Outbreak of Clostridium difficile 027 infection in Vienna, Austria 2008-2009

A Indra (Alexander.Indra@ages.at)1, S Huhulescu1, A Fiedler1, S Kernbichler1, M Blaschitz1, F Allerberger1
1. Austrian Agency for Health and Food Safety, Vienna, Austria

From November 2008 to 15 April 2009, 36 isolates of CD027 were identified in Austria, all originating from four hospitals in Vienna. All isolates were positive for toxin A, toxin B and the binary toxin, and showed a characteristic 18 bp deletion in the tcdC gene.

Clostridium difficile is an anaerobic spore-forming bacterium. Some strains may cause diarrhoea due to formation of toxins. Symptomatic C. difficile infection (CDI) is primarily linked with hospital admission and antibiotic treatment, although antibiotic exposure is neither necessary nor sufficient for CDI [1,2]. In Belgium, for instance, one third of CDI cases reported in the hospital surveillance system are not hospital-associated [3]. Symptoms range from mild diarrhoea to serious manifestations such as pseudomembranous colitis, toxic megacolon or perforation of the colon. C. difficile challenges hygiene standards as it forms spores. The risk of infection rises with increasing age, underlying disease and immunodeficiency [4].

In recent years, a particularly virulent strain, ribotype 027 (CD027), has emerged in a number of countries, particularly in connection with hospital outbreaks, but also in community-acquired diarrhoea cases [5]. The risk of serious disease and death associated with CD027 exceeds that of other C. difficile strains. The classical CD027 is characterised – among other things – by an increased production of toxins A and B, production of a binary toxin and resistance to newer fluoroquinolones such as moxifloxacin. The first three Austrian cases of CD027 occurred in 2006 and in March 2008 [6,7].

Since August 2006, the Austrian National Reference Centre for C. difficile has ribotyped approximately 2,700 human C. difficile isolates received from all nine Austrian provinces. In recent months, a drastic increase in CD027 cases has been noted, all originating from four hospitals in Vienna. From November 2008 to 15 April 2009, 36 isolates of CD027 were received at the National Reference Centre. The Figure summarises these C. difficile 027 cases by month of reception of the sample at the reference centre.

In contrast to the two isolates from March 2008, which were susceptible to fluoroquinolones, all 36 CD027-isolates cultured since November 2008 showed in vitro resistance against moxifloxacin. Five of the 36 isolates also showed in vitro resistance against clindamycin (with minimum inhibitory concentrations (MICs) of ≥256 µg/ml), 14 of the 32 isolates showed intermediate susceptibility for clindamycin (MICs of 4 µg/ml), and 13 isolates were susceptible (MICs of 2 µg/ml). All isolates were positive for toxin A, toxin B and the binary toxin, and showed a characteristic 18 bp deletion in the tcdC gene. For 28 of 36 recent PCR-ribotype 027 cases basic demographic data were available. Of those, 17 were female and the median age was 80 years (range: 60-97 years). At least four of the 28 cases were fatal.

CDI is not a reportable disease in Austria. Hospital discharge data indicate a significant increase of CDI during the last years, from 777 cases (54 deaths) in 2003 to 997 cases (80 deaths) in 2004, 1,453 cases (88 deaths) in 2005, 2,192 cases (150 deaths) in 2006, and 2,761 cases (219 deaths) in 2007. While the increase in incidence of CDI in Austria over the last years is not due to CD027, the Austrian Agency for Health and Food Safety has nevertheless advised hospitals to intensify the monitoring of CDI. Increased attention should be given to possible cases of nosocomial diarrhoea, particularly after antibiotic treatment. Clinical microbiology departments are asked to submit isolates from all cases with severe manifestations and on suspicion of an outbreak.
References


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