JOHNS HOPKINS MEDICINE
eLITERATURE REVIEW
eInfections Review
Presented by
The Johns Hopkins University
School of Medicine
Supported by
an Educational Grant from
ViroPharma, Inc.

April 2008: VOLUME 1, NUMBER 10

Infectious Diarrhea in Adults

In this Issue...

Cases of infectious diarrhea, especially from food and water-borne sources, are common in adults, and particularly in travelers and immunocompromised individuals. In this issue we focus on new data in 4 areas within this broad field, reviewing: the pros and cons of a new medication (rifaximin) for the treatment of travelers' diarrhea, the domestic outbreak potential of enterotoxigenic *Escherichia coli* (ETEC), post-infectious complications of infectious diarrhea, and the etiologies of bacterial diarrhea in HIV-infected populations.

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- **COMMENTARY** from our Guest Author
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- **POST-INFECTIOUS IRRITABLE BOWEL SYNDROME**
- **ENTEROTOXIGENIC *E. COLI* AS A CAUSE OF LARGE FOOD-BORNE OUTBREAKS IN THE UNITED STATES**
- **CAUSES OF BACTERIAL DIARRHEA IN INDIVIDUALS WITH HIV INFECTION**

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Beth D Kirkpatrick, MD has disclosed that she serves as a principal investigator for ACE Biosciences.

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LEARNING OBJECTIVES

At the conclusion of this activity, participants should be able to:

- Describe to colleagues the indications and limitations of rifaximin use for the prevention of travelers' diarrhea
- Identify for colleagues which patients are at risk for post-infectious irritable bowel syndrome after a gastrointestinal infection
- Describe to colleagues the most common enteric bacterial pathogens in persons with HIV infection

COMMENTARY

Approximately 211-375 million cases of infectious diarrhea occur in the United States each year, resulting in 900,000 hospitalizations and 6000 deaths.[1] Food-borne diseases contribute significantly to these numbers, and are estimated to cause 6-81 million illnesses annually, with 325,000 hospitalizations and 5000 deaths.[2] Our understanding of the etiologies of community-acquired diarrhea is limited to those causative agents which can be detected with available diagnostic microbiologic cultures and assays, and may under-represent both the overall incidence of diarrhea due to infectious agents as well as the role of specific pathogens. Using the CDC's FoodNet database, pathogens currently identified as causing the most diarrhea include *Salmonella*, *Campylobacter*, *Shigella*, *E. coli* 0157:H7 and *Cryptosporidium*.[3] Etiologic agents of diarrhea for which adequate diagnostic tests are not readily available include all strains of pathogenic *E. coli*, the noroviruses, and toxigenic strains of both *Staphylococcus aureus* and *Clostridium perfringens*.[1]

Infectious diarrhea and gastroenteritis caused by food and water contamination are an ongoing concern, particularly with increasing rates of antibiotic resistance and the growing importation of fresh fruits and vegetables. Recent outbreaks have implicated tomatoes (*Shigella flexneri*), spinach (*E. coli* 0157:H7), and delicatessen food (enterotoxigenic *E. coli* [ETEC]).[4,5] Nevertheless, the incidence of *Campylobacter*, *E. coli* 0157:H7, *Salmonella*, and *Yersinia* infections have decreased substantially between 1996-2003 according to FoodNet surveillance data.[3] Fluoroquinolone use in poultry has now also been discontinued in the US, and should decrease the food-borne transmission of quinolone-resistant strains of enteric bacteria.[6] Concurrently, other transmission methods of enteric agents are becoming increasingly recognized, including zoonotic routes (pets, petting zoos, farm animals, wildlife), recreational water sources, and sexual transmission.

ETEC is a common cause of travelers' diarrhea, but is not part of the standard diagnostic workup for infectious diarrhea. As described in the article by Beatty et al (reviewed herein), ETEC may also be an under-recognized cause of large domestic outbreaks in the United States. This pathogen is responsible for up to 30-40% of travelers' diarrhea (TD) cases, although rates vary by geographic region.[7] A new non-absorbable antibiotic (rifaximin) for

the treatment of traveler's diarrhea is additionally reviewed below (Taylor et al).

Whether acquired domestically or while traveling, post-infectious sequelae of food-borne diseases and infectious diarrhea are an increasing area of interest. Reactive arthritis, Guillain-Barre syndrome, and post-infectious irritable bowel syndrome have occurred following infection with *Salmonella*, *Shigella* and *Campylobacter*. Post-infectious irritable bowel disease (PI-IBS) has been described since 1962, and it appears that this patient population differs from the larger group of individuals who suffer IBS from other causes.[8,9] The risk of PI-IBS appears to have re-invigorated the debate over use of prophylactic agents against traveler's diarrhea, although PI-IBS has less association with ETEC than with the "invasive" enteric bacteria, and prompt treatment of infection remains a reasonable approach. The meta-analysis of post-infectious bowel syndrome by Halvorson et al further provides further insight.

Infectious diarrhea in immunocompromised patients is associated with increased morbidity, and exhibits a more diverse range of pathogens than in immunocompetent individuals. Recent articles describe infectious causes of diarrhea in solid organ and stem cell transplant patients.[10,11] Bacterial causes of infectious diarrhea in persons with HIV infection have been evaluated by Sanchez and colleagues, reviewed herein.

Finally, exciting data on our understanding of infectious diarrhea will likely emerge from a new NIH-initiative to understand normal human flora "the microbiome," and its effect on human immunity, metabolism and nutrition.[12]

References

1. Thielman NM, Guerrant RL. Clinical practice. Acute infectious diarrhea. *N Engl J Med.* 2004;350(1):38-47.
2. Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. *Emerg Infect Dis.* 1999;5(5):607-625.
3. CDC: Preliminary FoodNet Data on the Incidence of Infection with Pathogens Transmitted Commonly Through Food. *MMWR Morb Mortal Wkly Rep.* 2006;(14):392-395.
4. Reller ME, Nelson JM, Molbak K, et al. A large, multiple-restaurant outbreak of infection with Shigella flexneri serotype 2a traced to tomatoes. *Clin Infect Dis.* 2006;42(2):163-169.
5. Ongoing multistate outbreak of Escherichia coli serotype O157:H7 infections associated with consumption of fresh spinach—United States, September 2006. *MMWR Morb Mortal Wkly Rep.* 2006;55(38):1045-1046.
6. Nelson JM, Chiller TM, Powers JH, Angulo FJ. Fluoroquinolone-resistant Campylobacter species and the withdrawal of fluoroquinolones from use in poultry: a public health success story. *Clin Infect Dis.* 2007;44(7):977-980.
7. Steffen R. Epidemiology of traveler's diarrhea. *Clin Infect Dis.* 2005;41 Suppl 8:S536-540.
8. Chaudhary NA, Truelove SC. The irritable colon syndrome. A study of the clinical features, predisposing causes, and prognosis in 130 cases. *Q J Med.* 1962;31:307-322.
9. DuPont AW. Postinfectious irritable bowel syndrome. *Clin Infect Dis.* 2008;46(4):594-599.
10. Kamboj M, Mihu CN, Sepkowitz K, Kernan NA, Papanicolaou GA. Work-up for infectious diarrhea after allogeneic hematopoietic stem cell transplantation: single specimen testing results in cost savings without compromising diagnostic yield. *Transpl Infect Dis.* 2007;9 (4):265-269
11. Arslan H, Inci EK, Azap OK, Karakayali H, Torgay A, Haberal M. Etiologic agents of diarrhea in solid organ recipients. *Transpl Infect Dis.* 2007;9(4):270-275.
12. Li M, Wang B, Zhang M, et al. Symbiotic gut microbes modulate human metabolic phenotypes. *Proc Natl Acad Sci U S A.* 2008;105(6):2117-2122.

RIFAXIMIN FOR THE TREATMENT OF TRAVELERS' DIARRHEA

Taylor DN, Bourgeois AL, Ericsson CD, et al. **A randomized, double-blind, multicenter study of rifaximin compared with placebo and with ciprofloxacin in the treatment of travelers' diarrhea.** *Am J Trop Med Hyg.* 2006;74(6):1060-6.

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Taylor et al. evaluated adult travelers to compare the efficacy of the non-absorbable antibiotic rifaximin to ciprofloxacin for treating travelers' diarrhea. Subjects were enrolled from travel health clinics in Peru, Guatemala, India, and Mexico. All subjects had acute diarrhea of less than 72 hours of duration, and were randomized in a 2:1:1 fashion to receive rifaximin (200 mg orally, three times daily), ciprofloxacin (500 mg orally, twice daily with a placebo once daily), or placebo (three times daily). All regimens were received for three days, and subjects documented all symptoms, including fever, in a daily diary for five days after dosing. Stool specimens for microbiologic analysis were collected pre- and post-dosing. The primary study endpoint was time to last unformed stool (TLUS), measured from the start of study medication. Secondary endpoints included "clinical wellness," treatment failures, and microbiologic eradication. Data was evaluated for all subjects, as well as two subgroups. The first compared subjects with microbiologically confirmed "non-invasive" pathogens (diarrheagenic *E. coli*, ETEC, and enteroaggregative *E. coli* [EAEC]) to groups with invasive pathogens (*Salmonella*, *Shigella*, *Campylobacter*) or no defined pathogen. The second subgroup evaluated subjects with no evidence of clinically invasive disease, defined as subjects without fever or blood in the stool. Rifaximin minimal inhibitory concentrations (MIC) were performed on microbiologic isolates obtained pre-and post-treatment with rifaximin.

Three hundred ninety-nine adult subjects were enrolled in the trial with similar baseline characteristics in each group. The isolation rate of enteric pathogens was also similar between centers (with the exception of Goa, India which had significantly more *Campylobacter* infections). No pathogen could be isolated in 36% of subjects. Overall, the primary endpoint of TLUS was significantly shorter in the ciprofloxacin (28.8 hours) and rifaximin (32 hours) groups (P values of 0.0003 and 0.0014, respectively) when compared to the placebo group (TLUS, 65.5 hours). There was no significant TLUS difference between the rifaximin and ciprofloxacin groups (P =0.35); however, more subjects in the rifaximin group had treatment failure (14.7%) than with ciprofloxacin (6.9%). Not surprisingly, in subjects with microbiologically confirmed non-invasive *E. coli* infections, the rifaximin and ciprofloxacin groups had comparable TLUS and percentages reaching clinical wellness. Data were similar for the subset of patients without fever or blood in the stool. In contrast, ciprofloxacin was superior for subjects with microbiologically-confirmed invasive pathogens. In this sub-analysis, 68% subjects receiving rifaximin reached "clinical wellness" with a TLUS of 43.7 hours, compared to the ciprofloxacin group, where 85.7% achieved wellness, with a 24.4 hour TLUS. Overall microbiologic eradication rates were 61.6% in the rifaximin group and 80.7% in the ciprofloxacin group. MIC data demonstrated that for subjects receiving rifaximin, 10 of 19 ETEC isolates recovered post-treatment had a ≥4-fold increase in the MIC to rifaximin, particularly in isolates with a relatively high baseline MIC of ≥4 µg/ml. Clinical response rates in subjects with these isolates did not differ, and adverse event rates between groups were similar.

This study demonstrates that rifaximin is a well-tolerated and effective oral antibiotic for the treatment of travelers' diarrhea in subjects infected with non-invasive pathogens (predominantly ETEC, EAEC) and for those in which a pathogen cannot be identified but is assumed to be the cause of diarrhea. The use of rifaximin in the prevention of travelers' diarrhea has also been recently published.[1] The benefits of this agent include its non-absorbable nature and the potential ability to avoid fluoroquinolones for the treatment of non-invasive pathogens (notably, 42% of the *Campylobacter* isolates in this trial were shown to have MIC ≥0.25 µg/ml to ciprofloxacin, reiterating that in some cases neither of these agents would be useful). In anticipation of concerns about rifaximin-induced resistance, the authors carefully point out that the clinical response rates of persons with non-invasive travelers' diarrhea taking rifaximin were similar regardless of MIC increases; however ETEC isolates with higher pre-treatment MICs were also more likely to persist post-therapy. The long term effect of rifaximin use on antimicrobial resistance remains to

be seen, and the acceptance of this agent by travelers' health clinics will be interesting to follow.

References

1. DuPont HL, Jiang ZD, Okhuysen PC, et al. A randomized, double-blind, placebo-controlled trial of rifaximin to prevent travelers' diarrhea. *Ann Intern Med.* 2005;142(10):805-12.

POST-INFECTIOUS IRRITABLE BOWEL SYNDROME

Halvorson HA, Schlett CD, Riddle MS. **Postinfectious irritable bowel syndrome—a meta-analysis.** *Am J Gastroenterol.* 2006;101(8):1894-1899.

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This meta-analysis by Halverson and colleagues was performed to better estimate the risk for post-infectious irritable bowel syndrome (PI-IBS) following episodes of acute gastroenteritis/diarrhea. Published studies evaluating persons with IBS after an acute intestinal illness were included in the meta-analysis if they met predetermined inclusion and exclusion criteria using standardized grading criteria and data abstraction. Subgroup analysis evaluated self-reported vs laboratory-confirmed infectious diarrhea.

One hundred-eighty-eight articles were evaluated and 8 studies involving adults, all published from 1999 through 2005, were chosen for the meta-analysis. Individual study sizes ranged from 97 to 584,626 subjects, and 6 of the 8 trials were cohort studies. In these studies, 42% of the overall population was male. Two studies were outbreak investigations (one *Shigella*, one *Salmonella*) and 2 others involved travelers. All studies demonstrated an increased risk (odds ratio greater than one), and 6 of the 8 studies had a significant positive association of IBS following gastrointestinal infection. The median prevalence of IBS in these patients was 9.8%, compared to 1.2% in the control group (p=0.01). The summary odds ratio by the Mantel-Haenszel model was 7.3 without significant heterogeneity. Laboratory-confirmed gastrointestinal infection had twice the odds of PI-IBS with a pooled odds ratio (OR) estimate of 10.9 (95% CI 6.7-17.1) vs 5.3 (95% CI 3.0-9.3) in those without microbiologically confirmed infection. Interestingly, male subjects were more likely to develop PI-IBS.

The link between infectious diarrhea/gastroenteritis and PI-IBS has received significant attention, and the association is slowly coming into better focus. This study reiterates the presence of PI-IBS as a distinct entity, rather than a subset of IBS. As shown in this work and that of others, subjects with PI-IBS differ from the general population of IBS patients. Specifically, PI-IBS appears to affect both genders equally, and is less likely to involve pre-existing psychiatric issues.[1] The association between antecedent GI infection and PI-IBS appears to exist in mixed populations, as well as travelers and those diagnosed as part of an enteric disease outbreak. The increased odds ratio found in subjects with laboratory-confirmed infections lends credence to the importance of invasive bacterial pathogens in PI-IBS. Additionally, other studies have suggested that protracted duration (>14 days) of initial illness (as a measure of severity) is a critical element in the development of PI-IBS.[2] Further work is needed to better understand the pathophysiology of PI-IBS as well as the role of specific pathogens. Until then, Halverson's report refocuses our attention on providing rapid and effective treatment of infectious diarrhea due to invasive enteric bacteria.

References

1. Spiller RC. Role of infection in irritable bowel syndrome. *J Gastroenterol.* 2007;42 Suppl 17:41-47.
2. DuPont AW. Postinfectious irritable bowel syndrome. *Clin Infect Dis.* 2008;46(4):594-9.

ENTEROTOXIGENIC *E. COLI* AS A CAUSE OF LARGE FOOD-BORNE OUTBREAKS IN THE UNITED STATES

Beatty ME, Adcock PM, Smith SW, et al. **Epidemic diarrhea due to enterotoxigenic Escherichia coli.** *Clin Infect Dis.* 2006; 42(3):329-34.

Beatty et al., investigating a large outbreak of acute diarrhea in Wisconsin, performed an embedded cohort study to determine the epidemiologic risk factors for gastrointestinal illness. The epidemic occurred in 1998 after complaints of acute gastroenteritis following events catered by a single delicatessen. A case was defined as an individual with diarrhea or abdominal pain after consumption of food provided by the delicatessen. Information on patients presenting with gastroenteritis was obtained from health departments and emergency rooms. A convenience sample of ill persons was interviewed, which included a food questionnaire. The cohort study evaluated persons who attended the largest events during the suspected time of transmission. Laboratory assessment of stool specimens were performed and included expanded bacterial cultures as well as polymerase chain reaction (PCR) to identify the heat-labile (LT) and heat-stabile (ST) toxins of ETEC, and serotyping for the O and H antigens. Food specimens and environmental cultures were also performed. Serologic responses to the *E. coli* O6 LPS (IgM, IgG) were measured and compared to controls. Univariate and multivariate analyses were performed, as well as manual stepwise logistical regression for exposure identification.

ETEC infection was linked to a single delicatessen which catered 539 events to 16,691 people over a 3-day time period. 405 events had guests that become ill. The cohort study determined that the median prevalence of illness among guests was 20%, with 3338 persons estimated to have met the case definition. Illness was characterized by a median incubation period of 50 hours and a median duration of diarrhea of 5 days. Foods common to all cohorts included prepared mayonnaise-based salads and hand-cut fruit. The delicatessen was found to have inadequate hand washing facilities as well as presumptively inadequate plumbing and refrigeration. Stool specimens from ill persons revealed LT and ST-producing ETEC, which was confirmed as an identical strain by pulsed-field gel electrophoresis. Elevated anti-O6 titers (>1:160) were found in 19 of 27 collected serum samples, but no elevated titers were found in control serum. The source of contamination was not determined.

This study represents one of the largest food-borne disease outbreaks reported to date in the United States. Based on an internal cohort study, the authors estimate that over 3300 subjects became ill due to food-borne ETEC. Because the infective dose of ETEC is large (>$10^{\wedge 8}$ CFU), food must be heavily contaminated to cause disease, and the epidemiologic findings clearly linked this outbreak to a single delicatessen. The outbreak occurred with the most common circulating strain of ETEC (serotype 0169), but a precise food source was not identified. The authors developed an anti-06 ELISA to guide diagnostics for this outbreak; however ETEC-specific diagnostics are not often part of the initial workup for food-borne outbreaks.

Although ETEC is generally considered the primary agent of travelers' diarrhea, food-borne outbreaks of ETEC in the United States have been reported. (The next largest outbreak of ETEC caused 452 cases and was described in 1980.[1]) This study reiterates that food-borne infectious diarrhea due to enterotoxigenic *E.coli* occurs domestically as well as abroad, and that ETEC infection should be considered if routine cultures are negative.

References

1. Taylor WR, Schell WL, Wells JG, et al. A foodborne outbreak of enterotoxigenic Escherichia coli diarrhea. *N Engl J Med.* 1982;306(18):1093-5.

CAUSES OF BACTERIAL DIARRHEA IN INDIVIDUALS WITH HIV INFECTION

Sanchez TH, Brooks JT, Sullivan PS, et al. **Bacterial diarrhea in persons with HIV infection, United States, 1992-2002.** *Clin Infect Dis.* 2005;41(11):1621-1627.

Sanchez et al., analyzed the incidence and etiologies of diarrheal illnesses caused by bacteria in a large cohort of HIV-infected individuals during 1992-2002. Data was extracted from the Adult/Adolescent Spectrum of HIV Disease Project, a collaboration between the Centers for Disease Control (CDC) and 11 state and local health departments. Clinical diagnosis, microbiology, and CD4+ counts were abstracted. Diarrhea was only included in the case definition if a bacterial etiology was microbiologically confirmed. Specific methods of culture or diagnosis were not recorded, and pathogenic *E. coli* were not captured. HIV-infected persons were classified as "clinical AIDS" (previous diagnosis of any opportunistic infection, regardless of CD4+ count), "immunologic AIDS" (no clinical AIDS, but CD4+ count <200 cells/mL or CD4+ <14%) or "HIV infection without AIDS" (all others). Changes in rates were examined using logistic regression analysis.

Data for 44,778 persons was evaluated and the subject characteristics (ethnicity, age, gender) reflected the US HIV-infected population. Eleven thousand three hundred twenty episodes of diarrhea were reported, with a mean annual incidence of bacterial diarrhea of 7.2 cases per 1000 person-years. Of these, 1091 (9.6%) had a confirmed bacterial origin. The most commonly identified pathogen was *Clostridium difficile*, accounting for 54% of all cases, followed by *Shigella* (14%), *Campylobacter* (13.8%), and *Salmonella* (7.4%). In subjects with "clinical AIDS", rates of infection with all bacterial pathogens were highest. For this group, *C. difficile* had an incidence of 9.6 per 1000 person-years, and infections from other bacteria 5.1 per 1000 person-years. Nevertheless, during the study period (1992-2002) rates from bacterial causes of diarrhea (not including *C. difficile*) fell significantly in all HIV-infected persons, from a rate of 5.6 cases per 1000 person-years to 1.9 (OR 0.3). Similarly, the overall rate of *C. difficile*-associated disease (CDAD) fell, but the decline was only significant in the "clinical AIDS" population.

This report identifies trends in the bacterial causes of diarrhea in HIV-infected persons and spans the time period pre-and post- use of highly active antiretroviral therapy (HAART). Reflective of the general success of HAART therapy, the overall rate of these infections has fallen in the post-HAART era. Notably, *C. difficile* constituted the largest percentage of the identified cases of bacterial diarrhea throughout the entire period of study. Interestingly, despite an overall increase in United States, rates of CDAD decreased in persons with the most advanced disease ("clinical AIDS").[1] Presumably this group of patients had both less exposure to *C. difficile* due to decreasing hospitalizations as well as reconstitutions due to HAART that permitted a better immunologic defense against clinical disease. *Shigella* species were the second most common pathogen. This contrasts with infections in the non-HIV general population, in which *Shigella* are less common than *Salmonella* and *Campylobacter*. Although 48% of the current study population were men who have sex with men (MSM), 75% of the *Shigella* cases occurred in MSM. These data reconfirm that *Shigella* species are a sexually-transmitted pathogen in HIV-infected MSM, as shown in recent outbreaks and case-control studies.[2,3]

References

1. Archibald LK, Banerjee SN, Jarvis WR. Secular trends in hospital-acquired Clostridium difficile disease in the United States, 1987-2001. *J Infect Dis.* 2004;189(9):1585-1589.
2. Morgan O, Crook P, Cheasty T, et al. Shigella sonnei outbreak among homosexual men, London. *Emerg Infect Dis.* 2006;12(9):1458-1460.
3. Aragon TJ, Vugia DJ, Shallow S, et al. Case-control study of shigellosis in San Francisco: the role of sexual transmission and HIV infection. *Clin Infect Dis.* 2007;44(3):327-34.

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At the conclusion of this activity, participants should be able to:

- Describe to colleagues the indications and limitations of rifaximin use for the prevention of travelers' diarrhea
- Identify for colleagues which patients are at risk for post-infectious irritable bowel syndrome after a gastrointestinal infection
- Describe to colleagues the most common enteric bacterial pathogens in persons with HIV infection

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- **Paul G. Auwaerter, MD** has disclosed that he has served as a consultant for Novartis, Pfizer, Ortho-McNeil, Schering-Plough, and Genzyme. He is on the Speakers' Bureau for Schering-Plough and has also disclosed that he is a Stock Shareholder for Johnson & Johnson.
- **Sara E. Cosgrove, MD, MS** has disclosed that she has received grants or research support from Merck and served on the Advisory Boards for Ortho-McNeil, Cadence Pharmaceuticals, and Theravance/Astellas.

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