Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

SMALL MAMMALS: RATS

Chromodacryorrhea

BASIC INFORMATION

DEFINITION

Porphyrin-pigmented tears secreted by the harderian glands of rats. Chromodacryorrhea literally means "excessive production of colored tears" (*chromo* Gk = color; *dacryo* Gk = gland; *rhea* = to pour out).

SYNONYM

Red tears

EPIDEMIOLOGY

SPECIES, AGE, SEX Besides rats, red-pigmented harderian gland secretions are seen in certain strains of inbred mice (e.g., C3H, A, I, JK, C57 mice), Syrian hamsters, Chinese hamsters *(Cricetulus griseus),* and deer mice *(Peromyscus leucopus).* Old and sick rats are most commonly affected.

RISK FACTORS

- Stress
- Overcrowding
- Poor husbandry

CONTAGION AND ZOONOSIS Sialodacryoadenitis virus (SDAV) can directly affect the harderian glands.

ASSOCIATED CONDITIONS AND DISORDERS Pain, stress, systemic infection (*Mycoplasma pneumoniae*, SDAV). Any disease that leads to depression and reduced grooming. Chronic physiological stress in rats is likely to cause chromodacryorrhea.

CLINICAL PRESENTATION

HISTORY, CHIEF COMPLAINT

- Sudden onset of red staining around the eyes and nostrils
- Labored breathing
- Reduced appetite
- Lethargy
- Recent purchase from a pet store

PHYSICAL EXAM FINDINGS

- Red staining around the eyes and nostrils, and occasionally the forepaws (from wiping the nares)
- Usually clinical signs are associated with nutritional deficiencies, chronic physiologic stress (e.g., disease), chronic light exposure, or dacryoadenitis.

ETIOLOGY AND PATHOPHYSIOLOGY

- Any disease or condition that results in chronic stress will result in chromodacryorrhea.
- Harderian glands of rodents with "red tears" exhibit a variety of histological autofluorescence patterns. In addition, their secretions are also affected by protoporphyrin binding to lipids, affecting fluorescence.
  - Inflammation of the harderian gland (i.e., dacroadenitis) causes an increase in secretions. The most common cause is infection with SDAV, a coronavirus of rats.
  - The tears are secreted via activation of the parasympathetic nervous system via muscarinic receptors. Anticholinergic drugs have been shown to block secretions.
  - Harderian glands' secretions predominantly contain lipids that act as pheromones. The presence of porphyrins in Harderian gland secretions is more the exception than the rule when describing these secretions in rodents. Generally, porphyrins give color to secretions.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Epistaxis
- Conjunctivitis

INITIAL DATABASE

- Wood's lamp examination reveals bright orange-red fluorescence; allows differentiation from dried blood
- Further diagnostics will dependent on the clinical signs and suspected primary underlying disease, such as respiratory tract disease (see Respiratory Tract Disease, Acute, and Respiratory Tract Disease, Chronic).

TREATMENT

THERAPEUTIC GOAL

Address specific underlying cause if known.

ACUTE GENERAL TREATMENT

- Depends on the underlying primary cause (e.g., nutritional deficiency, respiratory disease)
- If due to SDAV, clinical signs will persist for 1 week, then will resolve spontaneously. Mortality is low.

CHRONIC TREATMENT

- Maintain proper husbandry.

- Ensure proper diet.
- Minimize stress.

RECOMMENDED MONITORING

If clinical signs persist longer than 1 week and no specific underlying cause can be identified, recommend recheck appointment for further diagnostics.

PROGNOSIS AND OUTCOME

Prognosis depends on underlying cause. If clinical signs are due to stress or husbandry, the prognosis is excellent if properly addressed. For other causes, the prognosis will vary from poor to good.

PEARLS & CONSIDERATIONS

COMMENTS

Many clients will present in distress because their pet is "bleeding from the eyes." They will be relieved to know that their pet is not actually bleeding but will need to understand that this clinical sign can be an indicator of a greater underlying disease that warrants investigation.

CLIENT EDUCATION

Chromodacryorrhea is not an actual disease in most cases but an indicator for an underlying problem or stress.

SUGGESTED READING

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CROSS-REFERENCES TO OTHER SECTIONS

Respiratory Tract Disease, Acute
Respiratory Tract Disease, Chronic

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SMALL MAMMALS: RATS

Mammary and Pituitary Tumors

Client Education Sheet Available on Website

BASIC INFORMATION

DEFINITION

Mammary gland tumors are the most frequently occurring tumors in female rats. Histologically, most are mammary fibroadenomas, although adenocarcinomas are also seen.

SPECIAL SPECIES CONSIDERATION

Rats have 12 mammary glands along the mammary chain, which extends from the cervical region to the tail base. Mammary tumors can arise in any of these locations.

EPIDEMIOLOGY

SPECIES, AGE, SEX

- Older animals are most frequently affected (>1 year of age).
- Females are at higher risk than males; an incidence of 2% to 16% has been reported experimentally in male rats.

GENETICS AND BREED PREDISPOSITION In inbred rat strains susceptible to mammary tumors expression levels of several prolactin-regulated genes are significantly elevated (e.g., messenger RNA's encoding prolactin and its cell surface receptor are amplified) indicating the presence of increased prolactin signaling in the mammary glands of mammary tumor susceptible rat strains.

RISK FACTORS

- Sex: females at higher risk than males
- Age: higher risk at greater than 2 years of age
- Nutrition: increased incidence with high-fat diets, reduced incidence with food restriction
- Prolactin-secreting pituitary tumors: increased risk of mammary tumors
- Neuter status: decreased risk if ovariectomized by 90 days of age; suspected to also have decreased risk even if ovariectomized after 90 days of age, but this has not been proven.
- The frequency of mammary tumors and pituitary tumors is significantly lower in 18- to 24-month-old ovariectomized (4%) versus sexually intact (mammary tumors, 49%; pituitary tumors, 59%) rats. Therefore, the decreased frequency of mammary tumor development could be related to the decreased frequency of prolactin-secreting pituitary tumors.

ASSOCIATED CONDITIONS AND DISORDERS Prolactin-secreting pituitary tumors (see Risk Factors)

CLINICAL PRESENTATION

HISTORY, CHIEF COMPLAINT

- Rapidly growing mass in region of mammary gland tissue
- Bleeding and/or odor if secondarily infected or ulcerated

PHYSICAL EXAM FINDINGS

- Circumscribed, movable, firm, subcutaneous mass in the region of the mammary glands, which extends from the cervical region to the tail base.
- The overlying skin may be ulcerated or infected if the mass is large, or if the surface has been traumatized.

ETIOLOGY AND PATHOPHYSIOLOGY

- As with other species, most mammary gland development occurs during puberty primarily under influence of estrogen and pregnancy under the influence of progesterone and prolactin.
- Neutering sexually immature females removes estrogen influence during mammary growth and prevents mammary epithelial ductal elongation, bifurcation, and extension throughout the fat pad. Inhibition of ductal morphogenesis significantly reduces the risk of mammary tumors by limiting the amount of mammary tissue that develops.
- Estrogen is an important stimulator of prolactin secretion that acts directly on pituitary lactotrophs and via the hypothalamus. Experimentally prolactinomas can be induced in rats by chronic estrogen administration. In the mammary gland, prolactin stimulates alveolar epithelial proliferation with its fibrous connective tissue support structure.
- Aging female rats exhibit changes in estrous cycle and reproductive patterns. At 10-12 months of age, the once-regular ovulatory cycles gradually become lengthened and irregular and eventually develop into a prolonged period of constant estrus characterized by ovaries containing big follicles that secrete large quantities of estrogen. Neutering sexually mature females removes constant estrogen secretion and decreases prolactin secretion so benign mammary tumors either do not develop or do not increase in size.
- Female rats with benign mammary tumors have 27 times higher plasma levels of prolactin than 6-month-old virgin rats, and prolactin levels similar

to that of rats on the seventh day postpartum. Because of this, rats with prolactin-secreting pituitary tumors are at increased risk of mammary tumor development.
- In aging rats, prolactin secretion is increased and is reflected in the high blood prolactin level in both sexes. This change is due to a reduction of hypothalamic dopamine activity. The escape from hypothalamic inhibitory control leads to lactotroph hyperplasia and a high incidence of prolactin cell adenomas in old rats.
- Gene expression of spontaneous fibroadenomas and adenocarcinomas compared to a normal rat mammary gland in the same developmental state has shown that fibroadenomas do not progress to adenocarcinoma.
- Adenocarcinomas arise de-novo (i.e., without prior adenoma stage) and represent fewer than 10% of mammary tumors in pet rats.
- Fibroadenomas can reach 8-10 cm in diameter and do metastasize.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Dermal/subcutaneous abscess (e.g., from bite wounds, foreign body penetration)
- Neoplasia (e.g., lymphoma)
- Mastitis

INITIAL DATABASE

- Fine-needle aspirate: caution during interpretation because large mammary tumors may be necrotic and can be difficult to differentiate from an abscess on cytology
- Blood work: complete blood count and serum biochemistry screening as a preoperative workup
- Thoracic radiographs/CT: preoperative workup if underlying respiratory disease is suspected

ADVANCED OR CONFIRMATORY TESTING

Histopathologic examination

TREATMENT

THERAPEUTIC GOALS

- Stabilization of the patient if septic or has suffered blood loss
- Complete surgical removal of the tumor (mastectomy) and prevention of recurrence

ACUTE GENERAL TREATMENT

- If necessary, stabilization of the patient if mammary tumor is infected/sepsis is present, or if blood loss has ensued from ulceration of the mass.
- Complete surgical removal of the tumor and any ulcerated/infected skin or tissue is the treatment of choice.
  - Because tumors may be quite large, closure of dead space is important to prevent seroma formation.
  - Some masses are difficult to remove if they are in close association with the vulva.
- Concurrent ovariectomy is recommended if patient is stable.

CHRONIC TREATMENT

Because recurrence of mammary tumors at different locations can frequently occur, repeated surgical removal might be necessary.

POSSIBLE COMPLICATIONS

Rats are notorious for mutilation of surgical sites, so appropriate pain medication with monitoring is compulsory. Use of an E-collar is necessary in some cases to prevent mutilation.

RECOMMENDED MONITORING

Tumors are likely to recur in other mammary glands in both male and female rats, especially if ovariectomy is not performed at the same time. Constant monitoring and palpation of the mammary glands are important.

PROGNOSIS AND OUTCOME

- In general, survival following mastectomy is good.
- Quality of life is improved post mastectomy; however, controversy continues over whether tumor removal actually prolongs survival time.
- Death can occur with sepsis or blood loss if the mammary mass is not surgically removed and becomes ulcerated or infected.

CONTROVERSY

- To date, the only proven treatment and prevention of mammary tumor development consists of surgical removal of the tumor and ovariectomy. Other treatments have been discussed but have not proven to be effective in preventing recurrence of spontaneous tumors or in decreasing their size once present.
- Cabergoline is a prolactin inhibitor that suppresses pituitary prolactin secretion and can be given orally. It has been successfully used in the palliative treatment of a pituitary adenoma in a rat at a dose of 0.6 mg/kg PO q 72 h, and thus may be helpful in rats that have mammary tumor development secondary to prolactin-secreting pituitary tumors and in those unable to undergo ovariectomy.
- Gonadotropin-releasing hormone (GnRH) agonists
  - Deslorelin implants (4.7 mg) have been used experimentally to suppress estrus in rats for 1 year and may be useful in rats that cannot be ovariectomized.
  - Leuprolide acetate has been experimentally shown to suppress the ability of the pituitary-gonadal system to secrete gonadotropin and testosterone for over 5 weeks; similar to deslorelin because it may be useful in rats that cannot undergo ovariectomy
- Melatonin induces apoptosis of rat prolactin-secreting tumors. Experimentally, SC melatonin administration in experimentally induced tumor-bearing rats significantly increased survival time and reduced prolactin levels but did not change the mammary tumor growth rate.
- Tamoxifen: antiestrogen used in the treatment of human breast cancer. This agent would be useful only in mammary adenocarcinomas that are estrogen receptor positive. This drug is NOT recommended, given the low incidence of adenocarcinoma in rats and the fact that it has been shown to induce hepatic cancer and proliferation of the rat uterus.

PEARLS & CONSIDERATIONS

COMMENTS

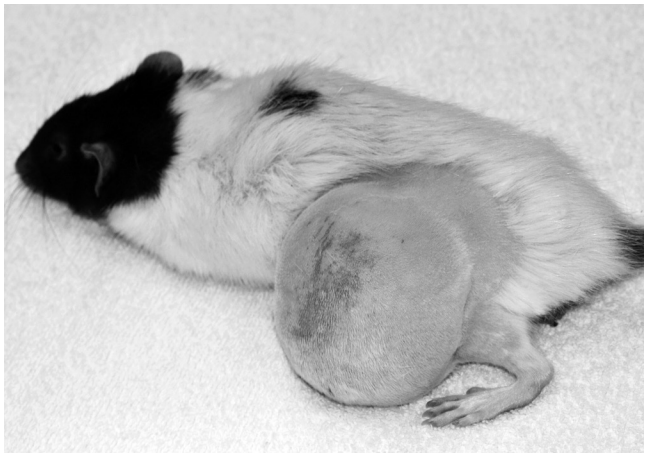
Histopathologic examination of removed tumors should be performed because spontaneous mammary adenocarcinoma has a 5%-10% incidence. Individual genetic variability and environmental factors such as nutrition and maternal effects in utero and during lactation most likely affect quantitative trait loci (QTL) that control susceptibility to mammary adenocarcinoma. However, the individual genetic traits and QTL that influence gene function have not yet been elucidated.

PREVENTION

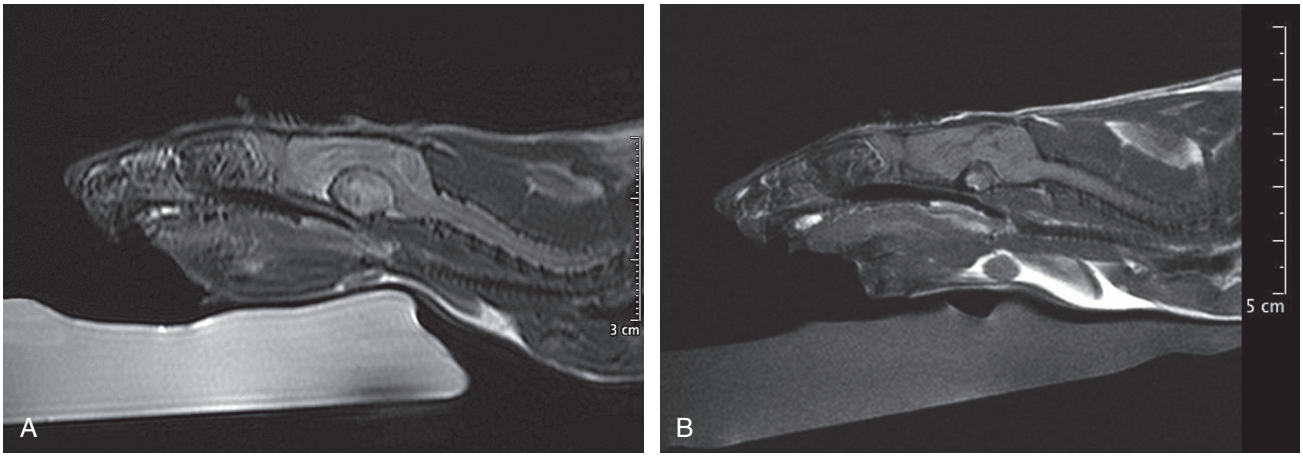
- Ovariectomy of female rats by 90 days of age
- Treatment of prolactin-secreting pituitary tumors

CLIENT EDUCATION

- All clients who own female rats should be educated about the importance of ovariectomy before 90 days of age.
- Additionally, clients should be educated on the importance of early detection and removal of tumors before they become too large to be safely removed surgically.



Mammary and Pituitary Tumors Large mammary fibroadenoma on a female rat. Note the close proximity to the left hind leg which impeded normal ambulation.



Mammary and Pituitary Tumors A, An MRI scan of the head of a 3-year-old rat showing a large pituitary tumor within the brain. **B,** An MRI scan of the same rat shown in A eight weeks after treatment with cabergoline. Note the significant shrinking of the tumor. *(Photo courtesy Jörg Mayer, The University of Georgia, Athens.)*

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SMALL MAMMALS: RATS

Renal Disease

BASIC INFORMATION

DEFINITION

Kidney or *renal disease* is a general term that describes any damage that reduces the functioning of the kidney.

SYNONYMS

- Kidney disease: renal disease, kidney disease, kidney failure, chronic renal failure, nephropathy, suppurative pyelonephritis, suppurative nephritis
- Chronic progressive nephrosis in rats: progressive renal disease in rats, progressive glomerulonephrosis, old rat nephropathy, glomerulosclerosis
- Nephrocalcinosis: renal tubular mineralization

EPIDEMIOLOGY

SPECIES, AGE, SEX

- Male rats develop a more severe form of chronic progressive nephrosis, usually earlier in life than females; lesions are more severe in rats over 12 months of age.
- Nephrocalcinosis is more common in females and can be found in animals as young as 7 weeks of age. Blood estrogen levels may play a role in that the disease can be prevented by ovariectomy and is induced in castrated male and female rats by estrogen administration.

GENETICS AND BREED PREDISPOSITION A significantly higher prevalence of chronic progressive nephrosis is seen in the Sprague-Dawley strain of rat. Osborne-Mendel and Buffalo strains are relatively insusceptible. Elevated prolactin levels are suspected of contributing to more severe disease.

RISK FACTORS

- Chronic progressive nephrosis: high-protein diets designed for superior body growth result in earlier onset of more severe disease
- Nephrocalcinosis: may be the result of a number of dietary factors, including magnesium deficiency, elevated dietary phosphorus or calcium, and diet preparations with a low calcium-to-phosphorus ratio
- Suppurative pyelonephritis/nephritis: isosthenuria, urolithiasis, and lower urinary tract infections

CLINICAL PRESENTATION

DISEASE FORMS/SUBTYPES

- Chronic progressive nephrosis
- Nephrocalcinosis
- Suppurative pyelonephritis/nephritis

HISTORY, CHIEF COMPLAINT

- Clinical signs vary with severity of kidney pathology:
  - Polydipsia and polyuria
  - Anorexia
  - Weight loss
  - Lethargy

PHYSICAL EXAM FINDINGS

- In any rat with renal disease, morbidity may vary from slight to none to significant, depending on the progression and severity of disease.
  - Lethargy
  - Weight loss
  - Cachexia
  - Dehydration
  - Poor fur quality
  - Abdominal pain
  - Diarrhea
  - Hypertension

ETIOLOGY AND PATHOPHYSIOLOGY

- Chronic progressive nephrosis
  - A high protein diet may acutely increase the glomerular filtration rate, possibly causing intraglomerular hypertension, which would lead to progressive loss of renal function.
  - Albuminuria not only serves as a marker of glomerular injury but is also associated with tubulointerstitial injury.
- Nephrocalcinosis
  - In rats, no single mechanism has been identified that explains the association between all dietary factors that have been related to the prevalence of nephrocalcinosis.
  - Nutritional studies have shown that diets high in phosphorus or low in calcium, with a net Ca:P molar ratio of less than 1.0, contribute to the development of nephrocalcinosis lesions. Increasing the calcium and phosphorus content and the Ca:P ratio to greater than 1.0 and closer to 1.3 markedly decreased the incidence and severity or prevented the occurrence of nephrocalcinosis lesions.
- Suppurative pyelonephritis/nephritis
  - Caused by various predominantly gram-negative bacterial organisms (e.g., *Pseudomonas*, *Escherichia coli*, *Proteus mirabilis*), which usually ascend to renal pelvis from lower urinary tract.
  - Chronic pyelonephritis is more common and frequently is clinically inapparent.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Hydronephrosis
- Neoplasia
- Polycystic kidney disease
- Calculi-associated obstructive disease
- Toxic nephrosis
- Ischemic injury

INITIAL DATABASE

- Urinalysis
  - Isosthenuria (normal specific gravity, 1.022-1.050)
  - Proteinuria (mild proteinuria is normal in rats)
  - Hematuria
  - Sediment analysis: casts, crystals, inflammatory or neoplastic cells, bacteria
- Urine culture
- Complete blood count: may be normal
  - Nonregenerative anemia
  - Leukocytosis
- Serum biochemistry profile
  - BUN elevation
  - Creatinine elevation
  - Hyperphosphatemia
  - Hypocalcemia or hypercalcemia
  - Hypokalemia or hyerpkalemia
  - Hypercholesterolemia
  - Hypoproteinemia
- Diagnostic imaging
  - Radiography: assess for increases or decreases in kidney size, radiopaque calculi within the urinary tract, abdominal masses associated with the urinary tract, and bladder distention.
  - Ultrasonography: discern size, contour, and texture of the kidneys, allowing for differentiation of focal versus diffuse disease

ADVANCED OR CONFIRMATORY TESTING

- Perform ultrasound-guided fine-needle aspiration for cytology
- Contrast urography
- Histopathologic examination

TREATMENT

THERAPEUTIC GOALS

- Delay progression of renal disease.
- Preserve overall patient well-being and quality of life.
- Promote diuresis and diminish the consequences of azotemia.
- Treat underlying or concurrent urinary tract infection,

ACUTE GENERAL TREATMENT

- Discontinue any potentially nephrotoxic drugs.
- Identify and treat any prerenal or postrenal abnormalities.
- Identify any treatable conditions such as urolithiasis or pyelonephritis.
- Fluid therapy
  - To induce diuresis and correct azotemia, electrolyte, and acid-base imbalances
  - Use of isotonic crystalloids
  - Subcutaneous administration: 60-100 mL/kg/d
  - Intravenous fluid therapy
    - Use lateral coccygeal or cephalic vein.
    - Maintenance fluids are 3-4 mL/kg/h.
  - Potassium supplementation of fluids based on blood potassium measurement
- Antibiotic therapy
  - Indicated for cases of suppurative pyelonephritis/nephritis and cystitis
  - Antibiotic selection should be based on culture and susceptibility whenever possible.
  - For empirical treatment, or for cases with negative urine culture, despite clinical suspicion, use antibiotics, which are effective against Gram-negative organisms and are renally excreted, to reach high tissue concentrations.
  - Amoxicillin/clavulanic acid 15-20 mg/kg PO q 8-12 h
  - Trimethoprim-sulfa 15-30 mg/kg PO q 12 h

- Enrofloxacin 10-20 mg/kg PO q 12-24 h
- If hyperphosphatemic, alter diet and initiate enteric phosphate binders:
  - Aluminum hydroxide 30-90 mg/kg/d, divided and administered with food
- Treat increased gastric acidity with H2 blockers:
  - Famotidine 0.5 mg/kg PO, SC q 24 h
  - Ranitidine 1-2 mg/kg PO, SC q 12 h
- Multivitamin supplementation is recommended because the excessive amount of urine produced by failing kidneys commonly results in loss of water-soluble vitamins.

CHRONIC TREATMENT

- Maintain long-term dialysis with maintenance subcutaneous fluid therapy (owners can be taught to do this at home): 60-100 mL/kg/d SC.
- Antibiotic therapy for chronic pyelonephritis should be at least 4-6 weeks.
- Dietary management: high protein appears to be the major cause of severe nephropathy, and the term *protein-overload nephropathy* is often used. Changing the source of protein to one such as soy protein, restricting caloric intake, or modifying the diet to decrease protein consumption could decrease the severity of nephropathy. Changing the diet so that the Ca:P ratio is greater than 1.0 and is closer to 1.3 may decrease the incidence and severity of nephrocalcinosis in rats.
- The hyperphosphatemia that occurs in chronic renal failure is closely related to dietary protein intake because protein-rich diets are also high in phosphorus.
- Consider use of omega-3 fatty acid supplements based on studies showing their beneficial effects in other species.

POSSIBLE COMPLICATIONS

- Anorexia
- Gastrointestinal ulceration
- Hyperphosphatemia
- Acidosis
- Anemia

RECOMMENDED MONITORING

- Overall condition and clinical response to therapy should be assessed in all patients with renal disease. Frequency of follow-up assessments varies with initial diagnosis and severity of disease. Periodic assessments for azotemia, anemia and phosphorus, and potassium and protein imbalances are recommended.
- Monitor body weight and condition, and adjust nutrition accordingly.
- Urinalysis and urine culture in patients being treated for pyelonephritis

PROGNOSIS AND OUTCOME

- With any diagnosis of renal insufficiency or failure, prognosis varies with severity of clinical pathologic findings, duration of disease, and severity of primary renal failure. If secondary to infection or obstructive disease, prognosis is determined by duration of the disease process and success in treatment—medical or surgical—of the underlying condition of secondary renal insufficiency.
- Depending on initial diagnosis, disease severity, and response to therapy, quality of life issues and euthanasia should be discussed with the owner in terms of any patient with renal disease.

CONTROVERSY

Hematology, clinical chemistry, and urinalysis values may vary significantly with strain or breed of animal, nutritional status, sex, sampling site or frequency, time of day, stressors, age, health status, drug exposure, and environment. Therefore, normal values are broad; these variables should be kept in mind when interpreting individual animal values.

PEARLS & CONSIDERATIONS

- Many different terms are used to describe renal function and its deterioration.
  - *Azotemia* refers to increased concentrations of urea nitrogen and creatinine and other nonproteinaceous nitrogenous waste products in the blood. *Renal azotemia* denotes azotemia caused by renal parenchymal changes.
  - Uremia is the presence of all urine constituents in the blood. Usually a toxic condition, it may occur secondary to renal failure or postrenal disorders, including urethral blockage.
  - Renal reserve may be thought of as the percentage of "extra" nephrons—those not necessary to maintain normal renal function. Although it probably varies from animal to animal, this value is greater than 50% in most mammals.
  - Renal insufficiency begins when the renal reserve is lost. Animals with renal insufficiency outwardly appear normal, but have a reduced capacity to compensate for stresses such as infection or dehydration and have lost urine concentrating ability.
  - Renal failure is a state of decreased renal function that allows persistent abnormalities (azotemia and inability to concentrate urine) to exist; it refers to a level of organ function rather than a specific disease entity. Acute renal failure generally refers to cases of sudden decline of glomerular filtration rate resulting in an accumulation of nitrogenous waste products and inability to maintain normal fluid balance. Chronic renal failure generally refers to an insidious onset with slow progression (usually months to years) of azotemia and inadequately concentrated urine.
- It is important to realize that most of the renal diseases discussed can manifest as varying stages of compromise in renal reserve, renal insufficiency, or renal failure. If or when the disease process progresses depends on variables such as the specific disease in question, environmental factors, and the individual animal itself.

COMMENTS

- NTP-2000 open formula is one diet available in laboratory medicine that is low in protein (14.0%) and has a Ca:P ratio approximating 1.3:1; it has been found to decrease the incidence of nephrocalcinosis in rats.
- Another laboratory rat diet, AIN-93G, has a lower phosphorus content (0.3%) and a higher Ca:P ratio and has been shown to lower the incidence of nephrocalcinosis.
- Dietary salt content has been found to have an effect on hypertension associated with hydronephrosis in rats. Hydronephrosis as a result of partial ureteral blockage led to increased blood pressure, which worsened significantly on a high-salt diet versus a low-salt diet.
- High levels of dietary soy isoflavones induced nephrocalcinosis formation, depending on the strain of laboratory rat.

CLIENT EDUCATION

Chronic renal failure requires continuous treatment and monitoring. Unless a specific underlying cause is diagnosed and treated successfully, treatment in many cases will be lifelong.

SUGGESTED READINGS

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SMALL MAMMALS: RATS

Respiratory Tract Disease, Acute

BASIC INFORMATION

DEFINITION

Acute bacterial pneumonia in rats is caused by subclinical infection with *Streptococcus pneumoniae* and/or *Corynebacterium kutscheri,* which develops into clinical pneumonia and/or septicemia secondary concurrent infection or immunosuppression.

SYNONYMS

- Pseudotuberculosis *(Corynebacterium kutscheri)*
- Pneumococcal infection *(Streptococcus pneumoniae)*
- Diplococcal infection *(Streptococcus pneumoniae)*

EPIDEMIOLOGY

SPECIES, AGE, SEX Older animals are at increased risk for *C. kutscheri*. Younger animals are at increased risk for *S. pneumoniae*.

RISK FACTORS

- Concurrent infection with *Mycobacterium pulmonis* or CAR bacillus
- Immune suppression

CONTAGION AND ZOONOSIS

- *Corynebacterium kutscheri*
  - Gram-positive bacillus bacteria
  - Transmission probably occurs through direct contact or oronasal exposure
- *Streptococcus pneumoniae*
  - Alpha-hemolytic Gram-positive diplococcal bacteria
  - Transmission probably occurs through direct contact or oronasal exposure
  - Zoonotic potential

ASSOCIATED CONDITIONS AND DISORDERS Chronic respiratory disease (murine respiratory mycoplasmosis)

CLINICAL PRESENTATION

DISEASE FORMS/SUBTYPES With both of these bacterial infections, animals can have no apparent clinical signs or can have severe respiratory disease and/or acute death.

HISTORY, CHIEF COMPLAINT

- Acute death
- Labored breathing
- Sneezing
- Oculonasal discharge
- Lethargy
- Lameness
- Head tilt

PHYSICAL EXAM FINDINGS

- Dyspnea
- Tachypnea
- Cyanosis
- Rales
- Porphyrin epiphora (chromodacryorrhea)
- Nasal discharge—with or without porphyrin staining
- Muffled heart sounds
- Collapse, tachycardia, poor peripheral pulses if septic shock
- Torticollis and/or nystagmus if otitis media present
- Arthralgia if arthritis present

ETIOLOGY AND PATHOPHYSIOLOGY

- Both *S. pneumoniae* and *C. kutscheri* colonize the upper respiratory tract (nasopharynx and tympanic bulla with *S. pneumoniae* and oropharynx, cervical, and submandibular lymph nodes with *C. kutcheri*) and can remain subclinical in the absence of concurrent disease.
- Concurrent infection with other pathogens (see Respiratory Tract Disease, Chronic) and confounding stressors lead to immune suppression, triggering a latent infection to become clinical.
- Suppurative inflammation of the upper respiratory tract is followed by infection of the lower respiratory tract, leading to bronchopneumonia and pleuritis.
- Bacteremia can lead to infection in other organs such as arthritis, meningitis, pericarditis, hepatitis, splenitis, and peritonitis or acute death.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Respiratory signs
  - Congestive heart failure
  - Chronic respiratory disease (if owners have not been aware of respiratory disease)
- Acute death
  - Sepsis from other bacterial infections (e.g., salmonellosis)
- Otitis media
  - Extension of otitis externa

INITIAL DATABASE

- Thoracic radiographs/CT: findings consistent with pulmonary consolidation and/or pleural effusion
- Skull radiographs/CT/MRI: tympanic bullae sclerosis or effusion if otitis media is present
- Serologic testing: *C. kutscheri* (ELISA)
- Complete blood count: neutrophilia, neutropenia if septic
- Serum biochemistry: hypoglycemia if septic
- Brochoalveolar lavage:
  - Cytology, Gram stain
    - *S. pneumoniae*: encapsulated Gram-positive diplococci
    - *C. kutscheri*: slightly curved Gram-positive rods
  - Aerobic culture and sensitivity
- Submandibular lymph node aerobic culture for *C. kutscheri*: caution as nonclinical animals can harbor bacteria in these lymph nodes

ADVANCED OR CONFIRMATORY TESTING

- Histopathologic examination
- *C. kutscheri*: necrotizing and suppurative pulmonary lesions, fibrinopurulent fibrosis with intralesional bacterial colonies that are pathognomonic (diphtheroid appearance of the bacilli with "Chinese letter" configurations)

TREATMENT

THERAPEUTIC GOALS

- Stabilization of the septic patient
- Eradication of the bacterial infection
- Management of concurrent disease (see Respiratory Tract Disease, Chronic)

ACUTE GENERAL TREATMENT

- Oxygen therapy if patient is dyspneic and/or cyanotic
- Fluid therapy: may require intraosseous administration if patient is severely compromised
- Antibiotic therapy should be based on aerobic culture and sensitivity results:
  - *S. pneumoniae*: highly resistant strains are found in humans, so appropriate antibiotic use is extremely important
    - Amoxicillin/clavulanic acid 15-20 mg/kg PO, SC q 12 h
    - Azithromycin 15-30 mg/kg PO q 12 h
  - *C. kutscheri*
    - Amoxicillin/clavulanic acid 15-20 mg/kg PO, SC q 12 h
    - Ampicillin 20-50 mg/kg PO, SC, IM q 12 h
    - Chloramphenicol 30-50 mg/kg PO, SC, IM q 8-12 h
    - Doxycycline 5-10 mg/kg PO q 12 h

CHRONIC TREATMENT

See Respiratory Tract Disease, Chronic.

POSSIBLE COMPLICATIONS

Oral doxycycline should not be given with any dairy products or other products containing calcium because this will decrease its bioavailability.

RECOMMENDED MONITORING

- Patients with severe disease should be hospitalized until they are able to go home on oral medications.
- Patients should be closely monitored for signs of chronic respiratory disease.

PROGNOSIS AND OUTCOME

Little is known about the prognosis of pure acute bacterial pneumonia because co-infection with other respiratory pathogens is common, as are subclinical infections.

PEARLS & CONSIDERATIONS

PREVENTION

Because both of these bacteria can be present without causing clinical disease, preventive measures are focused on decreasing stress, avoiding immune suppressive drugs, and maintaining appropriate diet/husbandry to avoid conversion to clinical disease.

CLIENT EDUCATION

All clients owning rats should understand the frequency of respiratory disease in rats and the importance of proper housing (good ventilation, avoidance of crowding, avoidance of dusty bedding such as wood shavings) and close observation for any signs of respiratory disease, so that treatment can be administered as soon as possible.

SUGGESTED READINGS

Amao H, et al: Natural and subclinical Corynebacterium kutscheri infection in rats, Lab Anim Sci 45:11–14, 1995.

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Borkowski GL, et al: Diagnostic exercise: pneumonia and pleuritis in a rat [Streptococcus pneumoniae], Lab Anim Sci 40:323–325, 1990.

Corning BF, et al: Group G streptococcal lymphadenitis in rats, J Clin Microbiol 29: 2720–2723, 1991.

CROSS-REFERENCES TO OTHER SECTIONS

Respiratory Tract Disease, Chronic

AUTHOR: **NICOLE R. WYRE**

EDITOR: **CHRISTOPH MANS**

SMALL MAMMALS: RATS

Respiratory Tract Disease, Chronic

Client Education Sheet Available on Website

BASIC INFORMATION

DEFINITION

Chronic respiratory disease (CRD) in rats is a multifactorial respiratory tract infection caused primarily by *Mycoplasma pulmonis*, commonly in association with other concurrent infections, resulting in chronic bronchitis and bronchiectasis.

SYNONYMS

CRD, murine respiratory mycoplasmosis (MRM)

EPIDEMIOLOGY

SPECIES, AGE, SEX Older animals are at increased risk.

RISK FACTORS

- Immune status (e.g., age, genotype of certain rats)
- Concurrent diseases (e.g., diabetes mellitus, neoplasia)
- General ventilation of housing
- Ammonia levels in bedding
- Nutritional status (e.g., deficiency of vitamin A or E)
- Obesity

CONTAGION AND ZOONOSIS

- This disease complex is due to the synergism of several pathogens transmitted directly, through aerosol or in utero. The major pathogen is *Mycoplasma pulmonis*, but other pathogens involved in establishing infection include the following:
  - Cilia-associated respiratory bacillus (CAR bacillus, Gram-negative filamentous bacterium)
  - Sendai virus (paramyxovirus)
  - Sialodacryoadenitis virus (coronavirus)

ASSOCIATED CONDITIONS AND DISORDERS

- Otitis media and torticollis (secondary to *M. pulmonis* middle ear infection)
- Reduced fertility (secondary to *M. pulmonis* oophoritis and salpingitis infection)

CLINICAL PRESENTATION

HISTORY, CHIEF COMPLAINT

- Nasal discharge
- Sneezing
- Labored breathing
- Lethargy
- Head tilt

PHYSICAL EXAM FINDINGS

- Porphyrin epiphora (chromodacryorrhea)
- Nasal discharge—with or without porphyrin staining
- Dyspnea
- Tachypnea
- Rales
- Cyanosis
- Muffled heart sounds
- Torticollis and/or nystagmus with otitis media

ETIOLOGY AND PATHOPHYSIOLOGY

- *M. pulmonis* colonizes the epithelial cells of the respiratory tract, middle ear, and epithelia of female genital tract.
- Although *M. pulmonis* causes upper and lower respiratory system lesions, the primary lesion is subacute chronic bronchitis that resembles chronic obstructive respiratory disease in humans.
- CRD in rats is a chronic inflammatory condition resulting in the hypersecretion and impaired clearance of mucus in which elevated levels and activation of macrophages and neutrophils play an important role.
- Once established in the lower respiratory tract, chronic bronchitis and bronchiolitis develop and progress to bronchiectasis and bronchiolectasis. Collections of mucus, leukocytes, and cellular debris accumulate in the lumen due to ciliostasis. There may be rupture of the bronchiolar walls, releasing inflammatory cells, mucus, and debris into the adjacent parenchyma, and developing pulmonary abscessation.
- As the airways become filled with mucus, bronchiolar lumen diameter decreases and a biofilm develops over bronchiolar epithelium, protecting secondary bacterial invaders from immune defenses and most antibiotics.
- *M. pulmonis* also causes an atrophic rhinitis in which the nasal turbinates become inflamed with a mixed pyogranulomatous infiltrate. The rhinitis accounts for the upper respiratory signs seen in CRD of rats. Because rats are obligate nose breathers, rhinitis results in open mouth breathing, hypoxia, and its associated metabolic disorders such as respiratory acidosis and myocyte irritability.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Respiratory signs
  - Neoplasia (primary pulmonary or metastatic)
  - Acute bacterial pneumonia (see Respiratory Tract Disease, Acute)
  - Congestive heart failure
- Otitis media
  - Extension of otitis externa

INITIAL DATABASE

- Thoracic radiographs/CT: findings are consistent with bronchopneumonia, bronchitis, and/or atelectasis
- Skull radiographs/CT/MRI: tympanic bullae sclerosis or effusion if otitis media is present
- Complete blood count: may be normal or consistent with chronic inflammation (neutrophilia, monocytosis)
- Serologic testing: *M. pulmonis*, CAR bacillus, Sendai virus
- Bronchoalveolar lavage for PCR testing *(M. pulmonis)* and aerobic culture (for secondary bacterial pathogens). Culture for *M. pulmonis* requires special mycoplasma media.

ADVANCED OR CONFIRMATORY TESTING

- Histopathologic examination
  - Silver-impregnation staining needed to diagnose CAR bacillus co-infection

TREATMENT

THERAPEUTIC GOAL

Elimination of the disease is impossible. The goal of therapy is to improve the rat's quality of life by controlling secondary bacterial infections and preventing acute dyspneic episodes.

ACUTE GENERAL TREATMENT

- Oxygen therapy
- Fluid support if presence of secondary dehydration

CHRONIC TREATMENT

- Antibiotic therapy will not eliminate the pathogen. Antibiotic selection ideally is based on culture and sensitivity results.
- Doxycycline 5-10 mg/kg PO q 12 h: preferred antibiotic because it has additional antiinflammatory properties and is secreted by respiratory epithelial cells
- Enrofloxacin 10-20 mg/kg PO, IM, SC q 24 h: CAUTION with SC or IM injection as can cause severe pain and tissue necrosis. Dilute with sterile saline before injection.
- Tylosin 10 mg/kg PO, SC IM q 12-24 h: not recommended as use in drinking water because it may reduce water consumption
- Azithromycin 15-30 mg/kg PO q 24 h
- Nutritional support as animals may lose weight with chronic disease

DRUG INTERACTIONS

Oral doxycycline should not be given with any dairy or other products containing calcium because this will decrease its bioavailability.

RECOMMENDED MONITORING

- Respiratory rate and effort
- Body weight/condition
- Appetite

PROGNOSIS AND OUTCOME

Because many factors contribute to rat respiratory issues, the disease cannot be eliminated, but clinical signs may be ameliorated with antibiotics and supportive care.

CONTROVERSY

- Use of corticosteroids as anti-inflammatory agents has been recommended to decrease the inflammation. Most (experimental) studies in rats have found steroids do not affect signs, function, and indices of inflammation. There is also significant concern that corticosteroid efficacy will be accompanied by consequential impairment of the rat's immune defenses leading to fatal pulmonary abscessation and/or pneumonia.
- Use of bronchodilators (both oral and inhaled) has been recommended because these agents are helpful in humans with chronic bronchitis. Specific studies with bronchodilators have not been performed in rats with chronic respiratory disease, but they may be helpful.
- Nebulized hypertonic saline solution (7%) has been used successfully in humans with cystic fibrosis as a mucolytic agent. It breaks down the mucous biofilm and gives relief for ~8 hours.
- Concurrent nebulization with bronchodialators and/or antibiotics has been recommended to directly deliver medications. Specific studies using these nebulizations have not been performed in rats with chronic respiratory disease but may be helpful.

PEARLS & CONSIDERATIONS

PREVENTION

This disease complex *(M. pulmonis)* is thought to be ubiquitous in pet rats; thus prevention of infection is nearly impossible. Preventing contributing factors such as proper ventilation, bedding, and diet and decreasing stress can be helpful.

CLIENT EDUCATION

All clients who own rats should be educated about the ubiquitous nature of *M. pulmonis* and the importance of ventilation, low cage ammonia levels, avoidance of dusty bedding such as wood shavings, and appropriate nutrition in decreasing the potential severity of chronic respiratory disease in rats.

SUGGESTED READING

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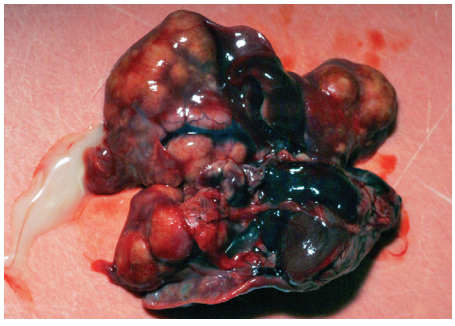
CROSS-REFERENCES TO OTHER SECTIONS

Respiratory Tract Disease, Acute

AUTHOR: **NICOLE R. WYRE**

EDITOR: **CHRISTOPH MANS**



Respiratory Tract Disease, Chronic Rat lungs, abscesses secondary to chronic infection.

Skin Diseases

BASIC INFORMATION

DEFINITION

Infectious and noninfectious diseases of the integument

SYNONYMS

Dermatitis, pyoderma, ulcerative dermatitis, ringworm, dermatophytosis, acariasis, ring tail, abscesses, bite wounds

EPIDEMIOLOGY

RISK FACTORS Inappropriate bedding (e.g., cedar, pine) can cause contact dermatitis.

CONTAGION AND ZOONOSIS

- Dermatophytes are potentially zoonotic.
- *Ornithonyssus bacoti* (tropical rat mite) is a zoonotic parasite.

ASSOCIATED CONDITIONS AND DISORDERS

- Conspecific trauma
- Nutritional deficiencies
- Chronic renal insufficiency

CLINICAL PRESENTATION

DISEASE FORMS/SUBTYPES

- Ectoparasitosis
- Bacterial dermatitis/ulcerative dermatitis
- Abscesses
- Dermatophytosis
- Ringtail
- Neoplasia

HISTORY, CHIEF COMPLAINT

- Skin wounds
- Rough hair coat
- Pruritus
- Hair loss
- Weight loss
- Lethargy
- Swellings on body
- Tail tip lesion

PHYSICAL EXAM FINDINGS Will vary depending on cause:

- Alopecia
- Pruritus
- Localized erythema
- Abrasions, excoriations, ulcerations
- Scaling, crusting
- Lichenification
- Cutaneous or subcutaneous masses

ETIOLOGY AND PATHOPHYSIOLOGY

- Bacterial dermatitis/ulcerative dermatitis
  - *Staphylococcus* spp.
  - Usually secondary to self-trauma due to pruritus from mites or pruritus/pain over skin of salivary glands during sialodacryoadenitis (SDA) virus infection; dermatophytosis, fight wounds
- Abscesses
  - *Staphylococcus aureus*, *Streptococcus* spp., *Pasteurella pneumotropica*, *Actinomyces bovis*
  - Often secondary to conspecific trauma
- Parasites
  - All ectoparasitic infections can be complicated by secondary infections and self-mutilation. These secondary complications need to be identified and treated.
  - Rat fur mite *(Radfordia ensifera)*: common; mild infestation produces few ill effects, but heavy infestation causes pruritus, leading to self-traumatization, and ulcerative dermatitis. Transmission is by direct contact.
  - Sarcoptic mites (e.g., *Sarcoptes scabiei, Sarcoptes anacanthos, Trixacarus diversus*): less common. Transmission is by direct contact. Leads to pruritus, crusting, and hyperkeratosis. Animals with clinical signs are often immune compromised.
  - *Notoedres muris*: causes typical papulous lesions on ear pinnae
  - Tropical rat mite *(Ornithonyssus bacoti)*: Blood sucking mite; opportunistic ectoparasite. It spends a relatively short time on a host (usually at night) and penetrates the skin for feeding only. Cause severe pruritus. Animals appear nervous, particular in evening hours and at night. Severe infestations can cause anemia, debilitation, and death.
  - Demodectic mites *(Demodex ratti, Demodex norvegicus, Demodex ratticola)*: rare
  - Lice *(Polyplax serrata, Polyplax spinulosa)*: Common; blood sucking lice. Located mainly at neck, at shoulders, and over back; poor fur condition and pruritus, which leads to self-mutilation
  - Pinworms *(Syphacia obvelata)*: perianal pruritus and tail base mutilation
- Dermatophytosis
  - *Microsporum* spp., *Trichophyton mentagrophytes*
  - Clinical signs vary: alopecia, erythema, dandruff formation. Animals usually are not pruritic, unless secondary bacterial infection present.
  - Immune deficiency or stress may be underlying cause in chronic cases.
- Neoplasia: fibroadenoma of the mammary glands (most common), mammary adenocarcinoma, lymphoma, etc. (see Mammary and Pituitary Tumors)
- Ringtail
  - Occurs in young rats (7-19 days) and is characterized by dry skin and formation of annular constrictions, which might progress to swelling, and tissue necrosis. Autoamputation might occur.
  - Low environmental relative humidity (less than 20%-40%) appears to be the cause; it is more often seen in rats housed in hanging cages and is rarely seen in pet rats.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Alopecia: trauma, dermatophytosis, chronic kidney disease, nutritional deficiency (low protein), neoplasia, barbering (behavorial)
- Ulcerative and crusting lesions: self-trauma, due to mites, secondary bacterial infections, fight wounds, neoplasia
- Pruritus: mites, secondary bacterial infections
- Crusting or flaking of skin: dermatophytosis, mites, nutritional deficiencies
- Cutaneous masses: neoplasia, inflammation, abscesses
- Localized erythema or pododermatitis: contact allergy, contact irritation (cleaners), trauma from bedding/cage material

INITIAL DATABASE

- Full dietary history
- Dermatologic examination
  - Skin scraping (sedation or general anesthesia may be required)
  - Acetate tape preparation
  - Impression smears
- Fine-needle aspirate and cytology of cutaneous and subcutaneous masses
- Dermatophyte culture
- Bacterial culture and sensitivity

ADVANCED OR CONFIRMATORY TESTING

- Serum biochemistry: if underlying organ disease is suspected
- Radiographs: to rule out underlying skeletal abnormalities (e.g., osteoarthritis; osteomyelitis) in cases of pododermatitis
- Biopsy and histopathologic examination of skin lesion

TREATMENT

THERAPEUTIC GOALS

- Eliminate pruritus and discomfort.

- Treat primary and secondary infections.
- Promote healing of skin lesions.

ACUTE GENERAL TREATMENT

- If animal is self-mutilating: shorten and blunt nail tips. In severe case, temporarily apply bandages to hindfeet. Apply E-collar to prevent removal of bandages.
- Ectoparasites
  - Ivermectin 0.2-0.4 mg/kg SC, PO q 7-14 d
  - Selamectin 10-25 mg/kg topically q 21-28 d
  - Treat until clinical signs are resolved and no more parasites are found on the animals.
  - Treat in-contact animals.
  - Treat the environment to prevent reinfection: regular bedding changes and cage cleaning. Discard cage furnishing that cannot be disinfected (e.g., wood-based furnishing).
- Bacterial dermatitis/ulcerative dermatitis
  - If indicated, provide systemic antibiotic therapy based on culture and sensitivity whenever possible
  - Start empirical treatment pending culture and sensitivity:
    - Cephalexin 30 mg/kg PO q 12 h
    - Amoxicillin/clavulanic acid 15-20 mg/kg PO q 12 h
    - Trimethoprim-sulfa 15-30 mg/kg PO q 12 h
    - Chloramphenicol 30-50 mg/kg PO q 8-12 h
    - Enrofloxacin 10-20 mg/kg PO, q 12-24 h
- Skin abscesses
  - Lance, débride, and flush or remove in toto if possible.
  - If indicated, provide systemic antibiotic therapy based on culture and sensitivity whenever possible.
- Dermatophytosis
  - Systemic antifungal therapy
    - Terbinafine 20-30 mg/kg PO q 24 h
    - Itraconazole 5-10 mg/kg PO q 24 h
  - Topical antifungal therapy
    - Enilconazole (1:50, emulsion as spray or moist wipe)
    - Miconazole/chlorhexidine shampoos
    - Lime sulfur dips (1:40, q 7 d)
    - Used alone or in combination with systemic therapy
    - Used preferably in cases of suspected dermatophytosis, while dermatophyte culture results are awaited
  - Environmental decontamination: frequent damp mopping of hard surfaces rather than sweeping can reduce environmental spread of spores; 1:10 bleach solution can be used to clean environment. Contact time: 10 minutes
- Monitoring: once-weekly dermatophyte test medium (DTM) cultures. Discontinue treatment when two consecutive negative cultures are obtained.
- Antihistamines
  - Diphenhydramine 1-2 mg/kg PO q 12 h
  - Hydroxyzine 2 mg/kg PO q 8-12 h
- Antiinflammatory drugs: meloxicam 0.3-0.5 mg/kg PO, SC q 12-24 h
- Neoplasia: surgical mass removal (see Mammary and Pituitary Tumors)
- Nutritional deficiency: improve diet; provide access to commercial pelleted diet

CHRONIC TREATMENT

Dermatophytosis will often require long-term therapy.

RECOMMENDED MONITORING

- Resolution of clinical signs
- Repeated evaluation for presence of ectoparasites
- Weekly DTM cultures for dermatophytosis cases

PROGNOSIS AND OUTCOME

Good to fair

PEARLS & CONSIDERATIONS

PREVENTION

- Provision of a commercial diet
- Quarantine all new incoming animals for a minimum of 30 days before allowing contact with other animals.

CLIENT EDUCATION

Dermatophytes are contagious; clients should seek medical advice if lesions are found on humans in the household.

SUGGESTED READINGS

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Honma M, et al: Plantar decubitus ulcers in rats and rabbits, Jikken Dobutsu 38:253–258, 1989.

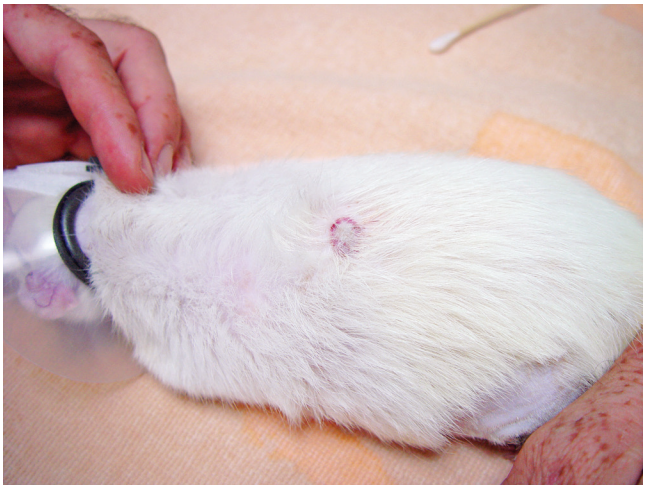
Taylor DK, et al: Lanolin as a treatment option for ringtail in transgenic rats, J Am Assoc Lab Anim Sci 45:83–87, 2006.

CROSS-REFERENCES TO OTHER SECTIONS

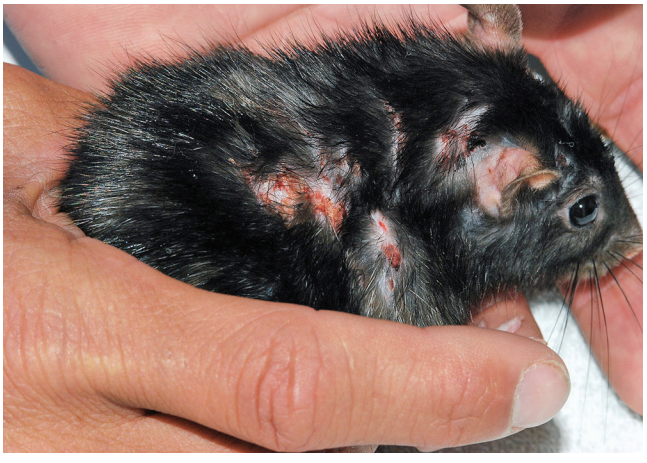
Mammary and Pituitary Tumors

AUTHOR: **CHRISTOPH MANS**

EDITOR: **THOMAS M. DONNELLY**



Skin Disease This skin lesion in a rat was caused by a subcutaneous injection of enrofloxacin; always dilute the drug if it needs to be injected SC or IM. *(Photo courtesy Jörg Mayer, The University of Georgia, Athens.)*



Skin Disease Skin lesions located over the shoulder and neck area, which were induced by fighting with cage mates. Isolation of the rat led to complete resolution of the skin lesions. Self-trauma, secondary to ectoparasite induced pruritus, can present in similar fashion.