Acinetobacter spp are problematic nosocomial pathogens often with multi resistance to different antimicrobial classes. Until recently, carbapenems remained active against nearly all Acinetobacter spp isolates, but resistance is increasingly reported from many countries, caused by acquired metallo-β-lactamases (MBLs) or OXA-type (class D) carbapenemases [1]. MBLs are particularly feared because of their ability to hydrolyse virtually all β lactams, including carbapenems, but this potential also exists among OXA-type carbapenemases. With the increase in resistance, resistance surveillance has become increasingly important. The rapid global emergence of Acinetobacter strains resistant to all β lactams, including carbapenems, illustrates the potential of this organism to respond swiftly to changes in selective environmental pressure. Upregulation of innate resistance mechanisms and acquisition of foreign determinants are critical skills that have brought A. baumannii great respect. The first identified OXA type enzymes with carbapenem hydrolyzing activity was from a clinical Acinetobacter strain isolated in 1985 from Edinburg, Scotland[132]. This plasmid encoded resistance determinant (initially named ARA-1) was found to be transferable, and the gene was later sequenced and named bla_{OXA-23}[132]. Previous reports have indicated that in UK OXA-23 and OXA-51 are most frequently detected in Acinetobacter [133]. OXA-23 gene is one of the most prevalent carbapenemases encoding genes reported worldwide, which can be located on chromosome or plasmids [134]. Similarly in this study all the strains were found to be positive for OXA-23. Similarly OXA-58 is also globally scattered among Acinetobacter islates. OXA-58 was identified more recently and similar to OXA-23 often plasmid mediated, which may explain its wide spread distribution. OXA-58 may be present along with OXA-23 which is responsible for reduced susceptibility to carbapenem group of drugs. NDM-1 metallo-β-lactamase was detected recently among Enterobacteriaceae and also in Acinetobacter, especially in
India and Pakistan [135]. A recent study in India showed the coexistence of OXA-23 and NDM-1 in clinical strains of Acinetobacter [136]. Similarly in our study we observed the coexistence of OXA-23 and NDM-1 gene. In a recent study in India NDM-1 gene was found in Acinetobacter, where all NDM-1 producing Acinetobacter were lacking OXA-58 [137] but in our study we found coexistence of NDM-1 and OXA-58. We also found presence of all three classes of genes in some strains. Hence use of multiplex PCR is quite convincing in simultaneous detection of different classes of carbapenemases genes. Even for epidemiologic surveys multiplex PCR technique may be very helpful and reduce the cost and duration of multiple PCR reactions. With increase in drug resistance in Acinetobacter, resistance surveillance has become increasingly important. Hence both the phenotypic and genotypic methods are important to detect the carbapenem resistance in Acinetobacter and techniques like Multiplex PCR would help to monitor the emergence and spread of carbapenem resistant Acinetobacter.