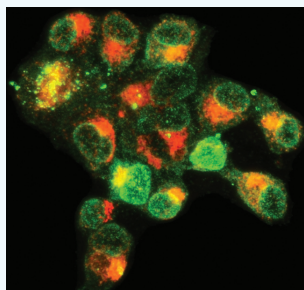


■ Article

Managing Side Effects of the Novel Taxane Cabazitaxel in Castrate-Resistant Prostate Cancer

Susan Doyle-Lindrud, DNP, AOCNP®



© iStockphoto.com/Dlumen

Cabazitaxel, a novel taxane, was approved in June 2010 by the U.S. Food and Drug Administration for treatment of metastatic castrate-resistant prostate cancer (mCRPC) in men previously treated with docetaxel. In TROPIC (N = 755), an open-label, randomized, phase III trial, cabazitaxel (plus prednisone) was associated with improvement in median overall survival compared with mitoxantrone plus prednisone (15.1 versus 12.7 months, $p < 0.0001$) in patients with mCRPC who had progressed following docetaxel-based regimens. That corresponds to a 30% relative reduction in risk of death compared with the mitoxantrone regimen. In addition, significant benefit existed in median progression-free survival with cabazitaxel versus the mitoxantrone regimen (2.8 versus 1.4 months, $p < 0.0001$). Most common adverse events (AEs)

associated with cabazitaxel were hematologic; the rates (all grade) of neutropenia, leukopenia, and anemia were greater than 90%. Diarrhea, fatigue, asthenia, and back pain were the most common grade 3 or higher nonhematologic AEs. Because expected AEs from cabazitaxel therapy can delay or even interrupt treatment, oncology nurses need to be aware of those risks and their management. This article reviews the vital role of nurses in identifying patients at high risk for AEs associated with cabazitaxel therapy and reviews strategies for prevention and management of symptoms.

Susan Doyle-Lindrud, DNP, AOCNP®, is the director of the oncology program in the School of Nursing at Columbia University in New York, NY. Editorial support was provided by Susan DePetris, PhD, of Phase Five Communications Inc., supported by sanofi-aventis U.S. LLC, a Sanofi company. The author was fully responsible for all content and editorial decisions and received no financial support or other form of compensation related to the development of the article. The content of this article has been reviewed by independent peer reviewers to ensure that it is balanced, objective, and free from commercial bias. No financial relationships relevant to the content of this article have been disclosed by the independent peer reviewers or editorial staff. Mention of specific products and opinions related to those products do not indicate or imply endorsement by the *Clinical Journal of Oncology Nursing* or the Oncology Nursing Society. Doyle-Lindrud can be reached at smd9@columbia.edu, with copy to editor at CJONEditor@ons.org. (First submission July 2011. Revision submitted August 2011. Accepted for publication August 28, 2011.)

Digital Object Identifier:10.1188/12.CJON.286-291

Prostate cancer is the most common type of nonskin cancer in American men (American Cancer Society, 2012; National Cancer Institute [NCI], 2011c). Based on the NCI (2011c) Surveillance, Epidemiology and End Result data, about one in six men will be diagnosed with prostate cancer in their lifetime (median age at diagnosis = 67 years) and 1 in 36 will die of the disease. An estimated 241,740 new cases of prostate cancer will be diagnosed and about 28,170 prostate cancer-related deaths will occur in the United States in 2012 (American Cancer Society, 2012).

Because of the widespread use of the prostate-specific antigen, a protein produced by the prostate gland, as a marker to detect prostate cancer, an increasing proportion of prostate cancers are localized at the time of diagnosis (Lu-Yao & Greenberg, 1994). From 2000–2007, 80% of men diagnosed with prostate cancer were diagnosed with localized disease, 11% with regional disease, and 4% with distant metastases (NCI, 2011a). For most men diagnosed with localized disease, potentially curative treatment options exist (Walczak & Carducci, 2007).

However, men who have advanced or recurrent disease still do not have such options.

Men with advanced prostate cancer often are treated with hormonal therapy, also known as androgen deprivation therapy (ADT), which consists of a luteinizing hormone-releasing hormone agonist or antagonist, to lower the production of testosterone to castrate levels. That drop in testosterone usually slows or stops the growth of prostate cancer for a period of time (Sharifi, Gulley, & Dahut, 2005; Walsh, DeWeese, & Eisenberger, 2001). Most men initially respond to ADT, but eventually the disease becomes resistant, known as castrate-resistant prostate cancer (CRPC) (Walsh et al., 2001). Standard treatment options for metastatic (m)CRPC include chemotherapy, second-line hormonal therapy, or a vaccine therapy, sipuleucel-T (National Comprehensive Cancer Network [NCCN], 2012e). Prostate cancer was considered a chemoresistant disease until the mid-1990s, at which time mitoxantrone with prednisone was shown to have a palliative role for men with mCRPC in improving quality of life (Tannock et al., 1996). However, no difference in