Spotted fever in East Gippsland, Victoria: a previously unrecognised focus of rickettsial infection

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ABSTRACT A new focus of spotted fever group rickettsial infection has been recognised in East Gippsland, Victoria. Seven cases have been identified among Melbourne residents after they holidayed in the area. The infections were confirmed serologically. The precise identity of the *Rickettsia* has not been determined.

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There are three endemic human rickettsial infections in Australia: murine typhus[1] transmitted by flea bite or inhalation of infected flea faeces; scrub typhus[2] transmitted by the bite of larval forms of trombiculid mites; and Queensland tick typhus[3] transmitted by tick bite. Each represents a major subgroup within the genus *Rickettsia*. Queensland tick typhus was first defined as a clinical entity by Andrews et al. in soldiers on the Atherton Tableland,[3] and the organism they recovered has been shown to belong to the spotted fever group of rickettsiae[4] which is transmitted principally by ticks. The spotted fever group contains a number of species with geographically defined locations on all continents except Antarctica.[5] Since its description in 1946 small numbers of cases of Queensland tick typhus have been reported along the length of the Pacific coast of Queensland and New South Wales as far south as Sydney.[6] Recently a spotted fever like illness has been reported from Flinders Island, Bass Strait.[7,8] It is yet to be established whether Flinders Island spotted fever is caused by *Rickettsia australis*, the aetiological agent of Queensland tick typhus. The case fatality rate is not precisely known, but the first death attributable to Queensland tick typhus has now been described.[9]

The spotted fevers are characterised by a history of tick bite or exposure to a tick infested locality. The development of a local area of inflammation at the site of the recent attachment of the tick often proceeds to necrosis — an eschar with associated local lymphadenopathy. The sudden onset of fever, usually with recurrent chills, early prostration, myalgia and headache, which are followed after several days by the development of a maculopapular rash on the trunk, head, neck and limbs are the major features of this group of infections. In some cases there may be a slight dry cough of delayed onset. A distinctive and characteristic yet inconstant feature of the rash is involvement of the palms and soles. Delirium is common and is held to be due to vasculitis which is itself a universal phenomenon in this infection. Cardiovascular collapse is seen in more severe cases. The case fatality rate varies according to age, and the infecting species. The best studied disease in the spotted fever group is Rocky Mountain spotted fever of which over 500 cases are reported each year in the United States.[10,11]

Effective treatment of spotted fevers is available with tetracyclines or, in severe or complex cases, chloramphenicol.

There are close similarities in vegetation and fauna related to the geologically recent land bridge between Victoria and Tasmania. This includes the area now known as Flinders Island. We reasoned that cases of spotted fever would occur in people travelling through or residing in East Gippsland, Victoria. In November 1987, infectious disease physicians who meet as the Melbourne Infectious Diseases Group were alerted to this possibility by one of us (B W D). We now report the occurrence of seven cases of spotted fever from this previously unrecognised location for this infection.

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Methods

The case definition was drawn from the original description of Andrews et al.[3] and from the work of Stewart.[7] A case met the definition when there was a clear history of sojourn in East Gippsland, a history of fever of abrupt onset, myalgia, headache and a maculopapular rash on the trunk and limbs. The presence of maculopapules on the palms and soles was considered to be characteristic, but their absence (or non-recording) did not exclude the diagnosis. A history of tick bite was considered valuable but not essential, as was the presence of an eschar. The diagnosis was established when alternative diagnoses could be excluded, and there was evidence of seroconversion or a fourfold rise in antibodies or a single serum showed a titre of at least 1:28 to *R. australis* antigen in an indirect immunofluorescence test as previously described.[8] In some cases attempts were made to demonstrate a rickettsaemia by inoculation of freshly drawn blood into guinea pigs, young and new born mice and Buffalo green monkey kidney cell lines. Antibodies to *Borrelia burgdorferi* and Ross River virus were tested by immunofluorescence and enzyme-linked immunosorbent assay (ELISA), respectively, according to published methods.[12-14]

Clinical records

Between December 1987 and January 1989 (two summer seasons) seven cases meeting the case description were detected in Melbourne residents who had travelled to East Gippsland. All cases developed in the summer or autumn. Several other cases of spotted fever were also seen during this time but were not included as they were acquired elsewhere. All cases were seen by either B W D or S R G; Cases 2, 3, 4 and 5 were managed by M I McD and J K McD and the other cases were managed by A P Y and R R D.

Case 1

On December 8, 1987, while camping at Wingan Inlet in the Croajingalong National Park, a 28-year-old artist noticed a tick attached to her left foot, just above the great toe web. She removed the tick with her fingers. She remained well until December 17 when she felt tired, developed shivers and aching joints and muscles. She remained in bed for the next three days with repeated evening chills. On December 20 she

developed a non-pruritic, non-vesicular maculopapular rash on the legs. She was referred by her local doctor to Fairfield Hospital the next day, December 21.

On examination her temperature was 37.5°C and quickly settled. There was a blotchy maculopapular rash (3 to 4 mm in diameter) on the trunk, arms, legs and face. The macules were noted to be tender and were present faintly on the palms and soles. There were two lesions on the left foot, one was a healing cut which had preceded the holiday and the other was a purplish bruise without necrosis, at the site of the tick bite. Liver function tests were all within the reference range, although the C-reactive protein level was elevated to 73 mg/L (reference value, <10 mg/L). The haemoglobin level was 118 g/L (reference range, 115–165 g/L), the white cell count was 3.8×10^9/L ($4–11 \times 10^9$/L), and the platelet count was 248×10^9/L ($150–400 \times 10^9$/L). There were 36% segmented neutrophils, 14% band forms, 5% monocytes and 46% lymphocytes. There was a mild left shift. She became afebrile the day after admission and no antibiotics were prescribed. She was discharged on December 23 feeling very well.

Cases 2, 3, 4, 5 (Family "M")

These patients were members of a family of five who holidayed with another family (Family "J") at Lakes Entrance between April 7 and April 12, 1988. The families stayed in adjacent holiday cabins some 2 km northeast of the township. The activities of the two families were identical with the exceptions detailed below.

Case 2

A 37-year-old chemical salesman, who had been in New Guinea and Japan in mid March 1988, returned to Melbourne by March 19 and proceeded with his family and Family "J" to Lakes Entrance on April 7. On the afternoon of April 11, Family "J" returned to Melbourne and Family "M" took a bush stroll through an area which was known to be tick infested. All the family wore tracksuits or long trousers and pullovers, except for Case 2 who wore shorts. No ticks were noted. Family "M" returned to Melbourne that evening.

On April 16, while on a business trip to Sydney, the patient noted a tender "blind pimple" on the back of his right leg just above the popliteal fossa. He developed a headache and myalgia on April 19 and by the following day noticed more pain in the lesion on his leg. There was now another similar lesion on the front of his left leg and he noticed enlarged inguinal nodes. By April 22 he had rigors and a fever of up to 39°C and remained febrile for another four days. His blood film at the time showed no toxic changes with a haemoglobin level of 155 g/L and a white cell count of 7×10^9/L. He had conjunctival injection and a maculopapular rash (diameter 3 to 4 mm) from April 23. Several "blisters" were sensed inside his mouth. He took flucloxacillin (500 mg every six hours) for four days from April 18. On April 24 he had an episode of delirium.

On April 26 he was feeling better and examination showed a marked maculopapular rash on the trunk, limbs, face, palms and soles. There was a 5 mm diameter area of necrosis in the skin just above the right popliteal fossa and another similar lesion on the lateral side of his left thigh. Both lesions were surrounded by a small zone of erythema and were not purulent. The inguinal glands were bilaterally enlarged and minimally tender. He became afebrile from April 27.

Case 3

The 11-year-old daughter of Case 2 became ill on April 20 with headache. Within two days she had developed chills, fever and sweats and went to bed. Her throat became very sore. Her blood film on April 22 showed no toxic changes with a haemoglobin level of 125 g/L and a white cell count of 5.2×10^9/L. On April 24 she was delirious and had a generalised maculopapular rash which started in the interscapular region. On examination on April 26 she had four small (4 to 5 mm) papules on her soft palate and right buccal mucosa (Figure 1). There was slight tenderness and enlargement of the infra-auricular nodes on the right. The faded exanthem was noted to also include the palms and soles. There was a small non-tender blackened 3 mm diameter lesion on the skin of the neck over the right sternocleidomastoid muscle (Figure 2). She took flucloxacillin (250 mg every six hours) for four days from April 18 and became afebrile from April 27.

Case 4

The 13-year-old son of Case 2 developed a red "pimple" on his anterior abdominal wall on April 16. On April 22 he became more unwell with a dry cough, headache and nausea followed by chills and fever up to 38.5°C. His blood film at this time showed a haemoglobin level of 104 g/L and a white cell count of 5.8×10^9/L with no toxic changes. There was evidence of iron deficiency which was shown some months later to be caused by a bleeding Meckel's diverticulum. The abdominal lesion blackened in the centre on the following day and one day later showed a central crater. On examination in addition to a faded macular rash, there was a 10 mm diameter eschar on the anterior abdominal wall, a few palatal petechiae and a few faint palmar macules. He took flucloxacillin (250 mg every six hours) for four days from April 18 and became afebrile from May 3.

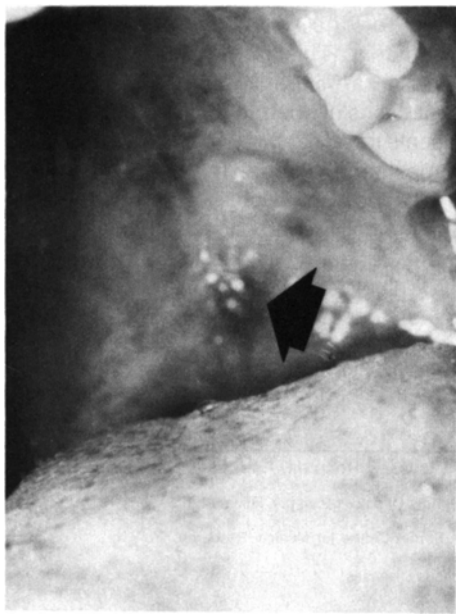

FIGURE 1: Oral lesions in Case 3.

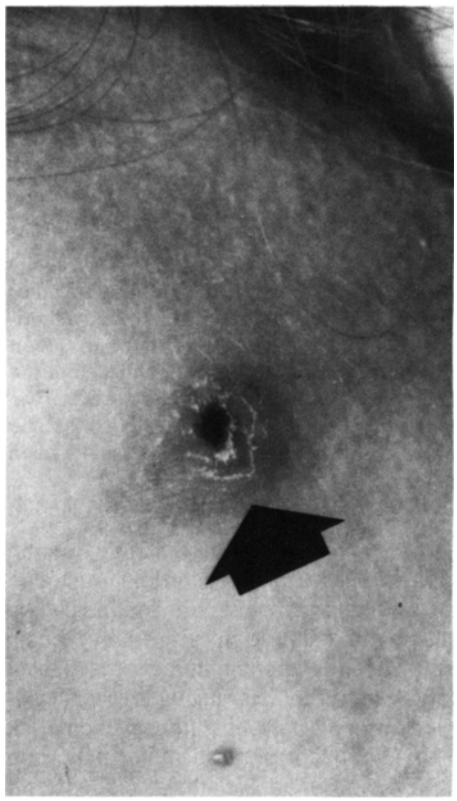

FIGURE 2: Eschar on neck, Case 3.

Case 5

The 36-year-old wife of Case 2 developed an "itchy pimple" on her right ankle on April 16. This lesion rapidly blackened over the next two to three days and additional lesions developed on both ankles and also on the upper abdomen in the right subcostal area. Fever and rigors developed on April 17. When she consulted her local doctor the following day, she had a marked headache, generalised myalgia and had developed a generalised, non-pruritic, maculopapular rash. Her blood film on April 22 showed no toxic changes with a haemoglobin level of 155 g/L and a white cell count of 7×10^9/L. She took flucloxacillin (250 mg every six hours) for four days from April 18 and was afebrile from April 22. Exami-

nation on April 26 revealed multiple eschars on the ankles and a large 20 mm by 8 mm eschar below the right subcostal margin. There was a fading maculopapular rash on the trunk and limbs.

Family "J" was interviewed subsequently and serial serum specimens obtained. There was no history of febrile illness after the holiday and all members remained seronegative.

Case 6

An 18-year-old female student returned from a two-week bushwalking trip with school friends in the Croajingalong National Park on December 22, 1988. The walking route followed a coastal track from Point Hicks to Mallacoota. On December 13, at Wingan Inlet, a tick was removed from her scalp and a second tick was removed at Shipwreck Creek on December 16. On December 19 she noticed a lump behind her right ear which continued to enlarge over the next four days. On December 24 she developed fever, severe headache and painful ankles. The following day she developed a rash and was admitted to Fairfield Hospital. Her admission temperature was 38.9°C. She did not appear toxic. There was a maculopapular rash, each lesion being 3 to 5 mm in diameter, on her trunk, buttocks and limbs. There was a tender enlargement of the postauricular, occipital and tonsillar nodes, more marked on the right. The liver and spleen were not tender or enlarged. The ankles were minimally tender to touch but were not warm, reddened or swollen. There was faint bruising at the sites of the two tick bites.

Liver function test results were within the reference range and C-reactive protein level was 15 mg/L. The haemoglobin level was 127 g/L, the white cell count was 7.8×10^9/L, and the platelet count was 250×10^9/L. The differential white cell count showed 75% segmented neutrophils, 7% band forms, 4% monocytes and 14% lymphocytes. There was a slight left shift and an occasional atypical lymphocyte. After blood was drawn for attempted rickettsial isolation she was commenced on doxycycline (100 mg twice a day) for seven days. The fever persisted on December 26 and 27, and she became afebrile on December 29, 60 hours after commencement of doxycycline. Her symptoms also rapidly improved and she was discharged well on December 30.

Case 7

A 17-year-old male student had been bushwalking in the Croajingalong National Park near Mallacoota from December 30, 1988 to January 8, 1989. On January 4 a tick was extracted with forceps from behind the left ear. By January 8 he noticed a tender lump at the site of the tick bite. On the morning of January 11 he awoke with a fever and was perspiring heavily. His throat was sore. That day he was prescribed oral penicillin by his local doctor. There was no history of penicillin allergy. On the evening of January 12 he developed a rash on the face, trunk and limbs. He also had a headache, myalgia and had transient diarrhoea. His temperature was measured at over 39°C.

On admission to Fairfield Hospital on January 14, his temperature was 40.2°C and he looked ill. There was a generalised, non-pruritic, erythematous, maculopapular rash, most marked on the extremities and the palms. The right postauricular node was enlarged and tender and there was a "bruise" at the site of the tick bite. The liver and spleen were not tender or enlarged. The liver function tests were mildly abnormal: the aspartate aminotransferase level was 155 U/L (<40 U/L reference value), the alanine aminotransferase level was 130 U/L (<50 U/L), the alkaline phosphatase level was 130 U/L (<95 U/L), the γ glutamyl transferase level was 65 U/L (<50 U/L), the total bilirubin level was 19 μmol/L (<17 μmol/L) and the C-reactive protein level was 126 mg/L. The haemoglobin level was 144 g/L, the white cell count was 5.9×10^9/L, and the platelet count was 113×10^9/L The differential white cell count showed 58% segmented neutrophils, 15% band forms and 27% lymphocytes. There was a slight left shift and an occasional atypical lymphocyte. After blood was drawn for conventional and rickettsial cultures the patient was commenced on doxycycline (100 mg twice a day). The following day he felt better although he had a late afternoon rigor. He was discharged on January 16 and was well at review on January 23.

None of these seven cases showed serological evidence of infection with *Borrelia burgdorferi* or Ross River virus.

Discussion

The clinical patterns in these patients show many similarities with each other and with described cases of both Queensland tick typhus[3] and Flinders Island spotted fever.[7] All cases occurred in the summer or autumn, typical of the seasonal pattern seen on Flinders Island. The cluster of cases in one family was unusual. Of the five members of that family, only one, a 9-year-old girl, did not become ill. None of Family "J" with whom they holidayed became ill, or showed serological evidence of rickettsial infection. However Family "J" had returned home a few hours earlier and had not ventured into the bushland location where the ticks were known to occur. This suggests that exposure of Family "M" to the agent occurred on the afternoon of the last day of their holiday.

All patients in this series became ill 6 to 11 days after the exposure to ticks or a location where ticks were known to occur. Only three patients (Cases 1, 6 and 7) had a clear history of tick bite. It is noteworthy that these patients developed neither the more advanced local necrosis nor the multiplicity of eschars that was seen in Family "M". If ticks are the responsible vectors for this disease, then perhaps the duration of attachment is important in determining whether local necrosis occurs, as those patients who noticed ticks had them removed as soon as they were detected. This would be consistent with our observations that the illness was more severe in Family "M", in whom ticks were not noted. In four cases there was a local lesion, enlarged local nodes or a skin lesion, which immediately preceded the systemic illness.

In all of our patients there was a relatively abrupt onset of fever and chills with true rigors within the first day or two. Early prostration, headache, myalgia and transient joint pains were usual. In some cases the headache was severe enough to cause the clinicians to consider the possibility of a central nervous infection, although the neck was always supple. Medical advice was often not sought immediately as the illness was considered to be the 'flu. A dry cough was noted in only one patient, a few days after the onset of fever.

The maculopapular, non-urticarial, non-desquamating rash appeared between two and five days after the onset of fever. The rash was present on the palms and soles in all cases. There was no vesiculation. The rash involved the periphery as much as the trunk. Conjunctival macules and injection occurred in three cases. Two cases reported soreness inside the mouth or throat. Transient pain without swelling or diffuse reddening in one or two joints was seen in Case 4.

There was erythema surrounding the fully developed eschars in Cases 2, 3, 4 and 5 and a tender local lymphadenopathy

TABLE: Results of serological investigations (microimmunofluorescence)

Case	Days from onset	Titres of antibody to *Rickettsia australis*
1	3	<64
	34	512
2	6	<64
	41	256
3	6	<64
	41	256
4	6	<64
	41	256
	118	512
5	6	<64
	41	256
6	2	64
	4	64
	7	128
7	4	<64
	6	<64
	13	512

was noted in five patients. Lymphangitis was not noted. The common sites for tick bites and eschars included the legs and the head or neck.

All patients showed elevated titres of antibody to *R. australis* and, except in Case 6, an unequivocal seroconversion, occurring as early as 10 days after onset. Antibody was readily detectable for up to 118 days after onset of the illness (Table). Although this illness is clinically consistent with an infection from the spotted fever group, no infective agent was recovered. In these patients, whose illnesses closely resembled spotted fever clinically, we consider it unlikely that the serological reaction with *R. australis* was caused by cross-reactions with any organisms other than rickettsiae. Serological cross-reactions within the spotted fever group of rickettsiae do occur. Significantly, all seven patients had almost identical titres when *R. conorii* (bio-Mérieux, Charbonnières les Bains, France) was used as antigen. However, the precise identification now depends on the isolation of the agent from the patients. The identity of the ticks involved in these cases also remains unknown. It is known that the common tick species biting humans in Queensland is *Ixodes holocyclus*[15] and that this species occurs in Victoria.[16] This and *I. tasmanii* have been confirmed to harbour *R. australis* in a study from Queensland.[17] However a recent field trip to East Gippsland (November 1989) demonstrated that *I. cornuatus* was the tick most commonly found on man; *I. holocyclus* was not detected (Stephen R Graves, unpublished observations).

Beta-lactam antibiotics were given to five patients although we do not believe they materially affected the course of the illness in these cases. Doxycycline was given to Cases 6 and 7 and probably modified the course of their illness.

The differential diagnosis of spotted fever is that of febrile exanthem. There are a number of illnesses which may be confused with this infection and these may be distinguished to some degree by their clinical features. Murine typhus (*R. typhi*), once a common disease in cities and towns in South Australia, Western Australia and Queensland[1,18,19] is now rare, perhaps because of effective rat and mouse control. There is no eschar and specific serology is required to distinguish this infection from the spotted fevers. Scrub typhus (*R. tsutsugamushi*) is known to occur in coastal north Queensland[20] and may be accompanied by an eschar. There is usually a history of exposure to scrub country. Serologic confirmation is required for a definitive diagnosis.[21] More usually the clinician will have to consider other serious infections. Meningococcal septicaemia is most commonly encountered in young children. Haemorrhagic skin lesions are common when the infection is fully established, but in the first 24 hours an erythematous maculopapular rash like early measles may be seen. There is usually an initial malaise and low grade fever, and the patient will appear both pallid and toxic. The relatively short prodrome and the rapid transition of the rash to a more purpuric form are prominent features. Blood cultures followed by empirical antibiotic therapy may be necessary in some cases until the precise diagnosis can be determined by further epidemiological, clinical and laboratory assessment.

The virus that causes Ross River virus infection is mosquito transmitted. It is a milder condition and has more prominent joint symptoms. We suspect that cases of spotted fever have been confused with Ross River virus infection and that the opposite may also occur. In Ross River virus infection, the rash usually spares the head and neck and the polyarthritis, while symmetrical in distribution, is less often symmetrical in intensity, and may be accompanied by joint swelling. An eschar would be unexpected in Ross River virus infection. Repeated chills and rigors would be unusual and the fever does not last more than a few days. Palmar and plantar macules may be seen in both Ross River virus infection and in the spotted fevers. Serological confirmation by the demonstration of Ross River virus specific IgM is available.[22]

Measles, rubella, leptospirosis and secondary syphilis may at times require careful distinction from spotted fever, while infectious mononucleosis should rarely be confused on clinical grounds.

The management of tick bites requires some elaboration. The tick should be removed with care so as to avoid crushing the tick or causing its contents to be inoculated into the patient's skin or into the eye. The majority of ticks can be removed by the application of forceps to the front of the head, close to the point of attachment to the skin. With the tick firmly grasped, sustained, gentle traction will extract the entire animal painlessly from the skin. Torsion should not be applied. Usually the mouth parts remain intact and no further exploration is necessary. The skin and forceps should be washed and disinfected afterwards.[23] It may be advisable to take a serum sample for storage.

The patient should be instructed to report any fever which might develop in the following 7 to 21 days. Cases considered to be consistent with spotted fever should be treated with doxycycline (100 mg twice a day) for 5 to 10 days, although there is, as yet, insufficient evidence to indicate that tetracyclines have a clear and beneficial effect on this particular rickettsiosis. Many patients appear to recover without antibiotics and this may be a prudent course to adopt in mildly ill children, especially in those who do not yet have their permanent front teeth. Hypotension, delirium, pregnancy, bleeding or evidence of renal impairment should be considered indications for admission to hospital. In general, serological evidence of infection is evident from 7 to 14 days after the onset of the illness and it is best to confirm the diagnosis by examining serial serum samples. Because the aetiological agent has not been isolated we have chosen to use *R. australis* as the antigen in the microimmunofluorescence test. However improvements in our understanding of this infection may lead to additional antigens being used.

Additional cases

During the preparation of this paper two additional cases came to our attention and are reported here because of their unique features. On October 31, 1989, a 31-year-old woman from Eden, New South Wales, presented to her doctor with a five-day history of fever, headache and myalgia. One day later she developed a maculopapular rash on the trunk and limbs as well as some papules which became vesicular and resembled the lesions of varicella. She had an incontrovertible history of previous varicella. There was a history of a tick bite occurring 17 days before the onset of the illness. The tick bite site on her abdomen was now a reddened 2 cm diameter scaly patch. Extensive investigations including blood, urine and skin cultures gave normal results. The full blood examination showed a haemoglobin level of 136 gm/L, and a white cell count of 5.2×10^9/L. Differential count showed 72% neutrophils, 21% lymphocytes, and 7% monocytes. The blood film was reported as normal. The screen for infectious mononucleosis was negative and the

anti-streptolysin O antibody level was not elevated. The patient received an initial dose of procaine penicillin followed by amoxicillin by mouth. She quickly improved and was well at review on November 14. Serum taken at this time showed *R. australis* microimmunofluorescence titres of 512 (total immunoglobulin) and 256 (IgM). This case indicates that spotted fever extends to the southernmost part of coastal New South Wales. The presence of a varicelliform rash (well described in Queensland tick typhus) suggests a strong relationship between that disease and the disease reported from Victoria.

A 24-year-old veterinarian reported his illness in September 1989. In the first week of August he had camped at Refuge Cove and Sealers Cove, Wilson's Promontory, South Gippsland. At that time he detached a tick from his eyelid. Five days later he developed inflammation at the site and was prescribed cephalexin. Two days later he developed fever and headache and a day later came out in a rash. The rash involved the trunk and limbs. He responded promptly to tetracycline. Although seroconversion was not detected his *R. australis* microimmunofluorescence titres were 512 (total immunoglobulin) and 256 (IgM) 23 days after onset. This case indicates that infections can occur in southern Australia in winter and further extends the range south and west on continential Australia.

Conclusion

We consider tick transmitted rickettsial infections of humans to be extant along the entire east coast of Australia. The illness is distinctive. The clinical picture is of fever, myalgia, and headache with a generalised maculopapular rash commonly with involvement of the palms and soles. Delirium and delayed onset of cough are seen in some cases. A history of exposure to coastal areas or other locations where ticks are prevalent; or a history of tick bite or the presence of an unexplained bruise, especially with local lymphadenopathy; or an eschar should arouse clinical suspicion. It would be reasonable to use empirical doxycyline therapy in these cases, subject to the usual contraindications. Serious long-term sequelae and case fatality have not been described in this infection. Confirmation of the diagnosis is available through the use of spotted fever group rickettsial serology.

References

1. Hone FS. A series of cases closely resembling typhus fever. *Med J Aust* 1922; 1: 1-13.
2. Langan AM, Mathew RY. The establishment of "Mossman", "Coastal", and other previously unclassified fevers of North Queensland as endemic typhus. *Med J Aust* 1935; 2: 145-148.
3. Andrew R, Bonin JM, Williams S. Tick typhus in North Queensland. *Med J Aust* 1946; 2: 253-258.
4. Plotz H, Smadel JE, Bennett BL, et al. North Queensland tick typhus: studies of the aetiological agent and its relation to other rickettsial diseases. *Med J Aust* 1946; 2: 263-268.
5. Marchette NJ. The tick borne rickettsiae of the spotted fever group or tick typhus group. In: Marchette NJ, ed. Ecological relationships and evolution of the rickettsiae. Vol 1. Chapter 5. Baton Rouge: CRC Press, 1982: 75-112.
6. Campbell RW, Abeywickrema P, Fenton C. Queensland tick typhus in Sydney: a new endemic focus. *Med J Aust* 1979; 1: 350-351.
7. Stewart R. "Flinders Island spotted fever": a newly recognised endemic focus of tick typhus in Bass Strait. Part 1. Clinical and epidemiological features. *Med J Aust* 1991; 154: 94-99.
8. Graves S, Dwyer B, McColl D, McDade J. "Flinders Island spotted fever": a newly recognised endemic focus of tick typhus in Bass Strait. Part 2. Serological investigations. *Med J Aust* 1991; 154: 99-104.
9. Sexton DJ, King G, Dwyer B. Fatal Queensland tick typhus. *J Infect Dis* 1990; 162: 779-780.
10. Woodward TE. Rickettsial diseases. In: Weatherall DJ, Ledingham JGG, Warrell DA, eds. Oxford textbook of medicine. Chapter 5. Oxford: Oxford University Press, 1987: 341-355.
11. Anonymous. Rocky Mountain spotted fever — United States, 1988. *MMWR* 1989; 38: 513-515.
12. Russel H, Sampson JS, Schmid GP, et al. Enzyme-linked immunosorbent assay and indirect immunofluorescence for Lyme disease. *J Infect Dis* 1984; 149: 465-470.
13. Vejtorp M, Leerhoy J. Comparison of the sensitivity of ELISA and HAI tests for routine diagnosis of rubella. *Acta Pathol Microbiol Scand [B]* 1980; 88: 349-350.
14. Carter IWJ, Smythe LD, Fraser JRE, et al. Detection of Ross River Virus immunoglobulin M by enzyme-linked immunosorbent assay using antibody class capture and comparison with other methods. *Pathology* 1985; 17: 503-508.
15. Domrow R & Derrick EH. *Ixodes holocyclus*, the man biting tick in southeast Queensland. *Aust J Sci* 1965; 27: 234-235.
16. Roberts FH. Australian ticks. Melbourne: CSIRO, 1970: 1-267.
17. Campbell RW, Domrow R. Rickettsiosis in Australia: isolation of *Rickettsia tsutsugamushi* and *Rickettsia australis* from naturally infected arthropods. *Trans R Soc Trop Med Hyg* 1974; 68: 397-402.
18. Saint EG, Drummond AF, Thorburn IO. Murine typhus in Western Australia. *Med J Aust* 1954; 2: 731-737.
19. Derrick EH, Pope JH. Murine typhus, mice, rats and fleas on the Darling Downs. *Med J Aust* 1960; 2: 924-928.
20. Doherty RL. A clinical study of scrub typhus in North Queensland. *Med J Aust* 1956; 2: 212-220.
21. Brown GW, Shirai A, Rogers C, Groves M. Diagnostic criteria for scrub typhus: probability values for immunofluorescent antibody and Proteus OXK agglutinin titers. *Am J Trop Med Hyg* 1983; 32: 1101-1107.
22. Fraser JRE. Epidemic polyarthritis and Ross River Virus disease. *Clin Rheum Dis* 1986; 12: 369-388.
23. Needham GR. Evaluation of five popular methods for tick removal. *Pediatrics* 1985; 75: 997-1002.

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David Mitchell Ross

MB BS, FRACP

Physician, died August 4, 1990, at the age of 88. Born on July 13, 1902 near Grafton, NSW; educated at Sydney Grammar School; Exhibition in Medicine, University of Sydney (MB BS, 1925). Fellowship of the Royal Australasian College of Physicians, 1963. Hospital appointments at Royal Prince Alfred Hospital, Sydney, 1926–1927, and Royal Hospital for Women Sydney, 1928 (resident medical officer); Royal Prince Alfred Hospital (honorary associate physician, 1947–1965).

David Mitchell Ross was the sixth of seven children of Thomas and Christine Ross. His parents both came from Scotland. Thomas Ross emigrated to Australia as a school teacher and taught in various parts of New South Wales and Christine came from Aberdeen to marry him. She had two brothers, both doctors. Of their seven children, five graduated in medicine from the University of Sydney. David's sisters, Mona, Marjory and Heather became noted pathologists in Sydney. David and brother Alec followed their example.

In 1930 David joined the late Tom Nelson in general practice at Ashfield where he gained practical medical experience over the next decade.

In 1933 he married Sheila Waugh. They had three daughters Susan (Dr Beal), Sandra (Mrs Bates) and Jane (Mrs Sherrard). Their marriage of 57 years was continuously happy and mutually supportive.

David enjoyed sport. He was a University of Sydney tennis "blue" and had a golf handicap of six.

During his War service with 2/7 Australian General Hospital he worked with Alex Sinclair who developed his interest in psychiatry. Following the war he spent six months working at a psychiatric hospital in Goulburn and then worked at the Repatriation General Hospital, Concord, as a psychiatrist. David then began private practice as a consultant physician in his chosen pioneering fields of psychosomatic medicine and allergy in which he made significant personal contributions over a 30-year period.

His life was dedicated to medicine and his patients. His practice in psychosomatic medicine was unique at that time and was based on his war time psychiatric training and experience of people and their problems in general practice. He fulfilled the combined roles of physician, psychiatrist and philosopher on which Hippocratic medicine was based. This was during the years of development of subspecialities and narrowing fields of practice which increased his value to his colleagues and patients. He was one of the last few dedicated general physicians.

David was an outstanding teacher and tutor. He tutored in medicine at St Andrew's College, Sydney, and helped many students to gain teaching hospital residencies and advised them in their future careers. Many leading specialists have written expressing their gratitude to him in this regard.

He was chosen as one of the three lecturers of the Royal Australasian College of Physicians in the first of the Advanced Courses in Medicine in Singapore in the early 1960s. His enthusiasm for teaching, his dry humour and friendship endeared him to everyone there.

David Ross was essentially a good man, tolerant, humane, with a generous, warm personality. He was a family man, making no demands on his children but demanding the best of himself. His many friendships were lasting. He loved his wife, family and home. During the last decade he became almost blind and deaf and suffered greatly. He never complained, remaining cheerful to the end. He loved life itself and maintained an interest in local and world affairs. Fortunately he was able to remain at home due to the constant devotion of his wife, Sheila. Our sympathy goes to her and her family.

STANLEY GOULSTON