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CPE Colonization - Once A Carrier Always A Carrier? Response to Lewis and Bart

To the Editor—We read with interest the studies by Lewis et al[1] and Bart et al[2] on efforts to predict clearance of gastrointestinal colonization with carbapenemase-producing *Enterobacteriaceae* (CPE) based on follow-up rectal screens. The authors are to be congratulated for trying to address an important infection prevention issue, ie, whether there is a time when contact precautions, specifically isolation, can be discontinued. Both studies report that a significant proportion of patients who had negative CPE surveillance cultures following their initial positive screen remained colonized with CPE at follow-up screening: 13% (36 of 276) and 33% (7 of 31) in the studies by in the study by Bart et al and Lewis et al, respectively. Indeed, even after further screening, CPE was detected in 2 patients who had been found to be negative on 3 occasions following their initial positive screen.[1] It is also important to note that no single culture-based screening method routinely employed has the high level of sensitivity required to detect all genotypes of CPE, particularly those displaying low-level resistance.[3] Therefore, the rates of recurrent carriage may, in fact, be underestimated. Bart et al attempt to identify those risk factors that correlate with CPE recurrence. However, given the relatively small numbers of patients identified and the limitations of this study, further work is certainly needed in this area.

Recent guidelines on infection prevention and control measures to reduce transmission of multidrug-resistant Gram-negative bacteria in hospitalized patients highlight the dearth of good evidence and confirm that no consensus exists on when contact precautions may be discontinued.[4] Furthermore, it is mistaken to make assumptions based upon other multidrug-resistant organisms, specifically methicillin-resistant *Staphylococcus aureus* (MRSA), where recognized decolonization regimens are commonly used because such treatment often assists in shortening the duration of carriage. In addition, for MRSA, the major reservoir of colonization is the skin and nasal mucosa, whereas for CPE and vancomycin-resistant enterococci (VRE), the gastrointestinal tract is the important reservoir. Although guidelines suggest that 3 consecutive negative swabs may allow for discontinuation of contact precautions in patients with VRE, this may be difficult to achieve in practice given the gastrointestinal reservoir.[5] In a study on a renal unit, 64% of patients remained positive for VRE when 3 or more follow-up rectal screening specimens were taken.[6] However, many of these were patients with chronic renal failure, which, in addition to other factors, may help explain this statistic. It is possible that, for groups with similar risk factors, the same difficulties arise with regard to persistent CPE carriage.

Much remains unknown about the natural epidemiology of patients with CPE and specifically when, and if, some patients ever lose the organism. Factors governing this condition are likely to be complex and include the underlying condition of the patient; the setting in which the patient is being cared for; recent, current, and the future administration of antibiotics and other drugs; and the complex milieu of the intestinal microbiome, which is dynamic and is likely to have an important impact on colonization. To date, we have largely relied on cultures to determine changes in the epidemiology of colonized patients with CPE, but the relationship between CPE and the remainder of the intestinal flora is likely to be complex. We need to apply meta-genomic approaches to explore that relationship and how it might affect the dynamic of CPE colonization.[7] As our knowledge regarding possible exploitation and restoration of the intestinal microbiome develops, so

too may our thinking regarding management of patients colonized with CPE.[8]

Prospective studies using innovative laboratory techniques are needed on patients with CPE during outbreaks and in endemic settings over periods of time, while in the hospital and after discharge. These studies will help us further understand the natural history of CPE colonization and determine the host/patient, environmental, and microbial factors that may help us predict which patients remain transiently, intermittently, or permanently colonized. This work is required to scientifically inform infection prevention and control strategies on the use of contact precautions and to facilitate risk stratification of patients previously identified as CPE colonized. This research becomes increasingly important as the number of patients with CPE increases, especially in settings where single room/isolation facilities are limited.

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A Fatal Case of Nosocomial Legionnaires' Disease: Implications From an Extensive Environmental Investigation and Isolation of the Bacterium From Blood Culture

To the Editor—Legionella pneumophila (*Lp*) serogroup 1 is a well-known cause of hospital-acquired pneumonia. Extrapulmonary manifestations as well as *Lp* bacteremic pneumonia are rare and occur mainly in immune-compromised subjects.[1–5]

Potable water has been implicated in nosocomial cases of *Lp* infection via inhalation of contaminated aerosols from hot-water systems.[6] Culture is the gold standard for the diagnosis of *Legionella* infection, even though culture-proven legionellosis cases are not frequently reported and documented cases of bacteria isolation from the blood are even less common.[7]

On January 5, 2014, a 58-year-old woman with a clinical history of alcoholic liver cirrhosis was admitted for worsening dyspnea to the Emergency Department of IRCCS AOU San Martino–IST Hospital, Genoa, Italy. On admission, the patient presented peripheral edema and signs of portal hypertension. Chest and abdominal radiograph did not detect pleuroparenchymal lung lesions.

The same day, the patient was transferred to the gastroenterologic unit where she stayed until February 6, 2014. She underwent 3 paracentesis procedures, a transjugular intrahepatic portosystemic shunt, and 3 angiography controls because of a progressive deterioration in liver function.

On February 6, 2014, the patient experienced a severe respiratory failure due to acute pulmonary edema. She was intubated and chest radiograph (Figure 1) detected pleuroparenchymal lung lesions compatible with pneumonia.

Blood cultures (Bactec Plus aerobic; BD) were performed and the patient was empirically treated with piperacillin-tazobactam. No specific diagnostic test for *Legionella* (culture, urinary antigen, serology) was performed. On February 7, 2014, the patient died because of respiratory and multiorgan failure.

After 6 days, blood culture became positive and subculture on blood agar (Kima; Megalab) and chocolate-enriched agar plates (Kima) was performed, showing the growth of colonies