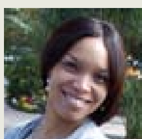
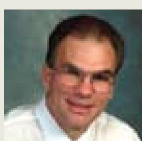
ONCOLOGY NURSE ADVISOR FORUM

Our Consultants

Ann J. Brady, RN, BSN, *symptom management care coordinator at the Cancer Center, Huntington Hospital, Pasadena, California.*

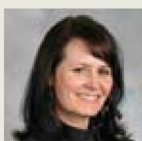


Jia R. Conway, DNP, FNP-BC, NP-C, *oncology nurse practitioner at Cancer Care Associates of York in York, Pennsylvania.*



Donald R. Fleming, MD, *hematologist/oncologist, Cancer Care Center, Davis Memorial Hospital, Elkins, West Virginia.*

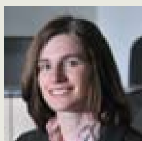
Karen MacDonald, RN, BSN, CPON, *pediatric oncology nurse, William Beaumont Hospital, Royal Oak, Michigan.*



Kerstin L. McSteen, BSN, MSN, ACHPN, CNS-BC, *clinical nurse specialist, palliative care consult service, Abbott Northwestern Hospital, Allina Health System, Minneapolis, Minnesota.*

K. Lynne Quinn, RN, MSN, CRNP, AOCNP, *director of radiation oncology, Bryn Mawr Hospital and Bryn Mawr Health Center, Bryn Mawr, Pennsylvania.*

Barbara B. Rogers, CRNP, MN, AOCN, ANP-BC, *Fox Chase Cancer Center, Philadelphia, Pennsylvania.*



Lisa A. Thompson, PharmD, *assistant professor of clinical pharmacy, University of Colorado School of Pharmacy, Aurora.*

Rosemarie A. Tucci, RN, MSN, *manager for oncology research & data services, Lankenau Hospital, Wynnewood, Pennsylvania.*

QUESTIONS & ANSWERS

CHEMOTHERAPY AGENTS AND RADIOSENSITIVITY

How do certain chemotherapy agents make tumors more radiosensitive? — Barry A. Cochran

Radiation therapy uses high energy x-rays or gamma rays and particles such as neutrons and electrons to kill cancer cells. It works by damaging DNA, causing the cells to stop dividing or die. When the damaged cells die, they are broken down and eliminated by the body's natural processes. Systemic chemotherapy uses anticancer drugs that enter the bloodstream and reach all areas of the body. Research has found that certain chemotherapy drugs can potentiate radiation therapy when used at the same time. Although concurrent therapy may increase side effects, studies have shown it improves survival for patients with certain cancers.

The combination of chemotherapy and radiation therapy given at the same time is sometimes called chemoradiation or radiochemotherapy. For some types of cancers, this combination treatment may kill more cancer cells and increase the likelihood of a cure, but it can also cause more side effects. Researchers are currently studying radiosensitizers and radioprotectors, chemicals that modify a cell's response to radiation. Radiosensitizers are drugs that make cancer cells more sensitive to the effects of radiation therapy. In addition, some anticancer drugs such as fluorouracil (Adrucil, 5-FU) and cisplatin (Platinol-AQ) make cancer cells more sensitive to radiation therapy. Chemoradiation can help the radiation work more efficiently and can also reduce the chance that the cancer will spread. Chemoradiation may be used by itself or before or after surgery. — K. Lynne Quinn, RN, MSN, CRNP, AOCNP

BEVACIZUMAB USE AND THROMBOEMBOLIC EVENTS

Is bevacizumab (Avastin) use in patients with metastatic colorectal cancer (mCRC) or non-small cell lung cancer (NSCLC) related to a higher incidence of thromboembolic events?

Clinical trials performed with bevacizumab demonstrated that the incidence of grade 3-4 venous thromboembolic events in patients with metastatic colorectal cancer (mCRC) or non-small cell lung cancer (NSCLC) is higher in those who receive bevacizumab in combination with chemotherapy than in those who receive chemotherapy alone (Avastin [package insert]; Nat Biotechnol. *2004;22[10]:1198.) There was also evidence of an increased risk of developing a second subsequent thromboembolic event in patients with mCRC receiving bevacizumab and chemotherapy compared with those receiving chemotherapy alone. One study of 53 patients (14%) on bolus-intravenous fluorouracil (IFL) plus bevacizumab arm and 30 patients (8%) on the bolus-IFL plus placebo arm received full dose warfarin following a venous thromboembolic event. Of*

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these patients studied, a secondary embolic event occurred in 21% (11/53) of patients receiving bolus-IFL plus bevacizumab and 3% (1/30) of patients receiving bolus-IFL alone. Whether possible drug-related inflammation leading to embolic disease is limited to patients with colorectal cancer who are receiving 5-fluorouracil-based therapy is still under investigation. As many as 5% of all patients using bevacizumab are at increased risk of thromboembolic events; however, the increased risk does not impact the basic risk/benefit ratio of the drug. The proactive approach for use of bevacizumab must be a balance between risk factors and benefit, and clinicians should be mindful that this risk does exist in patients who already have a heightened risk of thromboembolic events as a result of malignancy alone. — Jiajoyce R. Conway, DNP, FNP-BC, NP-C

GENOTYPE TESTING NOT WARRANTED

Is CYP2D6 genotyping for tamoxifen (Nolvadex) sensitivity before use of the drug beneficial?

*The significance of CYP2D6 genotyping and tamoxifen relates to the metabolite endoxifen, which is extremely important to the overall anticancer effects of tamoxifen (*Clinical Oncology News*. 2011;06:02). Endoxifen is predominantly formed by the CYP2D6 enzyme-mediated oxidation of N-desmethyltamoxifen. There has been controversy as to whether or not women receiving tamoxifen who either carry genetic variants associated with low or absent CYP2D6 activity, or who receive concomitant medications known to inhibit CYP2D6 activity have significantly lower levels of endoxifen, worse relapse-free time, and worse disease-free survival. Recent data from retrospective analyses of the ATAC study, which included 9,366 postmenopausal women with invasive breast cancer, that focused on subsets of 615 of 3,125 women who had received anastrozole and 588 of 3,116 women who had received tamoxifen demonstrated no evidence that CYP2D6 genotyping is an effective predictor of recurrence in either arm. The study also demonstrated that there was no strong evidence for avoiding CYP2D6 inhibitors such as fluoxetine, paroxetine, quinidine, and buproprion. The lack of standardization for CYP2D6 phenotype testing and variations of use with concomitant CYP2D6 inhibitors allows for weaknesses in the validity of this information. Overall consensus that remains from the recent San Antonio Breast Cancer Symposium thought leaders is that there are no data to warrant routine genotype testing for postmenopausal women taking tamoxifen; however, there should still be some degree of caution and avoidance of potent CYP2D6 inhibitors in women treated with tamoxifen. — Jiajoyce R. Conway, DNP, FNP-BC, NP-C* ■

Do you have a clinical challenge to share with your colleagues?

If you would welcome another perspective on how to manage a patient, or have a question about managing drug therapies in your patients, write us and we'll forward your query to one of our consultants and publish the response in *Oncology Nurse Advisor*.

SEND TO
Oncology Nurse Advisor
25 Philips Parkway, Suite 105
Montvale, NJ 07645

FAX
201-391-0880

E-MAIL
editor.ona@haymarketmedia.com

NAME DEGREES (RN, NP)

ADDRESS

CITY STATE ZIP

PHONE FAX

E-MAIL 0411