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

Uri Burstyn, bvSc

Case Description—A 1-year-old sexually intact female domestic shorthair cat was evaluated because of an 8-week history of pronounced mammary gland hyperplasia that had progressed to mastitis and abscessation of the mammary glands since parturition 7 days earlier. The cat was anorectic, was febrile, and had signs of discomfort. Its kittens were weak and appeared to have difficulty nursing.

Clinical Findings—Physical examination revealed pyrexia, mastitis with abscessation in the 6 caudal mammary glands, skin ulceration over the nipples, and areas of skin necrosis over the abscessed mammary glands. A CBC revealed nonregenerative anemia and leukocytosis with a left shift (2.160 × 10⁹ band cells/L) and toxic changes. Mastitis and incipient septicemia were considered the most likely causes. The history of mammary gland hyperplasia since the second week of pregnancy suggested a diagnosis of fibroadenomatous hyperplasia that predisposed the cat to subsequent mastitis.

Treatment and Outcome—Surgical drainage of the abscessed mammary glands, debridement of necrotic skin, and placement of a Penrose drain resulted in rapid improvement in clinical status. Broad-spectrum antimicrobial treatment (amoxicillin-clavulanic acid) was prescribed, and the cat was discharged from the hospital. Mastitis and fibroadenomatous mammary gland hyperplasia resolved rapidly afterward.

Clinical Relevance—Management of abscessed mammary glands through surgical drainage and drain placement is an option for treatment of cats with complications of fibroadenomatous hyperplasia. In the cat of this report, the treatment approach resulted in rapid resolution of mastitis, was less invasive than mastectomy, and avoided the potential complications of treatment with a progesterone-receptor antagonist. (J Am Vet Med Assoc 2010;236:326-329)

A 1-year-old sexually intact female domestic shorthair cat weighing 3.6 kg (7.92 lb) was evaluated because of a 5-day history of grossly enlarged mammary glands, difficulty nursing, anorexia, and signs of progressive discomfort. The cat had delivered 3 kittens 7 days earlier. During the first 1 or 2 weeks of the cat’s pregnancy, the owner had noticed the mammary glands were much larger than usual and were firm and cool to the touch. Ulceration of the skin covering the glands was noticed in late pregnancy. After parturition, the mammary glands changed in shape and increased in size, and the area of skin ulceration also grew. The kittens reportedly appeared bright, alert, and responsive, but membranes and mildly delayed skin-tent response. Two of the kittens appeared bright, alert, and responsive, but the third was weak with obtunded mentation. Physical examination of the cat revealed mild tachypnea (60 breaths/min; reference limits, 16 to 40 breaths/min) and hyperthermia (rectal temperature, 41.3°C [106.3°F]; reference limits, 38.2° to 38.8°C [100.8° to 101.8°F]).

When admitted to the hospital, the cat appeared thin (body condition score, 3/9) and was judged as 5% to 7% dehydrated on the basis of tacky oral mucus membranes and mildly delayed skin-tent response. Two of the kittens appeared bright, alert, and responsive, but the third was weak with obtunded mentation. Physical examination of the cat revealed mild tachypnea (60 breaths/min; reference limits, 16 to 40 breaths/min) and hyperthermia (rectal temperature, 41.3°C [106.3°F]; reference limits, 38.2° to 38.8°C [100.8° to 101.8°F]).

From the Vancouver Animal Emergency Clinic, 1590 W 4th Ave, Vancouver, BC V6J 1L7, Canada.

Address correspondence to Dr. Burstyn (uburstyn@gmail.com).

Abbreviation

<table>
<thead>
<tr>
<th>FAH</th>
<th>Fibroadenomatous hyperplasia</th>
</tr>
</thead>
</table>

Systemic arterial blood pressure was 118 mm Hg as determined by use of Doppler ultrasonography.

All other findings were unremarkable except for the mammary glands. All 8 mammary glands were enlarged: both inguinal, all 4 abdominal (caudal and cranial), and both thoracic glands. The inguinal mammary glands were the most severely affected and measured 10 to 12 cm in diameter. The cranial and caudal abdominal glands were asymmetric and measured 8 to 10 cm in diameter. The thoracic mammary glands were also asymmetric, with the diameters of the left and right glands measuring 3 to 4 cm and 2 to 3 cm, respectively. The inguinal and cranial and caudal abdominal glands had prominent dilated veins and were firm, fluctuant, and hot to the touch. The skin of the mammary glands was markedly stretched and had a thin, fragile appearance. Multiple pockets of fluid could be seen and palpated through the skin, and skin ulceration was evident around the nipples. An oval area of skin approximately 5 cm long and overlying the right caudal abdominal mammary gland was black with purple edges and was hard and cool to the touch. A 2-cm area of skin with a similar appearance was evident over the right inguinal mammary gland. The right thoracic mammary gland
was normal in appearance, whereas the left thoracic mammary gland was mildly enlarged in comparison. Signs of discomfort were evident during ambulation and during palpation of the mammary glands.

Examination of a lateral survey radiograph disclosed a homogeneous fluid opacity in the 3 caudal pairs of mammary glands. The body wall appeared intact, and no radiographic abnormalities were detected in the thorax and abdomen. A blood sample was collected and submitted to a private laboratory for a CBC and serum biochemical analysis. Results of the CBC included leukocytosis (18.0 × 10³ WBCs/mL; reference limits, 4.0 × 10³ WBCs/mL to 16.0 × 10³ WBCs/mL) and neutrophilia (13.86 × 10³ cells/mL; reference limits, 2.70 × 10³ cells/mL to 10.56 × 10³ cells/mL), with an increase in the number of band cells (2.16 × 10³ cells/mL; reference limits, 0.00 × 10³ cells/mL to 0.10 × 10³ cells/mL) with moderate toxic change as well as lymphopenia (1.08 × 10³ cells/mL; reference limits, 1.77 × 10³ cells/mL to 6.56 × 10³ cells/mL). The cat was anemic (4.6 × 10¹¹ RBCs/mL; reference limits, 6.3 × 10¹¹ RBCs/mL to 10.8 × 10¹¹ RBCs/mL), with a low Hct value (23%; reference limits, 32% to 99%), low hemoglobin concentration (70 g/L; reference limits, 104 to 160 g/L), and low mean RBC hemoglobin concentration (303 g/L; reference limits, 306 to 352 g/L). A few Döhle bodies were detected, and moderate echinocytosis was evident, with a reticulocyte count < 1%. Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentration (55 µmol/L; reference limits, 28 to 76 µmol/L). Concentration of biochemistry revealed a low serum creatinine (832 µmol/L; reference limits, 71 to 203 µmol/L). Platelets were clumped but adequate. Results of serum biochemical analysis revealed a low serum creatinine concentra...
Discussion

Fibroadenomatous hyperplasia, also described as fibroepithelial hyperplasia, is a progesterone-induced proliferation of the epithelium of mammary gland ducts and benign stromal proliferation that is manifested as marked, generalized enlargement of 1 or more mammary glands.\(^2,3\) The condition reportedly develops in young queens during early pregnancy, pseudopregnancy, or after receipt of progestogen injections. It can also develop in male cats treated with progesterone, particularly megestrol acetate.\(^1,4\) Results of studies\(^3,4\) in dogs suggest that progesterone stimulates a local release of growth hormone and insulin-like growth factors in mammary glands, which can cause tissue hyperplasia. The age of the cat reported here, its reproductive status, and its history of developing a marked degree of mammary hyperplasia in the first 2 weeks of pregnancy are all consistent with a diagnosis of FAH.

Mammary hypertrophy in early pregnancy with no corresponding clinical signs is not characteristic of mastitis. Mastitis is an uncommon condition in cats, usually developing in the peri-parturient period and often developing in association with trauma to the mammary glands.\(^7\) Common clinical signs include swollen, firm mammary gland tissue; ulceration; arexia; cachexia; pyrexia; abnormal glandular discharge; and hungry or weak kittens. In cats with severe mammary hypertrophy, abscessed mammary glands can develop.\(^7,8\) Skin ulceration secondary to FAH has been described\(^9,14\) and is likely to have contributed to the development of mastitis in the cat reported here. The fact that the kittens were only able to nurse at the thoracic mammary glands suggested that release of milk was also affected. Skin trauma and altered lactation create an ideal environment for an ascending infection.\(^8\)

Exogenous progesterone administration in cats has been implicated in the development of mammary neoplasia as well as FAH.\(^9\) Therefore, it is important to recognize that FAH is a clinical syndrome caused by an exaggerated tissue response to circulating hormones rather than the loss of regulation of cell growth that characterizes neoplasia.\(^1,2,3,11\)

Two approaches to managing FAH have been described: control of underlying hormonal stimulation and ablation of the target tissues. Medical treatment with the progesterone-receptor antagonist aglepristone has been used successfully to resolve the clinical signs of FAH. In 2 studies, 7 of 7 cats\(^13\) and 21 of 22 cats\(^13\) with FAH had improvement after 1 to 4 weeks of SC administration of aglepristone. The incidence of reported adverse effects was low and included transient pruritus at the injection site, abortion in pregnant queens, and sudden death attributed to acute heart failure and pulmonary arterial thrombus (1 cat). Two cats were pregnant at the time of treatment and aborted their litters 2 to 5 days after the first injection. Both cats subsequently developed endometritis secondary to incomplete emptying of the uterus but were successfully treated with ovariohysterectomy.\(^2\) Fibroadenomatous hyperplasia can redevelop when aglepristone treatment is discontinued but progestin treatment is continued,\(^13\) suggesting a multimodal approach may be needed to achieve permanent resolution of this condition. Because of the role of progestins in the development of FAH and the progestin-sensitizing effect of estrogen on mammary tissues,\(^2\) ovariohysterectomy has been recommended as a stand-alone treatment or as an adjunct to other approaches. However, data regarding the effectiveness of ovariohysterectomy as a stand-alone treatment are conflicting. Resolution of clinical signs after ovariohysterectomy has been reported,\(^2,3,4\) but results of other studies\(^1,3,13\) suggest ovariohysterectomy is not effective on its own.

Removal of the affected mammary glands by mastectomy has been recommended as the surgical treatment of choice for FAH,\(^1,3,14\) but redevelopment of FAH in untreated glands has been reported.\(^2,15\) The condition can also spontaneously regress in some situations,\(^2\) although the reason for spontaneous regression has not been identified. These findings suggest that the removal of the underlying source of progesterone is important in achieving long-term resolution of FAH.

In the cat reported here, a marked improvement in clinical status was achieved within 12 hours after admission by following the basic principles of abscess management (ie, drainage, debridement, and appropriate antimicrobial treatment). Ideally, choice of antimicrobials should be based on the results of bacterial culture and antimicrobial susceptibility testing, but in cats with impending sepsis, empirical selection of an antimicrobial is warranted until laboratory results are received.\(^8\) The cat's owner had financial concerns that precluded performance of bacterial culture and susceptibility testing, so a broad-spectrum antimicrobial\(^8\) with a label claim for abscess treatment was used.

Resolution of the clinical signs of FAH after discharge from the hospital may have been attributable to a combination of 2 factors: disruption of mammary tissue by severe abscessation and change in the cat's hormonal status after parturition. Given the limited understanding of this disease, it is impossible to predict the degree to which either of these factors contributed to resolution of FAH or the probability of redevelopment. Ovariohysterectomy and subsequent cessation of progesterone administration were recommended to minimize the risk of recurrence.\(^13\) In the author's opinion, it is inadvisable to use a queen that has had FAH for breeding purposes because of the possibility of disease recurrence and because there may be a heritable component to the condition. To the best of the author's knowledge, no studies have been conducted to examine the heritability of FAH, which would be an interesting topic for investigation.

The method of managing abscessation and mastitis secondary to FAH reported here led to a rapid improvement of the clinical status in the affected cat and prevented further progression to sepsis. Treatment by surgical drainage may be used as a less invasive alternative to mastectomy in managing queens with mastitis and abscessation secondary to FAH.

---

a. Sandoz Canada, Boucherville, QC, Canada.
b. Normosol-R, Baxter, Mississauga, ON, Canada.
c. Abbott Laboratories, Vancouver, BC, Canada.
d. Covidiem, St-Laurent, QC, Canada.
e. Prolene, Johnson & Johnson, Calgary, AB, Canada.
f. Clavamox, Pfizer Animal Health, Pointe Claire, QC, Canada.
g. Baytril, Bayer, Toronto, ON, Canada.
References

From this month’s AJVR

Evaluation of diffusion of triamcinolone acetonide from the distal interphalangeal joint into the navicular bursa in horses
Mary Boyce et al

Objective—To determine whether triamcinolone acetonide diffuses from the distal interphalangeal joint (DIPJ) to the navicular bursa, diffusion is direct or systemic, and addition of sodium hyaluronan has an effect on diffusion in horses.

Animals—11 adult horses without forelimb lameness.

Procedures—1 randomly chosen forelimb DIPJ of each horse received an injection of 10 mg of triamcinolone acetonide plus 20 mg of sodium hyaluronan (group 1), and the contralateral forelimb DIPJ received an injection of 10 mg of triamcinolone acetonide plus 2 mL of lactated Ringer’s solution (group 2). Synovial fluid samples were taken from both forelimb navicular bursae and 1 hind limb navicular bursa (systemic control group) at 6 hours. Triamcinolone acetonide concentrations in synovial fluid were quantified by use of high-performance liquid chromatography plus tandem mass spectrometry. Data were logarithmically transformed, and contrast analysis was performed on the 3 groups.

Results—Triamcinolone acetonide was detected in navicular bursal samples in all groups. Groups 1 and 2 had significantly greater concentrations of triamcinolone acetonide than the systemic control group. There was no significant difference between groups 1 and 2.

Conclusions and Clinical Relevance—Triamcinolone acetonide diffused directly from the DIPJ into the navicular bursa in clinically normal horses, and diffusion was not affected by addition of hyaluronan. Injection into the DIPJ with triamcinolone acetonide or a triamcinolone acetonide–hyaluronan combination can potentially be used for treatment of navicular syndrome, but further studies are needed to determine whether triamcinolone acetonide diffuses similarly in horses with navicular syndrome. (Am J Vet Res 2010;71:169–175)