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Hypertension in the Oncology Setting

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Case Presentation

K.A. is a 56-year-old African American female with a history of colorectal carcinoma with metastasis to her liver. She presents to the clinic for her fifth cycle of biweekly 5-fluorouracil, leucovorin, and oxaliplatin (FOLFOX) and bevacizumab (Avastin®, Genentech BioOncology, South San Francisco, CA). Overall, she has been tolerating the treatment with minimal toxicity.

Past medical history is significant for obesity, prediabetes, and hypertension (HTN). Her blood pressure (BP) is controlled in a range of 130/80 mmHg by hydrochlorothiazide 25 mg daily. Review of systems is significant for fatigue lasting several days following chemotherapy and occasional dyspnea on exertion. K.A. attributes the symptoms to deconditioning and chemotherapy. She denies history of dizziness, lightheadedness, headache, chest pain, or cough.

Since the initiation of K.A.'s chemotherapy, her vital signs have been stable with the exception of her BP measurements. Prior to her initial infusion, her BP was 136/84 mmHg. During the three subsequent cycles, her measurements elevated gradually to 140/88 mmHg, 146/92 mmHg, and 154/94 mmHg, respectively. Today her BP is 162/92 mmHg.

K.A. has a BP monitor at home and was asked to record her BP, after being seated for five minutes, daily for the next several days. She was instructed to contact the clinic to report the measurements and to call sooner if she became symptomatic. K.A.'s measurements revealed BPs averaging 154/96 mmHg.

Because K.A. already was taking the maximum recommended daily dose of hydrochlorothiazide, she was given a prescription for lisinopril 10 mg daily and instructed to continue to monitor her BP daily. The primary care provider was advised of the addition of the medication.

Definition

By definition, BP is the product of cardiac output and peripheral vascular resistance (BP = cardiac output x peripheral resistance). HTN is caused by an increase in cardiac output, peripheral resistance, or both. Cardiac output may be increased by any condition that raises heart rate or stroke volume, whereas peripheral resistance is increased by any factor that raises blood viscosity or reduces vessel diameter (Brashers, Haak, & Richardson, 1998). Therefore, variation in extracellular fluid volume, the contractile state of the heart, and vascular tone determine the variation in BP level (Schwartz & Sheps, 2004).

Two types of HTN exist: primary and secondary. Primary HTN, also known as essential HTN, is of unknown cause. Secondary HTN, on the other hand, results from an underlying, identifiable, and often correctable cause. Some causes of secondary HTN include renovascular disease, polycystic renal disease, and pharmacologic or nonpharmacologic medication side effects (Onusko, 2003). Only 5%–10% of HTN cases are believed to result from secondary causes. A diagnosis of HTN is based on the average of two or more properly measured, elevated BP readings on each of two or more office visits (National Heart, Lung, and Blood Institute, 2003). See Table 1 for classification of adult BPs.

Oncologists use the Common Terminology Criteria for Adverse Events as developed by the National Cancer Institute to report and grade symptoms. This system classifies HTN somewhat differently than that outlined by HTN specialists and is explained in Table 2.

Incidence and Epidemiology

HTN has been found in more than half of people older than 65 years (Kaplan, 1998b). As many as 65 million individuals with HTN currently take antihypertensive medications or have received recommendations from a physician to initiate treatment (Centers for Disease Control and Prevention, 2004). This equates to about one in three U.S. adults with high BP.

The American Cancer Society (2005) estimated that one in three Americans can expect to be diagnosed with cancer in their lifetime. The overlap between these two serious and often chronic conditions is significant.

Pathophysiology

Several hypotheses for the pathogenesis of essential HTN exist. Some of these include high dietary sodium intake and defects in renal sodium excretion, increases in blood volume, inappropriate autoregulation, overstimulation of sympathetic neural fibers in the heart and vessels, and hormonal inhibition of sodium-potassium transport across cell walls in the kidneys and blood vessels (Brashers et al., 1998).

Secondary HTN is caused by a systemic disease process that raises either peripheral vascular resistance or cardiac output. Renal, endocrine, vascular, and neurologic disorders; acute stresses (e.g., surgery, hyperventilation); and drugs may elevate BP. If the cause of the elevation is removed before permanent structural changes occur, BP should return to normal (Onusko, 2003).

Patients with cancer are at risk for HTN secondary to a number of causes, including

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