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Epidemiology and clinical features of flea-borne rickettsioses

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Abstract

Murine typhus is a flea-borne rickettsiosis caused by *Rickettsia typhi*. The disease is endemic to tropical and subtropical regions throughout the world. It is likely vastly under-recognized as a cause of febrile illness. Rats serve as the classic reservoir and transmit the bacterium to their fleas –*Xenopsylla cheopis*. In North America, an alternate cycle of transmission has been identified involving opossums and *Ctenocephalides felis*. Disease manifests as an undifferentiated febrile illness, similar to a host of other community acquired or vector-borne infectious diseases. Accompanying symptoms often include headache, myalgias, nausea, and vomiting. On physical exam, rash is present in only 50%. When rash is absent, clinicians often neglect to consider a rickettsial illness. Laboratory abnormalities include elevated hepatic transaminases, thrombocytopenia, and hyponatremia. Serology is the mainstay of diagnosis, and doxycycline is the medication of choice. *Rickettsia felis*, transmitted by *Ct. felis*, is described as causing a febrile illness similar to murine typhus and other rickettsioses. More recent descriptions identifying the presence of *R. felis* DNA within febrile and afebrile subjects in sub-Saharan Africa, in those with malaria, and in a variety of environmental samples has raised intriguing questions regarding the pathogenicity of the organism. Other *R. felis*-like organisms have been detected and isolated from fleas, but the role of these organisms in human disease is currently unknown.