ISSN: 2329-6771

Precision Medicine in Oncology: From Genomics to Target

Maria Klepper*

Department of Molecular Medicine, Baylor College of Medicine, Houston, USA

Introduction

Precision medicine in oncology, often referred to as personalized medicine, is an approach to cancer treatment and care that takes into account the individual genetic, molecular, and clinical characteristics of a patient and their tumor. The goal of precision medicine is to tailor cancer diagnosis, treatment, and management to maximize effectiveness and minimize side effects. While most previous studies have focused on targeting cancer cells with a dismal prognosis, novel therapies targeting stromal components are currently being evaluated in preclinical and clinical studies, and are already showing improved efficacies. As such, they may offer better means to eliminate the disease effectively [1].

Description

A cornerstone of precision medicine in oncology is the analysis of the patient's tumor at the genomic level. This involves identifying specific genetic mutations, alterations, and markers within the tumor DNA. Technologies like Next-Generation Sequencing (NGS) enable comprehensive genomic profiling. A cornerstone of precision medicine in oncology is the analysis of the patient's tumor at the genomic level. This involves identifying specific genetic mutations, alterations, and markers within the tumor DNA. Technologies like nextgeneration sequencing (NGS) enable comprehensive genomic profiling. Once specific genetic abnormalities are identified, targeted therapies are employed. These are drugs that precisely target the molecular pathways responsible for cancer growth. For example, tyrosine kinase inhibitors and immune checkpoint inhibitors are used based on the tumor's genetic profile. Very little information is accessible about the wellbeing and adequacy of inoculations for immunosuppressed patients. As a result, immune compromised kids are under-inoculated and helpless against immunization preventable diseases. It is extremely challenging to concentrate on the adequacy of inoculations for uncommon illnesses, for example, paediatric CML because of the tiny example size. Organic boundaries exhibiting a defensive impact in sound people may not be extrapolated to immune compromised people.

Lower reactions to immunization in immunosuppressed people contrasted and solid individuals are normal, and little information exists on the sturdiness of the reaction. Concerning live immunizations, with a couple of special cases, these are by and large viewed as contraindicated in immunosuppressed people due to somewhere safe and secure worries. The pervasiveness of youngsters with CML is continually expanding as the sickness turns out to be more treatable. Biomarkers are molecular or genetic indicators that help predict how a patient will respond to a specific treatment. They can also be used for prognosis and to anticipate potential side effects. The purpose of the study was to investigate the effectiveness of Human Growth Hormone (hGH) therapy in CML children receiving imatinib and to assess the GHRH-GH-IGF1

*Address for Correspondence: Maria Klepper, Department of Molecular Medicine, Baylor College of Medicine, Houston, USA, E-mail: klepper@gmail.com

Copyright: © 2023 Klepper M. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Received: 27 June, 2023, Manuscript No. Jio-23-115238; Editor assigned: 29 June, 2023, Pre QC No. P-115238; Reviewed: 12 July, 2023, QC No. Q-115238; Revised: 19 July, 2023, Manuscript No. R-115238; Published: 26 July, 2023, DOI: 10.37421/2329-6771.2023.12.440

axis in these patients. Twenty CML patients who received imatinib for duration longer than six months and experienced growth retardation were included.

The growth hormone stimulation assays were used to evaluate the GHRH-GH-IGF1 axis. The IGF-1 generation test was used to assess GH insensitivity. 15.2 years was the mean age at inclusion. Imagine therapy lasted an average of 5.7 years. Since Imatinib treatment began, the average height SDS has decreased by about 0.95 (p = 0.008). IGF-1 SDS was negative two in each case. 71.4% of patients who were stimulated with GHRH-Arginine showed an inadequate GH response. With glucagon stimulation, all patients demonstrated stimulable, albeit delayed, GH responses. GH insensitivity affected 20% of the patients. Four individuals received hGH treatment for a mean of 5.75 months; they saw improvements in their IGF-1 levels and growth rates of 0.21 to 0.86 cm per month. Imatinib causes a neurosecretory deficit in GH secretion that is acquired. Improvements in growth rate and normalisation of IGF-1 are brought about by growth hormone therapy [2,3].

In adults with Chronic Myeloid Leukaemia (CML) treated with tyrosine kinase inhibitors, evidence-based recommendations have been produced, but it has been difficult to create similar recommendations in paediatrics due to the rarity of this leukaemia in children and adolescents. However, the choice of a TKI continues to be based on clinical experience in adults because there aren't enough data on efficacy and safety unique to paediatric patients. Here, we offer four case studies that show typical yet difficult problems with paediatric CML treatment (suboptimal response, poor treatment adherence, growth retardation, and presentation in advanced phases). Additional key challenges that call for future clinical research through international collaboration are the lack of expertise with very young children, the transition of teenagers to adult medicine, and the aim of obtaining treatment-free remission for this uncommon malignancy.

Between 2 and 3% of leukaemias in children under the age of 15 and 9% in adolescents between the ages of 15 and 19 are CML. There are several distinctions between the diagnosis and treatment of CML in children, adolescents, and young adults compared to adults. This review describes the underlying disease's diagnosis and treatment, as well as potential difficulties. Only 2–3% of all leukaemia in juvenile children are Chronic Myeloid Leukaemia (CML). The existence of the genetic markers for CML, the Philadelphia chromosome and the BCR-ABL fusion, is essential for targeted molecular therapy with tyrosine kinase inhibitors, which has taken the place of Hematopoietic Stem Cell Transplantation (HSCT) as the accepted first-line therapy. The disease affects a small percentage of children, and despite clinical and molecular similarities to CML in adults, a separate therapy is required because affected children have lengthy life expectancies and unique developmental traits. Growth retardation is brought on by imagine in kids with Chronic Myeloid Leukaemia (CML) [4,5].

Conclusion

Precision medicine extends to the overall care of the patient, addressing not only treatment but also supportive care, survivorship, and the patient's unique needs and preferences. Precision medicine has fueled the development of clinical trials that target specific patient subpopulations with particular genetic mutations or biomarkers. These trials aim to test novel therapies in the most relevant patient groups. The likelihood of survival for those with Chronic Myeloid Leukaemia (CML) in children, adolescents, and young adults has significantly increased in recent years. Tyrosine Kinase Inhibitors (TKI), specific medications created for CML patients, were first made available in England in 2001. According on the year of diagnosis, we quantify the trends in the "cure" proportion here.

Acknowledgement

None.

Conflict of Interest

None.

References

- Mehraj, Umar, Rais A. Ganai, Muzafar A. Macha and Abid Hamid, et al. "The tumor microenvironment as driver of stemness and therapeutic resistance in breast cancer: New challenges and therapeutic opportunities." *Cell Oncol* (2021): 1-21.
- Ebrahimi, Safieh and Seyed Isaac Hashemy. "MicroRNA-mediated redox regulation modulates therapy resistance in cancer cells: Clinical perspectives." *Cell Oncol* 42 (2019): 131-141.

- Sun, Xuan, Robert CG Martin, Qianqian Zheng and Russell Farmer, et al. "Druginduced expression of EpCAM contributes to therapy resistance in esophageal adenocarcinoma." *Cell Oncol* 41 (2018): 651-662.
- Lei, Jonathan T., Xuxu Gou and Matthew J. Ellis. "ESR1 fusions drive endocrine therapy resistance and metastasis in breast cancer." MCO 5 (2018): e1526005.
- Roy, Souvick, Madhabananda Kar, Shomereeta Roy and Arka Saha, et al. "Role of β-catenin in cisplatin resistance, relapse and prognosis of head and neck squamous cell carcinoma." *Cell Oncol* 41 (2018): 185-200.

How to cite this article: Klepper, Maria. "Precision Medicine in Oncology: From Genomics to Target." J Integr Oncol 12 (2023): 440.