ABSTRACT

*Acinetobacter baumannii* has emerged as one of the most troublesome pathogens for health care institutions globally causing pneumonia, blood stream infections, wound infections and urinary tract infections. Its clinical significance has been propelled by its remarkable ability to up regulate or acquire resistance determinants, making it one of the organisms threatening the current antibiotic era. *A. baumannii* strains resistant to all known antibiotics have now been reported, signifying a sentinel event that should be acted on promptly by the international health care community.

Different clinical and environment strains of Acinetobacter were collected and subjected antimicrobial susceptibility testing. Strains which were found showing resistance to imipenem by both disk diffusion and minimum inhibitory concentration (MIC) were analysed for presence of oxacillinase genes i.e. OXA-23, OXA-58 (class D) and metallo-β-lactamase gene i.e. New Delhi metallo-β-lactamase i.e. NDM-1 (class B) by using multiplex PCR. Total number of Acinetobacter isolated were 195, of which 175 were clinical and 20 were environmental samples. Among 195 strains of Acinetobacter collected, 61(31.28%) strains (all clinical) were imipenem resistant by disk diffusion method. Of these imipenem resistant strains, 45 (23%) were further confirmed to be imipenem resistant by agar dilution method (MIC). Among thersa 45 strains resistant to imipenem by MIC detection, 14 (31%) were positive for NDM-1 gene, 19 (42.2%) were positive for OXA – 58 gene and all strains 45 (100%) were found positive for OXA – 23 gene. This study emphasizes the dissemination of OXA cabapenemase genes and its co existence with NDM-1 genes in carbapenem resistant Acinetobacter isolates.

*Acinetobacter baumannii* is a multidrug-resistant (MDR) nosocomial pathogen for which immunotherapeutic alternatives are needed. It was previously identified a surface autotransporter of *A. baumannii*, Ata, that bound to various
extracellular matrix/basal membrane proteins and was required for full virulence, biofilm formation, and the adhesion of \textit{A. baumannii} to collagen type IV. In the present study it was shown that Ata binding to collagen type IV was inhibited by antibodies to Ata. In addition, in the presence of complement and polymorphonuclear cells (PMNs), antibodies to Ata were highly opsonic against \textit{A. baumannii} ATCC 17978 and showed low to moderate killing activity against four heterologous \textit{A. baumannii} strains, whereas in the absence of PMNs, antibody to Ata efficiently promoted complement dependent bactericidal killing of all of the tested \textit{A. baumannii} isolates. Using a pneumonia model of infection in both immunocompetent and immunocompromised mice, we found that, compared to normal rabbit sera, antisera to Ata significantly reduced the levels of \textit{A. baumannii} ATCC 17978 and two MDR strains in the lungs of infected mice. The ability of Ata to engender anti-adhesive, bactericidal, opsonophagocytic, and protective antibodies validates its potential use as an antigenic target against MDR \textit{A. baumannii} infections.