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Letter to the Editor

High mortality outbreak of carbapenem-resistant *Pseudomonas aeruginosa* infection in a Brazilian pediatric oncology hospital



Dear Editor,

Carbapenem-resistant *Pseudomonas aeruginosa* is an important nosocomial pathogen associated with high mortality rates and can persist in moist environments in hospitals. Several outbreaks have been associated with environmental contamination, contaminated medical equipment, and healthcare cross-transmission. The production of acquired metallo- β -lactamase SPM-1 has been frequently associated to these infections.

Here, we report an outbreak of carbapenem-resistant *P. aeruginosa* encoding the *bla*_{SPM-1} M β L gene affecting nine

patients under cancer treatment admitted at the Institute of Pediatric Oncology from November 2011 to May 2012. Strains were isolated from blood ($n = 7$) and urine ($n = 2$) and identified by BD PhoenixTM Automated System (BD Biosciences, Sparks Md). DNA was extracted using the QIAamp DNA mini-kit (QIAGEN, CA) and tested for M β L genes by Multiplex real-time PCR (Rotor-gene-Q, Qiagen, CA)¹ followed by DNA sequencing (ABI sequencer, Applied Biosystems, USA). Clonal relatedness was evaluated by pulsed field gel electrophoresis (PFGE) using the *SpeI* restriction endonuclease (New England BioLabs, USA). The band patterns were analyzed using the Bionumerics Software 7.6 (Applied Maths, Belgium).

Table 1 – Clinical data of infection episodes caused by SPM-1 producing *Pseudomonas aeruginosa*.

Patient	Intensive care unit admission date	Diagnosis	Hemodialysis	Previously colonization by <i>Pseudomonas aeruginosa</i>	Date of positive isolation of <i>Pseudomonas aeruginosa</i>	Site of bacteria isolation	Death/discharge	Real-time PCR for <i>bla</i> _{SPM} gene	PFGE pattern
1	2011/10/31	Low level of consciousness convulsion and hyponatremia	No	No	2011/11/26	Urine	Discharge	Positive	B
2	2011/11/29	Tachypnea	No	No	2011/11/27	Blood	Death	Positive	C2
3	2011/11/04	Hypervolemia and respiratory distress	Yes	Yes	2011/12/01	Urine	Discharge	Positive	C5
4	2012/02/08	Gastrointestinal bleeding	No	Yes	2012/02/08	Blood	Death	Positive	C3
5	2012/02/16	Fever and dyspnea	Yes	No	2012/02/16	Blood	Death	Positive	C1
6	2012/01/30	Fever of unknown origin	No	No	2012/02/18	Blood	Death	Positive	Not typeable
7	2012/02/18	Veno-occlusive disease and respiratory failure	Yes	Yes	2012/02/28	Blood	Death	Positive	A1
8	2012/03/17	Septic shock	No	No	2012/03/27	Blood and catheter	Death	Positive	C
9	2012/04/26	Respiratory distress	No	No	2012/05/01	Blood and catheter	Death	Positive	C4

All nine *P. aeruginosa* isolates were resistant to all beta-lactams, fluoroquinolones, and aminoglycosides but susceptible to colistin. The samples were positive for the *bla*SPM-1 and negative for other M β L genes. PFGE demonstrated three different clusters, one of them was highly related to the endemic clone SPM-1 previously reported in the institution.² This fact reinforces that a genomic variety recently observed among various SPM-1-producing *P. aeruginosa* isolates is result of the accumulation of mutations along the time and the endemic clone was now adapted to spread in-hospital ambient.³

Three patients were previously colonized by *P. aeruginosa* and one had a contaminated prosthesis before the infection episode. Out of the nine patients, those who had the strain isolated from blood died (Table 1). All affected patients were critically ill and neutropenic. The majority of them presented septic shock and acute respiratory syndrome. Thus, we think these factors, combined with the lack of therapeutic options for these infections, could explain the observed high mortality rate.

Although no common source could be identified, and given the limited therapeutic options for these infections, there is an urgent need for a better adherence of infection control practices in addition to investigate SPM-producing strains in routine diagnostic bacteriology to prevent further dissemination of this challenging pathogen.

Conflicts of interest

The authors declare no conflicts of interest.

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