CASE REPORTS

A case of murine typhus in Queensland

Stephen R Graves, Joan Banks, Brian Dwyer and Geoff K King

Objective: **To present a case of murine (endemic) typhus, the first to be reported within the last 30 years in Australia.**

Clinical features: **A 17-year-old pregnant woman presented with a viral-like illness and later developed a spotted rash, fever and headache.**

Investigation and outcome: **Sera taken on Day 7 and Day 30 of the illness showed seroconversion to *Proteus* OX19 (Weil–Felix) and to *Rickettsia typhi* (by immunofluorescence), indicating recent infection with *Rickettsia* of the typhus group. Her illness was clinically compatible with murine typhus. She responded well to erythromycin and delivered a normal infant at term.**

Conclusion: **Infection with *Rickettsia typhi* (murine typhus) still occurs in Australia. It can be diagnosed by means of specific serological tests for rickettsial disease, which are superior to the non-specific Weil–Felix test.**

(Med J Aust 1992; 156: 650-651)

Murine typhus is known to occur in Australia and was once a commonly reported infection in several Australian States but is rarely diagnosed now. The clinical symptoms are not unambiguous and laboratory (serological) tests are not generally available.

We wish to report a case of murine (or endemic) typhus in a patient from north Queensland.

Clinical record

In February 1990, a 17-year-old Caucasian primagravid woman (23 weeks) presented to the Accident and Emergency section of Mossman Hospital after two days of aches and pains, coryza and cough. The presumptive diagnosis was a viral infection. Four days later she returned with persistent symptoms and complained of a sore throat and joint pains (elbows, wrists, knees and ankles). On the next day (Day 7 of illness) she had fever, headache, anorexia and a spotted rash.

She was admitted to hospital, acute phase serum was taken and she was treated with erythromycin, 250 mg four times a day for seven days. The following laboratory investigations were undertaken at that time: full blood examination (normal); urea and electrolytes (normal); liver function tests (raised alanine aminotransferase and aspartate aminotransferase); and leptospiral cultures (negative). She was discharged from hospital after three days, and when seen at outpatients (Day 14) was described as "good as new".

She ultimately had a normal spontaneous vaginal delivery at term. The placenta was normal and the puerperium uncomplicated. A six-week check of mother and child was uneventful.

The acute (Day 7) and convalescent (Day 30) sera were sent to Fairfield Infectious Diseases Hospital, Victoria, where full, specific serological tests for rickettsial disease were undertaken. The results indicated that the patient had murine (endemic) typhus caused by *Rickettsia typhi* (also known as *Rickettsia mooseri*) (Table). A seroconversion to *R. typhi* is good evidence that the patient had murine typhus.

Weil–Felix reaction, a non-specific test for rickettsial disease, which uses *Proteus* sp. antigens, also supported the diagnosis of murine typhus. The acute serum was negative for all three *Proteus* antigens (OX2, OX19, OXK), whereas the convalescent serum was positive for OX19 (titre >1/640) but negative for OX2 and OXK.

Serological investigation of Day 7 sera gave negative results for other diseases, including leptospirosis; Lyme disease; influenza A and B; parainfluenza 1, 2, and 3; adenovirus; respiratory syncytial virus; mumps; Q fever; *Chlamydia* (group antigen); and *Mycoplasma pneumoniae* (Queensland Health, Laboratory of Microbiology and Pathology). Unfortunately, the Day 30 sera was anticomplimentary and could not be tested.

TABLE: Specific rickettsial serology

Serum	Specific immunofluorescence serology (titre)		
	R. typhi (murine typhus)	*R. australis* (Queensland tick typhus)	*R. tsutsugamushi* (scrub typhus)
Acute (Day 7)	< 1/64	< 1/64	< 1/64
Convalescent (Day 30)	1/256	< 1/64	< 1/64

Discussion

Murine typhus was first described in 1922, in Adelaide, by an Australian, Hone, who considered it to be a different disease to epidemic typhus.[1] The next year Maxcy and Havens in the United States described a similar series of cases of mild typhus. They also considered it to be distinct from epidemic typhus.[2] Hone published further reports on this disease.[3,4] Wheatland, in 1926, reported an epidemic of this disease in Toowoomba, Queensland, which was associated with a mouse plague.[5] A further epidemic, also associated with a mouse plague in the same location, was reported by Derrick and Pope in 1960.[6]

The rickettsial organism causing murine typhus was isolated from patients in Queensland by Funder and Jackson in 1946 and shown to be distinct from that (*R. australis*) responsible for Queensland tick typhus.[7] The latter disease had only recently been recognised as a separate entity.[8,9] Derrick has provided a fascinating history of the early investigations into the various fevers (including murine typhus) of north Queensland and their differentiation.[10]

Murine typhus has also been reported in Western Australia,[11] and a positive reaction for *R. typhi* was detected in three out of 76 sera taken from asymptomatic persons on Flinders Island in Bass Strait.[12] It seems likely that murine typhus is Australia-wide.

The main features of the illness are fever, headache, macular rash (without eschar) and respiratory symptoms. The disease is acquired by one of three possible routes: (i) by the bite of a rickettsia-infected rodent flea and the regurgitation of the fleas' intestinal contents into the patient; (ii) by the bite of a rickettsia-infected rodent flea and the contamination of the wound with the flea's faeces; and (iii) by the inhalation of rickettsia-infected faeces of the rodent flea. In our patient we cannot be certain which route the infection took, but the absence of flea bites and the prominent respiratory symptoms early in the illness would be consistent with infection by inhalation of *R. typhi*-contaminated faeces from fleas of rats or mice.

Murine typhus responds well to a variety of antibiotics but doxycycline is probably the drug of choice. Although the disease is rarely reported in Australia, rural people are probably at most risk, particularly during periods of rat or mice plagues. With excessive rodent numbers, and rodent deaths, their ectoparasites will seek other

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hosts, including humans. Cases of murine typhus have also occurred among persons demolishing rodent-infested buildings presumably because aerosols of rickettsia-infected rodent flea faeces were generated. Prevention of disease involves exclusion of rats and mice from the human environment, a situation more readily achieved in urban than rural areas.

Diagnosis is by serological means, detecting a seroconversion or rise in antibody titre between two serum specimens taken 7 to 10 days apart. The traditional Weil–Felix test is now not recommended because it lacks both sensitivity and specificity. It can give a positive result in non-rickettsial diseases and, even in rickettsial disease, sera positive for OX2 and/or OX19 can indicate a spotted fever group rickettsial disease (e.g. Queensland tick typhus) or a typhus group rickettsial disease (e.g. epidemic typhus or murine typhus). The best available serological test for diagnosing murine typhus is indirect immunofluorescence using *R. typhi* fixed onto a glass slide. Such slides are commercially available (bio-Merieux, Charbonnières les Bains, France).

In conclusion, murine (endemic) typhus should be considered in patients with environmental exposure to rats and mice (and hence their ectoparasites) in whom a febrile illness occurs in association with a macular rash. The diagnosis can be confirmed by specific serology.

Serological diagnosis of rickettsial disease

The serology laboratory in the Clinical Pathology Laboratory at Fairfield Infectious Diseases Hospital, Fairfield, VIC 3078 (Tel. (03) 280 2516) and Queensland Health, Laboratory of Microbiology and Pathology, Brisbane, QLD 4000 (Tel. (07) 224 5420) are now able to undertake serological diagnosis of all four rickettsial diseases that occur in Australia — murine typhus (*R. typhi*), scrub typhus (*R. tsutsugamushi*), Queensland tick typhus (*R. australis*), and Flinders Island spotted fever (agent isolated but not yet identified, but antibodies cross-react with *R. australis*). Medical practitioners requiring these tests for diagnostic purposes may send paired (acute and convalescent) sera to these laboratories.

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(Received 2 Sep, accepted 11 Nov 1991)

Rabies

A second Australian case, with a long incubation period

(see also page 596)

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Objective: **The description of a second case of rabies in Australia, stressing the clinical features and that long incubation periods are possible.**

Clinical features: **A 10-year-old Vietnamese girl presented with fever, shoulder pain, subcutaneous emphysema, swallowing difficulty and agitation. After a period of maniacal behaviour all peripheral and central nervous system function was lost.**

Intervention and outcome: **Despite maximal intensive care, the patient died. The diagnosis of rabies was made at autopsy.**

Conclusions: **Rabies occurs in Australia and needs to be considered in the differential diagnosis of acute encephalitis and/or the Guillain–Barré syndrome. Incubation periods of more than six years can occur.**

(Med J Aust 1992; 156: 651-654)

Rabies has been recognised for at least 4000 years.[1] In 1985 over 25 000 cases were reported to the World Health Organization, but this is probably an underestimate of its incidence with as many as 50 000 deaths from rabies occurring each year in India alone.[2] The first case of rabies in Australia was described in 1988, and as is commonly the case when rabies occurs in developed countries the diagnosis was made at autopsy.[3]

We report a second Australian case where the rarity of this condition in Australia and the absence of recent potential exposure resulted in the diagnosis again being delayed until autopsy.

Clinical record

In November 1990 a 10-year-old Vietnamese girl presented to the accident and emergency department at Westmead Hospital with pain involving the left arm, shoulder and chest. This had begun 30 hours previously while she was riding a bicycle, but there was no history of trauma. The pain had become progressively more severe and seemed worse with breathing. It had not responded to paracetamol or traditional Vietnamese treatments with coins and herbal tea.

Examination revealed her to be very distressed by pain but less so if talked to or otherwise distracted. Tenderness was found in the left paracervical muscles, the left pectoral muscles, over the left side of her chest and in the left upper limb, but there were no objective signs of inflammation and no abnormalities on general physical examination. Her chest radiograph and electrocardiogram were normal. The pain was thought to be musculoskeletal and she was discharged and prescribed naproxen.

Eighteen hours later she returned in an ambulance in a state of extreme agitation. She described intermittent spasms of pain lasting seconds which involved her whole body but were centred in the left shoulder region. She also complained of difficulty in breathing, a blocked nose and a lump in her throat. She drank copious fluids but would then spit them out saying this eased her throat. She vomited "coffee ground" material. Although at times orientated in time, place and person she appeared disturbed, talking about her body image, her dirty hands and the "bad earth" inside her body.

On examination her temperature was 38.7°C, the

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