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Molecular characterization of *clostridium difficile* isolated in Hong Kong SAR

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Background: *Clostridium difficile* is a common cause of pseudomenbranous colitis and more often, antibiotic-associated diarrhea, and remains the most common cause of nosocomial diarrhea (15-39%). In 2003, a nosocomial outbreak caused by a hypervirulent strain of *C. difficile* ribotype 027 was first identified in Quebec, Canada. This strain has now been documented in outbreaks across the US and European countries. The aim of this study was to characterize the toxigenic profile and genotype of *C. difficile* isolated from hospitalized patients in Hong Kong.

Methods: At total of 204 isolates of *C. difficile* from diarrheal stool samples of hospitalized patients in Hong Kong during the period from January 2009 to December 2009 were recruited in this study. The presence of genes encoding toxin A (*TcdA*), toxin B (*TcdB*) and binary toxins (*cdt*A and *cdt*B) were detected by multiplex PCR. Single-locus sequence typing of the surface layer protein A gene (slpAST*)* was also performed for genotyping of these *C. difficile* isolates.

Results: Amongst these 204 *C. difficile* isolates, 35 (17.16%) strains were found to be non-toxigenic, 151 (74.02%) strains were toxin A + B+, 4 (1.96%) strains possessed toxin A, B and binary toxins, and 5 (2,45%) strains were toxin A-B+. Using slpAST, most isolates were identified as PCR-ribotype 002 (32.8%). The second commonest *C. difficile* strain was og39 (7.8%), followed by ribotype 018 (5.9%), ribotype 010 (4.9%), ribotype 066 (4.9%), ribotype 014 (3.9%), ribotype 017 (2.9%), and ribotype 001 (2.0%).

Conclusion: Ribotype 002 was the predominant genotype in Hong Kong and no ribotype 027 was detected in this study. Although molecular typing is not necessary for routine diagnosis, it is useful in epidemiology studies and recognizing the high-risk strain like ribotype 027 which would facilitate strategic infection control interventions especially during outbreak.

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Virulence genotypes and phylogenetic distribution between upper and lower community-onset urinary tract infections caused by *Escherichia coli*

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Background: The aim of this study was to find the prevalence difference of virulence genes between upper urinary tract infection (UUTI) uropathogenic *E. coli* (UPEC) and lower urinary tract infection (LUTI) UPEC among community-onset UPEC (CO-UPEC).

Methods: We conducted a case-control study of patients with positive UPEC between 2006 and 2007 at a teaching hospital in South Korea. CO-UPEC was the isolated from the patients from whom the specimen was obtained within 2 calendar days of hospitalization or at outpatient department. We divided CO-UPEC into UUTI- and LUTI-UPEC. All isolates underwent PCR-based microarray for phylotyping and detection of 11 virulence factors (VFs), including adhesins (*prf, sfa, drb, prsGj96, papGad*), toxins (*hly, cnf1*), siderophores (*iroN, aer*), capsule (*kpsMT*), and other (*ompT*). The virulence score was defined as the number of virulence genes identified. We also collected the rectal commensal *E. coli* (RCEC) of healthy people.

Results: A total of 548 CO-UPEC (UUTI-UPEC, n = 324; LUTI-UPEC, n = 224) and 183 RCEC isolates were collected. Compared with LUTI-UPEC, UUTI-UPEC showed higher virulence scores (4.38 ± 1.94 vs. 3.23 ± 2.41, $p < 0.001$) and more frequent traits in some VFs (*prf*, Odd ratio = 2.47 [95% confidence interval = 1.98 - 3.08]; *aer*, 1.31 [1.07 – 1.60]; *kspMT*, 1.76 [1.46 - 2.13]; *ompT*, 1.75 [1.45 - 2.12]; *papGad*, 3.11 [2.40 – 4.03]) but less frequent traits in some VFs(*drb*, 0.73 [0.56 -0.97]; *prsGj96*, 0.63 [0.48 – 0.83]). RCEC had less VFs than UUTI- and LUTI-UPEC. Phylogenic group B2 was more prevalent in UUTI-UPEC than LUTI-UPEC (58.3% vs. 44.2%, $p < 0.001$), however LUTI-UPEC belonged to phylogenic group A and B1 more frequently (13.8% vs. 4.0% and 9.8% vs. 4.0%, respectively, $p < 0.001$).

Conclusion: Even though UTI-UPECs previously considered as same entity, UUTI-UPEC was more virulent than LUTI-UPEC.

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