Since the first cases of Acquired Immunodeficiency Syndrome (AIDS) were reported in 1981, infection with Human Immunodeficiency Virus (HIV) has grown to pandemic proportions, resulting in an estimated 65 million infections and 25 million deaths. Fusion and entry of Human Immunodeficiency Virus type 1 (HIV-1) into target cell requires the binding of the external envelope glycoprotein gp 120 to both the CD4 molecule and one of several chemokine receptors, which function as co-receptors. Chemokine receptor-2 (CCR2) is a co-receptor for the entry of human immunodeficiency virus-1 (HIV-1) into the target cells. Genetic polymorphism of CCR2 human chemokine receptors have been associated with resistance during HIV-1 infection and disease progression. The protective effect of mutant alleles at these loci has important implications in AIDS pathogenesis. CCR2 is a member of the superfamily of seven transmembrane domain G protein-coupled receptors, the largest receptor superfamily in the human genome. CCR2 acts as a receptor for MCP-1 (CC chemokine) and as a co-receptor for HIV-1 cell-target entry. The G-to-A transition at position 190 characterizes the CCR2-641 mutation, causing valine to isoleucine substitution in codon 64. This mutation has been identified as an important factor for delaying progression to AIDS. In this study samples already collected from HIV screening centres, at the provincial hospitals in all the eight provinces of Kenya were analyzed. Peripheral blood mononuclear cells (PBMCs) were extracted from whole blood. Genomic deoxyribonucleic acid (DNA) was extracted from PBMCs. The genotypes of CCR2-641 were determined by Polymerase Chain Reaction (PCR) using specific primers. All the PCR amplicons were analyzed by gel electrophoresis. CCR2 PCR-amplified genomic regions were subjected to restriction fragment length polymorphism (RFLP) analysis with the digestion of restriction enzyme endonuclease BsaBI to differentiate between the mutant and the wild type CCR2 genes. Allele and genotype frequencies from each province were calculated by direct counting. In this study, the presences of CCR2-641 allelic variants in HIV-1 positive samples in the Kenyan provinces were analyzed. One hundred and eighteen samples were genotyped for the CCR2-641 mutation by PCRRFLP among which 4(3.4%) were homozygous mutants (mut/mut) and 21(17.8%) were heterozygous (wt/mut) for the CCR2-641 polymorphism, with the remaining 93(78.8) samples being wild-type homozygote (wt/wt) giving the frequency of the CCR2-641 allele in this survey as 0.21. Identifying HIV CCR2-641 allelic variants and their distribution may help understand the burden and course of the disease which may be important in clinical decision making.