LETTERS TO THE EDITOR

Aedes Mosquito Larva in the Hospital: A Note From Thailand

To the Editor—Mosquito-borne infections are increasingly important. To control mosquito-borne infections, mosquito control is needed. The *Aedes* mosquito is an important vector for many diseases including the Zika virus and dengue fever. The survey of *Aedes* mosquito larva is a routine public health practice in tropical countries.[1] Mosquito larvae can be found in many urban buildings. Hospitals can also be the setting of habitat for the *Aedes* mosquito, but this fact is mentioned infrequently. Here, I report and discuss the data from an *Aedes* mosquito larva survey in hospitals in an endemic area of Thailand (western region, 7 provinces) during the rainy season, May–June 2015. Overall, 30 hospitals were surveyed, and *Aedes* mosquito vector larva were detected in 16 hospitals (53.3%). The percentage of water-holding containers infested with larvae ranged from 0 to 30. The number of larvae-positive containers per 100 containers inspected at hospitals, the Breteaux index, ranged from 0 to 20.59. Based on these data, many hospitals can be considered the source for *Aedes*-borne infectious disease. Indeed, an important role of the medical center is to provide health care to and promote health within the community. Mosquito control in the hospital is usually a forgotten issue, but it can pose a significant problem if there is a healthcare-associated outbreak of mosquito-borne infection in the region. In the era of emerging and remerging mosquito-borne infections (eg, Zika virus infection, dengue and others), mosquito control in the hospital is necessary[2] and represents an emerging and important issue in the field of infection control and hospital epidemiology.

Viroj Wiwanitkit, MD

Affiliation: Chulalongkorn, University Bangkok, Bankok, Thailand.

Address correspondence to Professor Viroj Wiwanitkit, MD, Department of Laboratory Medicine, Faculty of Medicine, Chulalongkorn, University Bangkok, Bankok, Thailand 10330 (wviroj@yahoo.com).

Infect Control Hosp Epidemiol 2016;37:1519

 0899-823X/2016/3712-0024. DOI: 10.1017/ice.2016.212

REFERENCES

1. Benelli G, Mehlhorn H. Declining malaria, rising of dengue and Zika virus: insights for mosquito vector control. *Parasitol Res* 2016;115:1747–1754.
2. Wiwanitkit V. Zika virus infection: control and prevention. *J Chin Med Assoc* 2016;79:409.

Can Fecal Microbiota Transplantation (FMT) Eradicate Fecal Colonization With Vancomycin-Resistant Enterococci (VRE)?

To the Editor—Recently, fecal microbiota transplantation (FMT) has been attempted to eliminate colonization with multidrug-resistant organisms (MDROs), and case reports have shown considerable success.[1] In addition, FMT was effective for reducing antibiotic resistant genes in patients with recurrent *Clostridium difficile* infection (CDI),[2] which may have a significant role in MDRO decolonization. However, publication bias could exist against studies with negative findings for MDRO decolonization, and clinical evidence for extending the application of FMT remains sparse. We performed FMT to eradicate long-term vancomycin-resistant enterococci (VRE) colonization in 3 patients. Prolonged VRE colonization was documented by repeated rectal swab cultures (positive VRE on at least 4 consecutive swabs taken 1 week apart). Of these 3 patients, 2 had recurrent CDI and were treated with oral metronidazole and vancomycin (Table 1, cases 1 and 2). Another patient (case 3) remained in the hospital for isolation purposes due to VRE carriage after completion of treatment. Vancomycin resistance was confirmed using chromogenic agar and polymerase chain reaction. All cases were *Enterococcus faecium* with the *vanA* gene. Voluntary informed consent was obtained for FMT. Stools were donated by the patient's granddaughter (case 1, 18 years old), daughter (case 2, 45 years old), and son (case 3, 50 years old), respectively. The donors were healthy and no problems were identified on pre-donation screening tests; they were all negative for stool VRE. Oral vancomycin treatment was discontinued on the day before FMT (case 1 and 2), and the donor stool (100 g) in normal saline (200 mL) was transplanted to the patient via retention enema after environmental disinfection. All patients were able to retain the infusate for at least 1 hour, and there were no adverse events. In case 3, a second FMT was performed 1 day after the first FMT. All antibiotics were stopped after the FMT was conducted except in case 2, in which the patient developed pneumonia 15 days after FMT and piperacillin-tazobactam was given for 2 weeks. Patient characteristics and outcomes are summarized in Table 1. Patients 1 and 2 experienced resolution of CDI symptoms without recurrence during admission. After transferring to other facilities, 2 patients were lost to follow-up. We did not recruit additional patients because FMT did not shorten the duration of VRE carriage in these 3 patients.

Our data are supported by the Jang et al[3] case report in which FMT was performed twice to control refractory CDI. Their patient was also colonized with VRE and, despite

TABLE 1. Clinical Characteristics and Outcomes of Patients with Vancomycin-Resistant Enterococci Colonization Who Underwent Fecal Microbiota Transplantation

Case	Age, y	Sex	Route of FMT	Reason for Admission	Comorbidities	Discharged From the Hospital	Antibiotics After FMT	Duration From FMT to the Last Positive VRE
1	79	F	Enema	CDI	HTN	Yes[a]	No	12 weeks
2	72	F	Enema	Pyogenic spondylitis	DM, HTN, CDI	No[b]	Yes[c]	10 weeks
3	73	F	Enema (twice)	Septic arthritis	RA	No[d]	No	21 weeks

NOTE. FMT, fecal microbiota transplantation; CDI, *Clostridium difficile* infection; HTN, hypertension; DM, diabetes mellitus; RA, rheumatoid arthritis; VRE, vancomycin-resistant enterococci.

[a]Patient 1 was discharged from the hospital 1 month after FMT. She cleared VRE colonization 15 weeks after FMT (3 consecutive negative VRE cultures a minimum of 1 week apart at the outpatient clinic).

[b]Patent 2 was transferred to a long-term care facility 10 weeks after FMT and was lost to follow-up.

[c]Patient 2 developed pneumonia 15 days after FMT, and piperacillin-tazobactam was given for 2 weeks.

[d]Patient 3 was transferred to a long-term care facility 21 weeks after FMT and no further rectal VRE cultures were performed.

resolution of CDI, rectal VRE carriage persisted for at least 3 months. Although we used the lower delivery route (enema), Jang et al transplanted stool via both enema and the upper route (nasoduodenal tube).[3]

Ubeda et al[4] showed that reintroduction of a diverse intestinal microbiota to heavily VRE-colonized mice could eliminate VRE from the gut. Specifically, the presence of *Barnesiella* species in the intestinal tract was able to confer resistance to VRE in patients undergoing allogeneic hematopoietic stem cell transplantation.[4] Stielfel et al[5] reported that cephalosporinase-producing *Bacteroides thetaiotaomicron* prevented overgrowth of VRE and *C. difficile* in cephalosporin-treated mice. Defined microbiota transplant instead of whole stool may lead to more successful outcomes. Although these data are promising, it is not clear whether single bacterial species transfers would work in the human gastrointestinal tract.

In an industry-sponsored trial using an experimental microbiota suspension, 8 of 11 patients (73%) became VRE negative 1–6 months following FMT by enema.[6] However, the patients could have experienced spontaneous eradication. Because clearance of VRE varies widely, occurring after a median time of <3 months after discharge from the hospital without FMT and 6.5 months after antibiotics that promote VRE are discontinued,[7,8] it remains unclear whether FMT reduces the duration of VRE colonization.

Although FMT may contribute to shrinkage of the gut resistome, it does not seem to effectively shorten the duration of VRE carriage and may not be justified for the clearance of fecal colonization with VRE. Ongoing clinical trials will help resolve this issue and should help identify more effective methods of FMT against VRE colonization.

ACKNOWLEDGMENTS

Financial support. No financial support was provided relevant to this article.

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article.

Kyung Mok Sohn;[1]
Shinhye Cheon;[1]
Yeon-Sook Kim[1]

Affiliation: Division of Infectious Diseases, Chungnam National University Hospital, Chungnam National University School of Medicine, Daejeon, South Korea.

Address correspondence to Kyung Mok Sohn, MD, Division of Infectious Diseases, Chungnam National University Hospital, Chungnam National University School of Medicine, 282 Munhwa-ro, Jung-gu, Daejeon 35015, Republic of Korea (medone@cnuh.co.kr).

Infect Control Hosp Epidemiol 2016;37:1519–1521

 0899-823X/2016/3712-0025. DOI: 10.1017/ice.2016.229

REFERENCES

1. Young VB, Hayden MK. Environmental management in the gut: fecal transplantation to restore the intestinal ecosystem. *Infect Dis* (Lond) 2016;48:593–595.
2. Millan B, Park H, Hotte N, et al. Fecal microbial transplants reduce antibiotic-resistant genes in patients with recurrent *Clostridium difficile* infection. *Clin Infect Dis* 2016;62:1479–1486.
3. Jang MO, An JH, Jung SI, Park KH. Refractory *Clostridium difficile* infection cured with fecal microbiota transplantation in vancomycin-resistant *Enterococcus* colonized patient. *Intest Res* 2015;13:80–84.
4. Ubeda C, Bucci V, Caballero S, et al. Intestinal microbiota containing *Barnesiella* species cures vancomycin-resistant *Enterococcus faecium* colonization. *Infect Immun* 2013;81:965–973.
5. Stiefel U, Nerandzic MM, Pultz MJ, Donskey CJ. Gastrointestinal colonization with a cephalosporinase-producing *Bacteroides* species preserves colonization resistance against vancomycin-resistant Enterococcus and *Clostridium difficile* in cephalosporin-treated mice. *Antimicrob Agents Chemother* 2014;58:4535–4542.
6. Dubberke E, Jones C. Clearance of vancomycin-resistant Enterococcus concomitant with administration of a microbiota-based drug targeted at recurrent *Clostridium difficile* infection. In: 25th European Congress of Clinical Microbiology and Infectious Diseases (ECCMID), April 25–28 2015. Copenhagen, Denmark: Poster 0912.

7. Sohn KM, Peck KR, Joo EJ, et al. Duration of colonization and risk factors for prolonged carriage of vancomycin-resistant enterococci after discharge from the hospital. *Int J Infect Dis* 2013;17:e240–e246.
8. Shenoy ES, Paras ML, Noubary F, Walensky RP, Hooper DC. Natural history of colonization with methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant Enterococcus (VRE): a systematic review. *BMC Infectious Dis* 2014;14:177.

Increase in Prevalence of KPC-2–Producing *Klebsiella pneumoniae* Recovered From Respiratory Secretions of Intensive Care Patients—*Getting a Free Ride* on a Menacing Colistin Resistance

To the Editor—In recent years, carbapenem-resistant Enterobacteriaceae (CRE) have emerged as important nosocomial pathogens, while *Klebsiella pneumoniae* carbapenemase (KPC) has been the main carbapenem-resistant mechanism among CRE.[1,2]

Polymyxins (polymyxin B and colistin) have been widely applied because they are among the few agents that remain effective against multidrug-resistant Gram-negative bacteria such as carbapenem-resistant *Pseudomonas aeruginosa*, carbapenem-resistant *Acinetobacter* spp., and KPC producers.[3] Although their clinical usefulness is increasing in most treatment strategies, the emergence of colistin resistance has been detected worldwide.[4–6]

To date, risk factors for KPC infection (eg, from prior intestinal colonization) have been extensively studied, but the definitive role of the presence of KPC producers from airways has remained uncertain.[7] The widespread dissemination of colistin resistance among KPC producers has strong implications regarding colistin's utility, mainly in the setting of continuous selective pressure from the high-level use of the same, mostly in intensive care units. Therefore, a retrospective survey from January to June, 2016, including adult intensive care patients in a tertiary hospital in Porto Alegre, Southern Brazil, was conducted to assess the cumulative prevalence of KPC-producers, *Acinetobacter* spp., and *Pseudomonas aeruginosa* isolates recovered from endotracheal secretions of mechanically ventilated patients. Additionally, for these bacterial isolates, including >1 per patient from the same clinical site, susceptibility to colistin was evaluated to monitor how quickly colistin resistance could spread over the time.

Identification of bacterial species as well as carbapenem and colistin resistance was initially detected using an automated broth microdilution system (MicroScan; Beckman Coulter, Brea, CA), followed by confirmation with Etest (AB Biodisk, Solna, Sweden). For enterobacterial species, a synergistic test was applied using phenyl-boronic acid to detect KPC and ethylenediaminetetraacetic acid to detect New Delhi metallo-betalactamase, followed by gene detection by PCR analysis as described elsewhere.[8]

In total, 94 distinct patients were enrolled during the study period. Among the species considered in this study, only *Acinetobacter baumannii*, *P. aeruginosa* and KPC-2–producing *Klebsiella pneumoniae* (KPC-2-*Kp*) were detected. Cumulative prevalences for these isolates are shown in Figure 1A. Notably, the cumulative prevalences remained stable for *A. baumannii* (27.9% ± 2.74) and *P. aeruginosa* (28.9% ± 3.74) over the study period. No colistin resistance was observed over the follow-up period despite the high consumption of this class of drugs in this nosocomial setting over the same period of this survey (data not shown). In contrast, KPC-2-*Kp* (14.3% ± 5.27) showed a significant increase in prevalence during this period, ranging from 10.5% in January to 20.2% in June (odds ratio, 2.15; 95% confidence interval, 0.4–10.1).

A total of 25 KPC-2-*Kp* isolates, recovered from 94 patients, were included over the study period: 4 isolates presenting no colistin resistant were recovered in the initial period (January) and 11 colistin-resistant isolates (11 of 25, 44%) were found in the final period (January to June). The linear trend shows that colistin resistance among KPC-2-*Kp* is increasing drastically at a steady rate (Figure 1B).

In this study, endotracheal secretions presenting $\geq 10^5$ CFU/mL were selected to better reflect the influence of the use of colistin. Colistin is used to treat carbapenem-resistant *P. aeruginosa* and carbapenem-resistant *A. baumannii*, which contribute ~33.5% and 97.5% of carbapenem resistance, respectively; these isolates were more prevalent from respiratory secretions of ICU patients during the same evaluated period. As previously reported by Lee et al,[9] the emergence of colistin resistance among KPC-2-*Kp* isolates, which was also found in this study, may be explained by acquisition and development of resistance in the same isolate initially susceptible, or reinfection with a resistant isolate from a heterogeneous bacterial population under antibiotic selective pressure, as previously shown for fosfomycin[10] or polymyxin B when rectal surveillance KPC-2-*Kp* isolates were evaluated.[6] Although the impact of the presence of KPC on patient outcome has not been evaluated in this study, interest in the early diagnosis and prompt treatment of airway infections is growing, particularly concerning multidrug-resistant pathogens, such as KPC-producers, in the effort to reduce rates of inappropriate empirical therapy.[7]

An ideal control is one that, in addition to preventing the spread of multidrug-resistant organisms in the environment, can also stagnate (and eliminate, if possible) its antimicrobial resistance levels. This is a fact. However, controlling these 2 fronts is not easy, and finding the balance between them to promote patient safety may be the greatest challenge of our time.

In conclusion, an increase in prevalence of KPC-2-*Kp* at a clinical site originally with low prevalence is reported in

https://doi.org/10.1017/ice.2016.229 Published online by Cambridge University Press