Changes in Cancer Treatment

Mabs, Mibs, Mids, Nabs, and Nibs

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KEYWORDS

• Monoclonal antibody • Nanoparticle albumin-bound drug • Tyrosine kinase inhibitor
• Immunomodulatory drug • Proteasome inhibitor • Cancer therapeutics
• Targeted therapy • Nursing care

KEY POINTS

- Stay abreast on latest cancer treatments; cancer treatments have evolved from genocidal to specific cellular, molecular, and genetic targeting approaches (targeted therapies) to kill cancer cells.
- Manage the side effects of cancer treatments; this is essential to adherence and a successful completion of planned targeted therapy.
- Educate patients and their caregivers on treatment-related information to empower them in becoming active participants of the cancer journey.

Cancer therapeutics has changed at a rapid pace over the past two decades. Cancer treatment changes are undoubtedly driven by the recent advances in the understanding of cancer pathobiology, leading to molecular classifications of various cancer types instead of organ-based cancer classifications.[1] Scientific advances and discoveries have led to an improved therapeutic approach and eventually forged a new pathway to cure cancer, leaving behind the primitive genocidal and toxic approach of cancer treatment. Moreover, improved understanding of the pathobiology of cancer at the molecular and genetic levels catalyzed the rapid changes in cancer therapeutics, ushering in the era of targeted therapies. Since the Food and Drug Administration (FDA) approval of the first monoclonal antibody, rituxi*mab* (Mab) for non-Hodgkin lymphoma in 1997,[2] first proteasome inhibitor (PI), bortezo*mib* (Mib) for myeloma in 2003,[3–5] first immunomodulatory drug, lenalido*mid*e (Mid) for myeloma in 2006,[6] first nanoparticle albumin-bound drug, nab-paclitaxel (Nabs) for breast cancer in 2005,[7,8] and first tyrosine kinase inhibitor (TKI), imati*nib* (Nib) for chronic myelogenous leukemia (CML) in 2001,[9] cancer therapeutics has been growing in an unprecedented

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fashion, targeting specific gene mutation, protein dysfunction and dysregulation, intracellular signaling pathways, and immune modulation to name a few.

In myeloma (cancer of plasma cells) alone, four new drugs with different classifications and mechanisms of action have been approved by the FDA in 2015. This is an unprecedented, record-breaking year for drug approval for one type of cancer. These new drug approvals in 2015 included a Mib (ixazomib[10]), two Mabs (daratumumab[11] and elotuzumab[12]), and a histone deacetylase inhibitor (panobinostat).[13] In 2012, a second-generation Mib (carfilzomib)[14] and a third-generation Mid (pomalidomide[15]) were also granted FDA approval as treatment of myeloma. In patients diagnosed with myeloma, a plethora of treatment options provides hope and opportunities, but they come with clinical challenges for oncology nurses and all other oncology clinicians in terms of meeting the educational needs and treatment preferences of patients diagnosed with cancer, especially when treatment options are equivocal.

Patients newly diagnosed with cancer require immediate treatment in certain circumstances because of organ damage and these patients clearly have information needs related to disease, treatment, and side effects, which must be provided to patients and families to empower them throughout their cancer journey.[16] This article addresses the need of novice and experienced oncology nurses alike for treatment-related information and evidence-based nursing care of patients diagnosed with cancer throughout the cancer treatment continuum, emphasizing novel and breakthrough targeted cancer therapies.

MONOCLONAL ANTIBODIES

In 1986 the first commercially available Mab, orthoclone OKT3 (muromonab-CD3), was initially approved by the FDA for use in preventing kidney transplant rejection.[17] Twelve years later, the FDA approved rituximab as the first Mab for the treatment of B-cell lymphoma. Rituximab is a genetically engineered chimeric (murine-human) Mab directed against CD20 antigen found on the surface of normal and malignant B cells.[18,19] Rituximab attacks cancer cells after binding with CD20 B cells by a variety of mechanisms including cell cycle arrest; direct induction of apoptosis (program cell death); and sensitization to cytotoxic (cell killing) drugs, complement-dependent cytotoxicity, and antibody-dependent cellular cytotoxicity.[20] **Fig. 1** illustrates the mechanism of actions of rituximab. Since its first approval in 1997, rituximab has been combined with various other chemotherapeutic or biologic agents in the treatment

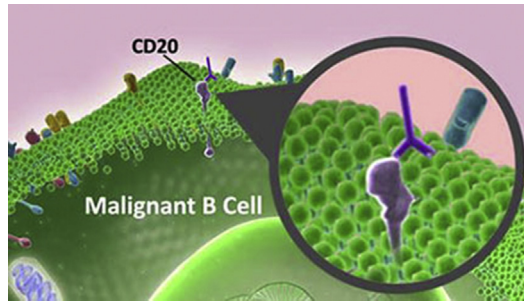

Fig. 1. Rituximab's mechanism of action: binds to CD20 receptor of B cells and once bound, the drug activates the immune system to attack the malignant B cells. (*From* National Cancer Institute. Using the immune system in the fight against cancer: discovery of rituximab. Available at: https://www.cancer.gov/research/progress/discovery/blood-cancer. Accessed March 3, 2016.)