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# Culture Negative Disseminated Septic Polyarthritis Complicating Rat-Bite Fever in an Immunocompetent Adult

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## Abstract

Rat-bite is uncommon worldwide, excluding developing countries. The history of a bite is usually missed if not meticulously asked for in history, proper exposure and examination. Today we report a case of culture-negative rat-bite fever with poly septic arthritis resembling a rheumatoid arthritis-like picture in a 64-year-old gentleman. The presentation at first imitated an arthritis-like picture which was polyarthritis affecting the right ankle, right knee, right wrist, right proximal interphalangeal, left metacarpophalangeal and proximal interphalangeal joints.

## Keywords

Rat-Bite, Fever, Rat-Bite Fever, Polyarthritis, Culture-Negative, Rheumatoid Arthritis Mimickers

## 1. Introduction

Rat bite fever (RBF) is a rare disease and is commonly under-diagnosed. However, its actual incidence is unknown because RBF is not a nationally notifiable disease, and many cases go undiagnosed since these bacteria are difficult to identify and are likely to respond to empiric antibiotic therapy. However, the mortality rate of untreated cases ranges from 7% to 13% and for cases complicated by endocarditis it can be up to 53% [1] [2].

It is commonly caused by *Streptobacillus moniliformis*, *Streptobacillus notomytis*, or *Spirillum minus*.

*S. moniliformis* causes most cases of the disease in the United States. *S. minus* causes RBF primarily in Asia, although it is probably present worldwide. Infec-

tion with *S. notomytis* has only rarely been reported.

The risk of contracting RBF after a rat bite is reported to be 10% [3]. RBF is most likely to be seen in those who are living in low socioeconomics with the demography of exposure changing to include pet store workers and laboratory technicians as there is an increase in the prevalence of use of rats in research.

Most rats are asymptomatic; however, they may occasionally demonstrate disease signs. The rate of nasopharyngeal carriage of *S. moniliformis* by rats (even healthy laboratory rats) is quite variable, reportedly as high as 100% [4].

*S. moniliformis* is commonly found in rats' nasal and oropharyngeal flora and probably other rodents.

Infection with *S. moniliformis* can result from a bite or scratch from an infected or colonised rat, or from handling rats at home or the workplace (e.g., in laboratories or pet stores).

Oral contact, such as kissing rat pets, can also spread disease. RBF patients make up about 30% of those who do not claim to have been bitten or scratched [5]. Infection with *S. moniliformis* can also occur after ingesting food or water contaminated with infected rat faeces [6] [7].

RBF causes an array of complications, with bacteraemia being the most common complication. Other serious complications include meningitis [8], endocarditis (including prosthetic valve endocarditis) [9], myocarditis, pneumonia, focal abscesses, septic arthritis [10] [11], and multi-organ failure.

In 2003, there were two occurrences of fulminant sepsis and death in individuals who had previously been healthy; one was brought on by a rat bite in a pet shop, and the other was probably caused by a sick pet rat.

Our objective is to shed light on a rat-bit fever as a causative agent for polyarthritis, especially with negative cultures in the setting of the proper history of a rat bite.

## 2. Case Report

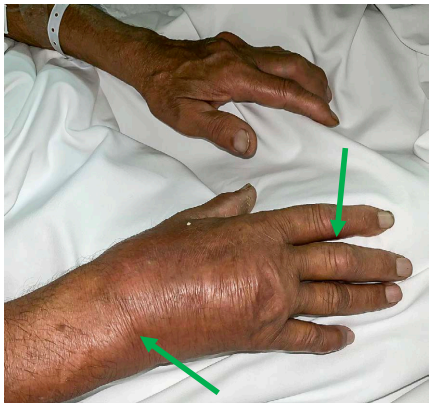
Mr. J 64-years-old gentleman was admitted with four days history of asymmetrical polyarthritis of the right ankle, right knee, right wrist, right proximal interphalangeal, left metacarpophalangeal and left proximal interphalangeal joints, with the joints being stiff and painful. The patient stated all his symptoms started around eight days before the admission but were progressive with an increase in severity that made him immobile. He denied any illicit drug use or recent travel. Past medical history showed that the patient was hypertensive, not on regular medications and has not been followed up in any clinic or hospital for the past 20 years. There was no family history of any autoimmune or rheumatological disease.

On examination, the patient was afebrile (36.8˚C), with a Blood pressure of 140/80 mm Hg, Heart rate of 62 BPM. His physical examination showed; An ill-looking patient, cardiopulmonary audible and S2 with a systolic murmur (mitral regurge), the abdomen was soft and lax, and no organomegaly was felt, with no lymphadenopathy present, and his respiratory examination was unre-

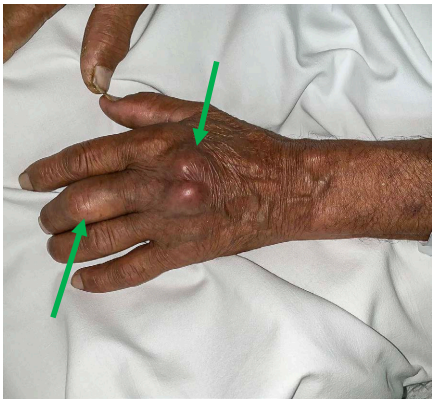
markable.

The patient had no rashes or other dermatological findings.

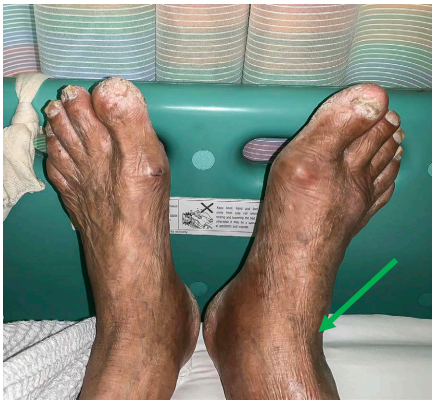
The most striking feature of his physical examination was bilateral asymmetrical joint swelling and effusion affecting the right ankle, right knee, right wrist, right proximal interphalangeal, left metacarpophalangeal and left proximal interphalangeal joints, as seen in Figures 1(a)-(c).



(a)



(b)



(c)

**Figure 1.** (a) Right hand showing right wrist swelling with proximal interphalangeal joint involvement; (b) Left wrist and proximal interphalangeal joint swellings and two soft tissues swelling on the wrist; (c) Right ankle swelling.

Upon further questioning, the patient admitted that a rat bit him while in his home eight days before the admission, and he killed and threw out the rat.

The rat bite is on the base of the left big toe, as seen in Figure 2.

On admission, the patient's labs showed a picture of a high leucocytic count, anaemia with acute kidney injury, hyponatremia and mild hepatic transaminitis, as shown in Table 1.

A transthoracic echocardiography was done that elicited a mild mitral regurge, however no evidence of vegetations and endocarditis was ruled out. Patient was started on *Piperacillin/Tazobactam*, *Daptomycin and Levofloxacillin,* however, there was no improvement in his symptoms.

We consulted the preventive medicine and public health department regarding rabies post-exposure prophylaxis, and they stated, "There is no need no initiate it as rabies prevalence in Kuwait is non-existent". Further work up was done as shown in Table 2.

We discussed the case with the microbiology team, and we requested specific blood cultures for rat bite organisms (*S. moniliformis*, *S. notomytis* and *S. minus*), however, blood cultures showed no growth. 16S rDNA testing was not available.

With the liver impairment and transaminitis, an ultrasound abdomen and pelvis were done and showed a picture of parenchymal liver disease.

Synovial fluid from the right knee was aspirated and showed the following results in Table 3.

Joint aspiration is shown in Figure 3.

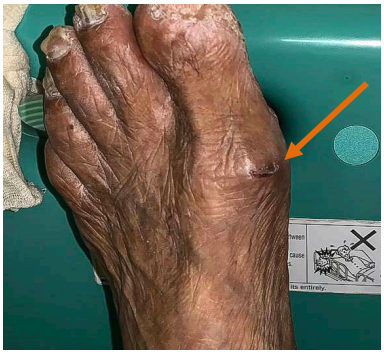


**Figure 2.** Rat bite on the base of left big toe.

**Figure 3.** Joint aspiration from right Knee.

Table 1. Lab results on admission.

| Lab name | Result<br>Normal Range |
|---|---|
| White cell count | $15.3 \times 10^9$<br>$3.7 - 11 \times 10^9$ |
| Neutrophilic count | 13.4<br>1.7 - 7.1 |
| Hemoglobin | 108<br>130 - 170 g/L |
| Platelets | 218<br>150 - 440 |
| Urea | 25.8 mmol/L<br>2.9 - 7.5 mmol/L |
| Creatinine | 387 μmol/L<br>71 - 115 μmol/L |
| Sodium | 127 mmol/L<br>136 - 146 mmol/L |
| Alkaline phosphatase | 193 IU/L<br>56 - 119 IU/L |
| Alanine aminotransferase (ALT) | 72 IU/L<br>10 - 60 IU/L |
| Aspartate aminotransferase (AST) | 58 IU/L<br>10 - 42 IU/L |
| Gamma glutamyl transferase (GGT) | 67 IU/L<br>12 - 64 IU/L |
| Total Bilirubin | 41.4 μmol/L<br>3 - 20 μmol/L |
| Direct Bilirubin | 25.5 μmol/L<br>0 - 5 μmol/L |
| C-reactive protein (CRP) | 370 mg/L<br>0 - 8 mg/L |
| Procalcitonin (PCT) | 19.430 ng/ml<br>0.02 - 0.046 ng/ml |
| Erythrocyte sedimentation rate (ESR) | 65 mm/hr<br>0 - 20 mm/hr |

**Table 2.** Further laboratory work up.

| Lab | Result |
|---|---|
| Rheumatoid Factor | Negative |
| Anti-cyclic citrullinated peptides (Anti-CCP) | Negative |
| Anti nuclear antibody (ANA) | Negative |
| Complement Levels (C3, C4) | Normal |
| Anti-Smooth muscle antibodies | Negative |
| Anti Liver, Kidney, Microsomal (Anti-LKM) | Negative |
| Hepatitis B | Negative |
| Hepatitis C | Negative |
| HIV | Negative |
| Neisseria gonorrhea | Negative |
| Brucella | Negative |
| Salmonella | Negative |
| Leptospira | Negative |
| PPD | Negative |
| QuantiFERON (T-Spot) | Non-reactive |

**Table 3.** Synovial fluid analysis.

| Lab | Result |
|---|---|
| Stain | No staining by gram stain |
| Cultrue | No growth |
| Cell count & differential | WBC $60 \times 10^6$<br>Mainly neutrophils 98% |

An MRI of both knees shows the following findings:

"The hallmark of the examination is the presence of distended both knee joint capsules by joint collection more on the right side eliciting low to intermediate T1 and high T2 signal intensity associated with evident thickening of the covering synovium which shows heterogeneous enhancement in the post-contrast study. osteoarthritic changes of the knee joints were also noted more on the left side. In view of multi-joint affection, the MRI findings are impressive of subacute polyarticular septic arthritis of both knee joints".

After eight days of antibiotics, patient has shown no to little improvement with exacerbation of his acute liver injury most likely due to *Daptomycin* so it was stopped, and the antibiotic regimen was changed to *Ceftriaxone*, *Vancomycin* and *prednisolone* 20 mg once daily to reduce the duration of symptoms and reduction in inflammatory markers with improvement in laboratory findings and inflammatory markers.

A repeat joint aspiration showed improvement in the leucocytic count with

white blood cells with less joint swelling and the patient being vitally stable and afebrile. The patient has finished *Ceftriaxone* for four weeks, improved and travelled back to his home country.

## 3. Discussion

In this case report, we present a challenging case; RBF is difficult to diagnose with a multitude of complications and multiple causative organisms. The history of a rat bite or a scratch is monumental for the diagnosis as the cultures are usually negative in RBF [12].

In a case report titled "Rat Bite Fever Resembling Rheumatoid Arthritis" by Ripa Akter, Paul Boland, Peter Daley, Proton Rahman, and Nayef Al Ghanim they state, aside from direct rat bites or scratches, rodent predators like cats, dogs, and pigs can also bite or scratch humans and transmit infection [13]. Rats typically have *Streptobacillus moniliformis* in their nasopharynx. This bacterium is also colonized by other rodents, including mice, guinea pigs, ferrets, squirrels, and gerbils [14]. RBF can also be brought on by ingesting contaminated food products, as was mentioned in Haverhill, Massachusetts, in 1926. RBF has been linked to farmers who drank unpasteurized milk, according to reports.

Owners of pets, youngsters, and individuals who work in pet businesses or laboratories that employ animals for research are more likely to contract this vi-rus within 3 to 10 days of exposure, 90% of people develop fever, which may follow a recurring infection. The extremities frequently exhibit a maculopapular, petechial, or purpuric rash, and a leukocytoclastic vasculitis is compatible with a biopsy result [15] [16]. The hands, wrists, elbows, knees, and sternoclavicular and sacroiliac joints are frequently affected by migrating polyarthritis [17] [18] [19].

Surgical debridement may be necessary in some cases of *Streptobacillus moniliformis* septic mono-arthritis [20] [21].

RBF's clinical symptoms vary according to the etiologic agent (*S. moniliformis or S. minus*) [22].

*S. moniliformis* in infants and adults, the clinical symptoms of RBF caused by *S. moniliformis* can range from a mild flu-like illness to fulminant sepsis [23] [24]. RBF has a roughly 13% death rate in untreated patients [25].

For individuals with infection due to *S. moniliformis*, the incubation period is typically less than seven days following exposure.

Symptoms start abruptly with fever, myalgias, migratory arthralgias, vomiting, pharyngitis, and headache. By the time symptoms develop due to a bite or scratch, the wound has usually resolved and there is no regional adenopathy. Individuals who acquire RBF through ingestion have more severe vomiting and an increased incidence of pharyngitis compared with those who acquire disease through a bite.

*S. minus*; In contrast to infection with *S. moniliformis*, RBF caused by *S. minus* has a longer incubation period (one to three weeks). In addition, the initial

wound may reappear at the onset of the systemic illness or persist with oedema and ulceration with associated regional adenopathy. Approximately 50% develop a red-brown macular rash. Arthritis is not a common clinical finding.

A Japanese woman who had a rat bite on her finger was diagnosed with *S. notomytis*, which led to her developing fever, dermatitis, and polyarthritis. RBF is typically diagnosed empirically since it is challenging to culture *S. moniliformis or S. notomytis*, and *S. minus* cannot be cultured.

There is no available serological test, however relevant specimens have been diagnosed via analysis of the 16S rDNA gene sequence [26] [27].

An empiric diagnosis of RBF is made in the patient with an unexplained febrile illness or sepsis and a history of rat exposure. This is particularly true in patients with a relapsing or intermittent fever pattern, a maculopapular rash, and/or polyarthritis or polyarthralgia (typically involving the knees and ankles).

Confirming a diagnosis of RBF is extremely difficult. If *S. moniliformis* or *S. notomytis* is felt to be the causative agent specimens of blood, synovial fluid, or aspirates from abscesses should be inoculated into bacteriologic media without sodium polyanethole sulfonate (SPS), such as an anaerobic culture bottle. SPS is present in most aerobic blood culture media, and it inhibits growth of the organism. The microbiology laboratory should be alerted so that specific media and culture conditions can be used to optimize the isolation of the organism.

As the aetiology of arthritis may be attributable to an immune-mediated mechanism rather than true infection with the organism, synovial fluid cultures can be negative even with appropriate diagnostic testing in cases of uncomplicated illness.

16S rDNA testing (if available) on appropriate specimens such as tissue (heart valve, bone) or synovial fluid may be useful for the diagnosis of *S. moniliformis* or *S. notomytis*. However, the sensitivity and clinical utility of such testing has not been proven. This type of testing is not available on routine blood samples.

*S. minus* is unable to be cultured, and a microbiologic diagnosis is made through demonstration of the organisms on examination of blood, exudate, or tissues using Giemsa or Wright stains, or dark-field microscopy.

Treatment of RBF; The management of patients with rat-bite fever (RBF) includes both local care of the wound in those reporting a bite, as well as appropriate antibiotics for individuals with clinical symptoms. This includes the management of the animal bite with copious irrigation of the wound and assessment regarding the need for tetanus or rabies post-exposure prophylaxis. Rats and other small rodents are rarely infected with rabies. If the animal is not available for testing, post-exposure prophylaxis for bites should be considered individually in consultation with public health authorities. Regarding antibiotic therapy, *penicillin* is the treatment of choice for RBF, and prompt therapy can prevent severe complications. The mortality rate of RBF is approximately 13% in untreated patients. Empiric therapy should be started immediately in patients with a compatible clinical presentation and exposure history since laboratory confirmation is difficult and may take several days. If the organism is detected, anti-

microbial susceptibility can be determined using standard testing.

There are no guidelines regarding the duration of RBF treatment, it is mostly guided by case reports and clinical experience. The uncomplicated disease consists of fever, myalgias, polyarthritis, polyarthralgia, vomiting, headache, and/or rash. In these individuals, the total duration of treatment is 14 days. Penicillin or ceftriaxone are the antimicrobial agents of choice, some patients are initially treated in the hospital with intravenous *penicillin G* (200,000 units every four hours) and, after clinical improvement, are then switched to intravenous *ceftriaxone* (1 gram daily) administered at home to complete the intravenous regimen. This is then followed by oral therapy to complete a 14-day course. Some prefer penicillin because there is broad clinical experience with this antibiotic, while others administer ceftriaxone because it is more easily administered, especially for individuals receiving outpatient intravenous therapy. For adults and children who are unable to take beta-lactam antibiotics, tetracyclines can be administered. Streptomycin and Gentamicin are alternative agents; however, their use is limited because other treatment options with less toxicity are available. Serious invasive infections complicating RBF are rare. These include meningitis, endocarditis (including prosthetic valve endocarditis), myocarditis, pneumonia, focal abscesses, bacteremia, pyogenic arthritis, and multiple organ failure. Although death has been reported in up to approximately 50%, the majority of these occurred in the absence of effective antimicrobial therapy [28].

## 4. Summary

Rat-bite fever is a rarely diagnosed illness caused by *streptobacillus moniliformis*, *Streptobacillus notomytis*, or *Spirillum minus*. With *S. moniliformis* being the most common causative organism in the United States and *S. minus* in Asia and *S. notomyt* is being the rarest of all three.

Infection with these organisms can result from scratches or bites from an infected or colonized rat, or from handling rats and home or workplace. For patients with infection due to *S. moniliformis* or *S. notomytis*, the incubation period is usually less than seven days after exposure. Symptoms start abruptly with fever, myalgias, migratory arthralgias, vomiting, pharyngitis, and headache. By the time symptoms develop due to a bite or scratch, the wound has usually resolved and there is no regional adenopathy.

There is a wide variety of complications that have been reported such as bacteremia, meningitis, discitis and endocarditis.

The diagnosis of RBF is mainly clinically as growing *S. moniliformis* is difficult and *S. minus* doesn't grow on artificial media. Also, there is no serological test available for RBF however there is an emerging role for 16s rDNA gene sequence on appropriate specimens.

Treatment of RBF starts from local wound care with Penicillin being the treatment of choice. Empiric therapy should be initiated in patients with appropriate clinical presentation and exposure. There are no specific guidelines for

treatment duration with only guidance being obtained from case reports and clinical experience.

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## Consent

Patient consent was obtained.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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