.

The following sections will delve into the existing literature on mammary congestion and various forms of mastitis, such as galactostasis, subclinical mastitis, acute mastitis, gangrenous mastitis, and chronic mastitis. In addition, this discussion will provide an overview of certain anatomical and physiological aspects of healthy mammary glands in queens, along with a description of normal clinical findings.

3.1. Mammary glands in queens

In queens, the mammary gland configuration typically consists of one pair of inguinal mammary glands, two pairs of abdominal mammary glands, and a single pair of thoracic mammary glands (Englar, 2019, Hughes, 2021, Johnston et al., 2001). In cats, the mammary gland structure comprises the mammary body and the teat. The mammary body consists of numerous lobes, lactiferous ducts, and sinuses. Each lobe contains numerous lobules housing milk secretory alveoli and multiple milk ducts lined with contractile

myoepithelial cells. The mammary teat is characterized by the presence of several sinuses and papillary ducts. These ducts are lined with squamous epithelium, corresponding to each galactophore system. The distal end of the teat is closed by a sphincter composed of three layers of circular muscle tissue and is equipped with multiple papillary orifices. Each mammary gland typically comprises 4–8 galactophore systems (Hughes, 2021, Johnston et al., 2001). Furthermore, it is worth noting that the cat mammary gland is considered to have terminal ductal lobular units (Hughes, 2021).

The mammary tissue of cats exhibits the expression of progesterone and estrogen receptors. During the prepubertal and postpubertal stages, mammary epithelial cell proliferation is stimulated by estrogen. In the diestrus phase and late gestation, mammary genesis development is stimulated by progesterone, somatotropic hormone (STH), and insulin-like growth factor 1 (IGF – 1)(Hughes, 2021).

Histological variations described in the bitch mammary glands during estrus can also be found in cats (Chandra et al., 2010). However, it is important to note that cats are seasonally polyestrous and have induced ovulation (Hughes, 2021). The available literature on the morpho-functional changes in queen mammary glands during gestation, lactation, and involution periods is limited (Hughes, 2021).

To ensure the healthy and robust development of kittens, mammary secretion provides essential nutrition and immunological support. It offers protection against infectious diseases through the presence of lactobacilli, which serve as a natural source of probiotics for the intestinal microflora of the kittens. Additionally, the mammary secretion contains leukocytes, immunocompetent cells, IgA, fatty acids, lysozyme, lactoferrin, proteins, cytokines, and other milk compounds that will help in the fight for homeostasis (Chastant-Maillard et al., 2017, Jiménez et al., 2013).

However, compared to the bitch milk heterogenic microbiological composition (Vasiu et al., 2021, Vasiu et al., 2020), there is limited literature available regarding the microbiome of queen's milk to date (Table 2). However, based on the available knowledge, the reported bacteria in the milk of queens include *Enterococcus faecalis*, *Enterococcus hirae*, *Escherichia coli*, *Lactobacillus plantarum*, *Staphylococcus aureus*, and *Staphylococcus simulans* (Akgül and Kaya, 2016, Demirel and Ergin, 2014, Jiménez et al., 2013, Roudebush and Wheeler, 1979, Wilson, 2013). In addition, *Toxoplasma gondii* has been identified and isolated from queen's milk as well (Park et al., 2007).

Table 2. Milk pathogens.

Pathogens	Healthy mammary glands	Acute mastitis	Gangrenous mastitis	Chronic mastitis	References
Escherichia coli		+	+		(Akgül and Kaya, 2016, Al-salihi et al., 2018, Roudebush and Wheeler, 1979, Wilson, 2013)
Enterococcus	+				(Jiménez et al., 2013)

faecalis					
Enterococcus hirae	+				(Jiménez et al., 2013)
Lactobacillus plantarum	+				(Jiménez et al., 2013)
Staphylococcus aureus			+		(Al-salihi et al., 2018, Demirel and Ergin, 2014, Wilson, 2013)
Staphylococcus simulans		+	+		(Akgül and Kaya, 2016)
Toxoplasma gondii				+	(Park et al., 2007)

3.2. Clinical assessment of the mammary chains in queens

Usually, queens have four pairs of mammary glands, with each gland being associated with a mammary teat (Englar, 2019, Hughes, 2021, Johnston et al., 2001). The hairs surrounding the mammary glands are usually concealed within the fur, requiring its removal or separation for examination, especially in prepubertal and nulliparous queens. Teats are more noticeable in queens that have previously queened (Englar, 2019).

It is important to palpate both mammary chains during the examination. The mammary tissue should feel soft and nonsensitive to the touch. In a healthy state, there should be no palpable masses within the mammary parenchyma. Any sign of local modification, including heat, redness, congestion, engorging, an abnormal discharge, or asymmetrical mammary glands may be indicative of inflammation in the mammary glands (Englar, 2019).

The teats need to be counted and inspected to ensure the integrity of their surfaces. The skin surrounding each teat should be free from any peri-areolar injuries, such as dermatitis. In lactating queens, it is important to manually squeeze each gland to assess milk production and consistency (Englar, 2019).

Additionally, when a mass is present within a gland, it is necessary to examine the inguinal and thoracic lymph nodes to confirm the absence of local metastasis.

3.3. Ultrasound of the queen's mammary glands

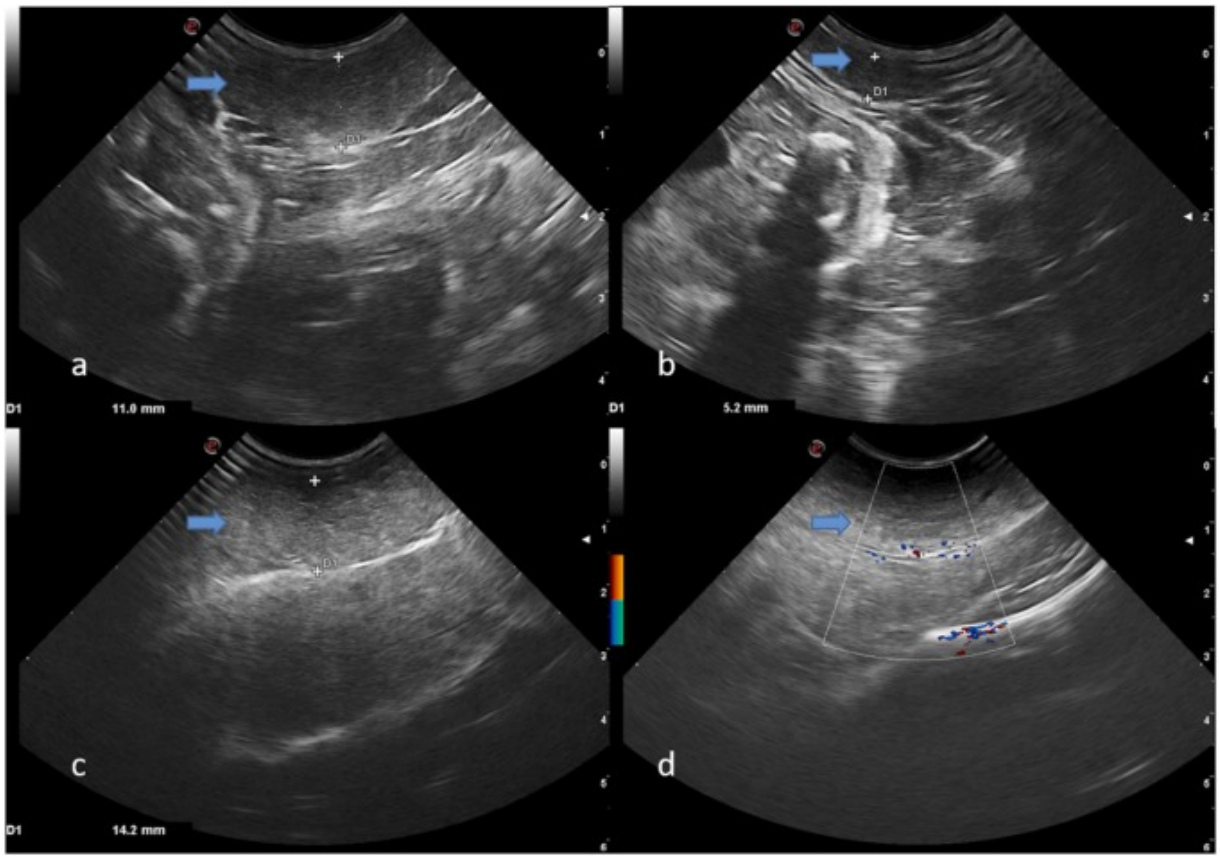
The mammary glands in queens have a limited amount of glandular tissue, which makes them unique to the species. As a result, the thickness of the mammary gland varies significantly depending on its physiological state (Table 3) (Payan-Carreira and Martins-Bessa, 2008). To conduct ultrasound (US) examinations, a 10–12 MHz linear transducer is commonly used.

Table 3. Ultrasound characteristics of the mammary glands in queens.

Physiological state of the mammary gland	Mammary gland dimensions (mm)	Echogenicity	Echostructure
Nulliparous	1.2–2.1	Hypoechogenic parenchyma	Homogenous with unidentifiable mammary glands ducts
Early gestation	2.2–3.0	Hypoechogenic parenchyma	Homogenous with a low density
Late gestation	6.5–8.7	Hyperechogenic parenchyma with hyperechogenic dorsal margins	Heterogeneous with a coarse granular pattern with small anechoic areas corresponding to the ducts of the mammary glands
Nursing glands	9.2–12.0	Isoechogenic parenchyma with hyperechogenic dorsal margins	Heterogeneous with a coarse granular pattern with well-defined anechoic structures corresponding to the ducts of the mammary glands
Non-nursing glands	3.3–6.1	Hypoechogenic parenchyma	Heterogeneous with a slight granular echo structure

Adapted after: (Payan-Carreira and Martins-Bessa, 2008).

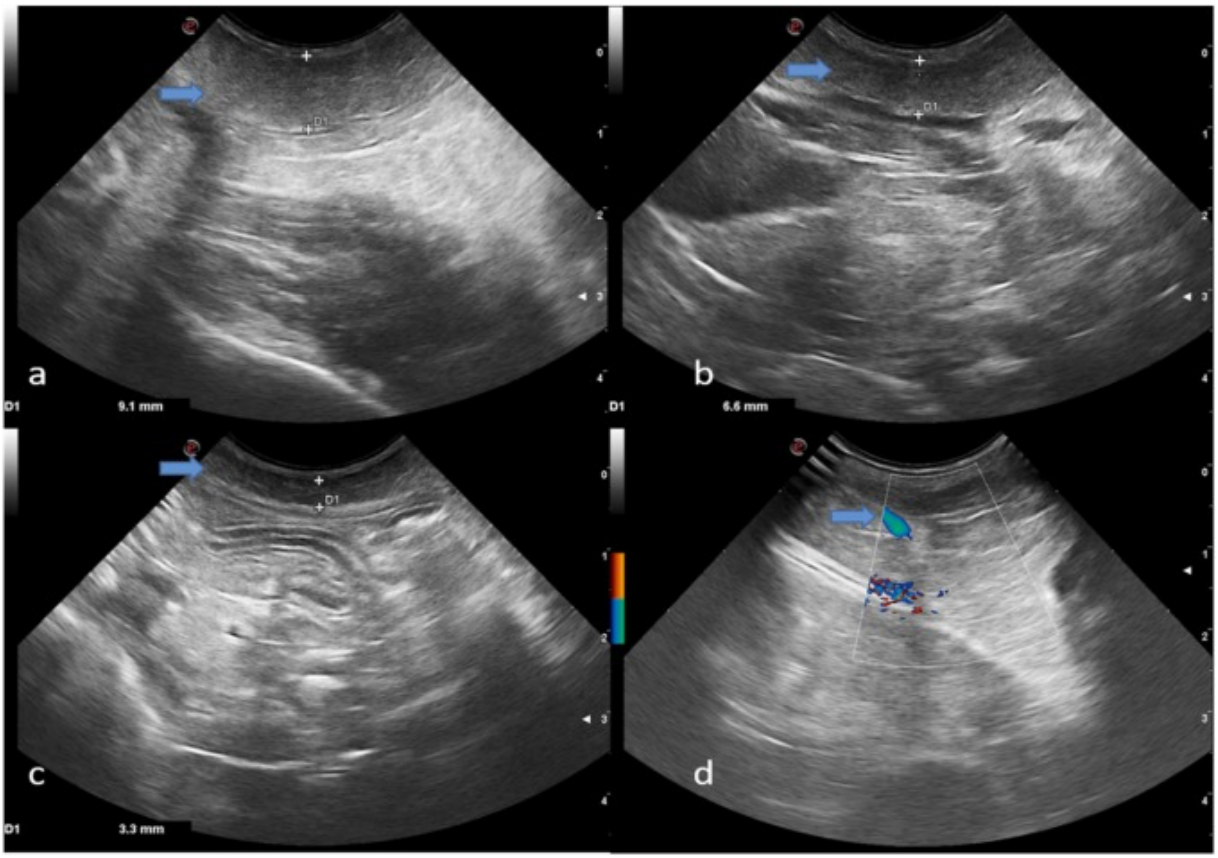
In unstimulated, intact, nulliparous, and adult queens, the abdominal wall is thicker than the mammary gland tissue. During lactation, the size of the glands is influenced by the presence of a suckling stimulus. In the first 40 days of gestation, there is a slight development of the mammary glands. After 40 days, substantial growth of glandular tissue occurs in late gestation, and the maximum mammary gland development is reached after queening (Fig. 1, Fig. 2, Fig. 3). Moreover, the puerperal non-lactating glands are smaller, similar to those in late gestation (Table 3). Furthermore, ductal structures can only be visualized during the periparturient period (Payan-Carreira and Martins-Bessa, 2008).



Download : Download high-res image (355KB)

Download : Download full-size image

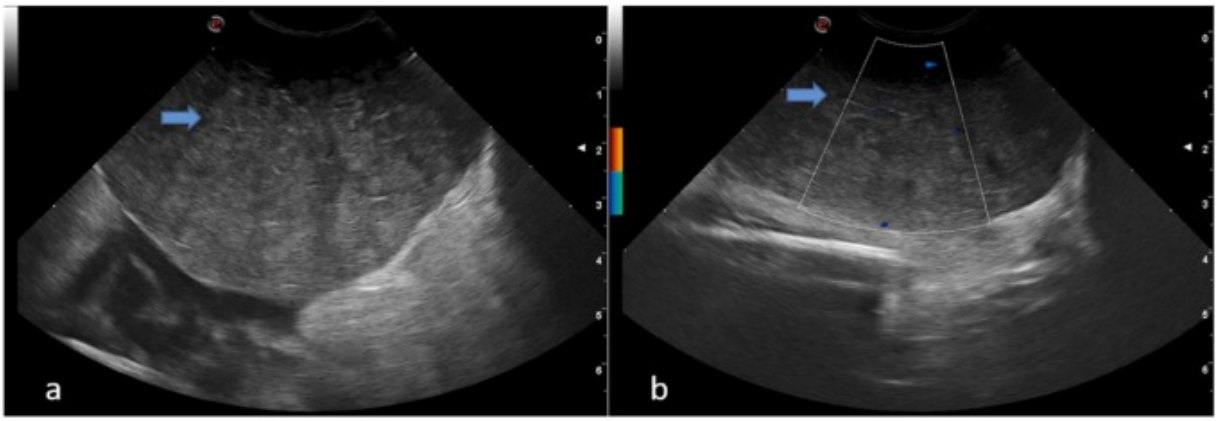
Fig. 1. ; Ultrasound depicting the presence of the mammary glands (blue arrows) in two DSH cats at 53–54 days of gestation. The mammary glands are physiologically enlarged and have characteristic moderate echogenicity (1a, b, c, d), blurred structure with visible increased flow in the Doppler examination (1d). Furthermore, small anechogenic areas are noticed in the mammary tissue, corresponding to ductal structures. The dorsal margins of the mammary glands are more hyperechoic and distinct than during the early stages of pregnancy. For the inguinal mammary glands, the parenchyma thickens varied between 11.0 mm (1a) and 14.2 mm (1c), while in the abdominal mammary glands, the thickness of the parenchyma was 5.2 mm (1b). Abbreviations: DSH - domestic shorthair cat.



Download : Download high-res image (399KB)

Download : Download full-size image

Fig. 2. Ultrasound depicting the presence of the mammary tissue (blue arrows) in one DSH, two weeks after weaning (2a, b, c), and in one DSH with pyometra (2d). The mammary glands are still enlarged with mixed echogenicity and blurred structure. Smaller ductal structures of the mammary tissue are still visible. Mammary gland capsules were emboldened, and parenchyma is also clearly visible (2a, b, c). Furthermore, the thickness of the mammary tissue varies among the glands of the same cat (2a, b, c), with 9.1 mm in the inguinal gland (2a), 6.6 mm in the caudal abdominal mammary gland (2b), and 3.3 mm in the cranial abdominal mammary gland (2c). In the second cat with pyometra (2d), the ultrasound revealed enlarged parenchyma with moderate echogenicity and visibly increased flow in the Doppler examination. Ductal structures of the mammary parenchyma were also noticed. Abbreviations: DSH - domestic shorthair cat.



Download : Download high-res image (163KB)

Download : Download full-size image

Fig. 3. Ultrasound depicting the presence of the mammary tissue (blue arrows) in one DSH with FEH (3a, b). The US revealed the presence of significantly enlarged, irregular heterogeneous mammary glands with visible increased flow in the Doppler examination (3b). Additionally, the mammary tissue acquires a more hypoechogenic pattern, and some small anechogenic areas are evidenced in the parenchyma, possibly corresponding to ductal structures of the mammary gland. Mammary gland capsules are also emboldened. The characteristic echogenicity of the distinct layers is lost with uneven shape and unclear edges. Abbreviations: DSH - domestic shorthair cat; FEH - fibroepithelial hyperplasia; US – ultrasound examination.

During US examination of the mammary tissue, it is important to carefully observe the characteristics of its margins and the surrounding healthy glandular tissue in a specific area. The appearance of the margins can provide valuable information for differentiating between benign and malignant lesions. Benign lesions typically exhibit regular and well-defined boundaries, uniform echogenicity within focal lesions, and a proper US image of the surrounding tissue. On the other hand, malignant changes are often characterized by irregular boundaries and echo distribution within the lesion's parenchyma. They may also show the presence of an areola surrounding the focal lesion with reduced echogenicity, resembling a "halo." Additionally, infiltration of the tissue bordering the lesion, indicated by the absence of clear boundaries, suggests the need for fine needle aspiration (FNA) for further evaluation. Furthermore, hyperemia, which refers to increased vascularity, can be observed in cases of mammary inflammation, indicating increased arterial and venous structures (Leong et al., 2018).

Furthermore, in cats during the postpartum period, abnormalities in the lactiferous ducts can be observed, such as dilatation of the ducts accompanied by thickened walls and contents. This may result in the accumulation of fluid within the inflamed area. The contents of these accumulations typically exhibit a hypoechoic and heterogeneous appearance, and the walls may appear irregularly thickened. It is also possible for the accumulations to form fistulas that connect with the skin or intramammary ducts (Leong et

al., 2018).

Finally, mammary US should be assessed in cases where kittens are not gaining weight, females experience discomfort during nursing, or the queens exhibit hyperthermia. By doing so, US evaluation can reveal certain findings such as hypoechogenic, coarse-grained heterogeneous areas within the mammary tissue, which may indicate acute septic mastitis. Hypoechogenic fluid pocket accumulations, suggestive of abscesses, can also be detected. Additionally, mammary fibrosis or other types of nodules may present as hyperechogenic areas with hypoechoic heterogeneity (Bassu, 2017, Davidson and Baker, 2009, Trasch et al., 2007, Trasch and Wehrend, 2008).

It should be remembered that the mammary US is undeniably valuable as a diagnostic tool. However, it should be noted that it is an additional examination, and a final diagnosis often requires combining it with the histopathological examination (Seweryn and Tworus, 2012).

3.4. Mastitis

3.4.1. Definition

Mastitis is characterized by inflammation of the glandular tissue and can affect one or multiple mammary glands simultaneously. A wide range of variations, from nonseptic galactostasis to septic, acute, gangrenous, or chronic inflammations, have been described, leading to a spectrum of asymptomatic (subclinical) to acute mastitis episodes (Johnston et al., 2001, Vasiu et al., 2020). Due to the limited amount of glandular tissue outside the periparturient period, mastitis primarily affects puerperal and lactating queens (Johnston et al., 2001, Root Kustritz, 2010).

3.4.2. Etiology and pathogenesis

However, the factors that contribute to the development of septic inflammations in the mammary glands are similar to those observed in other species (Vasiu et al., 2020). The most common route of infection is through the ascending path, where the infection spreads to the mammary tissue through the papillar opening of the teat or skin lesions on the mammary glands (Englar, 2019, Wallace, 1994, Zambelli, 2012). Skin and environmental bacterial strains are primarily responsible for causing mammary gland infections (Englar, 2019, Root Kustritz, 2010).

Furthermore, various factors can contribute to teats and mammary gland lesions, such as scratches from kittens during feeding, periodontitis followed by licking of the mammary glands, poor environmental hygiene, mammary gland congestion or galactostasis, and excessive manipulation of the mammary glands by humans. These factors can predispose the mammary glands to primary or secondary infections (Englar, 2019, Graham and Taylor, 2012, Zambelli, 2012). Moreover, in queens, several studies have described mastitis as being secondary to FEH (Chisholm, 1993, MacDougall, 2003, Payan-Carreira, 2013).

Moreover, mastitis can occur as a secondary condition to metritis, and in some cases, it can be diagnosed simultaneously, indicating the possibility of hematogenous bacterial translocation. Furthermore, systemic illnesses such as bacteremia, postpartum gastrointestinal inflammations resulting from placental or vulvar discharge and ingestion of meconium, and the presence of intestinal parasites that become active and migrate to the intestinal tract can also act as predisposing factors for septic inflammations in the mammary glands (Bassu, 2017, Wallace, 1994).

On the other hand, there is limited information available regarding the occurrence of subsequent mastitis episodes in future lactations of affected queens, as signaled in bitches, although this cannot be discarded (Bassu, 2017, Demirel and Ergin, 2014, Englar, 2019, Johnston et al., 2001, Wiebe and Howard, 2009).

In cases of nonseptic galactostasis, prolonged contact between the casein in the milk and the bloodstream can trigger a type one local hypersensitivity reaction. This reaction leads to the release of biogenic amines by basophils and mast cells. As a result, there is an increased presence of eosinophils in the affected mammary glands. Furthermore, if galactostasis is left unrecognized or untreated, septic inflammatory conditions can develop on top of the existing galactostasis (Day, 2008, Johnston et al., 2001, Vasiu et al., 2020).

3.4.3. Mammary congestion

Mammary glands that are congested appear swollen and sensitive, causing discomfort for the lactating queen. In addition, simple congestion of the glands can increase the risk of inflammation and secondary bacterial invasion (Zambelli, 2012).

Management of mammary congestion typically involves the use of warm water compresses and local massaging. Allowing the kittens to continue nursing is also important to avoid complications such as the development of galactostasis (Demirel and Ergin, 2014, Zambelli, 2012). However, due to the limited available data, further research is needed to provide a more comprehensive understanding of mammary congestion in queens.

3.4.4. Galactostasis

Nonseptic galactostasis, also known as aseptic mastitis, refers to the production and accumulation of milk in the mammary sinuses due to an imbalance between milk production and consumption. It is associated with inflammatory conditions but does not involve an infectious component such as fever or other systemic signs (Bassu, 2017, Holst, 2022, Zachary, 2017).

In queens, various factors can contribute to the development of nonseptic galactostasis, including neonatal mortality, small litter size, premature weaning, pseudopregnancy, mastitis, pain, or congenital abnormalities of the mammary duct. However, overproduction of milk may also occur without any identifiable cause. The thoracic mammary pair is reported to have a higher incidence of galactostasis (Demirel and Ergin, 2014, Holst, 2022, Martí, 2009, Orfanou et al., 2016, Prescott, 1972, Vasiu et al., 2020,

Wallace, 1994, Zambelli, 2012).

Mammary glands affected by galactostasis can exhibit swelling, pain, and firmness, causing visible discomfort when the queen is nursed (Holst, 2022, Wallace, 1994, Zambelli, 2012). If galactostasis is left unrecognized, it can lead to complications such as mammary fistula or the development of acute or chronic mastitis.

If nursing is still desired, warm water compresses, gentle stripping of the mammary glands, and encouraging the kittens to nurse can be helpful. On the other hand, if nursing is not desired, limiting food intake, applying cold water compresses, and administering diuretics may help alleviate inflammation and reduce lactation (Demirel and Ergin, 2014, Holst, 2022, Wallace, 1994, Wiebe and Howard, 2009, Zambelli, 2012).

However, given the scarce data available, future research should also focus on galactostasis in queens.

3.4.5. Subclinical and chronic mastitis

Subclinical cases of mastitis in cats refer to infections that lack obvious clinical signs of inflammation. Unfortunately, there is a lack of available data regarding subclinical mastitis in cats. However, it is important to consider that asymptomatic forms of mastitis can have significant consequences, including reduced weight gain in neonates, fetal or neonatal mortality, or even the loss of the entire litter (Englar, 2019, Holst, 2022).

The ascending route of infection is the primary cause of subclinical mammary gland infections in postpartum queens, which can be attributed to factors such as suckling kittens or damp and unsanitary environmental conditions (Barsanti, 2012, Bassu, 2017). Any litter in which there is excessive mortality or where the kittens fail to thrive should raise suspicion of subclinical mammary inflammation. However, it is still unclear whether subclinical cases are the cause or the result of a bacterial translocation between queens and kittens (Martí, 2009).

Chronic cases of mastitis in cats can occur as primary infections or as a result of untreated or undiagnosed acute mastitis. However, in queens, chronic mastitis is rarely observed, and there are only a few references acknowledging its presence. It is more commonly seen as an incidental finding in older cats. Furthermore, the mammary glands' modifications are discreet. However, in some cases, especially in geriatric cats, there may be swollen areas with hard nodules that can resemble neoplasms (Martí, 2009, Refai, 1988, Schäfer-Somi, 2018).

In cases of chronic mastitis, the mammary glands may feel firm and hard due to the formation of inflammatory connective tissue or small cystic dilations resulting from obstructed milk ducts (Bassu, 2017). Usually, no systemic illness is acknowledged (Refai, 1988); however, the kittens can fail to thrive (Bassu, 2017).

Additionally, there have been reports of nodular mastitis in a 16-year-old Japanese cat caused by an infection with *T. gondii* (Park et al., 2007). However, considering the systemic nature of toxoplasmosis, it is likely that the development of mastitis in this particular case was symptomatic, and necropsy confirmed the presence of toxoplasmosis spreading to other internal organs (Park et al., 2007).

In cases where subclinical or chronic mastitis is suspected, various laboratory tests should be performed to aid in the diagnosis. These tests may include measuring milk pH, conducting cytology and microbiology analysis (including susceptibility or blood cultures), performing a complete blood count (CBC), evaluating the acute phase protein response (APPs), in serum and/or milk, performing aspiration and cytology of masses, and utilizing US or X-ray imaging techniques (Bassu, 2017, Payan-Carreira, 2013, Vasiu et al., 2021) (Table 4).

Table 4. Clinical and laboratory findings in queens with different forms of mastitis.

Type of Mastitis	Clinical findings	Laboratory findings	References
Mammary congestion	*Queen* – local signs - distended and sensitive *mamma*; discomfort during lactation. *Kittens* – hungry.	*Milk analysis*: acidic milk pH (<6.5)*. *Milk smears*: few somatic cell counts, cellular debris and scattered neutrophils and macrophages*.	(Demirel and Ergin, 2014, Zambelli, 2012)
Galactostasis	*Queen* – local signs - swollen, painful and firm *mamma*; visible discomfort during nursing. *Kittens* – weight loss or failure to thrive.	*Milk analysis*: highly alkaline pH (<9.5)*. *Milk smears*: involve numerous eosinophils, degenerated neutrophils, epithelial, and foamy cells*.	(Holst, 2022, Wallace, 1994, Zambelli, 2012)
Subclinical mastitis	*Queen* – asymptomatic. *Kittens* - decreased weight gain; neonatal mortality or loss of entire litter (possibly developing septicemia).	Leukocytosis, anemia, an alkaline milk pH, folded levels of SAA**, CRP, increased milk count, pathogenic milk microorganisms*.	(Bassu, 2017, Englar, 2019, Holst, 2022, Vasiu et al., 2020)
Acute mastitis	*Queen* – local signs - hot, painful, distended, reddened *mamma*, abscessation, skin ulcers, purulent milk	*Milk analysis*: alkaline pH (>7); folded levels of	(Akgül and Kaya, 2016, Burstyn, 2010,

	secretion or agalactia. Systemic signs – fever, depression, inappetence, lying without moving, emesis, dehydration, tachypneic, tachycardia, hypotension, a delayed CRT, pale mucous membranes *Kittens* - failure to thrive, neonatal death.	APPs (i.e., SAA**, CRP)*· *Milk smears* involve numerous degenerated neutrophils and bacteria with phagocytosis on milk smears. *Milk microbiology*: isolation of pathogenic milk microorganisms. *Hematology*: hypochromic anemia, leukocytosis with lymphopenia, neutrophilia with left shift and toxic neutrophils. *Biochemistry*: decreased levels of ALT and creatinine^.	Johnston et al., 2001, Payan-Carreira, 2013, Root Kustritz, 2010, Wallace, 1994)
Gangrenous mastitis	*Queen* – local signs – cold, blackened, ulcerated, painful *mamma*; discolored, warm, or edematous *mamma*, with painful ectatic mammary veins; or *mammae* with large opened abscessed and necrotic mammary wounds with viscous and yellow or dark-brown milk secretion. Systemic signs – fever, vomiting, inappetence, polyuria, dehydration, delayed CRT, tachypnea, tachycardia, sepsis, SIRS or MODS. *Kittens* – failure to thrive, septicemia, neonatal death.	*Milk analysis*: alkaline pH (>7); folded levels of APPs (i.e., SAA**, CRP)*. *Milk smears* involve numerous degenerated neutrophils and bacteria with phagocytosis on milk smears. *Milk microbiology*: isolation of pathogenic milk microorganisms. *Hematology*: hypochromic anemia, leukocytosis with lymphopenia;	(Al-salihi et al., 2018, Demirel and Ergin, 2014, Roudebush and Wheeler, 1979, Tawfik et al., 2020, Wilson, 2013)
Chronic mastitis	Queen – local signs - swollen areas with hard nodules of the *mamma* or small cystic dilations. Systemic signs – absent. *Kittens* – failure to thrive.	*Milk analysis*: alkaline pH (>7); folded levels of APPs (i.e., SAA**, CRP)*. *Milk smears*: involve	(Bassu, 2017, Martí, 2009, Park et al., 2007, Refai, 1988, Schäfer-Somi, 2018, Vasiu et

				numerous degenerated neutrophils and bacteria with phagocytosis on milk smears. *Milk microbiology*: isolation of pathogenic milk microorganisms. *Hematology*: anemia with leukocytosis.	al., 2020)

Abbreviations: ALT, alanine transaminase; CRP, C-reactive protein; CRT, capillary refill time; MODS, multiple organ dysfunction syndrome; SAA, serum amyloid A; SIRS, systemic inflammatory response syndrome;

* Anecdotal data. Future research should focus on the specificity and sensibility of these parameters in lactating queens;

** SAA is the major APP in cats.

^Creatinine and ALT values can be decreased due to emaciation or muscle mass reduction in females with mastitis secondarily to FEH.

Specific abnormalities that may be identified in subclinical or chronic cases of inflammation include leukocytosis, anemia, elevated milk pH, abnormal levels of APPs (such as SAA, CRP, fibrinogen, etc.), with SAA as the major APPs in cats, an increased count of degenerated neutrophils with phagocytosis or foamy cells in milk cytology, the presence of pathogenic microorganisms in milk samples, and the presence of an inflammatory exudate within the tissues of chronically affected mammary glands (Bassu, 2017, Vasiu et al., 2021).

In the treatment of chronic mastitis, the choice of antibiotics should be based on the results of culture and susceptibility testing. It is important to consider the acidity of the milk, as some basic antimicrobials may undergo ion trapping, making them less effective. Additionally, the lipid solubility of some compounds, such as macrolides, should also be taken into account (Barsanti, 2012).

When administering antibiotic treatment for chronic mastitis in queens that are allowed to nurse, it is important to consider the potential impact on the health and development of the kittens, as antibiotics can readily pass through the milk. Therefore, using beta-lactam drugs (Table 5) as first-line antibiotics can be a safe choice for the kittens while they are suckling.

Table 5. Treatment in queen mastitis.

Drug class	Drug	Galactostasis	Acute mastitis	Gangrenous mastitis	References

Antiprolactinic					
	Cabergoline			5 μg/kg PO SID 5–7 days	(Ramsey, 2017, Wiebe and Howard, 2009)
Antibiotics					
Beta lactams[a]	Amoxicillin-clavulanic acid		20 mg BID PO 7 days	8.75–15 mg BID PO IM 21 days	(Burstyn, 2010, Demirel and Ergin, 2014, Wilson, 2013)
	Cefazolin		15–22 mg/kg SID IM IV 5 days		(Akgül and Kaya, 2016, Burstyn, 2010)
Gyrase inhibitors	Enrofloxacin[b]		5 mg/kg SID PO 7 days		(Burstyn, 2010)
Protein synthesis	Gentamycin[c]			5–10 mg/kg SID IM 7 days	(Roudebush and Wheeler, 1979)
Analgesics and anti-inflammatory drugs					
NSAIDs	Meloxicam		0.1 mg/kg SC SID		(Burstyn, 2010)
Opioid analgesic	Buprenorphine		0.02 mg/kg PO TID 2 days		(Burstyn, 2010)
Synthetic hormones	Oxytocin[c]	0.5–1 IU SC every 30 min			(Holst, 2022)

Abbreviations: BID, twice a day; IM, intramuscular(ly); IV, intravenous(ly); NSAIDs, Non-steroidal anti-inflammatory drugs; PO, per os; SID, once a day; SC, subcutaneous(ly). TID, three times a day;

[a] Depending on the severity of the affection, the antibiotic treatment may vary between 7 and 10 days or even up to 4 weeks (Holst, 2022);

[b] Antibiotics in this class should only be used if the sensibility test indicates so. In cats, irreversible retinal blindness has been reported (Ramsey, 2017);

[c] Do not use in patients where the risk of nephrotoxicity, ototoxicity, or ocular ulcers development is high (Ramsey, 2017);

[d] Oxytocin can also be administered by a nasal spray, which has a few minute's onset of action and may be administered into one nostril every 4–6 h (Holst, 2022, Wiebe and Howard, 2009).

However, due to the limited availability of data on subclinical and chronic mastitis in queens, it is imperative for future research to concentrate on providing more comprehensive descriptions of these two inflammatory conditions affecting the mammary glands.

3.4.6. Acute mastitis

In queens, acute mastitis is typically associated with lactation and the periparturient period, affecting one or multiple mammary glands simultaneously. It is accompanied by both local symptoms, such as hot, painful, swollen, and reddened mammary glands, as well as systemic signs of illness, including fever, depression, and even neonatal death (Holst, 2022, Johnston et al., 2001, Martí, 2009, Refai, 1988).

The primary cause of acute mastitis is generally ascending bacterial infections, such as *E. coli* or *S. simulans* (Akgül and Kaya, 2016). However, it can also be secondary to subclinical cases of mastitis or feline eosinophilic dermatitis with subsequent abscess formation (Burstyn, 2010, Chisholm, 1993, Holst, 2022, MacDougall, 2003).

3.4.6.1. Symptoms

In a 7-year-old Van cat, an acute case of mastitis caused by coinfection of *E. coli* and *S. simulans* was observed by Akgül and Kaya (2016). The cat exhibited bruising extending from the nipple to the surrounding mammary tissue, along with swelling, hardening, and severe local mammary pain. Additionally, difficulties were encountered in collecting milk, as the consistency appeared purulent with a yellowish color. The general signs of illness included loss of appetite, fatigue, depression, prolonged periods of lying without movement, and reluctance to nurse the kittens (Akgül and Kaya, 2016).

Moreover, in some cases, the sudden death of apparently healthy kittens may occur in the early stages of mastitis, with changes within the mammary glands becoming apparent later (Refai, 1988). Nevertheless, pyrexia, emesis, dehydration (indicated by xerostomia, persistent skin turgor, and sunken eyes), agalactia, failure to thrive, and weight loss in kittens, especially in immunosuppressed individuals, have also been reported (Akgül and Kaya, 2016, Holst, 2022, Johnston et al., 2001, Root Kustritz, 2010, Zambelli, 2012).

Another case involved a 1-year-old postpartum domestic shorthair cat (DSH), as described by Burstyn (2010). The cat had been experiencing FEH persisting for 8 weeks, which had complicated secondary mastitis and abscess formation. The mammary glands exhibited enlargement, measuring approximately 10–12 cm in diameter. They appeared asymmetric with a turgescent, venous appearance, and were hard and painful to the touch. Furthermore, fluctuating purulent pockets and skin ulcers were observed around the nipples, along with multiple peripapillary skin ulcers measuring 2–5 cm in size. There were also blue-to-black areas and fibrous septa separating the mammary tissue, creating potential fluid-filled pockets. The affected cat exhibited a fever (41.3 °C), lethargy, anorectic, dehydration, discomfort during nursing, and tachypneic (Burstyn, 2010). Some affected queens may become nonambulatory (cats are still able to move, wag their tails but are not strong enough to support their own weight and walk) and exhibit pale mucous membranes (Payan-Carreira, 2013).

Interestingly, another case involving FEH with secondary mastitis and ulcerations was documented in a 1.5-year-old neutered male DSH cat undergoing hormonal therapy (MacDougall, 2003).

In cases of septic mastitis, regardless of the underlying cause, the presence of septic shock is indicated by symptoms such as hyperventilation, tachycardia, hypotension, a delayed capillary refill time (CRT), and muscle weakness (Akgül and Kaya, 2016, Burstyn, 2010, Chisholm, 1993, Johnston et al., 2001, Zambelli, 2012).

3.4.6.2. Diagnostic measures

The diagnosis of mastitis relies on a combination of factors, including the patient's history, clinical evaluation, and laboratory assays. One of the early signs to watch out for is fever during the postpartum period, which can indicate the presence of mastitis or metritis. Therefore, it is advisable to encourage owners to monitor the rectal temperature of their cats daily throughout the lactation period (Johnston et al., 2001).

In the CBC, the presence of leukocytosis, neutrophilia with left shift and toxic neutrophils, and lymphopenia are indicative (Johnston et al., 2001, Root Kustritz, 2010). Anemia may be apparent in cases with mastitis secondarily to FEH (Burstyn, 2010, Payan-Carreira, 2013).

Blood biochemistry analysis can reveal decreased levels of creatinine and alanine aminotransferase (ALT) levels, particularly in emaciated or anorectic queens. In the context of mastitis, milk cytology examination often demonstrates the presence of degenerated neutrophils and bacteria on milk smears (Burstyn, 2010, Johnston et al., 2001, Root Kustritz, 2010, Wallace, 1994).

In cases where mastitis is secondary to FEH, imaging techniques such as X-rays or US can be valuable tools for identifying intramammary pockets filled with purulent exudate (Burstyn, 2010, Payan-Carreira, 2013).

3.4.6.3. Treatment

The treatment protocol for mastitis primarily involves antimicrobial therapy and analgesia (Table 5). In the initial stages, a broad-spectrum antibiotic should be administered, and subsequently, specific antibiotics can be chosen based on culture and sensitivity tests (Zambelli, 2012). Amoxicillin, amoxicillin with clavulanic acid, cephalexin, or erythromycin are highly recommended options as they are considered safe for neonates (Bassu, 2017, Englar, 2019, Holst, 2022, Johnston et al., 2001, Root Kustritz, 2010, Wallace, 1994, Wiebe and Howard, 2009, Zambelli, 2012). However, drugs such as chloramphenicol, doxycycline, tetracycline, or fluoroquinolones should only be used after separating the kittens from the queen (Holst, 2022, Johnston et al., 2001).

The choice of antimicrobial drug class for treating mastitis, particularly in cases of acute or chronic inflammation, is influenced by factors such as lipid solubility, pH, and the integrity of the blood–milk barrier. The milk is slightly more acidic than the serum, and as a result, weak bases with high lipid solubility tend to achieve higher therapeutic concentrations in the milk and are more likely to be excreted into it (Barsanti, 2012, Grundy, 2018, Martí, 2009, Root Kustritz, 2010).

In cases of septic shock and septicemia, it is crucial to initiate intravenous (IV) fluid therapy, and the affected queen should be treated as an intensive care patient, particularly if she has untreated FEH with secondary mastitis, skin ulcers, or abscess formation (Payan-Carreira, 2013).

For mild cases, warm water compresses or warm hydrotherapy can be applied to the mammary glands to promote drainage. Oxytocin may be administered SC every 30 min or intranasally (i.e., spray) every 4–6 h to stimulate milk secretion. It is beneficial to conduct queening in a familiar environment to facilitate milk release (Bassu, 2017), and the kittens should be allowed to suckle (Holst, 2022).

To address mastitis in nonlactating mammary glands, immersing the mammary glands in a diluted iodine povidone solution with warm water once daily can be effective (Holst, 2022, Johnston et al., 2001). Additionally, since nonlactating mammary glands have limited glandular tissue, any skin lesions present can be treated as superficial open skin lesions (Johnston et al., 2001, Payan-Carreira and Martins-Bessa, 2008). It is important to cover the affected and sensitive glands to prevent excoriations caused by nursing kittens (Root Kustritz, 2010). If it is necessary to stop milk production and lactation, the use of a dopamine agonist may be considered in certain cases (Table 5).

It should be noted that providing effective analgesia for queens can be challenging, as molecules are rapidly eliminated through milk, similar to what is observed in lactating bitches. However, like puppies, kittens can tolerate opioid analgesia, all these substances can pass through milk. If needed, the effects of opioids can be easily reversed in cases of lethargy or when kittens refuse to suckle (Mathews, 2008, Root Kustritz, 2010).

3.4.6.4. Prognosis

Mastitis can have a detrimental effect on lactogenesis, potentially leading to impaired milk ejection or a reduction in milk production (agalactia). However, if left untreated or undiagnosed, acute inflammations can progress to abscess formation or even gangrene (Demirel and Ergin, 2014, Holst, 2022).

3.4.7. Gangrenous mastitis

Mammary gangrene can occur as a primary condition or as a complication of other mammary inflammatory pathologies, such as abscess formation or subsequent tissue necrosis. It is characterized by local symptoms such as blackened, cold, or ulcerated mammary glands, along with severe systemic signs of illness, including sepsis (Johnston et al., 2001, Martí, 2009, Root Kustritz, 2010, Roudebush and Wheeler, 1979).

Various reports indicate that gangrenous mastitis predominantly affects young puerperal queens (typically between 1 and 3 years of age) within the first two weeks after queening (Al-salihi et al., 2018, Demirel and Ergin, 2014, Roudebush and Wheeler, 1979, Tawfik et al., 2020, Wilson, 2013).

However, the epidemiology of these reports is limited, making it difficult to determine if primiparous females are more affected compared to multiparous females. Additionally, while the affected breeds mentioned in the reports are DSH and Persian, it is important to note that these breeds are popular among

cat owners. Therefore, based solely on these few case reports, it is not possible to definitively conclude if these breeds are more susceptible to the condition or not.

Gangrenous lesions in queens have been reported to be caused by monoinfections with *S. aureus* or hemolytic *E. coli* (Demirel and Ergin, 2014, Roudebush and Wheeler, 1979) or coinfections involving both *E. coli* and *S. aureus* (Al-salihi et al., 2018, Wilson, 2013). However, the specific causative agent of the gangrenous lesion in the remaining case report mentioned (Tawfik et al., 2020) is not reported.

3.4.7.1. Symptoms

One week after parturition, a 1-year-old DSH, cat diagnosed with gangrenous mastitis caused by a monoinfection with hemolytic *E. coli* displayed various symptoms. These included acute vomiting, poor appetite, polyuria, dehydration, lethargy, muscle weakness, fever (39.5 °C), depression, and a whitish mucoid discharge on the perineal region. One mammary gland appeared enlarged, firm, painful, and discolored (Roudebush and Wheeler, 1979).

After 24 h, a distinct demarcation line between the adjacent mammary tissue and the surrounding tissue became evident, with a bluish-green coloration and an elevated fever (40.7 °C). By 72 h, the fever decreased (39.3 °C), the queen started to regain her appetite, and the affected mammary gland turned violet–black, clearly delineated from the surrounding healthy tissue. Furthermore, on the fifth day of hospitalization, the necrotic tissue of the mammary gland had completely separated, revealing a healthy bed of granulation tissue. The wound nearly healed completely within 14 days of the initial consultation (Roudebush and Wheeler, 1979).

In one case report, a 1-year-old primiparous mixed-breed cat, one week postpartum, was diagnosed with gangrenous mastitis caused by a monoinfection with *S. aureus*. The cat exhibited symptoms such as anorexia, dehydration, fever (40.2 °C), and tachypnea. One mammary gland appeared asymmetric, enlarged, hyperemic, warm, edematous, and painful with ectatic mammary veins. The cat also had a viscous and yellow mammary secretion (Demirel and Ergin, 2014).

In another case report, a 3-year-old DSH cat, 2 weeks postpartum, was diagnosed with gangrenous mastitis caused by coinfection of *E. coli* and *S. aureus*. The cat displayed symptoms such as anorexia, lethargy, weight loss, depression, fever (39.8 °C), tachycardia, tachypnea, dehydration, prolonged CRT, and pink mucous membranes. One mammary gland was swollen, firm, and warm. However, three days after the initial consultation, the affected mammary gland ruptured and was covered by a black necrotic skin flap. Additionally, all the other mammary glands were swollen and erythematous (Wilson, 2013).

Thus, as reported in one DSH (Al-salihi et al., 2018) and one Persian cat (Tawfik et al., 2020), in case of gangrenous mastitis caused by coinfections of *E. coli* and *S. aureus,* queens may present with a large open wound on the mammary gland with abscess and necrotic tissue. As a result, the females are reluctant to nurse, as they neglect the kittens and seek seclusion. Moreover, the milk secretion may have a dark-brown

color (Al-salihi et al., 2018).

If the untreated disease progresses, there is a possibility of developing endotoxemia in the event of an *E. coli* infection. Additionally, the development of systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome (MODS), and sepsis may occur (Al-salihi et al., 2018, Graham and Taylor, 2012, Root Kustritz, 2010).

3.4.7.2. Diagnostics

The diagnosis of the condition is established through a combination of historical information, clinical evaluation, and laboratory assays. Furthermore, in the mixed-breed queen, biochemistry assays yielded no significant findings. However, CBC revealed hypochromic anemia and leukocytosis with lymphopenia (Demirel and Ergin, 2014). On the other hand, in the DSH cat with gangrenous mastitis caused by hemolytic *E. coli*, there was mild leukocytosis with a left shift and the presence of toxic neutrophils. High numbers of degenerated neutrophils and bacteria were also observed on milk smears (Roudebush and Wheeler, 1979). Furthermore, additional complementary assays such as milk pH, microbiology, and the APPs response could be conducted using samples of milk and blood to further assist with the management of the case.

However, during the necropsy examination of the affected mammary tissue in the Persian cat with an open necrotic mammary wound, distinct observations were made. The tissue appeared dark red and exhibited multifocal to coalescing white, soft, and necrotic friable areas interspersed with a purulent exudate. Furthermore, multiple foci of liquefactive necrosis were identified. Additionally, the histopathological analysis demonstrated necrosis, tissue debris, bacterial colonies, and the presence of gas bubbles within the affected gland (Tawfik et al., 2020).

3.4.7.3. Treatment

Similar to acute mastitis, it is crucial to initiate aggressive IV fluid therapy as queens with gangrenous mastitis require intensive care. Antibiotic treatment is also essential. Therefore, pending the results of culture and sensitivity tests, empirical treatment with amoxicillin or clavulanate amoxicillin is advised (Table 5) (Al-salihi et al., 2018, Demirel and Ergin, 2014, Johnston et al., 2001, Wilson, 2013). In one case (Roudebush and Wheeler, 1979), a combination of penicillin and gentamicin was administered before the sensitivity results were available. However, it is not recommended to use gentamicin as the first-line therapeutic agent due to the potential for severe side effects (Ramsey, 2017), particularly in geriatric patients with underlying kidney, otic, or ocular impairments.

For local treatment, ethacridine powder (1 g/L water) can be utilized (Demirel and Ergin, 2014). Additionally, warm local compresses with Epsom salt, given once or twice daily (e.g., 1–2 spoons in 250 mL warm water), lavage with a diluted 0.9% betadine solution or warm water, or locally applied honey-embedded patches, given twice daily, are recommended (Johnston et al., 2001, Wilson, 2013). Treatment with cabergoline can be considered to suppress lactation while tamponing all mammary glands with Epsom salt and feeding wet

food are advisable measures (Wilson, 2013). It is important to cover the affected mammary glands to prevent excoriation of the friable tissue (Root Kustritz, 2010).

Furthermore, in complicated cases that necessitate surgical intervention, the treatment protocol involves debridement and removal of devitalized and necrotic tissues, followed by the placement of drainage tubes (Demirel and Ergin, 2014, Root Kustritz, 2010). Upon making the incisions, the discharge of seropurulent fluid may be observed (Demirel and Ergin, 2014). Additionally, after a few days of progression, areas with a diameter of 2–3 cm exhibiting a lack of substance may become evident, surrounding the devitalized necrotic-caseous tissues with hardened margins (Roudebush and Wheeler, 1979, Wilson, 2013).

Noteworthy, queens can endure severe necrotic processes while undergoing extensive wound healing through contraction and epithelization (Roudebush and Wheeler, 1979). Therefore, it is advisable not to hastily pursue radical surgical interventions, as the necrotic material typically undergoes a gradual process of sloughing and self-discarding (Roudebush and Wheeler, 1979, Wilson, 2013). However, in severe cases involving prolonged devitalized and necrotic tissue, it may be necessary to perform one or several mastectomies on the same patient (Demirel and Ergin, 2014).

3.4.7.4. Prognostic

Lastly, according to reports, the queen's health status should improve within 3–5 days of treatment. During this time, the females are expected to exhibit signs of brightness, alertness, responsiveness, a healthy appetite, and a stable mental state, with vital signs falling within the normal range (Al-salihi et al., 2018, Roudebush and Wheeler, 1979, Wilson, 2013).

Unfortunately, the prognosis is challenging to determine. Many cases have not been published, and despite all therapeutic measures, severe complications such as endotoxemia and sepsis may still arise, leading to a high mortality rate.

3.5. The nursing kittens' dilemma

Kittens should generally be allowed to nurse unless the queen is experiencing gangrenous mastitis or a mammary fistula, or if her health status does not permit it. Allowing kittens to consume antibiotic-treated milk can facilitate the drainage of the mammary gland and prevent episodes of galactostasis. Taking a precise and pragmatic approach to mastitis cases enables the queen to continue nursing. However, if antibiotic susceptibility results indicate the necessity of aminoglycosides, tetracycline, or chloramphenicol-based drugs, it is highly recommended to separate the kittens from the queen and utilize a milk replacer or a foster mother instead (Al-salihi et al., 2018, Englar, 2019, Holst, 2022, Johnston et al., 2001, Roudebush and Wheeler, 1979, Wiebe and Howard, 2009, Zambelli, 2012).

4. Conclusion

Mastitis in lactating queens should not be disregarded, as it can have negative implications for both the

queen and the nursing kittens. The present systematic review aims to consolidate the existing literature on mastitis in queens, enhancing our understanding of its clinical progression and treatment strategies. Additionally, it acknowledges the lack of information on this topic, which should be assessed in future studies.

Ethical approval

Not applicable.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of Competing Interest

The authors declare no potential conflicts of interest concerning this article's research, authorship, and/or publication. No conflict of interest exists.

Acknowledgments

The warmest thanks to DVM Ph.D. Professor Raul Alexandru Pop, and DVM Ph.D. students Gál Zoltán-Miklós and Ana Hîruța, for providing valuable support concerning the images.

Author contributions

All authors contributed to the literature review, drafting, and approval of the submission.

Informed consent

No animals or humans are identifiable within this publication; therefore, additional informed consent for publication was not required.

Appendix A. Supplementary material

Download : Download Acrobat PDF file (133KB)

Supplementary material

.

Recommended articles

References

Akgül and Kaya, 2016 Ö. Akgül, A. Kaya

Microbiological analysis of acute mastitis in a Van cat [Bir van kedisinde görülen akut mastitisin mikrobiyolojik analizi]

Kafkas Univ. Vet. Fak. Derg., 22 (2016), pp. 159-162, 10.9775/kvfd.2015.14082

View in Scopus Google Scholar

Al-salihi et al., 2018 K.A. Al-salihi, A.M.R. Al-yasari, H.A. Muhammid

Gangrenous mastitis in local short haired feline

Diyala J. Agric. Sci., 10 (2018), pp. 233-240

Google Scholar

Barsanti, 2012 J.A. Barsanti

Genitourinary Infections

C.E. Greene (Ed.), Infectious Diseases Of The Dog And Cat, Elsevier, Georgia (2012), pp. 1013-1044

Google Scholar

Bassu, 2017 G. Bassu

Mammary Gland Disorders: Agalactia, Galactostasis, and Mastitis

D.S. Greco, A.P. Davidson (Eds.), Blackwell's Five-Minute Veterinary Consult Clinical Companion - Small Animal Endocrinology and Reproduction, Wiley-Blackwell, Iowa (2017), pp. 393-407

Google Scholar

Biddle and Macintire, 2000 D. Biddle, K.D. Macintire

Obstetrical emergencies

Clin. Technol. Small Anim. Pr., 15 (2000), pp. 89-93, 10.1002/9781118327241.ch11

Google Scholar

Burstyn, 2010 U. Burstyn

Management of mastitis and abscessation of mammary glands secondary to fibroadenomatous hyperplasia in a primiparturient cat

J. Am. Vet. Med. Assoc., 236 (2010), pp. 326-329

CrossRef View in Scopus Google Scholar

Chandra et al., 2010 S.A. Chandra, J.M. Cline, R.R. Adler

Cyclic morphological changes in the beagle mammary gland

Toxicol. Pathol., 38 (2010), pp. 969-983, 10.1177/0192623310374327

View in Scopus Google Scholar

Chastant-Maillard et al., 2017 S. Chastant-Maillard, C. Aggouni, A. Albaret, A. Fournier, H. Mila

Canine and feline colostrum

Reprod. Domest. Anim., 52 (2017), pp. 148-152, 10.1111/rda.12830

View in Scopus Google Scholar

Chisholm, 1993 H. Chisholm

Massive mammary enlargement in a cat

Can. Vet. J., 34 (1993), p. 315

View in Scopus Google Scholar

Davidson and Baker, 2009 A.P. Davidson, T.W. Baker

Reproductive ultrasound of the bitch and queen

Top. Companion Anim. Med., 24 (2009), pp. 55-63, 10.1053/j.tcam.2008.11.002

View PDF View article View in Scopus Google Scholar

Day, 2008 J.M. Day

Immunopathological mechanisms, in: Day, J.M. (Ed.), clinical immunology of the dog and cat

Manson Publ. Ltd, Lond. (2008), pp. 61-74

CrossRef Google Scholar

Demirel and Ergin, 2014 M.A. Demirel, I. Ergin

Medical and surgical approach to gangrenous mastitis related to galactostasis in a cat

Acta Sci. Vet., 42 (2014), pp. 1-4

CrossRef Google Scholar

Englar, 2019 R.E. Englar

Abnormal Presentations of the Mammary Glands

R.E. Englar (Ed.), Common Clinical Presentations in Dogs and Cats, John Wiley & Sons, Inc, Hoboken, NJ, USA (2019), pp. 769-782, 10.1002/9781119414612.ch62

Google Scholar

Graham and Taylor, 2012 E.M. Graham, D.J. Taylor

Bacterial reproductive pathogens of cats and dogs

Vet. Clin. North Am. Small Anim. Pract., 42 (2012), pp. 561-582, 10.1016/j.cvsm.2012.01.013

View PDF View article View in Scopus Google Scholar

Grundy, 2018 Grundy, S.A., 2018. Metritis and Mastitis, in: Dobratz, K.J., Hopper, K., Rozanski, E., Silverstein, D.C. (Eds.), Textbook of Small Animal Emergency Medicine. John Wiley & Sons, New Jersey, pp. 791–794. https://doi.org/10.1002/9781119028994.ch122.

Google Scholar ↗

Holst, 2022 B.S. Holst

Feline breeding and pregnancy management: what is normal and when to intervene

J. Feline Med. Surg., 24 (2022), pp. 221-231, 10.1177/1098612x221079708 ↗

View in Scopus ↗ Google Scholar ↗

Hughes, 2021 K. Hughes

Comparative mammary gland postnatal development and tumourigenesis in the sheep, cow, cat and rabbit: exploring the menagerie

Semin. Cell Dev. Biol., 114 (2021), pp. 186-195, 10.1016/j.semcdb.2020.09.010 ↗

View PDF View article View in Scopus ↗ Google Scholar ↗

Jiménez et al., 2013 Jiménez, M.R., Martín, M.O., Quintana, J.E.A., Martínez, M.L.M., Saleta, Á.S., Barragán, M.A., Merino, M.V., Martell, B.F., Lloveras, T.C., Villosalda, L.F., Rodríguez, A.R., Puerta, B.J., López, J.J., Álvarez, F.L., Abuja, S.J.O., Pei, X.J., Gómez, R.M.J., Palacio, D.S., 2013. Mammalian milk microorganisms, compositions containing them and their use for the treatment of mastitis. US 8,557,560 B2.

Google Scholar ↗

Johnston et al., 2001 S.D. Johnston, M.V. Root Kustritz, P.S. Olson

The postpartum period in the cat

S.D. Johnston, M.V Root Kustritz, P.S. Olson (Eds.), Canine and Feline Theriogenology, Saunders, London, UK (2001), pp. 438-446

Google Scholar ↗

Jutkowitz, 2005 L.A. Jutkowitz

Reproductive emergencies

Vet. Clin. Small Anim. Pract., 35 (2005), pp. 397-420, 10.1016/j.cvsm.2004.10.006 ↗

View PDF View article View in Scopus ↗ Google Scholar ↗

Leong et al., 2018 P.W. Leong, N.C. Chotai, S. Kulkarni

Imaging features of inflammatory breast disorders: a pictorial essay

Korean J. Radiol., 19 (2018), pp. 5-14, 10.3348/kjr.2018.19.1.5 ↗

View in Scopus ↗ Google Scholar ↗

MacDougall, 2003 L.D. MacDougall

Mammary fibroadenomatous hyperplasia in a young cat attributed to treatment with megestrol acetate [Hyperplasie mammaire fibroadénomateuse chez un jeune chat soumis à un traitement à l'acétate de mégestrol acétate

Can. Vet. J., 44 (2003), pp. 227-229

View in Scopus ↗ Google Scholar ↗

Martí, 2009 Martí, J.A., 2009. Clinical aspects of mammary disease in the bitch and queen, in: Proceeding of the SEVC Southern European Veterinary Conference. Barcelona, pp. 1–11.

Google Scholar ↗

Mathews, 2008 K.A. Mathews

Pain management for the pregnant, lactating, and neonatal to pediatric cat and dog

Vet. Clin. North Am. Small Anim. Pract., 38 (2008), pp. 1291-1308, 10.1016/j.cvsm.2008.07.001 ↗

View in Scopus ↗ Google Scholar ↗

Orfanou et al., 2016 D.C. Orfanou, G.C. Fthenakis, V.S. Mavrogianni

Mastitis in lactating bitches, in: 8th International Symposium on Canine and Feline Reproduction ISCFR

Paris (2016), p. 183, 10.1371/journal.pone.0143930 ↗

Google Scholar ↗

Park et al., 2007 C.H. Park, H. Ikadai, E. Yoshida, H. Isomura, H. Inukai, T. Oyamada

Cutaneous toxoplasmosis in a female Japanese cat

Vet. Pathol., 44 (2007), pp. 683-687

CrossRef ↗ View in Scopus ↗ Google Scholar ↗

Payan-Carreira, 2013 Payan-Carreira, R., 2013. Feline Mammary Fibroepithelial Hyperplasia: A Clinical Approach, in: Payan-Careira, R. (Ed.), Insights from Veterinary Medicine. InTech, pp. 215–232. https://doi.org/10.5772/55550.

Google Scholar ↗

Payan-Carreira and Martins-Bessa, 2008 R. Payan-Carreira, A.C. Martins-Bessa

Ultrasonographic assessment of the feline mammary gland

J. Feline Med. Surg., 10 (2008), pp. 466-471, 10.1016/j.jfms.2008.03.006 ↗

View PDF View article View in Scopus ↗ Google Scholar ↗

Prescott, 1972 V.W. Prescott

Neonatal diseases in dogs and cats

Aust. Vet. J., 48 (1972), pp. 611-618

CrossRef ↗ View in Scopus ↗ Google Scholar ↗

Ramsey, 2017 Ramsey, I., 2017. BSAVA Small Animal Formulary, Part A: Canine and Feline, 9th ed. British Small Animal Veterinary Association, Cardiff.

Google Scholar ↗

Refai, 1988 M. Refai

Mastitis, aetiology, diagnosis and control

J. Egypt. Vet. Med. Assoc., 48 (1988), pp. 521-545

Google Scholar ↗

Root Kustritz, 2010 Root Kustritz, M.V., 2010. How do I treat mastitis in a nursing queen?, in: Root Kustritz, M. V. (Ed.), Clinical Canine and Feline Reproduction Evidence-Based Answers. Wiley-Blackwell, Hoboken, NJ, USA, pp. 249–250.

Google Scholar ↗

Roudebush and Wheeler, 1979 P. Roudebush, K. Wheeler

Peracute gangrenus mastitis in a cat

Feline Pr., 9 (1979), pp. 35-38

Google Scholar ↗

Schäfer-Somi, 2018 Schäfer-Somi, S., 2018. Mastopathies in aged bitches and queens, in: XIV Congress „Problems in Reproduction of Small Animals – Fertility, Pregnancy and Neonate". Wrocław.

Google Scholar ↗

Seweryn and Tworus, 2012 T. Seweryn, A.K. Tworus

Diagnostyka ultrasonograficzna ciąży u psów i kotów [Ultrasonography pregnancy diagnosis in dogs and cats]

Zycie Weter., 87 (2012), pp. 206-212

Google Scholar ↗

Tawfik et al., 2020 M. Tawfik, S. Oda, A. Khafaga

Pathological study of skin disorders in dogs and cats at Alexandria Governorate, Egypt

Alex. J. Vet. Sci., 65 (2020), pp. 66-75, 10.5455/ajvs.93531 ↗

Google Scholar ↗

Trasch and Wehrend, 2008 K. Trasch, A. Wehrend

Klinische, sonographische und mikrobiologische untersuchungen bei hündinnen mit akuter mastitis [Clinical, ultrasonographical and microbiological investigations in inflamed canine mammary glands]

Tierärztl. Prax., 36 (2008), pp. 191-199, 10.1017/CBO9781107415324.004 ↗

View in Scopus ↗ Google Scholar ↗

Trasch et al., 2007 K. Trasch, A. Wehrend, H. Bostedt

Ultrasonographic description of canine mastitis

Vet. Radiol. Ultrasound, 48 (2007), pp. 580-584, 10.1111/j.1740-8261.2007.00301.x ↗

View in Scopus ↗ Google Scholar ↗

Vasiu et al., 2020 I. Vasiu, R. Dąbrowski, A. Tvarijonaviciute

Lactation-related mammary gland pathologies-a neglected emergency in the bitch

Reprod. Domest. Anim., 56 (2020), pp. 208-230, 10.1111/rda.13866 ↗

Google Scholar ↗

Vasiu et al., 2021 I. Vasiu, G. Meroni, R. Dąbrowski, P.A. Martino, A. Tvarijonaviciute, M. Bochniarz, R.A. Pop, F.G. Brudaşcă, N.I. Fiţ

Aerobic Isolates from Gestational and Non-Gestational Lactating Bitches (Canis lupus familiaris)

Animals, 11 (2021), pp. 1-15, 10.3390/ani11113259 ↗

Google Scholar ↗

Wallace, 1994 M.S. Wallace

Management of parturition and problems of the periparturient period of dogs and cats

Semin. Vet. Med. Surg., 9 (1994), pp. 28-37

View in Scopus ↗ Google Scholar ↗

Wiebe and Howard, 2009 V.J. Wiebe, J.P. Howard

Pharmacologic advances in canine and feline reproduction

Top. Companion Anim. Med., 24 (2009), pp. 71-99, 10.1053/j.tcam.2008.12.004 ↗

View PDF View article View in Scopus ↗ Google Scholar ↗

Wilson, 2013 C.R. Wilson

Feline gangrenous mastitis [Mammite nécrotique féline]

Can. Vet. J., 54 (2013), pp. 292-294

View in Scopus ↗ Google Scholar ↗

Zachary, 2017 Zachary, F.J., 2017. Pathologic Basis of Veterinary Disease, 6th ed. Elsevier Inc., St. Louis.

Google Scholar ↗

Zambelli, 2012 Zambelli, D., 2012. Reproductive Physiology of Feline Pregnancy and Parturition and Conditions of the Periparturient Period, in: Lopate, C. (Ed.), Management of Pregnant and Neonatal Dogs, Cats, and Exotic Pets. Wiley-Blackwell, Hoboken, NJ, USA, pp. 43–54.

Google Scholar ↗

Cited by (0)