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Two sequential outbreaks caused by multidrug-resistant Acinetobacter baumannii isolates producing OXA-58 or OXA-72 oxacillinase in an intensive care unit in France

Madam,

Acinetobacter baumannii has emerged worldwide as an important nosocomial pathogen because of its ability to survive for long periods in dry environmental conditions, its capacity to acquire resistance to a variety of antimicrobial agents and its epidemiological complexity. 1 Carbapenems are among the drugs of choice for treatment of infections due to multidrug-resistant (MDR) A. baumannii isolates. However, resistance to these compounds is increasing and is often associated with acquired OXA-type carbapenemases. 2 During outbreaks, complete eradication and prevention of dissemination may require multiple interventions.

We report the dissemination of two carbapenem-resistant A. baumannii clones in a 14 bed medical–surgical intensive care unit (ICU) over a three-year period from October 2003 to September 2006. Evolution of the two outbreaks is shown in Figure 1. The first outbreak started in October 2003. The suspected index case was a colonised patient who had been admitted from another hospital. He developed a catheter infection with MDR A. baumannii isolate showing an intermediate resistance to imipenem [minimum inhibitory concentration (MIC) = 8 mg/L], and only susceptible to rifampicin, colistin and tobramycin. During the years preceding the admission of the index case, carbapenem non-susceptible strains had not been detected in the hospital. Despite a progressive implementation of multiple infection control measures, a total of 28 patients were found to be colonised or infected with MDR A. baumannii isolates over a 23-month period (Figure 1). The last case occurred in September 2005.

The second outbreak started in July 2005 after the transfer of an 80-years-old man from a hospital in Thailand to the ICU. The bronchial aspirate and the rectal swab yielded an isolate of MDR A. baumannii with a high level resistance to imipenem (MIC > 32 mg/L) and only susceptible to colistin and rifampicin. As with the first epidemic strain, despite reinforcement of isolation measures, enhanced cleaning of rooms, materials, and improved hand hygiene compliance, 15 additional cases were recorded during the 14 following months (Figure 1). In September 2006, the ICU was closed, cleaned, and bio–decontaminated with a hydrogen peroxide gaseous disinfection system (Clarus®R, Bioquell, Bonneuil-sur-Marne, France). Since then, no other cases of colonisation or infection by these MDR A. baumannii isolates have been reported. During both outbreaks, we observed long periods of time without colonised or infected patients.

During the two outbreaks, 352 environmental samples and samples from the hands of the healthcare workers were cultured. Only five yielded growth of A. baumannii with antibiotic susceptibility patterns consistent with those of the clinical MDR A. baumannii isolates. Four were found in the immediate environment of colonised patients and one was found on a ventilation grille located above a washbasin, a site inaccessible to the bio–cleaning. This is consistent with the ability of A. baumannii to persist for long periods on surfaces inaccessible to cleaning. The colonisation of patients and the spread into the environment are favoured by the formation of aerosols from such inaccessible reservoirs. 3 None of the healthcare workers was found to carry the MDR isolates on hands. Despite this negative screening, transfer by hands of healthcare workers is the most common route of cross–transmission and cannot be excluded from these outbreaks.

Random amplified polymorphic DNA and pulsed–field gel electrophoresis 4 identified the presence of two clones, with clone A predominating for two years before being replaced by clone B.

Screening for genes encoding oxacillinases and major metallo-β-lactamases by polymerase chain reaction (PCR) showed the presence in all the outbreak isolates of the blaOXA-51 gene, a naturally occurring oxacillinase in A. baumannii, and of genes encoding acquired oxacillinases. 5, 6 The blaOXA-58 gene was found in all isolates of clone A and the blaOXA-72 gene was found in all isolates of clone B. OXA-58 oxacillinase was first identified in an A. baumannii isolate in France and subsequently has been reported among A. baumannii isolates in several countries. 7 OXA-72 oxacillinase has been previously reported in several carbapenem–resistant A. baumannii isolates in Taiwan. 8 To our knowledge, this is the first report of a hospital outbreak of blaOXA-72 carrying A. baumannii in France.

It is noticeable that the disappearance of the first MDR strain followed promptly on the emergence of the second strain. To infer a causal relationship between these two events would be hazardous. Nonetheless, the hypothesis that it was only
Figure 1. Epidemic curve of the two outbreaks and implementation of infection control measures.

- □: cases of the first outbreak
- ▣: cases of the second outbreak

ICU, intensive care unit.

No. of colonised or infected patients
implementation of barrier measures that allowed eradication of the first strain is not consistent with the concomitant emergence of the second.

Finally, despite the implementation of multiple infection control measures, only bio-decontamination with hydrogen peroxide allowed the eradication of the second strain and ended the three-year period of MDR A. baumannii outbreak, underlining the epidemic potential of those bacteria and the problems associated with their environmental persistence.

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Ultrasound echocardiographic gel contamination by Burkholderia cepacia in an Italian hospital

Madam.

Ultrasound gel is a potential source of infection; contamination can occur at the time of manufacture with organisms that degrade parabens, which are commonly used as stabilising agents.1 Following the identification of a cluster of multidrug-resistant Gram-negative bacteria in a ward of the AOU Ospedali Riuniti in Ancona, Italy, an epidemiological survey took place. Microbiological monitoring of surfaces, equipment and substances used as antiseptics and gels for echocardiographic and electrocardiographic investigations was performed. The gels involved were composed of demineralised water and deionised water, thickening agent (Carbomer), complexing agent (EDTA), neutralising agent (sodium hydroxide), NaCl (electrocardiographic gel only) and high purity preservative Microcare IT (methylchloroisothiazolinone and methylisothiazolinone: MCI/MI) characterised by a broad spectrum of action for the control of bacteria, yeasts and moulds.

Three bottles (two opened, one sealed) were collected from the ward and cultures revealed contamination by Burkholderia cepacia. Twelve bottleneves belonging to the same batch, and from different batches circulating in the hospital, were cultured. The vials were simultaneously analysed by two laboratories; the results confirmed the presence of B. cepacia in every pack.

B. cepacia complex (Bcc), previously known as Pseudomonas cepacia, has been recognised as a cause of infection in hospitalised patients. The organisms are widespread in the environment, have minimal nutritional requirements and have the ability to survive in the hospital setting due to their resistance to many disinfectants and antiseptic solutions.2,3 B. cepacia can grow and proliferate in numerous aqueous products such as distilled water, tap water, povidone-iodine solutions, dialysis machines, ventilator temperature probes and ultrasound gel.2,4,5 Following these findings all the bottles of the ultrasound gel originating from the same manufacturer were identified and removed from use and a report was sent to the Ministry of Health. There was no colonisation or infection with B. cepacia in patients hospitalised in the ward during the investigation period. Although gel sterility is not essential for ultrasound examination of patients through intact skin, and low levels of relatively non-pathogenic bacteria pose little risk, contamination by a micro-organism such as B. cepacia needs to be taken seriously.1 As already observed, ultrasound gel may be a source of infection and it must be suspected and tested together with other products, particularly during an outbreak.1,2 The lack of established guidelines for the use of ultrasound gel in clinical practice is a well-known issue. Jacobson et al., on the basis of their experiences, made some suggestions for a better use of ultrasound gel, enhancing the importance of using only sterile gel in close proximity to susceptible sites such as mucous membranes during trans-oesophageal echocardiography.6 It is also suggested that routine sterility controls are introduced, even for usually non-sterile products, when used in critical areas, particularly when parabens are used as stabilising agents.

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