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Clarification of article on Clostridium difficileassociated colitis

In the article on *Clostridium difficile*—associated Lcolitis,1 the abstract in English suggested that vancomycin be used if metronidazole is ineffective; however, in French the choice is clindamycin. Because of the incidence of the problem in Quebec, this should be clarified. Use of cholestyramine resin as a treatment option was not mentioned, the role of proton pump inhibitors in predisposing patients to infection was not explained, and no mention was made of which cleaning products health care institutions could use to kill the spores.

> -Kenneth Brown MD, CCFP Borden, Ont by e-mail

Reference

1. Hull MW, Beck PL. Clostridium difficile-associated colitis. Can Fam Physician 2004:50:1536-45.

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e wish to thank Dr Brown for his interest in our article and to address the questions and concerns raised.

Apology. First, we wish to apologize for an error that occurred in the French translation of the abstract, which stated that clindamycin is the second-line agent for treatment of Clostridium difficile-associated diarrhea. As stated in the English text, second-line therapy when metronidazole is ineffective is oral vancomycin.

Proton Pump Inhibitors and Clostridium difficile. Although antibiotic therapy has been identified as a major risk factor for development of C difficile-associated diarrhea, other factors, such

as nasogastric tube placement, gastrointestinal procedures, and intensive care stay, have also been reported to contribute to disease development.1 Antacid therapy has also been considered a possible risk factor; numerous small case-control studies, however, have not confirmed this association.²⁻⁵ Since submission of our manuscript, newly published studies have provided evidence in support of this hypothesis. A case-control study involving 207 patients found an odds ratio (OR) of 2.5 (95% confidence interval [CI] 1.5-4.2) for proton pump inhibitors (PPIs) as a risk factor in development of disease. In addition, a logistic regression model taking into account antibiotic use, cytotoxic chemotherapy, and PPIs found an OR of 43.2 (CI 5.7-330.4) compared with an OR of 17.3 (CI 2.7-113.1) when only antibiotics and chemotherapy were considered.⁶ Similarly, a recent Canadian study used a cohort of inpatients and a separate case-control study to develop adjusted ORs of 2.1 (CI 1.2-3.5) and 2.7 (CI 1.4-5.2), respectively, for use of PPIs and risk of developing disease.7 Acid suppression has been associated with other infections of the gastrointestinal tract, and the authors of these recent studies have postulated that decreased stomach acid allows survival of the vegetative C difficile organism, and thus increases the risk of colonization and subsequent disease.

Use of cholestyramine for treatment of C difficile. Cholestyramine is an anion-exchange resin and is thought to be able to bind the secreted alpha and beta toxins that cause disease. Several case reports document successful use of cholestyramine in refractory relapses^{8,9}; however, further evidence to support its use is lacking, and the resin has also been shown to bind vancomycin, requiring the drugs to be given several hours apart.10 We believe its use is, therefore, limited, and did not include it

Disinfectants. The most recent Canadian Infection Control Guidelines for washing hands and disinfection in health care do not identify any

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specific disinfectant solutions for *C difficile* spores and recommend specialized interventions only in outbreaks. 11 Several solutions have been identified as effective at reducing environmental contamination. An unbuffered hypochlorite solution (500 ppm chlorine) was used in one study to decrease contamination from 31.4% to 16.5% of sampled sites.¹² Another study used a 0.04% formaldehyde and 0.03% glutaraldehyde solution to reduce environmental contamination from 13% to 3%.13 The American College of Gastroenterology Practice Guidelines for management of C difficile diarrhea recommend use of alkaline glutaraldehyde, sodium hypochlorite, or ethylene dioxide as effective disinfectants for vegetative and spore forms of C difficile.14 The Centre for Disease Control and Healthcare Infection Control Practices Advisory Committee guidelines for environmental infection control in health care facilities recommend disinfection with hypochlorite-based germicides in addition to meticulous cleaning to counter environmental contamination with C difficile within hospitals.15

> —Dr Mark Hull, MD, FRCP Vancouver, BC —Dr Paul Beck, PHD, MD, FRCP Calgary, Alta by e-mail

References

- $1.\ Bignardi\ GE.\ Risk\ factors\ for\ \textit{Clostridium\ difficile}\ infection.\ \textit{J\ Hosp\ Infect}\ 1998; 40 (1): 1-15.$
- 2. Shah S, Lewis A, Leopold D, Dunstan F, Woodhouse K. Gastric acid suppression does not promote clostridial diarrhoea in the elderly. QJM 2000;93(3):175-81.
- 3. Lai KK, Melvin ZS, Menard MJ, Kotilainen HR, Baker S. Clostridium difficile-associated diarrhea: epidemiology, risk factors, and infection control. Infect Control Hosp Epidemiol 1997:18(9):628-32.
- 4. Nelson DE, Auerbach SB, Baltch AL, Desjardin E, Beck-Sague C, Rheal C, et al. Epidemic Clostridium difficile-associated diarrhea: role of second- and third-generation cephalosporins, Infect Control Hosp Epidemiol 1994:15(2):88-94.
- 5. Brown E, Talbot GH, Axelrod P, Provencher M, Hoegg C. Risk factors for Clostridium difficile toxin-associated diarrhea. Infect Control Hosp Epidemiol 1990;11(6):283-90.
- 6. Cunningham R, Dale B, Undy B, Gaunt N. Proton pump inhibitors as a risk factor for Clostridium difficile diarrhoea. J Hosp Infect 2003;54(3):243-5.
- 7. Dial S, Alrasadi K, Manoukian C, Huang A, Menzies D. Risk of Clostridium difficile diarrhea among hospital inpatients prescribed proton pump inhibitors: cohort and case-control studies. CMAJ 2004;171(1):33-8.
- 8. Moncino MD, Falletta JM. Multiple relapses of Clostridium difficile-associated diarrhea in a cancer patient. Successful control with long-term cholestyramine therapy. Am J Pediatr Hematol Oncol 1992;14(4):361-4.
- 9. Kunimoto D, Thomson AB. Recurrent Clostridium difficile-associated colitis responding to cholestyramine. Digestion 1986;33(4):225-8.
- 10. Taylor NS, Bartlett JG. Binding of Clostridium difficile cytotoxin and vancomycin by anion-exchange resins. J Infect Dis 1980;141(1):92-7.
- 11. Hand washing, cleaning, disinfection and sterilization in health care. Can Commun Dis Rep 1998;24(Suppl 8):i-xi, 1-55 (Eng) i-xi, 1-57 (Fr).
- 12. Kaatz GW, Gitlin SD, Schaberg DR, Wilson KH, Kauffman CA, Seo SM, et al. Acquisition of Clostridium difficile from the hospital environment. Am J Epidemiol 1988;127(6):1289-94.

- 13. Struelens MJ, Maas A, Nonhoff C, Deplano A, Rost F, Serruys E, et al. Control of nosocomial transmission of Clostridium difficile based on sporadic case surveillance. Am J Med 1991;91(3B):138S-44S.
- 14. Fekety R. Guidelines for the diagnosis and management of Clostridium difficile-associated diarrhea and colitis. American College of Gastroenterology, Practice Parameters Committee, Am I Gastroenterol 1997:92(5):739-50.
- 15. Sehulster L. Chinn RY. Guidelines for environmental infection control in health-care facilities. Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR Recomm Rep 2003;52(RR-10):1-42.

Life cycle of family medicine doctors

n November 25 to 27, 2004, I, along with other Canadian family physicians, celebrated the 50th Anniversary of the College of Family Physicians of Canada.

In the past, I have reflected on the life cycle of the family as we care for our patients from birth to death. I deliver the newborn and support the grandparents as they deal with the "golden years."

As I was at the ceremony for our new Certificants, Fellows, and Life members, I thought about the life cycle of family medicine and of family doctors.

On this day, Dr W.D. (Doug) Armstrong (a past Family Physician of the Year) was receiving his life membership certificate. He had just retired from his family practice. He was my family physician and one of my mentors. I first spent time with him as a medical student, learning about family medicine in his office and at the Misericordia Hospital in Edmonton, Alta. Doug encouraged me to proceed with my career in family medicine.

On the day of the ceremony, as I received my Fellowship along with my classmates and colleagues, I pondered how we have taken over the role of our preceptors and mentors and how we are passing on the torch of family medicine as we teach and learn with medical students and family practice residents. This was just what our predecessors and mentors had wanted. They were the grandparents and we were the parents of the family medicine family.

On this same day, there were many new Certificants of our College. I had taught a number of them on their journey in family medicine, but for me, Dr Manickavasagam Sundaram's (Mani's) receiving his certification stood out. I had known him from his first days in medical school during his