In the April 2004 Test Your Knowledge, “Hypercalcemia of Malignancy: Part I” (Vol. 8, No. 2, pp. 209–210) included an overview of hypercalcemia with specific attention to the pathophysiologic mechanisms of the disease. This column is the second and final part of this series and will focus on patient assessment, medical management, and nursing interventions. Hypercalcemia is potentially life threatening because it can go unnoticed until it becomes severe. Untreated, patients with hypercalcemia have approximately a 50% mortality rate, with a median survival of less than three months after diagnosis of hypercalcemia of malignancy (Otto, 2001). Oncology nurses must assess for and manage this serious complication.

Case Study

Mr. C is a 62-year-old male who was diagnosed with stage IV squamous cell cancer of the lung. He arrives at the ambulatory clinic for his third course of palliative chemotherapy. His wife consults with the nursing staff this morning and reports that her husband’s confusion improved for two weeks following the last office visit; however, during the past week, he has been increasingly forgetful. Upon further discussion, Mrs. C reports that, in addition to the confusion, her husband is increasingly depressed and fatigued. Mr. C reportedly stayed in bed for the past week, he has been increasingly forgetful. Upon further discussion, Mrs. C reports that, in addition to the confusion, her husband is increasingly depressed and fatigued.

Adjusting the calcium according to the albumin is important because relative severity of hypercalcemia is underestimated if the correction is not performed. For every 0.8 mg/dl increase in albumin, the calciu, cell count would not be reflective of the calcium changes. Choices c and d, hyperphosphatemia and hypermagnesemia, are incorrect because both electrolyte levels would be decreased. Electrolyte changes frequently occur in conjunction with hypercalcemia. Antidiuretic hormone, normally responsible for urine concentration in the renal distal tubules, is impaired in the presence of an elevated calcium level. This results in polyuria with subsequent dehydration and a decrease in the glomerular filtration rate. Ultimately, the kidneys are unable to excrete the calcium. Polyuria causes the loss of other electrolytes, including magnesium and phosphorus. Because glomerular filtration is affected, BUN and creatinine should be monitored on an ongoing basis (Barnett, 1999). Mr. C’s BUN and creatinine levels increased from the previous visit, which can indicate a worsening of his hypercalcemia and renal function.

Discussion

1. What is Mr. C.’s corrected calcium?
   a. 13.3 mg/dl
   b. 14.0 mg/dl
   c. 14.3 mg/dl
   d. 15.1 mg/dl
   
2. Which laboratory change might you see in patients with hypercalcemia?
   a. Neutropenia
   b. Hypokalemia
   c. Hyperphosphatemia
   d. Hypermagnesemia

3. Further assessment in patients with hypercalcemia may reveal
   a. Tachycardia
   b. Hyperreflexia
   c. Constipation
   d. Signs of fluid overload

4. The best immediate treatment to correct Mr. C’s calcium and symptoms is
   a. Hydration and bisphosphonate infusion
   b. Chemotherapy administration and corticosteroids
   c. Growth factors to improve fatigue and minimize myelosuppression
   d. Hydration and observation

Question 1: The correct answer is choice c, 14.3 mg/dl. Choices a, 13.3 mg/dl, b, 14.0 mg/dl, and d, 15.1 mg/dl, are incorrect because of mathematical errors. Adjusting the calcium according to the albumin is the first step in assessing for hypercalcemia. About half of the circulating calcium is in the form of free (ionized) calcium, and nearly 45% is bound to plasma proteins such as albumin. Less than 5% is complexed with bicarbonate, citrate, or phosphate. For many patients, hypoalbuminemia ensues because of malnutrition and progressive disease. Adjusting the serum calcium according to the albumin is important because relative severity of hypercalcemia is underestimated if the correction is not performed. For every 0.8 mg/dl of albumin lost, a 0.8 mg/dl correction of calcium is created (Otto, 2001; Wichkam, 2000) (see Figure 1).

Therefore, in this case study, the measured total calcium concentration is 12.8 mg/dl + 0.8 x (4.0 – 2.1 g/dl) albumin, which is approximately 14.3 mg/dl.

Question 2: The correct answer is choice b, hypokalemia. Choice a, neutropenia, is incorrect because the white blood cell count would not be reflective of the calcium changes. Choices c and d, hyperphosphatemia and hypermagnesemia, are incorrect because both electrolyte levels would be decreased. Electrolyte changes frequently occur in conjunction with hypercalcemia. Antidiuretic hormone, normally responsible for urine concentration in the renal distal tubules, is impaired in the presence of an elevated calcium level. This results in polyuria with subsequent dehydration and a decrease in the glomerular filtration rate. Ultimately, the kidneys are unable to excrete the calcium. Polyuria causes the loss of other electrolytes, including magnesium and phosphorus. Because glomerular filtration is affected, BUN and creatinine should be monitored on an ongoing basis (Barnett, 1999). Mr. C’s BUN and creatinine levels increased from the previous visit, which can indicate a worsening of his hypercalcemia and renal function.

Question 3: The correct answer is choice c, constipation. The increased calcium causes delayed gastric emptying and delayed peristalsis from a decrease in smooth muscle contractility. Dehydration further potentiates the constipation. Choice a, tachycardia, is incorrect. Although electrocardiogram changes commonly occur.