Fig 1. Dendrogram and rep-PCR images of carbapenem-resistant isolates. American type culture collection (ATCC) isolate BAA-2146 was used was the control strain.

which aims to identify and contain CPE before symptomatic manifestation.[8] We believe that implementation of targeted surveillance culture for high-risk patients may expedite identification and isolation of silent CRE carriers and minimize horizontal transmission.[9] This measure should be coupled with enhancement of hand hygiene compliance.

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Vancomycin-resistant *Enterococcus* spp isolated from community acquired infections and colonizations in Querétaro City, Mexico

To the Editor:

Members of the genus *Enterococcus* are part of the human microbiota. They are found mainly in the gastrointestinal tract, but can also be detected colonizing the upper airways, the oral cavity, the skin, the vagina, and the urethra. Since the 1990s, enterococci have emerged as nosocomial pathogen, nowadays ranking as the third to fourth most common nosocomial agent worldwide; however, prevalence in Latin America has remained low. Enterococci have developed multidrug resistance (MDR) profiles, particularly to vancomycin (ie, that have become vancomycin-resistant enterococci [VRE]), that make them a global public health concern.[1,2] There have been reports of community-acquired (CA) infections and colonizations due to VRE.[3-6] The mechanism of spread in these cases is still unclear. It has been suggested that patients colonized while hospitalized could be the source of transmission.[3] Enterococci surveillance and monitoring studies have been scarce in México and have mainly focused on its nosocomial presence. Yet the studies that have been conducted have shown a significant increase in the rate of enterococcal nosocomial infections—and up to 26% frequency of VRE.[7,8] Moreover, a retrospective study held in Querétaro city[9] reported that 35% (*Enterococcus faecalis*) and 60% (*Enterococcus faecium*) of infecting enterococci strains were resistant to vancomycin, suggesting an incremental spread of VRE strains in this Mexican region. Thus, the aim of our study was to assess the frequency of CA VRE in Querétaro City, Mexico.

An observational study of enterococci-positive cultures identified in outpatients by 6 private clinical laboratories (authorized and certified by Mexican authorities) in Querétaro city, México, between August 2004 and November 2011 was carried out. For further analysis, samples classified as CA urinary tract infection (CUTI) (urine samples with a bacterial colony count $\geq$100,000 cfu/mL) and CA colonization (CAC) (from semen, female genital tract samples, and urethral discharge samples [none of the latter was part of segmented urine cultures]) were included. Enterococcal isolates were identified by conventional microbiologic testing.

Antibiotic susceptibility tests were done using either the Kirby-Bauer disk diffusion method or the automated MicroScan system (Siemens Healthcare Diagnostics, Deerfield, IL). Resistance to vancomycin was analyzed, as was resistance to ampicillin, penicillin, linezolid, high-level resistance (HLR) to gentamicin (500 μg), HLR to streptomycin (1,000 μg), erythromycin, rifampicin, ciprofloxacin, levofloxacin, and nitrofurantoin (tested only in urine cultures), based on Clinical and Laboratory Standards Institute (CSLI) references. MDR was determined based on the recently standardized international terminology recommended by the European Centre for Disease Prevention and Control and the Centers for Disease Control and Prevention.[10] Statistical analysis to determine differences between proportions was performed with the Fisher exact test; a P value $\leq .05$ was considered significant.

Data from a total of 312 enterococci isolates were included for analysis. Twenty-one VRE isolates (6.7%) were found. This frequency is higher than the up to 2% reported previously in the few studies assessing CA VRE.[3-6] Regarding Latin America, only a previous 2005 Mexican study had assessed community enterococci, finding no VRE isolates from urine specimens at that time.[7] This suggests that the appearance of VRE at the community level has recently emerged in this country. As for the type of sample source, urine (59.6%) was the prevailing source, although no statistical differences were found between the proportions by sample source between CUTI (6.4%) and CAC (7.1%). This suggests that these colonizing VRE, under certain host and/or environment conditions, could be causing infection. As for distribution by species, consistent with reports from different countries,[1,2] as well as a previous work in México that analyzed outpatient samples,[7] *E faecalis* were the predominant (96.8%) specie. In contrast, a very low prevalence of *E faecium* strains (2.2%) was observed. Despite its lower presence in the community, previous reports of CA VRE have showed that *E faecium* is the main enterococci species that displays resistance to vancomycin.[3-5] This is not consistent with what we observed in our work, because no statistically significant difference between species was found, suggesting that in our population, *E faecalis* is capable of developing resistance to vancomycin to a similar a degree as *E faecium*. Also, this species distribution strongly suggests that nosocomial strains could not be the main source of CA VRE in our work, because VRE *E faecium* is the most common nosocomial agent within the *Enterococcus* genus, both worldwide[1,2] and specifically in Querétaro City.[9] Thus, a plausible explanation for the presence of VRE in the community could be antibiotic misuse, which was identified in 2010 and that resulted in Mexican authorities establishing new guidelines to regulate the sale and dispensing of antibiotics.[11] It has previously been suggested that a predictor of VRE isolates in CA infections could be the use of antibiotics such as cephalosporins and fluoroquinolones within 3 months before their identification.[5]

The resistance profiles to the antimicrobials considered by the CSLI were compared between enterococcal CUTI and CAC. No statistical differences between proportions were observed in the resistance to each antibiotic, with the exception of HLR gentamicin (CUTI 41.5% vs CAC 26.8%; $P \leq .05$). Based on its therapeutic usefulness, antimicrobials were considered acceptable if $\leq$30% of the isolates were resistant to each. Nitrofurantoin (2.7%), linezolid (11.3%), and ampicillin (15%) met this criteria. The antibiotics with >30% resistant isolates were rifampicin (34.2%), ciprofloxacin (34.6%), HLR gentamicin (35.4%), levofloxacin (36.6%), HLR streptomycin (48.3%), and erythromycin (83.5%). Regarding ampicillin, resistance observed in our study was comparable to the 13% described in CA urinary infections in a previous Mexican work.[7] The resistance to β-lactam antibiotics observed can be considered still low. This is important because the use of this type of antibiotic, in synergistic combination with aminoglycosides, remains the first choice of treatment for severe enterococcal infections.[2] However, it is of concern that there was a lower sensibility to high doses of aminoglycosides. The effect of fewer therapeutic options derived from this trend toward increased resistance to HLR aminoglycosides has been pointed out by other researchers.[1,2]

A total of 134 isolates met the criteria for the MDR analysis, of which 128 (95.5%) were *E faecalis*. Twelve isolates (8.9%) were MDR (resistant to 3 or more antimicrobial categories). Resistance to 5 antimicrobial categories was the highest level of resistance. Within these MDR isolates, 5 (42%) were VRE and all belonged to the *E faecalis* species. As for the type of isolate, CUTI prevailed (9 isolates, 75%), although no statistical difference between proportions of CUTI and CAC was found. The proportion of VRE observed increased in relation to the resistance to a greater number of antimicrobial categories (16%, 33%, and 100% of the isolates resistant to 3, 4, and 5 antimicrobial categories were VRE, respectively).

It is important to emphasize that the presence of vancomycin resistance—together with resistance to other clinically useful antibiotics in CA enterococci strains—lessens the therapeutic options in case of infection.

Ours is the first report of VRE isolated from community samples in Latin America, and we showed a 6.7% frequency and up to 42% association to MDR, with *E faecalis* being the predominant species found. Our work suggests that the mechanisms for the selection of resistance to vancomycin are present in the community. This could be the result of antibiotic misuse in the country. Further epidemiologic studies regarding emergence of VRE at the community level should be done to identify risk factors and establish appropriate strategies of prevention, control, and eradication.

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Successful outbreak investigation of *Burkholderia cepacia* complex bacteremia in intensive care patients

To the Editor:

Bulkholderia cepacia complex (Bcc) is a group of ubiquitous gram-negative aerobic bacilli found in plants, soil, and moist environments. Bcc is well described as a cause of respiratory infections in patients with cystic fibrosis and chronic granulomatous disease. Bcc bacteremia and nosocomial pneumonia have also been observed in intensive care patients as sporadic cases or during outbreaks.

Here, we describe a small outbreak of nosocomial Bcc bacteremia in the 6-bed intensive care unit (ICU) of an 850-bed university-affiliated general hospital in Jerusalem, Israel. During July 2010, a patient admitted to our ICU because of congestive heart failure and pulmonary edema developed *Burkholderia* bacteremia. She was treated according to bacterial sensitivity with no attributable consequences. Several days later, a second patient in our ICU, hospitalized with severe immune thrombocytopenia and pulmonary hemorrhage, was found to have *Burkholderia* bacteremia. Repeated blood cultures were positive, and bacteremia resolved after an appropriate antibiotic was initiated. The patients had no known risk factors for *Burkholderia* infection and no obvious source for the bacteremia was found.

After the second case an outbreak investigation was conducted. We detected 2 more patients who had bacteremia with *Burkholderia* in 2010; 1 of these was a third patient in the same ICU 3 months earlier, and the fourth was a patient in our thoracic heart surgery ICU. We concluded that we were facing an outbreak of *Burkholderia* bacteremia.

Because *Burkholderia* is a waterborne and soilborne organism that can survive for a prolonged period in a moist environment, we sampled wet and humid products that had been used during care of the 2 index cases. We cultured inhalation solutions (ie, ipratropium bromide, albuterol, and water used in the nebulizer), insulin, heparin, erythromycin, piperacillin-tazobactam, water for injection, potassium chloride solution, hygiene products (eg, povidone, hand rub gel, chlorhexidine, and superoxide solution), and patient care products (eg, mouthwash and moisturizing cream).

All items were sterile except for the moisturizing cream of both patients, which was contaminated with *Burkholderia*. Each patient in our ICU had an individual cream tube. We examined the cream tubes of 2 other patients residing in our ICU at the same time and found that their cream was also contaminated with *Burkholderia*. Subsequently, we cultured 6 new, sealed cream tubes from our ICU and other departments in our hospital, and found 3 were contaminated with the same bacteria.

All clinical and environmental isolates of *Burkholderia* were isolated and identified by 16S ribosomal DNA sequencing at the Shaare Zedek Microbiology laboratory. For further identification, the specimens were sent to the Observatoire Cepacia, Laboratoire de Bactériologie-Hygiène Hôpital Purpan, Toulouse, France. Identification of the species level was performed by means of amplified 16S ribosomal DNA restriction analysis,[1] RecA species-specific polymerase chain reaction (PCR),[2] and/or RecA sequencing.[3] Genetic relatedness was assessed using PCR ribotyping.[4]

Blood isolates were identified as *B cenocepacia* IIIB (patient 1) and *B stabilis* (patient 2) (Table 1). Twelve cream samples were found to be contaminated with *Burkholderia* species, 9 with the same strain of *B contaminans*, and 3 with the same strain of *B cenocepacia* IIIB, which harbored the same PCR ribotype as the isolate from the moisturizing cream of patient 1. The moisturizing cream was withdrawn from all hospital departments and notification was made to the manufacturer and to the Israeli Ministry of Health. No new cases occurred. The manufacturer identified lapses in the water filtration system as the cause of contamination. Two-year follow-up showed no additional *Burkholderia* bacteremia in our institution.

We suspect the cause of the outbreak was moisturizing cream contaminated with at least 2-3 different *B cepacia* genomovars that had most probably occurred during manufacturing. Moisturizing cream was described once before as a cause of *Burkholderia* infections in an ICU.[5]

The strain in the first patient with *Burkholderia* bacteremia was identified as *B stabilis* by amplified 16S ribosomal DNA restriction analysis and RecA gene methods. Although this genomovar was not found in the moisturizing creams, we believe this case was also caused by using contaminated cream because of polygenomovar contamination of the cream boxes.

Although cosmetic products such as moisturizing cream are not required to be sterile if they are used solely topically, manufacturers are obligated to maintain high standards of control and are regulated by the Scientific Committee on Cosmetic Products and Non Food Products[6] (available online at http://ec.europa.eu/health/ph_risk/committees/sccp/documents/out242_en.pdf), and other national regulations. In the face of the numerous publications citing *Burkholderia* as a cause of outbreaks amongst vulnerable hospitalized patients, we believe that national and international regulations should require the absence of any *Burkholderia* species in products used for these patients. We also suggest avoiding the use of nonsterile cosmetics products in special populations, such as ICU patients and immune-compromised patients, while hospitalized.