Evaluating a chromogenic medium for extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae.

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ABSTRACT

OBJECTIVES: ESBL-producers are increasingly considered an infection control problem worldwide. Simple and specific means of screening for ESBL-producing Escherichia coli and K.pneumoniae are complicated by the fact that ESBL-producing Enterobacteriaceae may be mediated by other mechanisms. The purpose of this study was to evaluate the performance of a differential chromogenic screening medium selective for ESBL-producing enterobacteria using a large library of genetically characterised cephalosporin-resistant E. coli and K. pneumoniae.

METHODS: Isolates were from 12 medical centers across Canada. 213 E. coli and 17 K. pneumoniae with ESBL of AmpC mediated cephalosporin resistance were studied. E. coli and K. pneumoniae were screened using ceftazidime and/or ceftazidime plus 1 μM, 10 μM, 100 μM and 1000 μM of cefoxitin (CTM-K, SHV, TEM, OXA), AmpC-producing (E. coli 4/C, TEM-2 and 4 and 2 promoter mutations), 8 co-expressing cephalosporinases and AmpC+E. coli (4/C, TEM-15, 1/C, CTM-3, 2/C, CTM-3, 2/A and TEM-12, all with promoter/alteration mutations), 14 ESBL-K. pneumoniae (co-expression of CTM-K and SHV-2 and 2 promoter mutations). 91 AmpC+K. pneumoniae (30 μM and 0.1 μM of AmpC enzyme). These isolates were studied. 25% of E. coli had an atypical chromogenic reaction with a weak blue discoloration. All K. pneumoniae had a typical chromogenic reaction. 91 un inhibited AmpC producing hyperproducing organisms had >100 colonies/plate for TEM-12, the mechanism of cephalosporin resistance was the CTM-2 gene. One ESBL-E. coli was inhibited by the chromogenic assay. This isolate was ESBL-producing E. coli and K. pneumoniae with a cefoxitin MIC of 0.12 μg/mL and a ceftazidime MIC of 8 μg/mL.

RESULTS: For E. coli with ceftazidime and/or ceftazidime plus 1 μM, 10 μM, 100 μM and 1000 μM of cefoxitin (CTM-K, SHV, TEM, OXA), AmpC-producing (E. coli 4/C, TEM-15, 1/C, CTM-3, 2/A and TEM-12, all with promoter/alteration mutations), 14 ESBL-K. pneumoniae (co-expression of CTM-K and SHV-2 and 2 promoter mutations). 91 AmpC+K. pneumoniae (30 μM and 0.1 μM of AmpC enzyme). These isolates were studied. 25% of E. coli had an atypical chromogenic reaction with a weak blue discoloration. All K. pneumoniae had a typical chromogenic reaction. 91 un inhibited AmpC producing hyperproducing organisms had >100 colonies/plate for TEM-12, the mechanism of cephalosporin resistance was the CTM-2 gene. One ESBL-E. coli was inhibited by the chromogenic assay. This isolate was ESBL-producing E. coli and K. pneumoniae with a cefoxitin MIC of 0.12 μg/mL and a ceftazidime MIC of 8 μg/mL.

CONCLUSIONS: For all resistant E. coli (AmpC and ESBL), sensitivity for identification of ESBL-producing K. pneumoniae was 99.2% for ESBL-producers identified by cefoxitin and AmpC+K. pneumoniae was 99% compared to specificity 99.9% for ESBL-producers. The medium is specific for ESBL-producers than other references for the media (e.g. MacComb) with cefoxitin - 54.2% and other chromogenic media - 60.7% in one study (4).

ESBL-producers with low MICs to cefoxitin (e.g. TEM-12) may not be identified by this medium.

False positive results from AmpC producing E. coli may be due to non-ESBL l$:amacters (e.g. TEM-12, SHV) expressed in large quantities by the organism or porin alterations, or both. Such organisms may express an ESBL phenotype as has been observed by our group and others (3).

In Canada, where AmpC-producing E. coli constitute <50 of cefoxitin resistant isolates, a substantial reduction in workload would be expected, particularly in ESBL confirmation testing, if this medium was used for screening ESBL-producers when compared to the less specific method.

REFERENCES


CONCLUSIONS

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