Fatal Queensland Tick Typhus

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In previous reports of treatment failure in HIV patients infected with TB, noncompliance was suspected [1]. There is one case of relapse of TB after four-drug short-course therapy [8]. The isolates in that case were not phage typed but had identical susceptibility patterns.

The case reported here is unique in that failure of therapy was related to the emergence of a rifampin-resistant isolate with subsequent dissemination of disease. The current recommendations for treatment of TB in HIV-infected persons are isoniazid and rifampin with pyrazinamide given during the first 2 months of therapy [4]. The reason for pyrazinamide in the absence of primary drug resistance is unclear [5], but its addition to the initial drug regimen may prevent the development of a rifampin-resistant strain. Although the emergence of isoniazid- and rifampin-resistant strains of *M. tuberculosis* is undoubtedly rare, consideration should be given to prevention by the addition of another drug during the initial 2 months of treatment.

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Fatal Queensland Tick Typhus

COLLEAGUES—Queensland tick typhus (QTT), a tickborne rickettsial disease caused by *Rickettsia australis*, has been accepted as a distinctive clinical entity for >40 years. However, clinical data on this uniquely Australian disease are surprisingly limited. Only 9 cases have been published [1–5] since Andrew et al. [6] first described QTT in 12 Australian soldiers on training exercises in North Coastal Queensland during World War II. Death or serious complications have not previously been described even though only 1 patient received effective antirickettsial treatment. Thus authors of textbooks and many Australian clinicians assume QTT is always a mild disease.

In August 1989 a 68-year-old healthy white man from Mossman, Queensland, discovered a tick attached to his body. It was removed and not further identified. Oral amoxicillin was prescribed. Seven days later he experienced fever and chills followed by vomiting, headache, weakness, and a maculopapular rash. A generalized seizure occurred, and he was hospitalized. Thereafter his rash became petechial, and he was noted to have a crusted skin lesion on his forehead, thrombocytopenia, hypoprothrombinemia, acidosis, obtundation, progressive acute renal failure, bilateral pulmonary infiltrates (thought to be due to aspiration), and abnormal liver function tests. Penicillin, gentamicin, tetracycline, metronidazole, and, terminally, acyclovir were administered intravenously. He died of progressive multiple organ failure on the 14th day of illness. Sera obtained 7 and 14 days after onset later showed a microimmunofluorescence antibody (IFA) titer rise against *R. australis* antigen from <1:64 to 1:1024. During the same period, *Proteus* Ox-2 agglutinin titers rose from <1:40 to 1:640 and Ox-19 from 1:20 to 1:80, but his Ox-K titers remained negative (<1:20). Rickettsial isolation was not attempted nor was a glucose-6-phosphate dehydrogenase level determined. Paired serologic testing for Q fever, leptospirosis, dengue, measles, Ross River virus, and murine and scrub typhus was negative.

Q fever, murine typhus, scrub typhus, and QTT occur in Australia. The clinical and laboratory features of the latter three overlap considerably. All three may produce skin rash, myalgias, fever, headache, and thrombocytopenia. Murine typhus characteristically lacks an eschar; however, eschars may also be absent in some patients with scrub typhus [7] and in QTT [1–6]. Tick contact is not a consistently recognized feature of QTT; only 15 (76%) of the 21 previously reported cases had a history of tick bite. *Proteus* agglutinins do not reliably distinguish the three infections. For example, as many as half the patients with scrub typhus lack *Proteus* Ox-K agglutinins [8]; similarly, *Proteus* Ox-2 and Ox-19 agglutinin titers are not consistent markers of infection in spotted fever and typhus group infections. Early antimicrobial treatment may blunt *Proteus* agglutinin reactions. Specific rickettsial serologies such as the IFA test are necessary to distinguish these infections. Since QTT and scrub typhus can be fatal, therapy should never be withheld while diagnostic studies are undertaken.

Recent active surveillance efforts suggest that QTT is frequently not reported to Australian health departments. Furthermore, mild or atypical cases are likely not recognized or serologically confirmed

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(unpublished data). Thus the incidence of QTT may be higher and the complete clinical spectrum broader than previously reported.

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Posttraumatic Ankle Joint Infection with *Arcanobacterium haemolyticum*: A Case Report

COLLEAGUES—*Arcanobacterium haemolyticum*, recently reclassified from *Corynebacterium haemolyticum* [1], could easily be misidentified in the routine laboratory as a coryneform organism or a streptococcus. Difficulties in correctly identifying this organism may lead to failure to appreciate its disease-producing potential. We report the isolation of *A. haemolyticum* in pure culture from the ankle joint of an adult woman who developed infection after a kick on the medial malleolus.

A 24-year-old black woman presented to the trauma unit of our hospital with a painful swollen right ankle. She had been kicked over the medial malleolus by her boyfriend during an argument. There was a small abrasion in the medial malleolar region and radiographs revealed a bimalleolar fracture with talar shift (Weber B fracture). The fracture was set, a below-knee plaster cast applied, and the patient discharged.

A week later, at follow-up, the patient complained of severe pain over the ankle. Removal of the cast revealed a large septic wound over the medial malleolus (figure 1) and a swollen ankle joint. Exploration of the ankle joint under general anesthesia revealed a further fluctuant area over the lateral malleolus, which was incised and drained; the pus collected was submitted for microbiologic investigation.

The joint was irrigated with saline and the fractures transfixed with Kirschner wires. Subsequent wound healing was satisfactory, and 2 weeks later a successful skin graft was done.

A. haemolyticum was isolated from the pus specimen in pure culture. The organism yielded minute creamy-white nonhemolytic colonies on horse blood agar after overnight incubation in air with 6% CO_2 at 37°C.

After a further 24 h of incubation, the colonies demonstrated a narrow zone of β-hemolysis. Microscopic examination of a Gram's stained smear showed slender gram-positive bacilli that appeared to exhibit rudimentary branching. The catalase test was negative and no hydrogen sulfide was produced in the triple sugar iron tube. The organism was unable to liquefy gelatin, reduce nitrates to nitrites, or hydrolyse urea. Acid was produced in serum sugars from glucose, maltose, lactose, and sucrose. Xylose and mannitol were not fermented. Antibiotic disk sensitivity testing (Stokes' method) showed the isolate to be sensitive to penicillin, ampicillin, oxacillin, erythromycin, co-trimoxazole, chloramphenicol, clindamycin, cefamandole, fusidic acid, and vancomycin.

The patient was discharged after 2 more weeks of hospitalization and was seen at regular monthly visits. Nine months after the initial injury she had a stiff ankle with fibrous ankylosis and was ambulant using a walking stick.

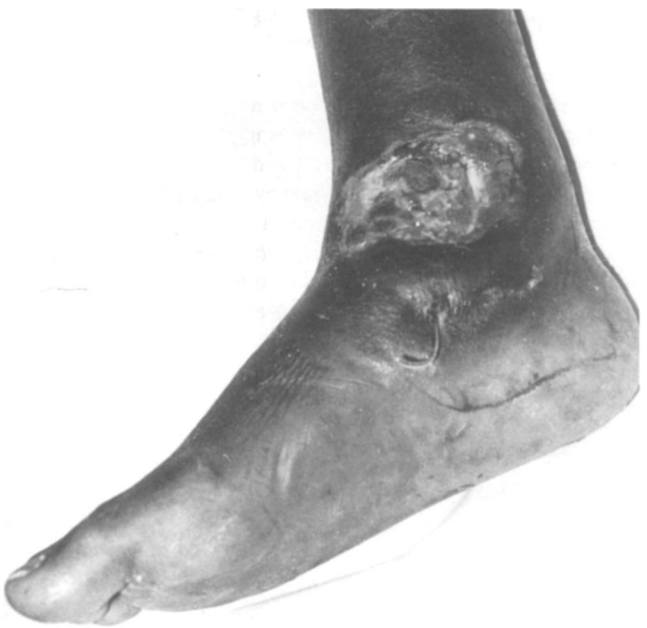

Figure 1. Large septic wound over medial malleolus.

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