

debilitating sequelae and can be life threatening.

Michael E. Schachter,*
Lindsay Wilcox,* Neil Rau,†
Deborah Yamamura,*‡
Shirley Brown,§
and Christine H. Lee*

*McMaster University, Hamilton, Ontario, Canada; †Halton Healthcare Services, Oakville-Trafalgar Memorial Site, Oakville, Ontario, Canada; ‡MDS Diagnostic Services, Toronto, Ontario, Canada; and §Toronto Public Health Laboratories, Toronto, Ontario, Canada

References

- Graves MH, Janda MJ. Rat-bite fever (*Streptobacillus moniliformis*): a potential emerging disease. *Int J Infect Dis.* 2001;5:151-4.
- Morse SS. Factors in the emergence of infectious disease. *Emerg Infect Dis.* 1995;1:7-15.
- Freels LK, Elliott SP. Rat bite fever: three case reports and a literature review. *Clin Pediatr (Phila).* 2004;43:291-5.
- Salmon RL, McEvoy MB. Rat-bite fevers. In: Zoonosis. Palmer SR, Soulsby S, Simpson DIH, editors. Toronto: Oxford Medical Publications; 1998. p. 187-90.
- Hambridge SJ, Ogle JW. Index of suspicion. Case 1. Diagnosis: rat-bite fever. *Pediatr Rev.* 2001;22:95-103.
- Parker F Jr, Hudson HP. The etiology of Haverhill fever (erythema arthriticum epidemicum). *Am J Pathol.* 1926;2:357-79.
- Washburn R. *Streptobacillus moniliformis* (Rat-bite fever). In: Mandell GL, Bennett JE, Dolin R, editors. Principles and practice of infectious diseases. 5th ed. Toronto: Churchill Livingstone Inc.; 2000. p. 2422-4.
- Lambe DW Jr, McPhedran AM, Mertz JA, Stewart P. *Streptobacillus moniliformis* isolated from a case of Haverhill fever: biochemical characterization and inhibitory effect of sodium polyanethol sulfonate. *Am J Clin Pathol.* 1973;60:854-60.
- Committee on Infectious Diseases, American Academy of Pediatrics. 2003 Red book: Report of the Committee on Infectious Diseases. 26th ed. Elk Grove Village, IL: The American Academy of Pediatrics; 2000. p. 482-3.

Address for correspondence: Christine H. Lee, Department of Pathology and Molecular Medicine, McMaster University, St. Joseph's Healthcare, 50 Charlton Ave East, Hamilton, Ontario L8N 4A6, Canada; email: cleec@mcmaster.ca

Cutaneous Injury and *Vibrio vulnificus* Infection

To the Editor: *Vibrio vulnificus* infection is transmitted by eating contaminated seafood or by exposure to seawater through an open wound (1). Among immunocompromised persons, especially those with chronic liver disease, *V. vulnificus* can cause a life-threatening illness characterized by blistering skin lesions, necrotizing fasciitis, and septic shock (2-5). However, the epidemiology and risk factors for severe forms of *V. vulnificus* infection among healthy persons are less well documented (4-6).

We conducted a retrospective clinical record review of *V. vulnificus* infections in persons admitted to all public hospitals from January 1, 2003, through August 31, 2005, in Hong Kong, which has a population of >6 million persons. We defined a case-patient as a patient with culture of *V. vulnificus* from any clinical specimen. A record search of clinical case notes was performed through a computerized clinical management system maintained by the Hospital Authority, which manages all public hospitals in Hong Kong. For each case-patient identified, we reviewed demographic data (age, sex, occupation, residence), clinical and laboratory data (date of onset, symptoms, laboratory investigation findings, diagnosis, outcome), and potential risk factors (past health and possible source of exposure) associated with the case. We compared previously healthy patients with patients who had predisposing medical conditions in terms of demographic profile, clinical signs and symptoms and outcome, and known exposure factors. Mann-Whitney U tests, χ^2 tests, or Fisher exact tests were used to detect significant differences ($\alpha = 0.05$).

We identified 29 cases over the 32-month study period. Twenty-two

(76%) patients had disease onset from May through August, the summer season in Hong Kong. Fifteen (52%) cases were in men, and the median age was 70 years (range 24-82 years). Fifteen (52%) patients had underlying illnesses that were known to predispose them to *V. vulnificus* infection, including chronic liver disease (30%), chronic renal failure (15%), diabetes mellitus (7%), and thalassemia major (3%). Fourteen (48%) patients were previously healthy. No significant differences in age and sex were found.

Among the 14 previously healthy patients, the consequences of *V. vulnificus* infection included necrotizing fasciitis (70%), severe cellulitis (7%), primary septicemia (14%), and gastroenteritis (7%). Two patients who had necrotizing fasciitis and 1 patient with primary septicemia died. Compared with patients with predisposing medical conditions, patients with a history of good health had a higher (but not significant) proportion of necrotizing fasciitis (70% vs 47%, $p = 0.12$), a lower proportion of septicemia (14% vs. 27%, $p = 0.26$), and an equal number of severe cases of cellulitis (7% vs. 7%). Furthermore, fewer patients with a history of good health died than did patients with predisposing illnesses (21% vs. 33%, $p = 0.25$). The median duration between symptom onset and admission for all patients was 1 day (range 0-3 days), with no significant difference between the 2 groups.

A history of cutaneous injury or a skin prick from a seafood part (e.g., fish fin, shrimp spine, or crab leg) was significantly more common among previously healthy patients than among patients with predisposing illnesses (70% vs. 27%, $p = 0.02$). Ten (83%) of the 12 previously healthy patients with necrotizing fasciitis and septicemia reported a history of cutaneous injury. The corresponding proportion was significantly lower (31%) among patients with predisposing medical conditions ($p = 0.01$). Among

all 29 patients, a history of eating raw oysters or other raw or undercooked seafood before illness onset was uncommon and was only reported by 1 patient. Although *V. vulnificus* has not been proven as the cause of gastroenteritis, Hseuh et al. have suggested that such results might have occurred because patients with diarrhea seldom sought care from a large teaching hospital or saved stool samples for investigation (7).

V. vulnificus infection was first reported in humans in 1979 (1). Since then, most case reports have focused on immunocompromised persons and their risk from eating raw oysters and their risk from eating raw oysters among (4–6). Our study found that a considerable proportion of *V. vulnificus* infections in Hong Kong occur among healthy persons. Furthermore, severe forms of the infection, such as necrotizing fasciitis and septicemia, are relatively common among healthy persons, although they may cause fewer deaths than they do among persons with predisposing medical conditions. Among healthy persons, *V. vulnificus* infection is most likely associated with a history of cutaneous injury caused by handling seafood, which can allow the bacteria to enter the body through an open wound. The risk of exposure is more important in this locality than in other areas where swimming or eating raw oysters and undercooked seafood are the major risk factors (4,6–8), possibly because fresh seafood is widely consumed, and seafood is easily accessible in wet markets in Hong Kong. Our study shows that the risk is higher during the summer, which is consistent with the fact that *V. vulnificus* is more active in warmer temperatures (9). We suggest that all persons, even healthy persons, exercise caution to avoid injury while handling seafood. Physicians should consider possible *V. vulnificus* infection when diagnosing a rapidly progressive skin and soft tissue infection in a healthy person who reports an injury from handling seafood.

**P.H. Chung,* S.K. Chuang,*
Thomas Tsang,* Lai Wai-man,†
Raymond Yung,‡ and Janice Lo‡
for the Collaborative Study Group
on *Vibrio vulnificus* Infection in
Hong Kong**

*Department of Health, Hong Kong Special Administrative Region, People's Republic of China; †Hospital Authority, Hong Kong Special Administrative Region, People's Republic of China; and ‡Centre for Health Protection, Hong Kong Special Administrative Region, People's Republic of China

References

1. Blake PA, Merson MH, Weaver RE, Hollis DG, Heublein PC. Disease caused by a marine *Vibrio*. Clinical characteristics and epidemiology. *N Engl J Med*. 1979;300:1–5.
2. Klontz KC, Lieb S, Schreiber M, Janowski H, Baldy L, Gunn RA. Syndromes of *Vibrio vulnificus* infections: clinical and epidemiologic features in Florida cases, 1981–1987. *Ann Intern Med*. 1988;109:318–23.
3. Mitra AK. *Vibrio vulnificus* infection: epidemiology, clinical presentation, and prevention. *South Med J*. 2004;97:118–9.
4. Gholami P, Lew SQ, Klontz KC. Raw shellfish consumption among renal disease patients. A risk factor for severe *Vibrio vulnificus* infection. *Am J Prev Med*. 1998;15:243–5.
5. Haq SM, Dayal HH. Chronic liver disease and consumption of raw oysters: a potentially lethal combination—a review of *Vibrio vulnificus* septicemia. *Am J Gastroenterol*. 2005;100:1195–9.
6. Potasman I, Paz A, Odeh M. Infectious outbreaks associated with bivalve shellfish consumption: a worldwide perspective. *Clin Infect Dis*. 2002;35:921–8.
7. Hsueh PR, Lin CY, Tang HJ, Lee HC, Liu JW, Liu YC, et al. *Vibrio vulnificus* in Taiwan. *Emerg Infect Dis*. 2004;10:1363–8.
8. Tacket CO, Brenner F, Blake PA. Clinical features and an epidemiological study of *Vibrio vulnificus* infections. *J Infect Dis*. 1984;149:558–61.
9. Shapiro R, Altekruse S, Hutwagner S, Bishop R, Hammond R, Wilson S, et al. The role of Gulf Coast oysters harvested in warmer months in *Vibrio vulnificus* infections in the United States, 1988–1996. *J Infect Dis*. 1998;178:752–9.

Address for correspondence: P.H. Chung, Medical and Health Officer, Field Epidemiology Training Program, Surveillance and Epidemiology Branch, Centre for Health Protection, Department of Health, Hong Kong

Special Administrative Region, People's Republic of China; email: mo_fetp2@dh.gov.hk

Neorickettsia helminthoeca in Dog, Brazil

To the Editor: *Neorickettsia helminthoeca* causes salmon poisoning disease (SPD) in canids. SPD has been described only in the United States and the northwestern Pacific region of Canada (1). This report complements previous pathologic findings (2) and identifies SPD beyond the known disease-endemic region.

From 2001 to 2005, 20 dogs (5 mongrels and 15 beagles) showed pathologic lesions consistent with SPD. All beagles were born in coastal Florianópolis, Santa Catarina, Brazil, and later transferred to Maringá, Paraná, Brazil, for the last 3–4 years of life. Lymph nodes, spleen, liver, and intestines from 10 beagles were aseptically obtained at necropsy in Maringá and frozen at –20°C until used at the Johns Hopkins Medical Institutions in Baltimore, Maryland.

Genomic DNA was extracted from frozen tissues with QIAamp DNA Mini Kits (Qiagen, Valencia, CA, USA). DNA from *N. helminthoeca* and *Anaplasma phagocytophilum* was used as a positive control. Nuclease-free water was used as a negative control. We used gene-specific primers for *Neorickettsia* spp. 16S rRNA (*rrs*) (NeoSH-F; 5'-TAGGCCCGCGTTA-GATTAGCTTGT-3' and NeoSH-R; 5'-TACAACCCAAGGGCCTTCATCACT-3') and *N. helminthoeca* RNA polymerase β -subunit (*rpoB*) (NH-rpoB-F; 5'-TGTCTTCGAAGGCC-