

P0252

Paper Poster Session
Focus Acinetobacter

Acinetobacter baumannii isolated from rectal screening samples as part of the MagicBullet clinical trial: molecular epidemiology and susceptibility to carbapenems, tigecycline and colistin

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Background: MagicBullet is a multi-center European collaborative research project investigating two empirical antimicrobial regimens, i.e. colistin vs. meropenem, both combined with levofloxacin, to treat ventilator-associated pneumonia (VAP). The objective of our study was to investigate the molecular epidemiology, carbapenem, colistin and tigecycline susceptibility, and carbapenem-resistance determinants of *Acinetobacter baumannii* isolated from stool samples of VAP patients enrolled in MagicBullet.

Material/methods: The first *A. baumannii* isolates cultured from rectal screening samples were collected from VAP patients from 33 hospitals in Greece, Italy and Spain from 05/2012 to 10/2015. Species identification was performed by MALDI-TOF and *gyrB* multiplex PCR. Susceptibility testing was performed by Etest. Carbapenem-resistance determinants were identified by PCR and sequencing. Molecular epidemiology was investigated using rep-PCR (DiversiLab), with a similarity of $\geq 98.6\%$ interpreted as identical. International clones (IC) were identified with a similarity of $\geq 95\%$ compared to our reference database.

Results: Seventy *A. baumannii* isolates were collected from rectal screening samples from 68 patients hospitalized with VAP in 13 hospitals. All isolates were resistant to the carbapenems with MICs of > 32 mg/L (Table 1). Carbapenem resistance was always associated with acquired carbapenemases: OXA-23, OXA-40 and OXA-58 were identified in 54, 6 and 8 isolates, respectively. Furthermore, two isolates harboured both OXA-23 and OXA-58. In all instances OXA-23 and OXA-58 genes were associated with insertion elements IS*Aba1* and IS*Aba3*, respectively. The majority of isolates (74%) were resistant to tigecycline with MICs > 2 mg/L, while an equal number of isolates remained susceptible to colistin with MICs ≤ 2 mg/L. The majority of isolates (n=55) represented IC2, with four clonally related subtypes identified (C (n=39), D (n=2), E (n=12), F (n=2)). Subtype C comprised isolates originating from 11 hospitals enrolled in MagicBullet. IC1 was represented by 11 isolates (G (n=7), H (n=3), singleton (n=1)) from a single Spanish hospital and five Greek hospitals, respectively.

The remaining four isolates (A (n=2), I (n=2)), with subtype A originating from a hospital in Spain and subtype I originating from two hospitals in Greece, were unclustered.

Conclusions: Carbapenem resistance was universal in *A. baumannii* cultured from stool samples of VAP patients. While more than 70% of isolates showed elevated MICs to tigecycline, a similar percentage remained susceptible to colistin. Our susceptibility data highlight alarming resistance rates to last-resort drugs in commensal *A. baumannii* from VAP patients, including colistin. Rep-PCR confirms that IC2 is currently the predominant lineage and suggests the presence of an epidemic *A. baumannii* clone that has spread within Greece, Italy and Spain.

Table 1: Distribution of resistance and susceptibility to the tested antimicrobials in *A. baumannii* isolates.

	% susceptible	% resistant
Imipenem	0	100
Meropenem	0	100
Tigecycline*	26	74
Colistin	74	26

*EUCAST breakpoint Enterobacteriaceae