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INSIDE

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Leptospirosis During Adventure Travel

SPECIAL FEATURE

On aug. 20, 2000, the ecochallenge sabah 2000 expedition race began in Malaysian Borneo. This event attracted 76 four-person teams from 26 countries, and included 36 teams from the United States and five teams from Canada. The EcoChallenge race is an intense, multisport event requiring participants to trek through jungles, swim in open water, canoe and kayak in rivers and oceans, mountain bike, scuba dive, spelunk, and climb their way through 320 miles of Borneo wilderness. In the words of the EcoChallenge promotional material "competitors will navigate through ancient caves and paddle along winding rivers in indigenous Sampan canoes where herds of elephants, monkeys, crocodiles, and even the rare Sumatran rhino can be seen. Teams will trek and mountain bike along dense rain forest trails while orangutans and ancient tribes of once fierce headhunters will curiously watch their passing. Teams will sail through tropical seas to magical coral-fringed islands using the native Perahu outrigger canoes and even dive down to an underwater coral reef checkpoint. Teams will negotiate swift jungle rivers and rappel down cascading waterfalls using fixed ropes."[1] The event ran from Aug. 20 to Sept. 3 with participants taking anywhere from 6-12 days to complete the course, racing nonstop. Forty-four teams completed the race. Further details about EcoChallenge can be found at www.ecochallenge.com.

Beginning in early September, racers began to present with an acute febrile illness to health care professionals in their home countries. Reports to the GeoSentinel network[2] and to the Centers for Disease Control (CDC)[3] quickly began to accumulate, and helped to describe the nature of the illness and the extent of involvement. Of 153 athletes interviewed by the CDC up to late October, 68 (44%) met the case definition of an illness characterized by fever with at least two of the following symptoms: chills, myalgias, headache, diarrhea, or conjunctivitis (M Cetron, CDC, personal communication). Thirty-seven percent of case patients were hospitalized with no deaths occurring. The typical clinical syndrome included fever and myalgias with proteinuria, mildly elevated liver enzymes, and an increased serum CK level. Based on the characteristic clinical syndrome combined with positive serology in 13 of 27 (48%) U.S. case patients, a diagnosis of leptospirosis was made. Other clinically similar tropical diseases, including malaria, were ruled out. Among multiple potential exposures, participants encountered severely flooded rivers beginning on Aug. 25. Exposure during the river swim was significantly associated with illness.

COMMENT BY DAVID R. HILL, MD, DTM&H

Leptospirosis has been previously reviewed in *Travel Medicine Advisor Update*.[4-6] It is an uncommon, but well-recognized cause of acute febrile illness in both travelers and native inhabitants of temperate and tropical areas of the world.[7-9] *Leptospira* spp.

are motile spirochetes that infect both domestic and wild animals. Dogs, livestock, and rats are the most commonly infected animals throughout the world. Organisms can survive for long periods in the kidneys of animals and then are excreted in urine, contaminating water, mud, or moist soil. When humans come into contact with the organism by swimming in or drinking contaminated water, or being covered with mud, as can easily occur during adventure travel, organisms penetrate mucous membranes or cuts and abrasions and establish infection. Heavy rainfall facilitates the spread of organisms, because as water saturates the environment, leptospires become washed into surface water. During times of flooding, there have been well-documented increases in leptospirosis. Fiji and Thailand are currently experiencing outbreaks. Flooding in Nicaragua in 1995[10,11] and in Guatemala, Honduras, and Nicaragua in 1998, in association with Hurricane Mitch, led to a marked increase in cases. Leptospirosis is also well-recorded in adventure travelers occurring in river rafters in Thailand[7] and Costa Rica.[12] In the summer of 1998, triathletes in Illinois experienced leptospirosis following their swim in a rain-swollen lake.[5,13] The outbreak in triathletes affected 11% of participants; based on preliminary information, this current outbreak in Sabah had an extremely high attack rate of more than 40%.

Following an incubation period of four days to 2-3 weeks, the illness begins abruptly and is characterized by fever, chills, myalgias, and headache. Conjunctivitis, abdominal pain, vomiting, and diarrhea are also seen. A severe illness known as Weil's disease with renal and hepatic, and rarely pulmonary involvement, can be life threatening.

Although treatment of mild illness is controversial, the CDC was recommending therapy of mild disease with doxycycline 100 mg twice daily for a week.[3] Severely ill, hospitalized patients should be treated with intravenous penicillin.[14] Evidence from U.S. troops stationed in Panama indicated that prophylaxis with 200 mg of doxycycline weekly is effective.[15]

There are several points for travel medicine experts. The first point is to consider leptospirosis in the differential diagnosis of an acute febrile illness in returned travelers, particularly if they have had fresh water exposure through recreational activities such as diving, swimming, or river rafting, or through occupational exposure with work in rice fields, sewer systems, or with handling of potentially infected animals.

The second is to inform travelers of leptospirosis if their plans would put them at risk and to become involved as advisors to tour groups or adventure racing promoters to provide accurate information about tropical disease risk. There has to be improved communication between the travel industry and travel medicine providers. Organizers of adventure travel may not emphasize the risk of disease in favor of promoting the exciting nature of their trip. Indeed, leptospirosis had occurred several times previously in adventure racers, some of whom were racing again in 2000 EcoChallenge, but it does not appear that this disease was adequately considered by race organizers.[16] Racers may not focus on tropical disease risk during their period of intense training and be more concerned about accident or injury.

Third, if a traveler will be at risk for leptospirosis, they should consider prophylaxis with doxycycline 200 mg weekly. It will be of interest to see if EcoChallenge racers who may have been taking doxycycline either for chemoprophylaxis of leptospirosis or malaria were protected from illness.

Finally, the benefits of a global surveillance system for tropical or travel-related disease with rapid dissemination of information are clearly illustrated with this outbreak. Within days, the GeoSentinel network had identified cases presenting to three sites in three different countries. In conjunction with CDC and WHO, information was provided via tropical and travel medicine e-mail list serves and the Internet (CDC web site [www.cdc.gov/travel/], EcoChallenge web site, and ProMed[17]), so that physicians could identify illness and participants could receive appropriate evaluation and treatment. Earlier in 2000, this surveillance system helped to rapidly identify an outbreak of W-135 meningococcal disease in religious pilgrims who traveled to Mecca for the annual Hajj.[18] Thus, travel medicine health professionals should consider joining the American Society of Tropical Medicine and Hygiene (www.astmh.org) and/or the International Society of Travel Medicine (www.istm.org) so that they can participate in this global information network. ❖

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The Chief Resident Presenting with a Coral Cut Injury

CASE REPORT

By Marc A. Ciampi, MD, and Andre N. Sofair, MD, MPH

A 30-year-old previously healthy male presented with pain in his left knee and left groin. Five days prior to presentation, he had scraped his exposed left knee against a large brain coral while snorkeling in the Caribbean, near the island of Aruba. He noted local erythema, slight pruritus, and a small abrasion. After irrigating the wound, the patient used topical antibacterial cream daily. He remained afebrile with no evidence of local or systemic complaints. He denied significant pain, increasing erythema, or wound drainage. On the day of presentation, the patient developed notable groin tenderness and mild left-sided lumbar pain along with increasing knee pain. He denied fever, chills, night sweats, or fatigue. He had no significant past medical history and was taking no medications. He had no known allergies and denied recent ingestion of shellfish.

The patient was afebrile with normal vital signs and general appearance. Physical examination of his knee was notable for the presence of a raised, slightly erythematous rash showing a dermatograph of brain coral, and a 2 mm abrasion with no significant drainage or fluctuance. The joint was normal with no evidence of effusion or inflammation. The left leg revealed tender inguinal lymphadenopathy without lymphangitis. Abdominal examination showed mild left upper quadrant and left costovertebral angle tenderness; a spleen tip was not palpated.

He was empirically started on an oral fluoroquinolone antibiotic, levofloxacin, to treat his soft tissue infection. Oral doxycycline was added one day later to ensure adequate coverage of various marine microorganisms. He was treated for 10 days with resolution of his groin tenderness and back pain after two days of therapy. Over three weeks, the rash resolved completely without complications.

Discussion

Contact with coral or "coral cuts" may produce significant and sometimes dramatic cutaneous reactions.[1,2] Manifestations include localized erythema, urticara, and occasional pruritus.[3] The local reaction can be a response to coral nematocysts, contamination of the wound site with microparticulate coral and calcium carbonate, possible bacterial infection, or toxin effects.

Although on a worldwide basis staphylococci and streptococci remain the most common causes of soft tissue infections, vibrios, and some *Aeromonas* spp. are virulent waterborne organisms that may infect wounds sustained in a marine environment.[1-8] *Erysipelothrix rhusiopathiae*, coliforms such as *Escherichia coli*, and *Mycobacterium marinum*, *M. balnei*, or *Pseudomonas* spp. are also capable of producing localized infections after exposure to salt water.[6] Wound infections acquired in this environment may also be polymicrobial.[7,9]

Ecology and Epidemiology

The halophilic *Vibrio* spp. are naturally free-living aerobic inhabitants of marine environments. These

organisms have been found in Europe, Asia, Australia, South America, and North America.[10] In North America, they have been recovered from the waters of the Gulf coast, the entire East Coast from Florida to Maine, the California and Washington State coasts, and from the waters around Hawaii. Halophilic vibrios have been found in both water and marine sediments, adherent to plankton, or absorbed onto mollusks and crustaceans.[11] *Vibrio* spp. are taken up by filter-feeding molluscs such as oysters, clams, mussels, and scallops achieving concentrations as high as 10^6 bacteria per gram of oyster during periods of warm water temperatures. Bacteria are also found in the intestines of some estuarine fish, which may transport them between oyster beds or serve as a source of wound infections.[12]

Vibrio spp. reside in ocean water or marine estuaries within a wide range of salinity (1-34 parts per thousand). Organisms have been isolated from brackish lakes and even from the Great Salt Lake.[13] A salinity greater than 25 parts per thousand has adverse effects on their survival.

Intolerant of cold conditions, *Vibrio* spp. thrive during the summer and fall months, but they may also survive the winter months in marine sediment.[5,14-18] *Vibrio* spp. are found in zones where there is decreased dissolved oxygen concentrations, possibly reflecting increased nutrient concentrations in such areas. Vibrios are rarely found in the open ocean, likely due to colder water temperatures, the absence of nutrients, the higher hydrostatic pressures, and the relatively higher salinity.[18]

Vibrio infections are acquired either by the consumption of contaminated food and water or through skin and soft tissue injuries.[4] The primary food sources for acquisition are raw/undercooked oysters or other seafoods.[4,19] In those with skin and soft-tissue infections, nearly all report prior recreational or occupational exposure to sea water or marine organisms.[9]

Clinical Presentations

Three major presenting clinical syndromes have been described for vibrios including gastroenteritis, soft tissue infection, and septicemia. There have been additional case reports of vibrio-associated otitis media, pneumonitis, keratitis, meningitis, and endometritis.[4,13]

Soft tissue infections caused by noncholera vibrios may present as one of two distinct clinical entities, primary vibrio cellulitis, or secondary cellulitis following primary bacteremia.[4] Direct cutaneous inoculation from abrasions, lacerations, or puncture wounds may result in primary vibrio cellulitis. With the exception of *V. cholerae* O1, primary vibrio cellulitis has been associated with all known *Vibrio* spp.[1,5,20-23] In hospitalized patients with vibrio wound infections, the majority are caused by *V. vulnificus* (43%), followed by *V. parahaemolyticus* (29%) and *V. alginolyticus* (18%). The case fatality for *V. vulnificus* was 11%, and for *V. parahaemolyticus* it was 5%.[9]

Wound infections range from mild, limited disease to rapidly progressive, necrotizing infections.[9,10,13,19] Virulence may be related to the organisms' capsular polysaccharide and lipopolysaccharide. Many vibrios also produce degradative toxins and enzymes. These include chitinases, which allow vibrios to colonize the exoskeletons of marine zooplankton, as well as hemolysins and metalloproteases, which break down tissues at the site of colonization. Vibrios also produce siderophores that scavenge iron from host transport proteins, transferrin and lactoferrin. This may account for the increased virulence of *Vibrio* spp. in patients with iron overload states.[12]

Cellulitis usually occurs within 24-48 hours but can occur as early as four hours, or as late as 12 days after exposure.[20,24,25] Fever occurs in 45-80% of primary cellulitis cases.[4] Infected wounds are usually erythematous or ecchymotic, swollen and notably tender with little to no purulent discharge.[4,24] Vesicles or bullae with secondary necrotic centers and necrotizing fasciitis have also been described.[9,12,16,24,26]

Patients with a history of liver disease, renal disease, chronic illness or immunodeficient states are at considerably increased risk of generalized sepsis following cellulitis.[4,9,19,20,27-30] In cirrhosis, it has been suggested that porto-systemic shunting may allow vibrios to bypass the hepatic reticuloendothelial system. Additionally, liver disease predisposes such patients to complement deficiencies, impaired chemotaxis, and phagocytosis.[31] Iron overload states also contribute to fulminant vibrio infection. Increased bioavailability of free iron, found in patients with hemochromatosis, may stimulate bacterial growth and metabolism. In addition, iron overload may impair normal host phagocytic activity, increasing susceptibility to infections.[12,31,32] Septicemia occurs in 15% of patients with primary soft tissue infections and contributes to the high case-fatality rates. In those with bacteremia, the rate is 32%; without hematogenous involvement, the case fatality rate is only 1%.[9]

Secondary cellulitis in the setting of primary septicemia associated with *Vibrio* spp. carries a 32-50% mortality rate.[4,9,10,19,33] In these cases, there is usually a preceding history of having eaten raw/undercooked oysters or other seafood ingestion.[4,19] These individuals develop generalized, metastatic, macular, or papular lesions in the setting of primary bacteremia. Culture-positive cutaneous lesions have been reported with bacteremia caused by *V. vulnificus*, *V. cholerae* non-O1, *V. parahaemolyticus*, and rarely *V. alginolyticus*.[4,34]

Treatment of Coral-related Soft Tissue Infection

Local wound care should include soap and water, fol-

lowed by aggressive irrigation and debridement with saline solution and hydrogen peroxide to remove foreign material from the site and prevent secondary infection or granuloma formation.[35]

Given the potential polymicrobial nature of coral-related infections, broad-spectrum antibiotics should be considered. For those with obvious cellulitis, or as a prophylactic measure in those with abnormal immune systems, antibiotics with a spectrum of activity against staphylococci and streptococci should be used. In addition, antibiotics that are effective against halophilic *Vibrio* spp. should be administered.

Vibrio spp. frequently produce beta-lactamases and are often resistant to various beta-lactam antibiotics. The beta-lactam inhibitor, sulbactam, does not completely render these organisms susceptible to ampicillin. They are often resistant to cephalothin, cefuroxime, and cefoperazone, but sensitive to cefotaxime, ceftazidime, aztreonam, and imipenem.

Although the fluoroquinolones, ofloxacin and norfloxacin, are effective against these organisms, ciprofloxacin appears to have the greatest activity with an MIC_{90} of approximately 0.25 mg/L. Trimethoprim/sulfamethoxazole is effective, as are the tetracyclines and chloramphenicol.[36] For those with devitalized tissue or fasciitis, surgical intervention is indicated. This may be particularly important when dealing with infections caused by *Vibrio damsela*, which were reviewed in the July/August issue of *TMA Update* for our readers.[16,21,37]

(*Dr. Ciampi is a Clinical Instructor in Medicine at Yale University and Dr. Sofair is an Assistant Clinical Professor of Medicine at Yale University with the Emerging Pathogens Program, New Haven, Conn.*) ❖

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Typhoid Fever in a Child Returning from Pakistan

CASE REPORT

By Dana Shaked

A 10-year-old boy had been in excellent health and recently returned from a 10-week trip to Karachi, Pakistan. He had been born in Karachi where he lived until age 4. Since that time, both he and his family (parents and a sister aged 13) returned for visits about every year and a half. Approximately 10 days following this return trip, the patient experienced fevers reaching 40.5°C associated with chills, headaches, nausea, and vomiting. Prior to his departure from the United States, he received one dose of mefloquine malaria prophylaxis and the first dose of hepatitis A vaccine. He had not received malaria prophylaxis in the past and he had never received any form of typhoid vaccine. All his standard routine childhood immunizations were up-to date. There were no pets at home or any unusual contact with animals during his trip. The febrile episodes appeared responsive to acetaminophen.

The boy was taken to his pediatrician after four days of continued symptoms. His white blood count (WBC) was 8900 cells/µL and malaria smears were negative. No one else in his immediate or extended family had become symptomatic. He was then referred to Yale New Haven Hospital where his WBC count was 10,400 and the differential showed 67 band forms, 18 segmented neutrophils, 13 lymphocytes, and two monocytes with a sedimentation rate of 40 mm/h. Stool and urine cultures were repeated.

On arrival at our hospital, the patient was alert, oriented cooperative, and did not appear to be in acute distress. Dehydration was noted to be less than 5%. Body temperature was 40°C with shaking chills, a heart rate of 96 beats/min, and a respiratory rate of 22 breaths/min. Blood pressure was 110/70 mm Hg. There were no rashes noted except for truncal psoriasis that had been known to his dermatologist. There were no meningeal signs including nuchal rigidity nor evidence of lymphadenopathy. Lungs were clear and heart sounds were normal. His abdomen was soft, nontender, and was not distended. There was no guarding or rebound tenderness; no organomegaly was noted. There was full range of motion in all extremities.

On the day of admission, laboratory studies showed a WBC count of 8900, with 84% segmented neutrophils. Hemoglobin was 12.4 g/dL and hematocrit was 36%, with a platelet count of 261,000/µL. Serum electrolytes and liver function tests were all within normal limits and malaria smears were negative. Blood cultures grew Gram-negative rods identified as a group D *Salmonella* spp. (*Salmonella typhi*). Organisms were sensitive to all antibiotics tested including nalidixic acid, ampicillin, chloramphenicol, ceftriaxone, ciprofloxacin, and trimethoprim/sulfamethoxazole. The patient had been started on intravenous ceftriaxone and hydration to correct fluid losses from vomiting. The urine culture was negative and stool examinations revealed concomitant infection with *Blastocystis hominis* and *Entamoeba histolytica* trophozoites.

While on our ward, the patient experienced 3-4 loose nonbloody stools per day during the first few days and had one episode of vomiting following a heavy meal. Temperature continued to reach as high as 40°C several times each day. He defervesced after three days of intravenous antibiotics and was sent home on the fourth day with oral ciprofloxacin, 500 mg orally, twice a day, for 10 days to treat his typhoid fever, and oral metronidazole for seven days to treat his amebiasis. (*Ms. Shaked is a fourth-year student, Technion Faculty of Medicine, Haifa, Israel.*)

Editor's Comment—*The case presented by one of our visiting students at Yale should be viewed in light of the following data presented by our Associate Editor, Maria D. Mileno. She clearly indicates the relevance of travelers as the source of most imported typhoid fever in the*

United States. The site of travel is critical and the sources of imported typhoid fever are changing as are patterns of antibiotic sensitivity. ❖

New Sources and Spread of Multidrug Resistant *Salmonella typhi*

ABSTRACT & COMMENTARY

Synopsis: *A careful assessment of bacterial isolates obtained from patients with acute typhoid fever in the United States has shown clear shifts in the sources of this infection, as well as evolving and worrisome antimicrobial resistance patterns of Salmonella typhi.*

Source: Akers M-L, et al. Laboratory-based surveillance of *Salmonella* serotype *typhi* infections in the United States. *JAMA* 2000;283:2668-2673.

Comprehensive information on both the incidence and sources of antimicrobial resistant *S. typhi* isolates in the United States are presented in this article. Akers and associates review the recent surveillance data for typhoid fever (TF) cases and bacterial isolates reported to the Foodborne and Diarrheal diseases Branch of the U.S. Centers for Disease Control. Each year, approximately 16 million new cases of TF occur worldwide and about 300 laboratory-confirmed cases are reported in the United States. While the number of reported U.S. TF cases has remained fairly stable during the last 20 years, the proportion of TF cases ascribed to travel outside the United States has increased from 62% in 1975-1984 to 81% in the current study, which covers 1996-1997. During this same timeframe, the proportion of TF cases attributed to exposures in Mexico decreased from 46% in 1985 to 6%. In contrast, cases associated with exposures on the Indian subcontinent (India, Pakistan, and Bangladesh) increased from 25% in 1985 to 57% currently.

In a follow-up, this trend was confirmed in a presentation at the 38th meeting of the Infectious Diseases Society of America in New Orleans in September.[1] The data from the National Antimicrobial Resistance Monitoring System (NARMS) for enteric bacteria were obtained in a 1999 surveillance study in which up to 63% of travel-associated *Salmonella typhi* isolates were documented as acquired in India. From June 1996 to May 1997, 350 *S. typhi* isolates were tested for their antimicrobial sensitivity patterns. Seven percent were resistant to nalidixic acid, the parent drug for the fluoroquinolone class of antibiotics. This resistance pattern represents the potential harbinger of decreased ciprofloxacin sensitivity, which will have major clinical significance.

From 1999 to the present, ongoing surveillance detected 166 patients with TF. Two-thirds of cases were associated with travel. The mean age was 22, and 51% were female. Twenty-five percent were children. Approximately two-thirds were blood isolates and one-third were obtained from stool, with 3% of isolates from urine. Fully 19% of *S. typhi* isolates were resistant to nalidixic acid in the most recent study, although none were found to be resistant to ciprofloxacin or ceftriaxone. While there have been no reported U.S. cases of ciprofloxacin-resistant *S. typhi*, nalidixic acid resistance has clearly been increasing—that is not good news.

To make matters worse, a research letter published in the *Lancet* has already described increasing numbers of treatment failures using ciprofloxacin for the treatment of TF in the United Kingdom, even when strains appeared to be sensitive.[2] In 1998, strains with decreased sensitivity to ciprofloxacin accounted for 32 of 151 *S. typhi* isolates (21%). All strains with decreased sensitivity to ciprofloxacin were still fully sensitive to cephalosporin antibiotics, such as ceftriaxone or cefotaxime. The majority had been obtained from patients with a history of a recent return trip from India or Pakistan. Apparently, nalidixic acid-resistant strains of *S. typhi* with decreased susceptibility to ciprofloxacin are now endemic in both India and Pakistan.

COMMENT BY MARIA D. MILENO, MD

Multidrug resistance of *S. typhi* (MDRST) to traditional antimicrobial agents (i.e., ampicillin chloramphenicol, trimethoprim-sulfamethoxazole) has been increasing at an alarming pace. From 1985 to 1989, just 0.6% of U.S. strains represented MDRST. From 1996 to 1997, nearly one in every five *S. typhi* isolates (17%) were MDRST, and 7% (including 12 strains of MDRST) were also resistant to nalidixic acid, the parent drug for the fluoroquinolones, including ciprofloxacin. By 1999, resistance to nalidixic acid had reached 19% among U.S. isolates of *S. typhi*. Reports of TF treatment failures with ciprofloxacin from the United Kingdom suggest we may see this occurring soon in the United States. Where to look?

The answers are becoming clearer as the majority of reported TF cases in the United States are travel-associated and the majority (61%) of those are now clearly related to travel on the Indian subcontinent. Travel medicine consultants might consider offering typhoid vaccine, even to individuals embarking upon short stays in that region, given the high proportion of all cases originating

from there.

Children are at high risk for TF and those younger than 18 years accounted for 44% of all cases, including 42% of travel-associated cases in the earlier surveillance study. The median age of children in that group was 8 years. Neither MDRST nor nalidixic acid-resistant strains were more common in children than in adults; however, fluoroquinolones are not approved for use in children, which leaves us with limited therapeutic options for them.

During the pre-antibiotic era, TF case-fatality ratios approached 20%. Treatment with effective antimicrobial agents has reduced rates to less than 1%. Should antimicrobial therapeutic options fail, the more serious manifestations of TF may emerge. TF can include hepatitis, characterized by jaundice and elevated transaminase levels, which occur in approximately 10% of affected adults and children. On rare occasions, encephalopathy results. Patients with TF who have both jaundice and encephalopathy may present with a picture that mimics fulminant hepatic failure resulting from other conditions, notably hepatotropic viruses and drugs. This form of hepatitis is described in the reference originating from the Department of Gastroenterology at St. John's Medical College and Hospital, Bangalore, India.[3] ❖

References

1. Steinberg EB, et al. Antimicrobial resistance of *Salmonella typhi* in the United States—the National Antimicrobial Monitoring System, 1999. Abstracts of the IDSA Annual Meeting 2000, New Orleans, September 2000, page 40, abstract #3.
2. Threlfall J, et al. Ciprofloxacin-resistant *Salmonella typhi* and treatment failure. *Lancet* 1999;353: 1590-1591.
3. Kamath PS, et al. Differentiation of typhoid fever from fulminant hepatic failure in patients presenting with jaundice and encephalopathy. *Mayo Clin Proc* 2000;75: 462-466.

CME Questions

7. Reported typhoid fever in the United States has all of the following characteristics at present except one. Which statement is false?

a. Most *S. typhi* isolates are now resistant to nalidixic acid.
b. Travel-associated isolates in the United States now predominate.
c. Most travel-associated isolates originate from the Indian subcontinent.
d. Reported numbers of U.S. typhoid fever cases have remained relatively stable over the last 20 years.
e. The proportion of *S. typhi* isolates originating from Mexico has been steadily decreasing in the United States.

8. Which one of the following statements regarding vibrio infections is true?

a. *Vibrio* spp. are generally isolated from fast moving fresh water streams in the tropics.
b. Soft tissue infections are indolent and induce a granulomatous response to the causative bacteria
c. Iron overload states, such as cirrhosis, predispose to serious vibrio infections.
d. The first generation cephalosporins remain the agents of choice for serious vibrio infections.
e. Risk of vibrio infection is unrelated to seafood or shellfish ingestion.

9. Prevention of leptospirosis during travel can be attempted by which one of the following?

a. Weekly prophylaxis with doxycycline
b. Weekly prophylaxis with mefloquine
c. Receiving vaccination for hepatitis A and B simultaneously
d. Not drinking untreated mountain water
e. Not handling domestic animals