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Targeting Cell Signalling Pathways: A Short Review on Tyrosine Kinase Inhibitors

Tasleem[1*], Nasreen Sultana[2,3], Sushma Desai[4]
[1*]Associate Professor, Department of Pharmaceutical Chemistry
[2]Associate Professor, Department of Pharmaceutics
Dr. V. R. K institute of Pharmaceutical Sciences, Humayun Nagar, Mehdipatnam, Hyderabad, Telangana, India[1,2]
YNCliIT Research Institute, Mehdipatnam, Hyderabad, Telangana, India
[4]Research Scholar, Department of Pharmaceutical Sciences
Gitam School of Pharmacy, Rudraram, Telangana, Hyderabad, Telangana, India
Corresponding Author: Tasleem
Email.ID: fathima.tasleem@gmail.com

Abstract: *This short review discusses about tyrosine kinase inhibitors (TyKI) as a potential targeting cell signalling pathway blocker playing active role as anticancer drugs. We learn about various TyKI drugs developed for oncological conditions are BCR-ABL (B cell Receptor fusion Abelson) Inhibitors, EGFR(Epidermal Growth Factor Receptor) Inhibitors, VEGFR (Vascular Epidermal Growth Factor Receptor) Inhibitor and BTK (Bruton's Tyrosine Kinase) Inhibitors used successfully in chemotherapy. Like any anticancer drug class TyKI also suffer from therapeutic limitations due to adverse effects. we learn the benefits of TyKI over traditional drugs and its key clinical parameters required to start this therapy for maximizing its outcome.*

Keywords: Anticancer drugs, Tyrosine kinase inhibitors, cell signalling pathway, adverse effects.

I. INTRODUCTION

Tony Hunter is a pioneer in development of Tyrosine kinase inhibitors (TyKI) as early in 1979. It has become a breakthrough in cancer treatment switching from cytotoxic chemotherapy to targeted cell therapy in controlling the cancerous growth enhancing survival rate to almost 90% in patients has gained attention worldwide. With over 90 drugs are marketed under this class. Most of these drugs belong to BCS class II poorly aqueous soluble and highly permeable nature where an economical method development procedure is in need.

Mechanism of action of Tyrosine kinase Inhibitors work by phosphorylating the kinase receptors causing the alteration and disruption in cell growth cycle of cancerous cells causing cell growth cessation, migration, differentiation and finally cell apoptosis.

Based on the binding sites of receptors TyKI are classified as Type I-V as follows

TYPE I- ATP competitive binding observed.

TYPE II- DFG motif cause inactivation of kinases in ATP binding site.

TYPE III-bind exclusively to allosteric sites adjacent to ATP sites.

TYPE IV-bind far away from allosteric ATP sites.

TYPE V-these exhibit multi-site binding on receptor kinases.

However TyKI identified with these Common adverse effects listed as follows

Gastrointestinal region includes diarrhea, alteration of sense of taste, abdominal pain, constipation, hepatotoxicity, stomatitis and nausea.

Cardiovascular region includes hypertension, hypotension, congestive heart failure, Thrombosis, cardiac ischemia, Myocardial Infarction, strokes, QT region interval prolongation observed in ECG.

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Dermatological region includesSteven Johnson's syndrome, Toxic epidermal necrosis, drug rash observed with eosinophilia, cheilitis, facial edema, paronchia, pruritis, photosensitivity, xerosis,hair color changes, alopecia, maculopapular rashes and acute generalized exanthematous pustulosis.

Neurological region includes cognitive impairment, peripheral neuropathy, headaches and dizziness.

Musculoskeletalrelated are myalgia and arthralgia.

Hematologicalrelated are anemia, thrombocytopenia, neutropenia and bruising.

Ophthalmic related are keratitis, retinal pigment epithelial detachment, central serous retinopathy.

Renal related are proteinuria and kidney dysfunction.

Endocrine related are hypokalemia, thyroid dysfunction and lacrimation.

Respiratory related are epistaxis, rhinorrhea, dyspnea, interstitial pulmonary disease,Upper respiratory tract Infection, interstitial pulmonary disease.

General includes fatigue, fever, chills, Head-ache.

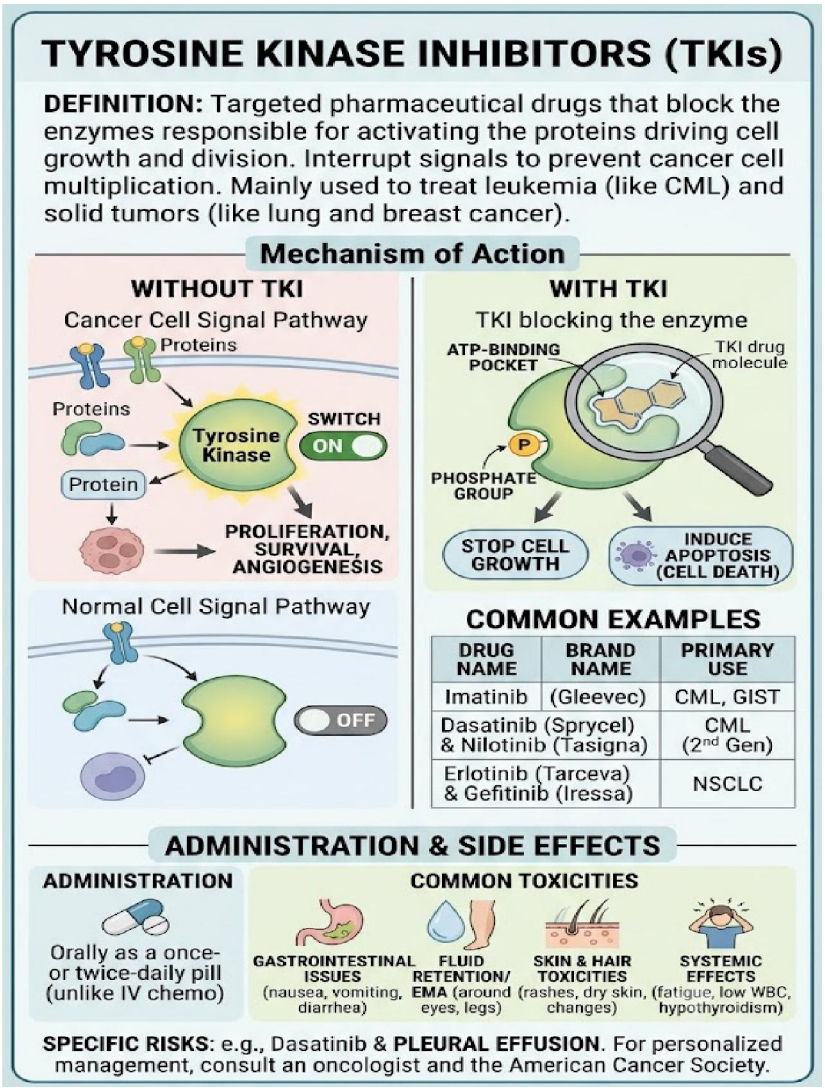

Fig 1: Diagrammatic Explanation of Tyrosine Kinase Inhibitors as Anticancer Agents.

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Table 1 target receptor binding by tyrosine kinase inhibitor and its application

Receptor	Tyrosine Kinase Inhibitors	Category
B-Cell Receptor-Abelson Inhibitors	Asciminib, Dasatinib, Imatinib, Nilotinib, Bosutinib, Ponatinib,	Acute Lymphoblastic Leukemia. Chronic Myeloid Leukemia.
Epidermal Growth Factor/ Human Epidermal Receptor Factor 2 Inhibitors	Erlotinib, Gefitinib, Afatinib, Osimertinib, Lapatinib, Neratinib, Tucatinib, Dacomitinib	Non-Small Cell Lung Cancer, Breast Cancer
Vascular Epidermal Growth Factor Receptor Inhibitors	Sunitinib, Sorafenib, Pazopanib, Axitinib, Cabozantinib, Lenvatinib, Regorafenib, Tivozanib	Renal Cell Carcinoma, Liver, Thyroid, GIST
Anaplastic lymphoma Kinase Receptor Inhibitor -Proto oncogene Inhibitors	Crizotinib, Ceritinib, Alectinib, Brigatinib, Lorlatinib, Repotrectinib	ALK-positive or ROS1-positive NSCLC
Bruton ‘s Tyrosine Kinase Inhibitors	Ibrutinib, Acalabrutinib, Zanubrutinib, Pirtobrutinib	Chronic Lymphocytic Leukemia (CLL), Mantle Cell Lymphoma
Janus Kinase Inhibitors	Ruxolitinib, Tofacitinib, Baricitinib, Upadacitinib, Fedratinib, Pacritinib	Myelofibrosis, Rheumatoid Arthritis, Dermatologic conditions
FMS like tyrosine kinase inhibitor - Proto oncogene Receptor Kinase Receptor Inhibitors	Midostaurin, Gilteritinib, Quizartinib, Avapritinib, Ripretinib	Acute Myeloid Leukemia, Gastrointestinal stromal tumor, Mastocytosis
Rearranged during Transfection Receptor/Mesenchymal epithelial Transition receptor / Fibroblast Growth Factor Receptor Inhibitors	Selpercatinib, Pralsetinib, Capmatinib, Tepotinib, Erdafitinib, Pemigatinib, Futibatinib	Rearranged during Transfection / Mesenchymal Epithelial Transition receptor inhibition driven Non-small cell lung cancer, Cholangiocarcinoma, Urothelial Cancer
Neurotropic Tyrosine Receptor Kinase Inhibitors	Larotrectinib, Entrectinib	NTRK fusion-positive solid tumors

II. CONCLUSION

With this brief information on Tyrosine kinase inhibitors class of Anticancer drugs. The drugs adverse effects can be more manageable in future by overcoming its biopharmaceutical challenges in developing pharmaceutical formulations other than oral formulations in a safe and economical way.

III. DECLARATION

AI generated image used for understanding the Tyrosine Kinase inhibitors using AI gemini.

REFERENCES

[1]. Byrd, J. C., Brown, J. R., O'Brien, S., et al. (2014). Ibrutinib versus ofatumumab in previously treated chronic lymphoid leukemia. *New England Journal of Medicine*, *371*(3), 213–223. https://doi.org/10.1056/nejmoa1400376

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[2]. Druker, B. J., Talpaz, M., Resta, D. J., et al. (2001). Efficacy and safety of a specific inhibitor of the BCR-ABL tyrosine kinase in chronic myeloid leukemia. *New England Journal of Medicine*, *344*(14), 1031–1037. https://doi.org/10.1056/nejm200104053441401

[3]. Druker, B. J., Guilhot, F., O'Brien, S. G., et al. (2006). Five-year follow-up of patients receiving imatinib for chronic myeloid leukemia. *New England Journal of Medicine*, *355*(23), 2408–2417. https://doi.org/10.1056/nejmoa062867

[4]. Estfan B, Byrne M, Kim R. Sorafenib in advanced hepatocellular carcinoma: hypertension as a potential surrogate marker for efficacy. Am J Clin Oncol. 2013 Aug;36(4):319-24.

[5]. Klümpen HJ, Samer CF, Mathijssen RH, Schellens JH, Gurney H. Moving towards dose individualization of tyrosine kinase inhibitors. Cancer Treat Rev. 2011 Jun;37(4):251-60.

[6]. Mok, T. S., Wu, Y.-L., Thongprasert, S., et al. (2009). Gefitinib or carboplatin–paclitaxel in pulmonary adenocarcinoma. *New England Journal of Medicine*, *361*(10), 947–957. https://doi.org/10.1056/nejmoa0810699

[7]. Motzer, R. J., Hutson, T. E., Tomczak, P., et al. (2007). Sunitinib versus interferon alfa in metastatic renal-cell carcinoma. *New England Journal of Medicine*, *356*(2), 115–124. https://doi.org/10.1056/nejmoa065044

[8]. Roskoski R. Properties of FDA-approved small molecule protein kinase inhibitors. Pharmacol Res. 2019 Jun;144:19-50.

[9]. Soria, J.-C., Ohe, Y., Vansteenkiste, J., et al. (2018). Osimertinib in untreated *EGFR*-mutated advanced non–small-cell lung cancer. *New England Journal of Medicine*, *378*(2), 113–125. https://doi.org/10.1056/nejmoa1713137

[10]. Shaw AT, Engelman JA. Ceritinib in ALK-rearranged non-small-cell lung cancer. N Engl J Med. 2014 Jun 26;370(26):2537-9.

[11]. Terada T, Noda S, Inui K. Management of dose variability and side effects for individualized cancer pharmacotherapy with tyrosine kinase inhibitors. Pharmacol Ther. 2015 Aug;152:125-34.

[12]. Varone F, Sgalla G, Iovene B, Bruni T, Richeldi L. Nintedanib for the treatment of idiopathic pulmonary fibrosis. Expert Opin Pharmacother. 2018 Feb;19(2):167-175.

[13]. Zeiser R, Andrlová H, Meiss F. Trametinib (GSK1120212). Recent Results Cancer Res. 2018;211:91-100.