EV0159

ePoster Viewing

Antimicrobials: mechanisms of action and resistance

Multidrug-resistant *Proteus mirabilis* in nursing homes

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Background: *Proteus mirabilis* is emerging cause of nosocomial infections particularly of wounds and urinary tract. The infections are often difficult to treat because of acquisition of various resistance mechanisms such as extended-spectrum beta-lactamases (ESBLs) or plasmid-mediated AmpC beta-lactamases. An increased frequency of *P. mirabilis* isolates resistant to expanded-spectrum cephalosporins was observed recently in a long-term-care facility in Zagreb. The aim of this study was the molecular characterization of resistance mechanisms to expanded-spectrum cephalosporins in *P. mirabilis* isolates from this nursing home.

Material and methods: Twenty strains collected from April 2013 till April 2014 showing, reduced susceptibility to ceftazidime were analysed. Antibiotic susceptibilities were determined by broth microdilution method according to CLSI. Inhibitor-based tests with clavulanate and phenylboronic acid (PBA) were performed to detect ESBLs extended-spectrum AmpC β-lactamases, respectively.Transfer of cefotaxime was tested by conjugation (broth mating method) using *E. coli* resistant to rifampicin. AmpC β-lactamases were characterized by PCR and sequencing of bla_{ampC} genes. Plasmids were characterized by PCR-based replicon typing (PBRT).

Results: Presence of an AmpC β-lactamase was confirmed in all strains by combined-disk test with phenylboronic acid. The strains were phenotypically negative for ESBLs. All strains were resistant to amoxicillin alone and combined with clavulanate, cefuroxime, cefotaxime, ceftriaxone, cefoxitin, gentamicin and ciprofloxacin, but susceptible to cefepime, piperacillin/tazobactam, imipenem, and meropenem. The strains showed variable susceptibility/resistance patterns to ceftazidime with MICs ranging from 16 to >128 mg/L . Cefotaxime resistance was not transferred to *E. coli* recipient strain by conjugation. PCR and sequencing using primers targeting *bla*cmy genes revealed CMY-16 β-lactamase. The strains harboured additional TEM beta-lactamase. The plasmid encoding CMY-16 did not belong to any known PBRT.

Conclusions: This is the first report of multidrug resistant *P. mirabilis* in a nursing home in Croatia. Cephalosporin resistance was due to plasmid-mediated AmpC β-lactamase CMY-16. Accurate and fast laboratory identification of plasmid-mediated AmpC beta-lactamases is important to avoid therapeutic failures. Some authorities recommend all expanded-spectrum cephalosporins to be reported as resistant regardless on the results of in vitro susceptibility testing. Genotyping of the isolates by pulsed-field gel electrophoresis is in progress to determine the possible clonal relatedness of the isolates.