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Médecine et maladies infectieuses 45 (2015) 133–138

Médecine et
maladies infectieuses

Original article

A comparative study of antimicrobial resistance rates and phylogenetic groups of community-acquired versus hospital-acquired invasive *Escherichia coli*

Comparaison de la résistance aux antibiotiques et des groupes phylogénétiques des souches d'Escherichia coli isolées d'infections invasive : origine communautaire et nosocomiale

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Received 7 November 2014; received in revised form 19 November 2014; accepted 27 January 2015

Available online 26 February 2015

Abstract

Objectives. – *Escherichia coli* is the leading cause of various infections, both in community and nosocomial settings. Our objective was to determine the antibiotic resistance rates and the phylogenetic groups of invasive *E. coli* and to assess the relationship between these characteristics according to the community or nosocomial origin of the strains.

Materials and methods. – One hundred non-redundant *E. coli* strains, causing invasive infections, were collected and investigated between 2010 and 2012. The phylogenetic groups were determined by triplex PCR. The statistical analysis was performed with Pearson χ^2 test and *P*-values below 0.05 were considered as statistically significant.

Results. – Sixty-three strains were community-acquired (CA) and 37 were hospital-acquired (HA). The resistance rates among CA and HA strains were respectively: cefotaxime (11.1/37.8%), ciprofloxacin (19/43.2%), amikacin (3.2/27.2%), and cotrimoxazole (42.8/64.8%). *E. coli* strains caused bacteremia (CA = 34.9%; HA = 83.7%), peritonitis (CA = 58.7%; HA = 13.5%), appendicitis (CA = 3.2%; HA = 2.7%), and cholecystitis (CA = 3.2%; HA = 0%). The distribution of phylogenetic groups among CA and HA strains was: A (25.4/18.9%), B1 (9.5/16.2%), B2 (23.8/37.8%), and D group (41.3/27%). High resistance rates to cefotaxime ($P = 0.02$), ciprofloxacin ($P = 0.01$), amikacin ($P = 0.001$), and cotrimoxazole ($P = 0.05$) were statistically significantly associated with a nosocomial origin.

Conclusion. – Our results prove the diversity of phylogroups among *E. coli* invasive strains whatever their origin, and a higher antibiotic resistance rate in nosocomial strains. An adequate use of antibiotics and applying strict hygiene measures would limit the transmission and selection of these bacteria in hospital as well as in community settings.

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Keywords: *Escherichia coli*; Community acquired infections; Nosocomial infections; Phylogenetic groups

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Résumé

Objectif. – Notre objectif était d'identifier les profils de résistance aux antibiotiques et les groupes phylogénétiques de souches d'*E. coli* responsables d'infections invasives et de les comparer selon leur origine communautaire ou nosocomiale.

Matériel et méthodes. – Entre 2010 et 2012, 100 souches non répétitives d'*E. coli* responsables d'infections invasives ont été collectées au laboratoire de microbiologie de l'hôpital Charles-Nicolle. Les groupes phylogénétiques ont été déterminés par la technique PCR triplex. L'étude statistique a été réalisée par le test χ^2 de Pearson.

Résultats. – Parmi les 100 souches, 63 étaient d'origine communautaire et 37 d'origine nosocomiale. Les taux de résistance aux antibiotiques chez les souches isolées d'infection communautaire et nosocomiale étaient respectivement : céfotaxime (11,1/37,8 %), ciprofloxacine (19/43,2 %), amikacine (3,2/27,2 %) et cotrimoxazole (42,8/64,8 %). Nos souches étaient responsables de bactériémie (CA = 34,9 % ; HA = 83,7 %), péritonite (CA = 58,7 % ; HA = 13,5 %), appendicite (CA = 3,2 % ; HA = 2,7 %) et de cholécystite (CA = 3,2 % ; HA = 0 %). La distribution des groupes phylogénétiques parmi les souches d'origines communautaires et nosocomiales était : A (25,4/18,9 %), B1 (9,5/16,2 %), B2 (23,8/37,8 %) et D (41,3/27 %). Une relation statistiquement significative a été retrouvée entre la résistance au céfotaxime ($p=0,02$), à l'amikacine ($p=0,001$), à la ciprofloxacine ($p=0,01$) et au cotrimoxazole ($p=0,05$) et l'origine nosocomiale des souches.

Conclusion. – Nos résultats montrent la diversité des groupes phylogénétiques des souches invasives d'*E. coli* dans le milieu hospitalier ainsi que dans la communauté. L'utilisation raisonnée et contrôlée des antibiotiques ainsi que l'application stricte des moyens d'hygiène permettraient de lutter contre la transmission et la sélection de ces bactéries aussi bien dans le milieu hospitalier que communautaire.

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Mots clés : *Escherichia coli* ; Groupes phylogénétiques ; Infections communautaires ; Infections nosocomiale

1. Introduction

E. coli is a commensal bacterium of the inhuman intestinal tract. It is the most frequently isolated gram-negative bacillus in community as well as in nosocomial-acquired infections [1]. Invasive infections caused by *E. coli* such as bacteremia, deep-seated infections, soft tissue infections, etc. are usually due to highly adapted clones with a great variety of virulence factors (VF) including adhesins, invasins, and protectins [2]. The results of previous phylogenetic analysis have shown that *E. coli* strains fall into 4 main phylogenetic groups: A, B1, B2, and D [3]. Group B2 and D strains carried various VF and to cause different extra-intestinal infections. *E. coli*, as most bacteria, has developed resistance to standard as well as to modern antibiotics with dramatic recent increases in resistance to 3rd generation cephalosporins and fluoroquinolones, resulting in serious therapeutic issues.

Our objective was to assess the antibiotic resistance rates and phylogenetic groups of invasive *E. coli* according to their community or nosocomial origin.

2. Materials and methods

2.1. Definitions

Community-acquired (CA) infections were defined as infections in which the onset of patient symptoms occurred before admission or within 48 h of admission to the hospital [4].

Hospital-acquired (HA) infections were defined as infections in which the onset of symptoms occurred more than 48 h after admission [4].

Invasive infection was defined as a localized or systemic inflammatory response in otherwise sterile anatomical sites [5].

2.2. Patients and bacterial isolates

2.2.1. Patients' data

The clinical data was recorded for each patient including: age, sex, ward, infection type, and nosocomial or community origin of the infection.

2.2.2. Bacterial isolates

One hundred invasive, consecutive, and non-repetitive *E. coli* isolates from 100 patients were collected from various specimens submitted to the Charles Nicole hospital clinical microbial laboratory in Tunis, between January 2010 and February 2012. Isolates were identified by the Api 20E system (bioMérieux, Marcy l'Étoile, France). Antimicrobial susceptibilities were determined using the standard disk diffusion method on Mueller Hinton agar according to Clinical Laboratory and Standards Institute guidelines (CLSI, 2010) [6]; and extended-spectrum β -lactamases (ESBL) production was detected by the double disk synergy test [7].

2.3. Phylogenetic analysis

Phylogenetic groups A, B1, B2, and D were determined using triplex PCR as previously described [3]. Each reaction was carried out by using a 20- μ l mixture containing 2 μ l of 10X *Taq* polymerase buffer, 20 pmol of each primer, each deoxynucleoside triphosphate was used at a concentration of 2 mM, 2.5 U of *Taq* polymerase (Biomatik), and 2 μ l of genomic DNA. The PCR was performed with a Perkin-Elmer GeneAmp 2400 thermal cycler under the following conditions: denaturation for 5 mins at 94 °C; 30 cycles of 30 s at 94 °C, 30 s at 55 °C, and 30 s at 72 °C, and a final extension step of 7 mins at 72 °C. The *chuA* and *yjaA* genes and TspE4.C2 DNA fragment were amplified. The primer pairs used were ChuA.1 (GACGAACCAACG-GTCAGGAT) and ChuA.2 (TGCCGCCAGTACCAAAGACA),

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