

Haverhill Fever with Spine Involvement

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ABSTRACT. Haverhill fever and rat-bite fever are closely-related syndromes caused by *Streptobacillus moniliformis*. This infection is characterized by the abrupt onset of fever with rigors, myalgias, headache, polyarthritis, and rash. We report a case of infection with *S. moniliformis* that manifested as acute polyarthritis with involvement of the spine. To our knowledge, involvement of the spine has not been reported previously with this infection. Diagnosis can be particularly difficult in the absence of fever or obvious exposure to rodents, as in our case. A high degree of awareness is necessary to make the diagnosis of this potentially fatal infection, which is easily treatable. (J Rheumatol 2006;33:1409–10)

Key Indexing Terms:

HAVERHILL FEVER

STREPTOBACILLUS MONILIFORMIS

RAT-BITE FEVER

Haverhill fever and rat-bite fever are closely-related syndromes caused by *Streptobacillus moniliformis*¹⁻³. This infection is characterized by the abrupt onset of fever with rigors, myalgias, headache, polyarthritis, and rash. We report a case of infection with *S. moniliformis* that manifested as acute polyarthritis with involvement of the spine.

CASE REPORT

A 68-year-old man was brought to the emergency room of a rural community hospital, with a 4-day history of progressive and diffuse joint pain and swelling. His history included osteoarthritis of the hands and knees, type 2 diabetes mellitus, and hypertension.

Four days prior to admission he began to experience pain in both knees and ankles that progressed quickly to swelling and restriction of movement of most of his peripheral joints accompanied by severe pain and stiffness of his neck and back. A petechial rash developed on the extensor surfaces of his lower extremities 2 days prior to admission. The severity of his joint symptoms rendered him bedridden. There was no history of fever, chills, vomiting, diarrhea, tick bite, dental work, travel, or intravenous drug use. He worked as a dairy farmer and lived with his wife on the farm. A 30-year-old nephew who lived with them had a similar illness of diffuse joint pain and swelling one week prior but symptoms were milder and resolved spontaneously.

Due to severe neck stiffness and the petechial rash, meningitis was suspected. Two sets of blood cultures were drawn and he was given intravenous (IV) ceftriaxone prior to transfer to our hospital for further management.

On admission he was in severe pain. He was afebrile and had a petechial rash over the extensor surfaces of his lower extremities. He had a symmetrical polyarthritis, with involvement of the proximal interphalangeal and metacarpophalangeal joints, wrists, elbows, shoulders, knees, and ankles. He also had severe pain and restriction of movement in his cervical and lumbar

spine. Joint examination was made difficult by severe pain. Peripheral white blood cell count was 17,100/mm³, with 91% polymorphonuclear leukocytes.

The left knee had a moderate effusion. Arthrocentesis revealed a total white blood cell count of 19,250/mm³, with 84% polymorphonuclear leukocytes. Calcium pyrophosphate dihydrate (CPPD) crystals were reported, although not specified as intracellular or extracellular. Gram stain of synovial fluid (SF) was negative. A diagnosis of acute polyarticular pseudogout was considered, but the presence of petechial rash did not support this.

Plain radiographs of the hands and knees revealed osteoarthritic changes with no chondrocalcinosis. Treatment with ibuprofen was started, but was switched to naproxen the next day. Blood and SF cultures from our hospital were negative. However, on the third hospital day, the microbiology laboratory of the community hospital at which the patient initially presented reported a Gram-negative bacillus growing in one of the anaerobic blood culture bottles. The culture was sent to our microbiology laboratory, where the pleomorphic, fastidious organism was presumptively identified as *S. moniliformis* and therapy with IV penicillin G was started. Bacterial identification was confirmed by the Central Public Health Laboratory (Toronto, ON). An echocardiogram was negative for valvular vegetations. The patient did not recall being bitten by a rat or other animal, but stated that there were rats around his farm, including in the milking stables. He admitted to drinking unpasteurized milk together with his nephew. His wife would not drink unpasteurized milk. After receiving 14 days of antibiotic treatment, joint pain and swelling improved remarkably.

DISCUSSION

Haverhill fever and rat-bite fever are 2 closely related syndromes caused by infection with *S. moniliformis*, a bacterium found in the oropharyngeal flora of rodents. Fifty percent to 100% of healthy wild and laboratory rats harbor *S. moniliformis* in the oropharynx¹⁻³. The infection is called rat-bite fever when the organism is transmitted to humans by a bite or scratch of rats, mice, squirrels, or carnivores that prey on those rodents, including cats, dogs, pigs, ferrets, and weasels³. The infection may also be acquired by handling dead rats with no apparent breach of intact skin. The risk of developing this infection after a bite or scratch is estimated to be around 10%².

Haverhill fever (erythema arthriticum epidemicum) occurs following ingestion of foods or liquids contaminated by rats or other infected animals. This name comes from the famous

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outbreak from contaminated milk that occurred in Haverhill, Massachusetts, USA, in 1926 and affected 86 people^{3,4}. More recently, 130 children were infected at a boarding school in Chelmsford, England, in 1983, probably from contaminated raw milk consumption^{5,6}. Since our patient's nephew had a similar illness of diffuse joint pains and swelling one week prior with milder symptoms and spontaneous resolution, and both admitted drinking unpasteurized milk, our patient and nephew might represent a "mini-outbreak." The farmer's wife avoided infection by refusing to drink unpasteurized milk. Clinically, Haverhill fever resembles rat-bite fever, but with more severe gastrointestinal symptoms and pharyngitis. Presumably, once ingested, *S. moniliformis* organisms gain access to peripheral circulation by penetrating gastrointestinal mucosa¹.

Apart from classical manifestations that include abrupt onset of fever, headache, myalgias, and various rashes, up to 50% of cases will develop a polyarthritis of the knees, shoulders, elbows, wrists, and small joints of the hands¹⁻³. In our case and another² the polyarthritis was symmetric; other cases are better characterized as asymmetric, migratory polyarthritis^{1,7}. Joint effusions are common and, as in our report, exceedingly painful. The arthritis is felt to be septic and not reactive; however, growing the bacteria from SF is uncommon^{1,3}. Involvement of the spine by *S. moniliformis* has never been reported in the literature to our knowledge. Our patient had severe pain and restriction of movement of his whole spine that mimicked meningitis and improved with antibiotic treatment.

Diagnosis of Haverhill fever is often difficult because of unfamiliarity with the disease and the nature of the causative organism. In our patient, early in the admission, the report of CPPD crystals in SF from the knee prompted the consideration of polyarticular pseudogout as the diagnosis. However, the intense spinal involvement and petechial rash did not fit this diagnosis. The presence of chondrocalcinosis, but with a joint fluid negative for CPPD crystals, confounded the diagnosis in another patient with rat-bite fever⁷. High prevalence of chondrocalcinosis in the elderly population is likely to compel a rheumatologist to consider this entity highly in the differential diagnosis. Indeed, we and others⁷ made the diagnosis only because blood or SF cultures were positive. *S. moniliformis* is a non-encapsulated, non-motile, Gram-negative bacillus that is highly fastidious and pleomorphic. Sodium polyanethol sulfonate, an anticoagulant commonly used in blood culture bottles, inhibits the growth of this organism and can delay or prevent diagnosis^{3,7}. Negative blood and synovial cultures obtained at our hospital were drawn after the patient received ceftriaxone for suspected meningitis.

Treatment of choice for *S. moniliformis* infection is IV penicillin G for 10 to 14 days. Most patients respond promptly to therapy. For individuals who appear well after 5 to 7 days, therapy can be completed with an additional week of oral penicillin. Tetracycline or doxycycline may be given to patients allergic to penicillin^{1,3,8}.

Generally, even without antimicrobial treatment, the symptoms slowly resolve over a period of 10 to 14 days. Mortality in untreated cases is around 10%, usually due to infective endocarditis^{3,9,10}. However, there have been recent reports of fatal *S. moniliformis* infections in patients with severe unsuspected infections⁸. Other reported complications include myocarditis, pericarditis, meningitis, and focal abscesses in a variety of organs. Chronic arthritis has been reported in a minority of cases^{2,3}.

Our patient represents a unique case of Haverhill fever because of involvement of the spine. This infection should be included in the differential diagnosis of a wide variety of clinical syndromes, especially in farmers, laboratory workers, and urban residents exposed to rats. High clinical suspicion and cooperation with the microbiologist is essential in making the diagnosis of this uncommon but readily treatable infection.

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