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Editorial

Precision Therapy in Oncology

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Cancer is second most common cause of death in the world. As the technology is advancing, the number of newly diagnosed cancer cases are on raise. Especially, in advanced stage. A decade back, the treatment for advanced cancers was primarily by chemotherapeutic agents. But now, targeted therapies are becoming increasingly popular.

Cancer chemotherapeutic agents act by destroying rapidly growing cells. Few of them cell cycle specific and few are cell cycle non specific. this results in killing of cancer and non cancerous cells resulting in severe toxicity.

Oncogenesis can be simplified as an imbalance between tumor stimulating growth factors and tumor suppressor factors.

Gene "driver" mutation is essential for tumor growth along with several factors like protein kinases, Growth factors (FDF), Hypoxia inducible factor (HIF), PDGF, TGF and VEGF to name few. Similarly tumor is suppressed by P53, which is tumor suppressor gene and T cell mediated cell death. The immune system detects and destroys abnormal cells and prevents the growth of many cancers. cancer cells have ways to avoid destruction by the immune system such genetic changes that make them less visible to the immune system, proteins on their surface that turn off immune cells. Dysregulation in either balancing factors results in tumor growth and spread.

The diversity of cancer types in combination with the understanding about driver mutations made oncology, a pioneer of precision medicine. As there are advancements in understanding cancer triggering pathways, role of molecular markers has become very important.

"One size fits all" approaches of cytotoxic chemotherapy is being rapidly replaced by more precise therapies using drugs and treatments based on a molecular diagnosis and tailored to individual Patient.

On one hand, there are approaches to disrupt pathways essential for cancer growth and survival and on the other hand attempts are being made to push the patient's immune system for a better response against malignant tumor cells.

Targeted therapies aim at the molecular receptors and directly effecting pathways involved in oncogenesis. Targeted therapy can only be expected when the mutation is confirmed. Protein kinases regulate key cellular processes, such as proliferation, survival and migration. Genes encoding for a number of protein kinases are mutated in one or several cancers. Few such mutations are seen in BRAF, EGFR KIT and PDGFRA.

several targeting molecules have been developed such as EGFR inhibitors (Geftinib, Erlotinib),ALK inhibitor (crizotinib), BRAF Inhibitor (Vemurafenib),VEGFR inhibiotrs (Bevacizumab), Drugs targeting BCR-ABL1 fusion protein (Imatinib), RAF inhibitor (sorafinib), Olaparib (PARP inhibitor)

An alternative approach is to affect the regulation of tumor suppressor gene and immunotherapy. Tumor suppressor genes can be regulated via chromatin modifiers. These reactivate the transcription of tumor suppressor genes, such as CDKN1A, p53 which can induce apoptosis, cell cycle arrest. Immunotherapy helps the immune system to better act against cancer. Several types

of immunotherapy agents have been developed such as immune check point inhibitors (PDL-1 inhibitors), Monoclonal antibodies and T cell transfer therapies and vaccines.

Many times a combination of chemotherapy and targeted therapy is used or single targeted therapy can be used depending on the cancer stage and patient condition. In conclusion, precision therapy using targeted and immunotherapy has rapidly changed the landscape of cancer therapy and the quality of patient life.