ISSN: 2593-9947
Journal of Clinical and Medical Sciences

OPEN ACCESS Freely available online

Perspective

Association of Bruton's Tyrosine Kinase Inhibitor with Tumour Lysis Syndrome

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DESCRIPTION

BTKis (Bruton's Tyrosine Kinase inhibitors) are used to treat B-cell leukemia as well as lymphomas such as chronic lymphocytic leukemia, mantle cell lymphoma, and Waldenström macroglobulinemia. An adverse effect related with this class is Tumor Lysis Syndrome (TLS).

BTKis is an important mediator in the B-cell antigen receptor and cytokine receptor pathways. This route is required for B-cell trafficking, chemotaxis, and adhesion and is activated by BTK activity *via* B-cell surface receptor signaling. BTK inhibitors acalabrutinib, ibrutinib, and zanubrutinib are approved, whereas evobrutinib, tolebrutinib, fenebrutinib, pirtobrutinib, spebrutinib, and tirabrutinib are still in clinical trials. They block BTK from executing its activity by building a covalent link with a cysteine residue in the enzyme's active site.

Tumour Lysis Syndrome (TLS) is a potentially fatal cancer chemotherapy-related oncologic emergency in which massive malignant cells die and rapidly degrade, releasing massive amounts of potassium, nucleic acids, cytokines, and phosphate into the systemic circulation either spontaneously or in response to therapy. The fast release of these intracellular components causes hyperkalaemia, hyperphosphatemia, hyperuricemia, and subsequent hypocalcaemia. These metabolic anomalies can cause significant morbidity, putting patients at risk for serious clinical outcomes such as cardiac arrhythmias, acute renal injury, fluid overload, pulmonary oedema, convulsions, and even death.

TLS is also a serious consequence of cancers such as acute lymphocytic leukemia, Burkitt's Leukemia (BL), advanced BL, Acute Myeloid Leukemia (AML), and advanced Diffuse Large B-Cell Lymphoma (DLBCL).

Only a few studies have addressed the safety profile of BTKis, with the bulk of them focused on cardiovascular related side events, pericarditis, skin cancer, and progressive multifocal leukoencephalopathy. Ibrutinib, one of the BTKis, has the potential to cause TLS. It has not been documented for acalabrutinib or zanubrutinib, though. Through disproportionality analysis in the Food and Drug Administration Adverse Event Reporting System (FAERS) database, this work attempts to investigate the possible signal between BTKis and TLS adverse events.

The occurrence of TLS, a haematooncologic emergency caused by the rapid breakdown of malignant cells usually triggered by this cytotoxic therapy, is not thoroughly documented. Hyperphosphatemia, hyperuricemia, hyperkalaemia, metabolic acidosis, and hypocalcaemia are all symptoms of TLS. It is caused by cancer cells rapidly decomposing and releasing intracellular components into the bloodstream such as nucleic acids, cytokines, potassium, and phosphate. However, it may be spontaneous in rapidly growing high-grade hematologic malignancies such as BL, AML, and anaplastic large T-cell or DLBCL. The type of cancer, the form and intensity of anticancer therapy, and the presence of pre-existing illnesses such as renal insufficiency all enhance the risk of TLS. As a result, TLS can cause abrupt renal failure, cardiac arrhythmias, and other complications. Our research focuses primarily on the potential signal of BTKis (ibrutinib, acalabrutinib, zanubrutinib) and TLS.

This method is used by regulatory bodies and pharmaceutical corporations to discover adverse events that newly marketed medications may cause. Though this technique may uncover a possible drug-AE link, it does not demonstrate a causal association, which can only be established through randomised controlled studies that aid in the discovery of putative AE mechanisms. BTKis have emerged as a promising treatment option for hematological cancers.

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Received: 03-Jun-2022, Manuscript No. JCMS-22-17607; **Editor assigned:** 07-Jun-2022, Pre QC No. JCMS-22-17607 (PQ); **Reviewed:** 20-Jun-2022, QC No JCMS-22-17607; **Revised:** 27-Jun-2022, Manuscript No. JCMS-22-17607 (R); **Published:** 04-Jul-2022, DOI: 10.35248/2593-9947.22.6.193.

Citation: Min P, Ghor P (2022) Association of Bruton's Tyrosine Kinase Inhibitor with Tumour Lysis Syndrome. J Clin Med. 6:193.