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# Fatal Rat Bite Fever in a Pet Shop Employee

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**Background** *Rat bite fever is a zoonotic disease that has been described in laboratory personnel as well as the general population.*
**Methods** *A 24-year-old male pet shop employee contracted the disease through a minor superficial finger wound on a contaminated rat cage. The disease progressed from a flu-like illness to endocarditis involving first the aortic valve and then the mitral valve and septum. Despite aggressive therapy including two surgical procedures, the patient died from sepsis and multi-organ system failure 59 days after initial injury.*
**Results** *This is the first reported case of rat-bite fever (RBF) in a pet shop work setting.*
**Conclusions** *Zoonotic infections may present a significant hazard to workers handling animals. Education on hazards of animal contact and other preventive measures are needed in small places of business like pet shops.* Am. J. Ind. Med. 45:357–360, 2004.


***KEY WORDS: rat-bite fever; endocarditis; Streptobaccillus moniliformis; zoonotic infections; pet stores***

## INTRODUCTION

Zoonoses have been long recognized as occupational diseases. Historically, hunters have been considered those at risk from animal-born diseases [Ramazzini, 1940]. With the advent of human culture, workers in other occupational settings, such as animal laboratories, slaughterhouses, and meat production facilities, veterinarians, and zookeepers have joined this list. With globalization of the economy and increased travel we can expect an increased number of people at risk as well as the spread and emergence of new zoonotic diseases in the United States.

We report a case of fatal bacterial endocarditis due to *Streptobaccillus moniliformis*. The patient was employed in a pet store, an occupational setting, which has not been previously reported to be associated with the risk of occupational endocarditis. The case illustrates the importance of appropriate protection of pet store workers and of early recognition by health care professionals.

Rat-bite fever (RBF) is a zoonotic disease, which is increasing in incidence [Graves and Janda, 2001]. The actual incidence of this disease may be underreported due to the relative difficulty of its diagnosis. There were 13 cases of RBF recorded in the US between 1958 and 1983. Of these, five cases were traced to rat bites in laboratory personal [Wullenweber, 1995]. A total of 18 cases of endocarditis, one of the most severe complications of RBF, were reported between 1915 and 2000. Of these, three cases were linked to rats at the workplace [Rupp, 1992; Rordorf et al., 2000]. No cases of RBF have so far been reported in a pet shop setting.

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## CASE REPORT

A 24-year-old previously healthy young man employed as a pet shop clerk scratched his left fifth finger on a screening of a cage housing rats. No actual rat bite occurred. He had been working in a pet shop for about 5 months prior to this injury. His typical duties included cleaning cages and filling cage feed trays.

Approximately 2–3 days following the injury, the initial scratch progressed into visible redness with blister formation. The patient reported lancing the blister himself. Also 4–5 days following the original injury, he developed flu-like symptoms including fevers, chills, malaise, and diffuse body aches. Two weeks following the injury he presented to the local hospital emergency room with persistent flu-like symptoms as well as left shoulder pain radiating to the left arm, shortness of breath, and vomiting.

The nursing intake assessment in the ER noted the patient to be in moderate general distress, mild respiratory distress, pale, and diaphoretic. The emergency department physician's note, although mentioning the patient's employment in a pet shop, did not make any reference to the work injury. The physician assessment found the patient uncomfortable but not toxic. His temperature was 104.5°F, blood pressure was 140/65, heart rate was 144, and respiratory rate was 24. Pulmonary and cardiac examinations were essentially normal except for tachycardia on the cardiac exam. No cardiac murmurs were noted at that time. Exam of extremities was noted as normal without any mention of the wound at the site of original injury. The white blood cell count was 5,700 with 91% polymorphonuclear cells; the creatine phosphokinase was 997 (range 25–180). The chest radiograph was interpreted as negative. Two sets of blood cultures were drawn. The initial assessment was 'fever of unknown origin.' The physician's note indicated that after 2 L of intravenous fluids the patient felt better and wanted to go home. The patient was discharged home, advised to take acetaminophen and seek follow up care at the County clinic the following Monday, 2 days following his evaluation in the emergency department.

Six days after the first ER visit the patient returned to the emergency department because of worsening shortness of breath and persistent fever. The physical exam at that time revealed moderate respiratory distress with accessory respiratory muscle use, rales at the lung bases, 2/6 cardiac murmur, and a red macular lesion on his left ankle. His white blood cell count was 16,100. Arterial blood gas analysis revealed significant hypoxemia ($PaO_2 = 51$ on room air) and troponin I was positive. A chest radiograph revealed cardiomegaly with pulmonary edema. He was admitted to the hospital and started on intravenous ampicillin with sulbactam. Cardiac echocardiogram demonstrated aortic valve regurgitation. The patient was diagnosed with bacterial endocarditis, admitted into the hospital, and on the same day underwent cardiac surgery consisting of debridement of a myocardial abscess, aortic root enlargement, and aortic valve replacement with a 21 mm St. Jude prosthesis.

Intra-operatively, it was noted that the patient had a small aorta, 300–400 cc of thick cloudy pericardial effusion, and large fixed vegetation encompassing the left coronary cusp. Much of the leaflet was destroyed with the process extending well into the septum and posterior wall burrowing deeply into the myocardium. He did well post-operatively and was discharged home 8 days after the surgery with a daily dose of 2 g intravenous ceftriaxone and oral coumadin.

On the day of discharge the patient remained afebrile the night before and during the day. He had no complaints other than being tired and was ambulating and performing activities of daily living independently. On the day of discharge his vital signs were: temperature = 36.7°C, blood pressure = 139/60, heart rate = 72, respiratory rate = 18, and arterial oxygen saturation = 93% breathing room air. The laboratory data on the day of discharge was as follows: hemoglobin = 9.4, hematocrit = 27.5, white blood count = 20,700, platelets count = 505,000, creatinine = 0.8, potassium = 3.7, prothrombin time = 22.2 with international normalized ratio = 3.31. At the time of discharge, the patient was afebrile for less than 48 hr and his white blood cell count on the day of discharge actually increased compared to the day before.

On the day following hospital discharge, the patient returned, in acute respiratory distress with hemoptysis. Evaluation at that time revealed severe mitral regurgitation on an echocardiogram. Chest computerized axial tomography showed a left pleural effusion and a right pulmonary infiltrate. He was admitted to the hospital but 2 days later transferred to another hospital in anticipation of more specialized surgical care required by the seriousness of his condition. Fourteen days following the first surgical procedure, the patient underwent a second procedure consisting of aortic valve replacement with a homograft, debridement of the myocardial abscess, and mitral valve repair.

The initial antibiotic treatment following the second hospital admission again included intravenous ampicillin with sulbactam. A variety of other antibiotic agents were used throughout the rest of the hospital course to provide the optimal coverage. Fluconazole was added to the antibiotic regimen to cover for possible fungal pneumonia.

Despite aggressive anti-microbial coverage, his condition continued to deteriorate and progressed to hypotension requiring management with an intra-aortic balloon counterpulsation pump and vasopressors. Multi-organ systems failure ensued and the patient died approximately 59 days following the initial contact with a rat cage.

The two blood cultures drawn during the first ER visit were negative. However, one of the two blood cultures obtained 6 days later when the patient returned to the hospital was positive for *S. moniliformis*. The first preliminary result on this blood culture was reported approximately 64 hours

after the specimen was submitted. At that time the report described the organism as a small, curved gram-negative rod and specified its antibiotic sensitivity profile. The final identification of the organism, though, did not come until 24 days after the culture was submitted to the lab. None of the other cultures, including the culture of the aortic valve leaflets and pericardial fluid yielded the growth of the organism. None of the gram stains of blood specimens revealed the organism. However, the report on the gram stain of the aortic valve tissue resected during the first surgical procedure alluded to suspected presence of gram-negative bacteria. The minimal inhibitory concentrations (MICs) for *S. moniliformis* isolate in this case were as follows: gentamycin, 2 μg/ml; ceftriaxone, 0.004 μg/ml, erythromycin, 4 μg/ml; tetracyclin, 0.19 μg/ml, penicillin G, <0.016 μg/ml.

## DISCUSSION

Dr. Shvartsblat did not participate in the care of the patient but reviewed medical records for this case as part of his involvement as a medical consultant to California OSHA.

This case illustrates that RBF can be a significant occupational disease and it is the first one known to occur in a pet shop as an occupational setting.

RBF typically presents as a local skin lesion, followed by a flu-like illness about 10 days after the initial injury. Endocarditis is one of the most severe complications of this zoonotic infection. Other possible complications of the disease include myocarditis, pericarditis, pneumonia, septic arthritis (usually large joints), and meningitis. Embolic events are rare but have been reported. Abscesses may form in virtually any organ of the body including the brain. Infections of pericardial effusion and amniotic fluid are possible. Two causative organisms have been identified: *Streptobaccillus moniliformis* and *Spirillum minus*; the former is much more common in the United States. There are two main routes of entry for the organism into the human body: through a break in the skin barrier or via the gastrointestinal tract.

Ingestion of *S. moniliformis* leads to the gastrointestinal form of the disease known as Haverhill Fever characterized by pharyngitis and vomiting. The origin of the name is the 1962 outbreak that occurred in Haverhill, Massachusetts due to milk contaminated with rat urine.

The other bacterium transmitted by rat bite is a Spirochete called *Spirillum minus*. The disease caused by this organism is known as Sodoku. It is predominant outside the United States and most commonly occurs in Asia.

This case illustrates that pet store employees are at risk of zoonoses such as RBF. Other zoonotic infections such as Salmonella may be contracted from handling reptiles and may constitute another important hazard for pet shop employees. Currently, there are no industry specific OSHA regulations addressing injury and illness prevention in animal handlers.

The diagnosis of this disease is straightforward when there is a high index of suspicion. Inadequate awareness of the potential biological hazards in animal handling and retailing on the part of the employee, the supervisor, and healthcare providers may have contributed to the delay of diagnosis and treatment and a poor outcome. Awareness of work supervisors and employees themselves must be an integral part of any injury and illness prevention program for employers involved in animal handling. The importance of early medical attention even for minor injuries such as superficial scratches must be stressed.

Health care providers who evaluate employees from pet shops, laboratories, zoos, or any other places where animal handling or contact occurs must be aware of the potential for zoonotic infections, possible exposure routes, and incubation periods.

Aggressive work-up should be initiated promptly when presenting history, symptoms, and signs warrant. The laboratory must be made aware of the presumption of zoonotic infection so that appropriate media are used to culture the specimens.

Although occupationally related cases of RBF have been reported in laboratory workers, this is the first reported case contracted in a pet shop. This case also demonstrates that a bite by an infected animal is not required to cause the disease. A superficial scratch is sufficient to provide a port of entry for this organism into the bloodstream and cause the devastating sequence of events that occurred here.

Due to the insidious and non-specific nature of symptoms of this disease at the onset, making an early diagnosis of *S. moniliformis* infection remains a challenge. Definitive diagnosis is further hampered by the fastidious nature and slow growth of this organism. Thus, the true prevalence of the disease may be underestimated by available reports. Furthermore, since this is not a reportable disease, there are no data available on its national incidence.

*S. moniliformis* is very sensitive to penicillin. In fact, *S. moniliformis* is sensitive to a variety of other antibiotics including penicillin, ampicillin, streptomycin, erythromycin, gentamycin, tetracycline, cefuroxime, and vancomycin [Wullenweber, 1995]. So, almost any initially chosen broad-spectrum coverage should provide adequate control of the infection with this organism. The recommended length of therapy depends on the site of infection. The recommended treatment for endocarditis caused by *S. moniliformis* isolate susceptible to 0.1 μg/ml penicillin concentration is 4.8 million U of procaine penicillin G administered daily intramuscularly for 4 weeks. More resistant isolates require 20 million U of daily intravenous administration of penicillin G for 4 weeks. Addition of streptomycin to the standard therapy is advised in the cases of cell wall-deficient L-form isolates [Rupp, 1992].

The choice of antibiotics used in this case was appropriate as an empiric therapy and should have provided adequate coverage were they initiated in the timely manner. The antibiotic sensitivities for the organism in this case were consistent with those reported previously for *S. moniliformis* isolates. Thus, earlier data reveals the following MICs for *S. moniliformis*: penicillin, $\leq$0.03 μg/ml; cephalothin, $\leq$0.03 μg/ml; ceftriaxone, $\leq$0.03 μg/ml; vancomycin, 0.5 μg/ml; tetracycline, 0.25 μg/ml; erythromycin, 2 μg/ml; streptomycin, 8 μg/ml; and gentamycin, 1 μg/ml [Rupp, 1992].

This case highlights the need for primary preventive measures at all work sites where animals are handled, including small retail shops. Measures should include wearing heavy-duty gloves during animal handling or cleaning of cages, regular hand washing after each animal handling, and keeping food supplies separate from the areas where animals are handled. Any bite or scratch must be promptly cleaned with antiseptic solution and the injured employee must be referred for evaluation to a qualified healthcare provider. A detailed occupational history focusing on the types of animals handled as well as detailed description of any recent injuries must be an integral part of the initial evaluation. Close follow up is necessary even for seemingly minor trauma.

Secondary prevention of RBF and other zoonotic infections acquired at the work place must involve raising awareness of this disease for patients and clinicians. Patients and physicians must appreciate that even minor wounds sustained in the course of animal handling may lead to dire consequences without proper treatment. Flu-like symptoms occurring shortly after such injuries should be promptly addressed and evaluated.

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