MAJOR ARTICLE

Outbreak of *Aeromonas hydrophila* Wound Infections Associated with Mud Football

Hassan Vally,[1,2] Amanda Whittle,[3] Scott Cameron,[2] Gary K. Dowse,[1] and Tony Watson[1]

[1]Communicable Disease Control Branch, Department of Health, Perth, [2]National Centre for Epidemiology and Population Health, Australian National University, Canberra, and [3]South West Population Health Unit, Department of Health, Bunbury, Australia

On 16 February 2002, a total of 26 people presented to the emergency department of the local hospital in the rural town of Collie in southwest Western Australia with many infected scratches and pustules distributed over their bodies. All of the patients had participated in a "mud football" competition the previous day, in which there had been ~100 participants. One patient required removal of an infected thumbnail, and another required surgical debridement of an infected toe. *Aeromonas hydrophila* was isolated from all 3 patients from whom swab specimens were obtained. To prepare the mud football fields, a paddock was irrigated with water that was pumped from an adjacent river during the 1-month period before the competition. *A. hydrophila* was subsequently isolated from a water sample obtained from the river. This is the first published report of an outbreak of *A. hydrophila* wound infections associated with exposure to mud.

Members of the genus *Aeromonas* are facultative anaerobic, nonsporulating gram-negative bacilli that are ubiquitous inhabitants of fresh and brackish water [1]. *Aeromonas* species have been found in a variety of aquatic environments, including lakes, rivers, streams, springs, rainwater, swimming pools, and seawater, and have also been isolated from tap water and soil [2–4]. These species have been recognized as pathogens of fish, reptiles, and amphibians for many decades, but it is only recently that they have been recognized as significant human pathogens [1]. In humans, infections caused by *Aeromonas* species generally result in either acute or chronic gastrointestinal illness, septicemia in immunosuppressed individuals, or water- or soil-associated traumatic wound infections [4, 5].

Aeromonas wound infections are most commonly caused by *Aeromonas hydrophila* and have been reported after accidental puncture of the skin followed by exposure to contaminated water or soil [4–8]. These infections occur sporadically and infrequently, and they are more common in warmer climates [5, 7]. Wound infections caused by *A. hydrophila* often progress rapidly and may require surgical debridement or the amputation of limbs or digits [6]. Fatal *Aeromonas* wound infections in healthy adults have also been reported [9, 10]. Treatment of *Aeromonas* wound infections is complicated by the fact that members of this genus are universally resistant to penicillin (the result of the presence of chromosomal β-lactamase), rendering standard empirical antibiotic treatment for common streptococcal or staphylococcal wound infections ineffective [11].

In this report, we describe an unusual outbreak of wound infections caused by *A. hydrophila* in individuals participating in a "mud football" competition in a small rural town in the southwest of Western Australia. An investigation was conducted to ensure that appropriate antibiotic therapy was administered to patients, to identify factors contributing to the outbreak, and to add to our knowledge of the clinical features of *A. hydrophila* wound infections.

PATIENTS AND METHODS

Background. Collie is a small rural town of ~8500 residents situated 200 km south of Perth, the capital of Western Australia. On Sunday, 17 February 2002, a total

Received 20 June 2003; accepted 3 December 2003; electronically published 29 March 2004.

Financial support: Western Australia Department of Health and Commonwealth Department of Health and Aging (jointly funded scholarship to H.V.).

Reprints or correspondence: Dr. Gary Dowse, Communicable Disease Control Branch, Dept. of Health, Western Australia, PO Box 8172, Perth Business Centre, Perth WA 6849, Australia (Gary.Dowse@health.wa.gov.au).

Clinical Infectious Diseases 2004; 38:1084–9

1058-4838/2004/3808-0007$15.00

of 26 persons who had participated in a charity mud football competition in Collie on the previous day presented to the emergency department at Collie Hospital with infected scratches and pustules over their torsos and limbs. Most persons reported 20–30 lesions, with some reporting >100 lesions. One patient required removal of an infected thumbnail at the emergency department, and another required surgical debridement of an infected toe in the hospital the next day. Swab samples were obtained from lesions of 2 patients at the emergency department, and *A. hydrophila* was identified from cultures of these swabs 2 days later. A third swab specimen obtained from the patient requiring surgical debridement also grew *A. hydrophila.* Anecdotally, at least 16 mud football players, in addition to the 26 who presented at the emergency department, visited their medical practitioners with similar symptoms, but no further data were obtained from these individuals.

Eleven adult and 4 youth teams consisting of a total of ~100 people were involved in the mud football competition. The games were played between 1:30 pm and 4:00 pm in the afternoon on a midsummer day in which the maximum temperature reached 26°C. Two football fields were used simultaneously for a round-robin competition, with each game consisting of two 15-min halves. A Rugby Union competition was played, which involved considerable physical contact, including players tackling and wrestling each other for the ball in the mud (figure 1). Children who did not participate in the mud football competition were provided with their own mud pool.

Case series. All 26 patients (or their parents) who presented to the emergency department at Collie Hospital on Sunday, 17 February, were interviewed. A questionnaire was used for the interview that addressed the clinical features and exposure of the patients to mud and to river water. Other data collected included the estimated number and location of skin lesions, other presenting symptoms, preexisting medical conditions, and current systemic antibiotic treatment. Patients who had been prescribed systemic antibiotics before the identification of *A. hydrophila* were advised to contact their medical practitioner to ensure that they were taking the recommended antibiotics for treatment of *Aeromonas* infection. These patients were also followed-up to determine whether their antibiotic treatment was altered.

Environmental investigation. An inspection of the mud football fields, the adjacent Collie River, and the irrigation equipment was performed by the local environmental health officer. In addition, a water sample was obtained from the river near the inlet pipe for the irrigation pump and tested for temperature, pH, and bacterial pathogens.

Laboratory methods. Swab samples of skin lesions were plated onto horse blood agar plates. After overnight incubation at 35°C, oxidase-positive colonies were further identified with the API 20E biochemical identification system (BioMerieux).

Figure 1. A game of mud football in Collie, Western Australia (15 February 2002; used by permission of Janine Kay, copyright 2002).

Antibiotic susceptibility testing of clinical isolates was completed using the NCCLS agar dilution method [12].

To test water samples for *Aeromonas* species, 100 mL of water was filtered through a 0.45-μm nitrocellulose membrane. This membrane was then placed on a horse blood agar plate containing ampicillin (5 mg/L). After incubation overnight at 37°C, oxidase-positive colonies were further identified with the API 20E biochemical identification system.

RESULTS

Public health management of the infection outbreak. After being notified of the outbreak of wound infections early on Monday, 18 February, the Collie environmental health officer compiled a list of mud football participants so that they could be telephoned, assessed, and advised about the appropriate management of their lesions. The next morning, putative *Aeromonas* species were reported by the Collie microbiology laboratory and sent to the Public Health Reference Laboratory in Perth for speciation and antibiotic susceptibility testing. The Communicable Disease Control Branch in Perth was notified by the Reference Laboratory of the cultures positive for *Aeromonas* species and began to coordinate the response to this outbreak with the regional public health unit.

The primary objective of the public health response was to ensure that all patients with *Aeromonas* infection were identified and provided with appropriate treatment and that medical practitioners and the public were alerted to the possibility of *Aeromonas* wound infections due to mud football or exposure to river water. Letters and *Aeromonas* infection fact sheets were faxed to local medical practitioners advising them to review their treatment of any mud football participants they had seen with skin infections, particularly any antibiotic therapy that was administered. The treatment recommended for suspected

Aeromonas skin infections was oral ciprofloxacin, oral trimethoprim-sulfamethoxazole, or intravenous ceftriaxone. Patients who had presented to the emergency department on Sunday were interviewed and were advised to contact their medical practitioner for reassessment of their antibiotic therapy. The Communicable Disease Control Branch also released a local media statement warning the public about the potential for serious infections after exposure to mud or untreated water supplies and e-mailed all local microbiology laboratories to advise them to be alert for the presence of *Aeromonas* species in wound isolates.

Antibiotic susceptibility. The resistance profiles of the 3 clinical isolates of *A. hydrophila* were identical. These isolates were found to be resistant to amoxicillin, meropenem, oral cephalosporins (cefaclor and cephalexin), cephalothin, and colistin and were susceptible to norfloxacin, ciprofloxacin, gentamicin, tobramycin, amikacin, trimethoprim, ceftriaxone, ceftazidime, amoxicillin–clavulanate potassium, ticarcillin disodium–clavulanate potassium, aztreonam, cefepime, and nitrofurantoin.

Case series. Eighteen male and 8 female participants presented to the emergency department on the Sunday after the mud football competition (table 1). The median age was 17 years (range, 3–43 years). Every patient was exposed to mud during the mud football competition. In addition, all of the patients were directly exposed to river water after mud exposure. Anecdotally, most patients were reported to have bathed in the river after playing in the mud; however, many patients also showered with river water before bathing in the river.

Table 1. Characteristics of patients presenting to the emergency department with *Aeromonas* skin infections associated with a game of "mud football."

Patient	Sex	Age in years	Preexisting cuts	Nonlesion symptoms[a]	Empirical antibiotic treatment	Definitive antibiotic treatment
1	M	19	Yes	Yes	Cephalexin	Not changed[b]
2	M	20	No	Yes	Cephalexin	Cotrimoxazole
3	M	17	No	No	None	None
4	F	40	No	No	Erythromycin	Ciprofloxacin
5	M	9	No	Yes	Erythromycin	Ciprofloxacin
6	F	39	No	Yes	Cephalexin	Ciprofloxacin
7	M	6	Yes	No	Erythromycin	Not changed[b]
8	M	9	Yes	Yes	Erythromycin	Cotrimoxazole
9	F	4	No	Yes	Flucloxicillin	Cotrimoxazole
10	M	17	No	Yes	Cephalexin	Cotrimoxazole
11	M	21	No	Yes	Erythromycin	Ciprofloxacin
12	M	41	No	Yes	Cephalexin	Not changed[b]
13	M	16	No	Yes	None	None
14	F	3	No	Yes	Cephalexin	Not changed[b]
15[c]	M	18	No	Yes[d]	Flucloxacillin	Ceftriaxone (iv)
16[c]	F	30	No	Yes	Cephalexin	Cotrimoxazole
17	M	16	No	Yes	Flucloxacillin	Cotrimoxazole
18[c]	F	43	No	Yes[e]	None	Ciprofloxacin
19	M	8	No	No	Erythromycin	Ciprofloxacin
20	M	11	No	Yes	Erythromycin	Ciprofloxacin
21	F	5	No	Yes	Flucloxacillin	Not changed[b]
22	F	7	No	Yes	Flucloxacillin	Not changed[b]
23	M	17	No	Yes	Cephalexin	Not changed[b]
24	M	16	No	Yes	Dicloxacillin	Ciprofloxacin
25	M	20	No	Yes	Dicloxacillin	Not changed
26	M	18	No	Yes	Erythromycin	Cotrimoxazole

[a] Included ⩾1 of the following: rash, malaise, myalgia, fever, rigors, headache, nausea, sore throat, and earache.
[b] Infection resolved.
[c] Cultures of swab specimens were positive for *Aeromonas hydrophila*.
[d] Surgical debridement of toe required.
[e] Thumbnail was removed.

The reported locations of lesions were the legs (77%), arms (58%), torso (35%), back (23%), chest (19%), buttocks (8%), feet (8%), head (4%), and face (4%). The emergency department physicians who treated these patients reported that only scratches and abrasions were infected (i.e., there were no infected lesions on intact skin) and that up to 50% of all scratches were infected in some patients (M. J. Birch and B. Saharay, personal communication).

Twenty-two players (85%) reported symptoms in addition to infected lesions, including rash (69%), malaise (46%), fever (35%), headache (35%), myalgia (31%), nausea (31%), rigors (8%), sore throat (4%), and earache (4%). Although rash was reported by a large number of patients, attending physicians did not substantiate this, suggesting that patients confused their numerous cuts and abrasions with the presence of a rash. None of the players who presented to the emergency department reported any immunocompromising illnesses.

Antibiotic therapy was prescribed for 23 (88%) of the 26 patients presenting to the Collie Hospital emergency department (table 1). In all of these patients, the empirically provided antibiotic therapy was unlikely to be effective against *A. hydrophila* infection. After reassessment of their clinical status, 15 (65%) of the 23 patients had their antibiotic regimen changed. The treatment in 8 patients was not changed, because their skin infections were resolving or had resolved. Of the 3 patients who were not initially prescribed antibiotics, 1 (patient 18) was provided ciprofloxacin after reassessment by her doctor. In addition, the patient who required toe surgery (patient 15) was administered intravenous ceftriaxone therapy after initially being treated with flucloxicillin by emergency department physicians.

Environmental investigation. The environmental health officer reported that the mud football fields were prepared by plowing them to a depth of 500–600 mm and then irrigating them with water from the adjacent Collie River with sprinklers. At this time, the Collie River was low and had pooled as a result of low rainfall levels (>30% below average; Bureau of Meteorology, Perth, Western Australia) during the previous 12 months. Water was pumped onto the fields with an irrigation pump and PVC pipes that formed part of an orchard irrigation system >25 years old. The fields were irrigated each evening for 1 month before the mud football competition, with the amount of watering increased a few days before the event to saturate the fields.

The fields were originally used to grow fruit trees, but these trees had been removed, and the fields had been fallow for 2 years. The soil consisted of pea gravel and contained a stubble that was a mixture of wild oats, wild turnips and radishes, field grasses, and weeds. *A. hydrophila* was cultured from the sample of river water obtained from near the irrigation pipe inlet at the time of the outbreak. The pH of the river water was 7.5, and the water temperature near the irrigation pipe was 23°C. The surface water temperature of the parts of the river that received more sun exposure was ~30°C.

DISCUSSION

This report is the first description, to our knowledge, of an outbreak of cutaneous wound infections attributable to *A. hydrophila*. Exposure to contaminated mud is likely to have been the source of infection, although exposure of skin lesions to contaminated river water may also have played a role in this outbreak. Assuming all patients presenting to the emergency department had *A. hydrophila* infections, as their clinical presentation suggested, the attack rate for this outbreak was at least 26%. Given that at least 16 other players with similar lesions were reported to have visited general practitioners, an attack rate of >40% is possible. Patients reported up to 100 infected lesions and pustules distributed over their body, and over one-half reported systemic symptoms, including fever, malaise, myalgia, headache, and nausea. Two patients also developed complications requiring surgical intervention.

We could identify only 1 other report in the English language literature of an outbreak of skin infections associated with exposure to mud [13]. In this outbreak, college students were reported to have developed perifolliculitis caused by Enterobacteriaceae after participation in a mud-wrestling social event. A subsequent case-control study indicated that trauma to the skin was a significant risk factor for infection after mud wrestling. Likewise, trauma to the skin is a well-documented risk factor for *Aeromonas* wound infections associated with exposure to water [6, 8]. In the current outbreak, multiple cuts and abrasions, caused primarily by gravel and stubble in the football fields, are likely to have played an important role in facilitating *Aeromonas* wound infections. The presence of a large number of lesions on the arms and legs, which would have been most frequently abraded during play, supports this hypothesis.

The method in which the mud was prepared for mud football probably played an important role in this outbreak. The playing field was irrigated with river water for a month before the competition, with watering increased a few days before the event to saturate the field. During this period, daytime temperatures were warm, with maximum temperatures generally >25°C, and this may have provided an ideal environment for the growth of *Aeromonas* species in the soil. A previous study has shown that *Aeromonas* species can grow rapidly in soil when conditions are favorable (and when nutrients are available) [14]. Survival curves in soil were characterized by a rapid increase in cell numbers by several logs that lasted 1–2 weeks after initial contamination of the soil. Despite a decrease in the number of viable cells that occurred after this period of rapid growth, all of the strains studied were still present 140 days

after initial contamination. Of importance, it was also shown that the virulence factors of *Aeromonas* species were preserved after growth in soil [14]. Unfortunately, in the current investigation, soil samples were not obtained at the time of the outbreak to confirm the presence of *Aeromonas* species in the mud football fields.

In attributing causes for the current outbreak, it is worthwhile to compare the inaugural mud football competition held in March 2001 (in which there were no adverse effects reported) with the one conducted in February 2002. Although numbers of *Aeromonas* species in natural aquatic environments normally increase in the summer [15], in the summer of 2001–2002, there had been below-average rainfall, and the river level was very low and had pooled, which may have further elevated the levels of *Aeromonas* species in the river at the time of the 2002 competition. In addition, in 2002, the football field was watered for a whole month, but in 2001, the field was only watered for a few days before the event, providing less of an opportunity for *Aeromonas* organisms to multiply. Furthermore, an old irrigation pump and piping was used to water the field in 2002, whereas watering in 2001 was completed manually with a free-standing pump and hose. Thus, the possibility that the irrigation system may also have been a source of *Aeromonas* species in this outbreak cannot be discounted, because this organism has been reported to adhere to water distribution pipe surfaces [16].

A major concern regarding *Aeromonas* infections is that they may mimic streptococcal or staphylococcal soft-tissue infections, because they are potentially highly pathogenic and are resistant to penicillin, ampicillin, flucloxacillin, carbenicillin, and cefazolin [7, 17, 18]. Consequently, the standard empirical antibiotic therapies for wound infections are ineffective against *Aeromonas* infection, for which the recommended therapy in Australia is cefotaxime, ceftriaxone, or ciprofloxacin [18]. The pathogenicity of *Aeromonas* infections appears to be due to the action of several extracellular toxins that result in a very short incubation period and rapid progression of infection [4, 19, 20]. Consequently, delays in the administration of appropriate antibiotics to individuals infected with *Aeromonas* species increase the risk of serious sequelae, especially in those who are immunocompromised [4]. Importantly, in this outbreak, antibiotic therapy was modified relatively early after the identification of *A. hydrophila* in wound cultures.

Fortunately, mud football competitions or other mud sports are infrequent events, and, on the basis of this outbreak, we suggest that they should be discouraged. However, if they are to be conducted, appropriate strategies to prevent wound infections should be adopted. Clearly, the soil from which mud is prepared should be as free as possible of abrasive material likely to cause cuts and scratches in participants. In addition, mud should be prepared using treated water, or at least water from flowing rivers, to minimize microbial contamination. It would also be advisable to water the fields as close in time as possible to the commencement of any event, so as to minimize the opportunity for bacteria to multiply. And finally, it is recommended that event organizers provide warm showers with treated water and disinfectant for immediate antiseptic treatment of wounds.

In conclusion, there are several public health lessons that stem from this outbreak. First, organizers and local municipalities should be aware of, and consider the risks of, wound infections associated with these types of events before giving approval for them to proceed. Second, organizers should provide participants with written advice alerting them to the risks, and recommended management, of wound infections. It is also important that doctors are educated to suspect *Aeromonas* species when there is potential contamination of a wound by water or soil. Finally, there is a need to develop safe mud-making guidelines for similar events.

Acknowledgments

We thank our colleagues at the South West Population Health Unit, the Environmental Health Division of the Department of Health, and at the PathCentre Division of Microbiology and Infectious Diseases in Western Australia, for their help with this investigation; Ros Rabjones, Colin Wheadon, and Anne Foyer, for their assistance with the outbreak investigation; and Professor Tom Riley, for reviewing the manuscript.

References

1. Mandell GL, Douglas RG, Bennett JE, Dolin R, eds. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. 5th ed. Philadelphia: Churchill Livingstone, **2000**.
2. Hazen TC, Fliermans CB, Hirsch RP, Esch GW. Prevalence and distribution of *Aeromonas hydrophila* in the United States. Appl Environ Microbiol **1978**; 36:731–8.
3. Burke V, Robinson J, Gracey M, Peterson D, Partridge K. Isolation of *Aeromonas hydrophila* from a metropolitan water supply: seasonal correlation with clinical isolates. Appl Environ Microbiol **1984**; 48:361–6.
4. Semel JD, Trenholme G. *Aeromonas hydrophila* water-associated traumatic wound infections: a review. J Trauma **1990**; 30:324–7.
5. Kelly KA, Koehler JM, Ashdown LR. Spectrum of extraintestinal disease due to *Aeromonas* species in tropical Queensland, Australia. Clin Infect Dis **1993**; 16:574–9.
6. Isaacs RD, Paviour SD, Bunker DE, Lang SD. Wound infection with aerogenic *Aeromonas* strains: a review of twenty-seven cases. Eur J Clin Microbiol Infect Dis **1988**; 7:355–60.
7. Gold WL, Salit IE. *Aeromonas hydrophila* infections of skin and soft tissue: report of 11 cases and review. Clin Infect Dis **1993**; 16:69–74.
8. Weber CA, Wertheimer SJ, Ognjan A. *Aeromonas hydrophila*—its implications in freshwater injuries. J Foot Ankle Surg **1995**; 34:442–6.
9. Fulghum DD, Linton WR, Taplin D. Fatal *Aeromonas hydrophila* infection of the skin. South Med J **1978**; 71:739–41.
10. Bloch T, Hochstetler M, Waller BF, Clark SA. *Aeromonas hydrophila* wound infection associated with myonecrosis and gas gangrene. Indiana Med **1987**; 80:1090–2.
11. Jones BL, Wilcox MH. *Aeromonas* infections and their treatment. J Antimicrob Chemother **1995**; 35:453–61.

12. NCCLS. Methods for dilution antimicrobial susceptibility tests for bacteria that grow aerobically. 6th ed. Approved standard M7-A6. Wayne, PA: NCCLS, **2003**.
13. Adler AI, Altman J. An outbreak of mud-wrestling–induced pustular dermatitis in college students: *Dermatitis palaestrae limosae.* JAMA **1993**; 269:502–4.
14. Brandi G, Sisti M, Schiavano GF, Salvaggio L, Albano A. Survival of *Aeromonas hydrophila, Aeromonas caviae* and *Aeromonas sobria* in soil. J Appl Bacteriol **1996**; 81:439–44.
15. Kaper JB, Lockman H, Colwell RR, Joseph SW. *Aeromonas hydrophila:* ecology and toxigenicity of isolates from an estuary. J Appl Bacteriol **1981**; 50:359–77.
16. Assanta MA, Roy D, Montpetit D. Adhesion of *Aeromonas hydrophila* to water distribution system pipes after different contact times. J Food Prot **1998**; 61:1321–9.
17. Bulger RJ, Sherris JC. The clinical significance of *Aeromonas hydrophila:* report of two cases. Arch Intern Med **1966**; 118:562–4.
18. Therapeutic guidelines: antibiotic. 12th ed. North Melbourne, Australia: Therapeutic Guidelines, **2003**.
19. Brook I, Rogers J, Rollins DM, Coolbaugh JC, Walker RI. Pathogenicity of *Aeromonas.* J Infect **1985**; 10:32–7.
20. Houston CW, Chopra AK, Rose JM, Kurosky A. Review of *Aeromonas* enterotoxins. Experientia **1991**; 47:424–6.