Correspondence

IMP-1 carbapenemase detected in an Acinetobacter clinical isolate from the UK

Luke Tysalla, Mark W. Stockdalea, Paul R. Chadwickb, Marie-France I. Palepoua, Kevin J. Townera, David M. Livermorea and Neil Woodforda*

aAntibiotic Resistance Monitoring and Reference Laboratory, Central Public Health Laboratory, London NW9 5HT; bMicrobiology Department, Salford Royal Hospitals NHS Trust, Hope Hospital, Salford M6 8HD; cPublic Health Laboratory, University Hospital, Nottingham NG7 2UH, UK

*Corresponding author. Tel: +44-20-8200-4400; Fax: +44-20-8358-3292; E-mail: nwoodford@phls.nhs.uk

Sir,

The emergence of carbapenemases in Acinetobacter spp. has been called a ‘global sentinel event’, warranting prompt epidemiological and microbiological interventions.1 In a recent survey of antimicrobial resistance among 595 clinical isolates of Acinetobacter spp. collected at 54 hospitals in the UK, 13 isolates resistant to either imipenem or meropenem, or to both agents, were found (MICs > 4 mg/L) (Henwood et al., submitted). Twelve of these isolates failed to yield PCR products with primers for the blaIMP, blaVIM and blatoXA genes known to encode carbapenemases, but one strain (A1411) gave a PCR product with primers specific for blaIMP alleles and yielded extracts that hydrolysed imipenem in a spectrophotometric assay. We report here details of the patient from whom the organism was isolated, and of the carbapenemase gene present.

The patient was a 45-year-old man who had been on home parenteral nutrition for 15 years for short bowel syndrome secondary to Crohn’s disease; he also had eczema, osteoporosis and ankylosing spondylitis. He had been admitted to the intensive care unit (ICU) for 3 weeks in October 1998 with severe osteomyelitis of the iliac crest, and was transferred to hospital contacts until April 2000, when he had an episode of hip sepsis. He was admitted in July 2000 for 3 days with suspected line infection and was treated empirically with vancomycin. Although the line infection was not confirmed, strain A1411 was isolated from one of six blood culture bottles. This organism was not thought to be clinically significant, but was identified subsequently as Acinetobacter junii (genomic species 5) by the tRNA fingerprinting technique.2 A. junii is an unusual pathogen, but has been isolated previously, albeit rarely, from blood cultures and central venous lines.3 The patient did not receive meropenem or imipenem during any of his admissions. Both carbapenems were stock items at that time, but hospital records showed only modest use of meropenem, comprising 131 defined daily doses on the ICU and 348 for the trust during the 1998/1999 financial year. Very little imipenem has been used at this hospital during the past 5 years, and the patient had no history of foreign travel or other links with the Far East.

Sequencing indicated that the 741 bp blaIMP allele from A1411 (GenBank accession number AY055216) encoded a polypeptide identical to classical IMP-1 enzyme, although the gene contained seven silent mutations in comparison with other blaIMP sequences available in the database (e.g. accession numbers AY223604, D29636 and S71932). Previously, IMP-1 enzyme has only been reported in scattered isolates of Pseudomonas aeruginosa, Serratia marcescens and Klebsiella pneumoniae in Japan, and in a single K. pneumoniae isolate from Singapore,4 whereas alleles specifying other IMP enzymes have been detected in isolates of P. aeruginosa, Acinetobacter baumannii, and various Enterobacteriaceae from continental Europe, Canada and the Far East. An 87 bp sequence upstream of the blaIMP start codon in A1411 was identical to part of attI, an integron-associated recombination site.5 Therefore, the carbapenemase allele is likely to be a cassette within a class 1 integron, as is blaIMP-1 in many Japanese isolates.6

Carbapenemases belonging to β-lactamase molecular classes A, B and D are emerging in Acinetobacter spp., Pseudomonas spp. and the Enterobacteriaceae.4 The sources of these resistance genes are unknown. The allelic nature of the enzymes, particularly those of the IMP and VIM families, is consistent with multiple gene escapes from undefined donor species, and strain A1411 may represent a new example of such an escape. A more worrying possibility is that IMP-1 has spread to the UK, but has remained undetected to date. In this context it should be emphasized that carriage of a carbapenemase gene does not always result in the expression of phenotypic carbapenem resistance.4 Strain A1411 had only borderline resistance to carbapenems. It was reported as susceptible to meropenem following disc susceptibility testing in the clinical laboratory, where imipenem was not tested. In the reference laboratory the strain was noted to show low-level meropenem resistance by an agar incorporation method (MIC 8 mg/L),
but remained susceptible to imipenem (MIC 2 mg/L), although the MIC was raised in comparison with the mode for the genus. The prevalence of _bla_{IMP-1}_ and other carbapenemase genes in apparently carbapenem-susceptible _Acinetobacter_ isolates should be established as a matter of priority.

**References**


