Comparative in vitro activity of temocillin, meropenem, ceftazidime and piperacillin/tazobactam against panel strains and clinical isolates of Burkholderia cepacia complex from 9 different genomovars.

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INTRODUCTION and OBJECTIVE

CF is caused by mutations in a gene encoding a protein named CFTR which functions as a chloride channel in epithelial membranes (Colins, 1992). The most dramatic changes are observed in CF airways causing chronic pulmonary infections with surprisingly few bacterial pathogens. Pseudomonas aeruginosa (most common isolate), Staphylococcus aureus, Stenotrophomonas maltophilia and Burkholderia cepacia [CFAR Annual Report, 2002].

The B. cepacia complex (Bcc) represents at least 9 distinct bacterial species or "genomovars". Bcc are found in soil and on plants. The identification of unique Bcc strains CF sputum isolates implies acquisition from unknown reservoirs. The global prevalence rate among CF patients is around 3% (with up to 8% in adults) (Saman and Siegel, 2003). Infections with Bcc are regarded as crucial for CF patients because in about one third of patients it causes a rapid decline of lung function, with as consequence, a dramatic reduction of life expectancy (up to 50%) (Corey and Farewell, 1996).

Although temocillin has already been used in a pilot clinical studies (Taylor et al., 1992) with success for the treatment of Bcc infections in CF patients, only a few in vitro susceptibility data are available.

Our aim was, therefore, to determine the MICs of antibiotic used in CF patients (meropenem [MER], ceftazidime [CTZ], and piperacillin/tazobactam [PTZ]) in comparison with that of temocillin (TMO) towards a well characterized panel of B. cepacia complex strains.

METHODS

MICs were measured by broth microdilution using the CLSI method. Pseudomonas aeruginosa ATCC 27853 and Escherichia coli ATCC 25922 were included as control strains. CLSI breakpoints for Enterobacteriaceae were used for Mer, CTZ, and PTZ and that of Fuchs et al. (1985) (CLSI Microb. 15:F-30) for TMO.

RESULTS

Figure 1 shows the MIC distributions of TMO, CTZ, MER, and PTZ against all 100 strains of B. cepacia complex. The red line correspond to the CLSI breakpoint for CTZ, MER, and PTZ and that of Fuchs et al. for TMO (pe thorax less values).

CONCLUSIONS

- TMO active against more B. cepacia complex strains compared to other clinically used β-lactams.
- These results, combined with those of pilot clinical studies, suggest a potential advantage of TMO in B. cepacia infected CF patients.

REFERENCE