Hyperglycemic-Inducing Neoadjuvant Agents Used in Treatment of Solid Tumors: A Review of the Literature

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Purpose/Objectives: To review the literature regarding the development of hyperglycemia associated with neoadjuvant agents used in the treatment of solid tumor cancers.

Data Sources: Research articles were obtained from PubMed, CINAHL®, and Cochrane Reviews. The following search terms were used alone and in combination: diabetes, glycemic control, chemotherapy, androgen deprivation therapy, interferon-alpha, immunosuppressants, cancer, neoplasms, and hyperglycemia.

Data Synthesis: Twenty-two studies were identified reporting the development of hyperglycemic events in patients who received a variety of chemotherapeutic agents.

Conclusions: Findings suggest patients are at risk for the development of hyperglycemia from certain chemotherapeutic agents. Docetaxel, everolimus, and temsirolimus alone or in combination with other agents can promote hyperglycemia. Androgen-deprivation therapy commonly used in prostate cancer, increases the risk for the development of hyperglycemia and diabetes.

Implications for Nursing: Oncology nurses play an important role in the identification and treatment of hyperglycemia in patients receiving chemotherapy. Future research is needed that focuses on the association between glycemic control and adverse outcomes in patients with a solid tumor cancer who are at risk for treatment-induced hyperglycemia.

Key Words: neoplasm; chemotherapy; hyperglycemia

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Patients with a solid tumor cancer are at risk for hyperglycemia (blood glucose > 126 mg/dL) during treatments. Hyperglycemia can contribute to the risk for adverse outcomes such as infections and nonmalignancy-related mortality (Ali et al., 2007; Fuji et al., 2007; Hammer et al., 2009; Storey & Von Ah, 2012). In addition, hyperglycemia may increase the risk for development of clinical toxicities, grade 4 neutropenia, neutropenic fever, sepsis, and neuropathy (Brunello, Kapoor, & Extermann, 2011). Hyperglycemia during cancer treatment is one of the clinical toxicities that can cause chemotherapy dose delays or reductions (Brunello et al., 2001; Richardson & Pollack, 2005). Hyperglycemia may decrease the response to chemotherapeutic agents (Zeng et al., 2010). Understanding the contributors to hyperglycemia in patients with a solid tumor cancer is essential to create interventions for improved outcomes.

In patients with a solid tumor cancer, many factors can contribute to hyperglycemia, including nutritional imbalances (Jenkins et al., 2002; Martin-Salces, de Paz, Canales, Mesejo, & Hernandez-Navarro, 2008), physical inactivity (Katz, 2007; Moien-Afshari et al., 2008), older age (Stookey, Pieper, & Cohen, 2004), high body mass index (Roumen, Blaak, & Corpeleijn, 2009), high stress levels (Godbout & Glaser, 2006), and infections (Turina, Christ-Crain, & Polk, 2006). These factors are also associated with the development of type 2 diabetes (T2D). Having preexisting T2D (the hallmark of which is hyperglycemia) is one factor that increases the risk for hyperglycemic events during cancer treatment (Fuji et al., 2007). About 18% of all individuals with cancer have preexisting diabetes at the time of diagnosis (Barone et al., 2008).

Patients do not have to have preexisting diabetes to encounter glycemic problems and related adverse outcomes while undergoing treatment for cancer. The current prevalence of hyperglycemia among patients with a solid tumor cancer with and without preexisting diabetes is currently unknown. One study that investigated allogeneic hematopoietic cell transplantation recipients found an overall median blood glucose of 133 mg/dl (hyperglycemic) among 1,175 patients, and blood glucose at a level greater than 200 mg/dl was related to an almost twofold increased risk for mortality compared to a level of 101–150 mg/dl ($p = 0.0009$) (Hammer et al., 2009). This population hinted at the potentially larger glycemic issue for all patients with