Acinetobacter sp. has emerged as a major hospital pathogen (8). The greatest concern has been the emergence of carbapenem resistance in Acinetobacter baumannii by the acquisition of OXA-type carbapenemase or metallo-β-lactamases, since few effective antimicrobial agents exist. Several mechanisms can underlie carbapenem resistance in A. baumannii (1), but less is known about carbapenem resistance in non-A. baumannii species (4). There have been only three reports about Acinetobacter soli (5, 6, 8), with no mention of carbapenem resistance. We isolated carbapenem-resistant A. soli from two Japanese patients with bloodstream infections.

In January and April 2011, carbapenem-resistant A. soli was isolated from blood cultures of two patients at the Tohoku University Hospital. A central venous catheter was in situ in both cases. The species was identified by partial sequencing of the rpoB gene (7). MICs were determined by the agar dilution method of the Clinical and Laboratory Standards Institute (2).

To detect OXA–51-like, OXA–23-like, OXA–24-like, and OXA–58-like carbapenemases and IMP–1–, IMP–2–, VIM–1–, VIM–2–, SIM–1–, and NDM–1–type metallo–β-lactamase genes, PCR was performed (3, 9). The proximity of ISAbal, ISAbal2, ISAbal3, and IS18 to blaOXA–58-like genes (9) and the carO (outer membrane protein) gene (1) was investigated by PCR. The OXA-type carbapenemase and metallo–β-lactamase genes were sequenced. Pulsed-field gel electrophoresis (PFGE) was done with the SmaI restriction enzyme (11). Isolates with >80% similarity were considered to be within the same cluster (10).

The MIC of imipenem was ≥16 μg/ml for both isolates (Table 1). PCR showed that one isolate possessed only the IMP–1 gene, while the other had both IMP–1 and OXA–58-like genes. No other carbapenem resistance genes were detected. The OXA–58-like carbapenemase gene was not linked to ISAbal, ISAbal2, ISAbal3, or IS18. Sequencing of the blaOXA–58-like, and blaIMP–1 genes yielded OXA–58 and IMP–1, respectively. Both isolates exhibited decreased expression of carO. Thus, the mechanism of resistance in one of these isolates could involve a synergistic interaction between IMP–1 expression and reduced expression of an outer membrane protein. The two isolates had different PFGE patterns (not shown).

Currently, 33 genomic species of the Acinetobacter genus have been identified by molecular methods (5). A. baumannii is generally the pathogen isolated most frequently in clinical settings, although it is difficult to perform accurate species identification at many institutions. Recently, sequencing has provided reliable identification of Acinetobacter isolates to the species level in laboratories (7), and severe infections caused by non-A. baumannii clinical isolates have been reported (5, 8). To our knowledge, however, carbapenem-resistant A. soli isolates have not been reported previously.

Three Acinetobacter isolates with imipenem MICs of ≥16 μg/ml were obtained from blood cultures at the Tohoku University Hospital over the past 5 years, and two of these isolates were identified as A. soli by partial rpoB gene sequencing. This indicates that carbapenem resistance is now present among clinical isolates of A. soli, and we should monitor its prevalence. The present findings emphasize the importance of performing accurate epidemiological investigation of non-A. baumannii species, including A. soli.

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### REFERENCES


