TO STUDY THE BREAKPOINT CLUSTER REGION-ABELSON GENE MUTATION AND THEIR RESISTANCE TO IMATINIB, A TYROSINE KINASE INHIBITOR IN CHRONIC MYELOID LEUKEMIA IN ASSAM, INDIA

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The TKI (tyrosine kinase inhibitor) Imatinib, which has improved the 5-year survival rate from less than 20% to over 90%, has drastically changed the therapy and natural history of CML (chronic myeloid leukemia) in recent years. The BCR-ABL (breakpoint cluster region-abelson) mutations and their TKI resistance mechanisms are discussed in this study. Following the completion of the IRIS (International Randomized Study), which was initiated in June 2000, Imatinib was approved by the Food and Drug Administration as the first-line treatment for CML in December 2002. Imatinib causes the BCR-ABL protein to switch to the inactive conformation, which causes leukemic cell death and growth arrest by binding to amino acids in the ABL kinase domain and blocking adenosine triphosphate binding. Patients receiving Next Generation TKIs experienced significantly fewer fatalities from CML. Asciminib, a next-generation BCR-ABL1 inhibitor, has been discovered to have substantial potential in the treatment of CML, in contrast to earlier approved TKIs. Nearly 50% of CML patients receiving frontline TKI treatment in 2021 will need a 10-year change in therapy. Ponatinib, a third-generation inhibitor, has demonstrated promising efficacy and tolerance to T35I in CML patients. This review also covers the response and resistance of the BCR-ABL mutation against generations of TKI, utilizing four case studies of CML patients. Exosome research in CML patients, digital PCR, gene editing techniques, and the new generation inhibitor Asciminib's position in the larger picture of CML will be determined by further research. Given the necessity of further therapeutic alternatives, these facts have been underlined in this study.