

Detection of ESBL among ampc producing enterobacteriaceae using inhibitor-based method

Sasirekha Bakthavatchalu^{1,&}, Uma Shakthivel¹, Tannu Mishra¹

¹Department of Microbiology, Centre for Post Graduate Studies, Jain University, Bangalore, Karnataka- 560 011, India.

&Corresponding author

Sasirekha Bakthavatchalu, Department of Microbiology, Centre for Post Graduate Studies, Jain University, Bangalore, Karnataka- 560 011, India

Abstract

Introduction: The occurrence of multiple β -lactamases among bacteria only limits the therapeutic options but also poses a challenge. A study using boronic acid (BA), an AmpC enzyme inhibitor, was designed to detect the combined expression of AmpC β -lactamases and extended-spectrum β -lactamases (ESBLs) in bacterial isolates further different phenotypic methods are compared to detect ESBL and AmpC.

Methods: A total of 259 clinical isolates of *Enterobacteriaceae* were isolated and screened for ESBL production by (i) CLSI double-disk diffusion method (ii) cefepime- clavulanic acid method (iii) boronic disk potentiation method. AmpC production was detected using cefoxitin alone and in combination with boronic acid and confirmation was done by three dimensional disk methods. Isolates were also subjected to detailed antibiotic susceptibility test.

Results: Among 259 isolates, 20.46% were coproducers of ESBL and AmpC, 26.45% were ESBL and 5.40% were AmpC. All of the 53 AmpC and ESBL coproducers were accurately detected by boronic acid disk potentiation method.

Conclusion: The BA disk test using Clinical and Laboratory Standards Institute methodology is simple and very efficient method that accurately detects the isolates that harbor both AmpCs and ESBLs.

Introduction

The rapid global dissemination of *Enterobacteriaceae* harboring plasmid-borne extended-spectrum β -lactamases (ESBLs) and plasmid-mediated AmpC β -lactamases represents a significant clinical threat [1,2]. ESBLs producing organism confer resistance to penicillin, cephalosporins, and monobactams. They cannot hydrolyze cephamycins and are inhibited by clavulanic acid (CA) [3]. Like ESBLs, plasmid-mediated AmpC β -lactamases have a broad substrate profile that includes penicillin, cephalosporins, and monobactams. In contrast to ESBLs, they hydrolyze cephamycins and are not inhibited by commercially available β -lactamase inhibitors [4,5]. Inappropriate use of cephalosporins in