ABSTRACT

Acinetobacter baumannii is a non-fermentative, non-motile, catalase-positive, non-oxidative, aerophilous and Gram-negative coccobacillus. It causes infections in immune compromised persons and the patients present in hospitals and intensive care units. It is an opportunistic pathogen accountable for diseases of the urinary tract, bloodstream, skin and other soft tissues. Various antibiotic resistance mechanisms in bacteria have been studied. It includes attainment of carbapenemase, changes in outer membrane proteins (OMPs), penicillin binding proteins (PBPs), efflux pumps, outer membrane vesicles (OMVs), phospholipases, permeability defects and modification of target sites. Outer membrane porin OprD mediated resistance occur due to different mechanisms including upsetting OprD promoter, early termination of OprD transcription process, reduced transcriptional expression via co-regulation with multi drug efflux pump, frame shift mutations and premature stop codons. Loss of OprD expression cause decreased susceptibility to carbapenems. The current study aims to determine the role of porin in resistance against imipenem in *Acinetobacter baumannii*. Imipenem disks were used to check the antibiotic resistance in environmental and clinical isolates. Environmental isolate showed larger zone of inhibition as compared to clinical isolate. OprD gene was PCR amplified and cloned in pTZ57R cloning vector. Sequence analysis showed the environmental isolate have 92% homology with other species of *Acinetobacter*. The nucleotide sequence was translated using EXPASY translate tool and molecular structure was build through homology modeling using SWISS MODEL protein server. This structure of oprD protein was compared with already reported 13TU mutant isolate. OprD protein of this mutant isolate showed the absence of pore in it used for the transport of antibiotics. This makes bacterial strain resistant to Imipenem.