KPC Antibiogram in a large teaching Brazilian Hospital


1 University of São Paulo, São Paulo, Brazil
2 Hospital das Clínicas, São Paulo, NA, Brazil
3 University of São Paulo, São Paulo, Brazil

Background: Describe susceptibility profile of KPC Klebsiella pneumoniae producers recovered in a teaching hospital from São Paulo.

Methods: We analyzed 27 isolates of KPC, one per patient, from July 2008 to April 2009. Identification was performed by GN1650 Cards Vitek™ (Biomerieux, Marcy l’Etoile, France); susceptibility test was performed by disk diffusion and the Hodge Test was done according to CLSI-M100S19 recommendations. blaKPC detection was done by PCR with previously described primers and Pulsed Field Gel Electrophoresis (PFGE) was also performed. Minimal Inhibitory Concentration (MIC) for polymyxin B, tigecycline and carbapenens was performed by Etest™ (Biomerieux, Marcy l’Etoile, France).

Results: KPC antibiogram showed multiple resistance as expected: 100% to ertapenem, 81.4% to meropenem, 77.7% to imipenem, 93.6% to amikacin; 82.5% to gentamicin, 89% to cefepime and cefazidime and 100% to ciprofloxacin, sulphamethoxazol/trimetropim, piperacillin/tazobactam and to aztreonam. MIC 50/90 were 2 mg/l and 3 mg/l to tigecyclin, and 2 mg/l and 48 mg/l to polymyxin B. PFGE showed that 89% belonged to the same molecular profile.

Conclusion: KPC isolates showed very few therapeutically options but resistance may emerge during treatment and its in vitro activity is not routinely recommended by CLSI.

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Decreased susceptibility to polymyxins emerging during treatment for carbapenem-resistant Enterobacter aero-
genesis infection


1 University of São Paulo, São Paulo, Brazil
2 Instituto Adolfo Lutz, São Paulo, Brazil

Background: Emergence of carbapenem resistant enterobacteriaceae (CRE) is worrisome and polymyxins are possible therapeutic options. The objective of this article is to describe emergency of polymyxins resistance during therapy.

Methods: A 30 years old male patient with bilioma after liver transplantation had eighteen successive cultures isolates of E. aerogenes (Ea) from blood and peritoneum fluid recovered during thirty days hospitalization. Identification was done by Vitek™ (Biomerieux) and antimicrobial susceptibility test were done by disk-diffusion (DD) and Etest™ (AB Biodisk) according to CLSI. Polymyxins agar dilution was also performed. All isolates were submitted to pulsed field gel electrophoresis analysis.

Results: In the third day of hospitalization the first blood culture was positive for Ea susceptible to carbapenens. MIC for polymyxin B was 1 mg/L and for colistin 0.5 mg/L. DD showed 14 mm of zone inhibition for colistin. After six days of meropenem therapy Eas isolates became resistant to carbapenens with MIC higher than 32 mg/L. Colistin therapy was initiated until patient’s death. The eight initials isolates recovered before colistin treatment had MIC less than 2 μg/ml for polymyxins and eight Ea isolates recovered after this period had MIC above 12 μg/ml for polymyxins. The fourteenth isolate, one day before the patient’s death, had MIC of 64 mg/L for polymyxin B and colistin. DD inhibition zone for colistin was 7 mm. Correlation between DD and MIC above 81g/ml for polymyxins was seen with disk inhibition zones inferior than 13 mm. All Ea belonged to the same clone.

Conclusion: CRE is a therapeutically and epidemiological challenge in every hospital. Colistin is one of the therapeutically options but resistance may emerge during treatment and its in vitro activity is not routinely recommended by CLSI.

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Outbreak of (OXA-66 carbapenemase) multidrug-resistant Acinetobacter baumannii in a Spanish tertiary-care hospital: Epidemiology and study of patient movements

F. Gonzalez, E. Culebras, J. Head, M. Gomez, G. Morales, J. Picazo

Hospital Clinico San Carlos, Madrid, Spain

Background: Acinetobacter baumannii is an increasingly common nosocomial pathogen. Carbapenems have been the agents of choice for severe Acinetobacter infections. We describe an outbreak of multidrug-resistant (MDR) A. baumannii that produced OXA-66 carbapenemase and was resistant to imipenem. We also analyze the relationship between the spread of this strain and patients' movements within the hospital.

Methods: Thirty-one isolates of A. baumannii with very similar susceptibility patterns from 15 patients, recovered in a 2 months period, were studied. We analyzed 8 more isolates recovered during the following year. ERIC-PCR and RAPD genotyping methods were used to define clusters of clonally related isolates. PFGE was used to confirm the results and to check the maintenance of the epidemic strain over the following year. Patterns of possible transmission were analyzed by recording patient movements within the hospitals. Antibiotic susceptibility testing to 28 agents was performed by microdilution and by E-test. The isolates were
screened by PCR analysis with primers specific for 6 carbapenemase genes. Amplification products were sequenced to determine the gene present.

Results: Twelve of the 15 patients studied were hospitalized at the ICU. The most frequent sites of isolation were the respiratory tract (16) and the blood (11). With the exception of colistin (0% resistance) there were no antibiotics with good activity against these isolates. Of the other antibiotics tested, ticagycline showed the best activity with an MIC90 value of 2 mg/L. The genotypic study revealed that the same strain was responsible for all the infections. All the isolates harboured the bla-OXA-51-like gene and the 6 of them chosen to sequence the gene were identical and 100% homologous with the bla-OXA-66 gene. PCR showed that the insertion sequence IS4aba1 was present upstream of the oxacillinase gene in all the isolates.

Conclusion: a) Molecular typing revealed that the outbreak detected in our hospital was due to a single A. baumannii clonal group. b) The epidemic strain of A. baumannii produces an OXA-66-IS4aba1 carbapenem-hydrolyzing oxacillinase. c) Even though the outbreak was controlled, and the number of isolates decreased significantly, the clone responsible of the outbreak persisted at the hospital during the following year.

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Online games teaching children hygiene and antibiotic resistance: Evaluation of the e-Bug games

D. Farrell1, P. Kostkova1,*, J. Weinberg1, D. Lecky2, C. McNulty2

1 City University, London, United Kingdom
2 Health Protection Agency, Gloucester, United Kingdom

Background: e-Bug is a EC-funded antibiotic, hygiene teaching resource aiming to reinforce an awareness of microbes, respiratory hygiene and prudent antibiotics use among junior and senior school children across Europe. e-Bug junior web games were developed for children age 9-12 years. The e-Bug junior game has a number of “levels” teaching the given set of learning outcomes (LOs). Player, shrunken inside human body, interacts with good and bad cartoon microbes (Fig 1) as well as antibiotics and viruses. Teaching the LOs is implemented through interaction with microbes (making yogurt (Fig 3), finishing course of antibiotics (Fig 2). Knowledge is tested seamlessly before and after each level in a Game Show style similar to the game ”Do you want to be a Millionaire?”

Methods: Evaluation was primarily conducted in the UK and online demonstrating statistically significant knowledge gain of the learning outcomes. This was complemented by focus groups and observational studies with 29 pupils taking part (and fully completing the pre and post questionnaire) from three schools. Before playing the game, only 4 pupils “agreed” that fungi were microbes while after playing 18. Smaller improvements were seen in other questions including: ”We use microbes to make things like bread and yogurt” (11 correct before, 23 correct after) and ”Soap can be used to wash away bad bugs” (20 before vs 24 after)

Conclusion: a) Molecular typing revealed that the outbreak detected in our hospital was due to a single A. baumannii clonal group. b) The epidemic strain of A. baumannii produces an OXA-66-IS4aba1 carbapenem-hydrolyzing oxacillinase. c) Even though the outbreak was controlled, and the number of isolates decreased significantly, the clone responsible of the outbreak persisted at the hospital during the following year.

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Antimicrobial utilization and susceptibility patterns of a sentinel group of bacterial isolates prior and subsequent to the introduction of Ertapenem to the hospital formulary

J. Araujo1,*, C. Rodriguez-Osorio1, E. Criollo-Mora1, A. Ramos-Hinojosa1, A. Macias Hernandez2, A. Ponce-de-Leon1, J. Sifuentes-Osorio1

1 INSTITUTO NACIONAL DE CIENCIAS MEDICAS Y NUTRICION SALVADOR ZUBIRAN, TLALPAN, Mexico
2 National Institute of Medical Sciences, Mexico City, Mexico

Background: It has been suggested that after the introduction of ertapenem into hospital use, the amount of other carbapenems and the rate of resistance to other antimicrobials decreases. We conducted a retrospective study to evaluate these outcomes.

Methods: We studied 48 months (two 24 month-periods) before (PRE) and after (POST) the introduction of ertapenem in August 2004. Antibiotic use was determined using the standard defined daily dose (DDD) per 1000 patient/days. Antimicrobial susceptibility testing to Escherichia coli, Pseudomonas aeruginosa, Klebsiella pneumoniae and Acinetobacter baumannii was performed with the Vitek automated system. We used chi-square for trend to compare rates of resistance and changes in antimicrobial use.

Results: In the PRE-period, we analyzed 4072 E. coli, 677 P. aeruginosa, 571 K. pneumoniae, and 67 A. baumannii isolates; in the POST 4648, 884, 559, and 109, respectively; antibiotic consumption was as follows: PRE-period: meropenem 89.9 DDD/1000/patients/days, antipseudomonal cephalosporins 90.9, and ceftriaxone 195.65; POST period: 75.3, 103.58, and 184.77, respectively. The rate of antimicrobial susceptibility was: E. coli, to meropenem in PRE-period 99.6% and in POST-period 98.05%, to ceftazidime 88.67% and 84.75% (p < 0.000), respectively; to P. aeruginosa, meropenem 67.41% and 61.74% (p = 0.004), respectively, ceftazidime 63.74% and 62.12% (p = 0.3388), respectively, and piperacillin/tazobactam 69.17% and 76.14% (p = 0.12), respectively.