

Ibrutinib: A New Targeted Therapy for Hematologic Cancers

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Background: Hematologic cancers can occur from the overactivity of Bruton's tyrosine kinase, a proto-oncogene in blood cell maturation. Ibrutinib, a new oral targeted therapy drug, is the first agent that binds to the Bruton's tyrosine kinases and inhibits overgrowth of B cells. In blocking this overgrowth, ibrutinib has been shown to achieve lengthy remissions for patients with mantle cell lymphoma (MCL) and chronic lymphocytic leukemia (CLL). Remissions are highly valued in these cancers; cure is rare in MCL, and CLL is incurable.

Objectives: This article reviews ibrutinib, its risks and benefits, and the role that oncology nurses play in educating patients and promoting drug adherence.

Methods: A comprehensive review of the literature was conducted using key words such as *ibrutinib*, *mantle cell lymphoma*, *chronic lymphocytic leukemia*, *tyrosine kinase inhibitor*, and *oral chemotherapy*.

Findings: Ibrutinib has been shown to be well tolerated, with manageable, low-grade toxicities compared to traditional cytotoxic agents. For all patients with a hematologic cancer, but particularly for the large proportion of older adults affected by hematologic malignancies, ibrutinib provides a new treatment option with a low toxicity profile.

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Unlike traditional cytotoxic chemotherapy, which affects tumor cells and healthy cells, the targeted therapy agent ibrutinib focuses on tumor cells (Wujcik, 2011). Ibrutinib is intended for select hematologic cancers and is a small-molecule Bruton's tyrosine kinase inhibitor (TKI) (Cameron & Sanford, 2014). Multiple TKIs are available, but ibrutinib is the first that is specific to Bruton's tyrosine kinase (Cameron & Sanford, 2014). Overactive Bruton's tyrosine kinases are proto-oncogenes that signal B cells to proliferate, differentiate, and survive, resulting in malignancy (Bhatt, Alejandro, Michael, & Ganetsky, 2014). B cells traditionally originate from stem cells in the bone marrow and mature into adaptive components in the humoral immune response (Manson & Porter, 2011). However, when the overactive Bruton's tyrosine kinases signal B cells' unregulated growth, the overabundant mutated B cells can lead to cancers such as mantle cell lymphoma (MCL), chronic lymphocytic leukemia (CLL), diffuse large B-cell lymphoma (DLBCL), multiple myeloma (MM), and Waldenström's macroglobulinemia (WM) (Bhatt et al., 2014; Treon et al., 2015).

Ibrutinib prevents the faulty Bruton's tyrosine kinases from being able to signal this tumor cell growth and division.

Indications

Ibrutinib is indicated and approved by the U.S. Food and Drug Administration (FDA) for MCL, CLL, and WM. It received accelerated FDA approval in November 2013 for patients with MCL who have received at least one prior therapy (FDA, 2013b). Ibrutinib received accelerated approval in February 2014 for patients with CLL who have received at least one prior therapy (FDA, 2014a). In July 2014, the FDA granted full approval for patients with CLL who have received at least one prior therapy and for patients with CLL with a chromosome 17p deletion who may or may not have received prior therapy (FDA, 2014b). Chromosome 17p deletion is associated with a poor prognosis in CLL (University of Texas MD Anderson Cancer Center, 2013). In January 2015, the FDA approved ibrutinib for the treatment of patients with WM (FDA, 2015).