

Inpatient Diabetes Mellitus in the Oncology Setting

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Case Study: A.B. is a 32-year-old Caucasian man with a significant, 12-year medical history of diabetes mellitus type I as well as a history of hypertension and hypercholesterolemia. He presented to his primary care doctor with blurred vision and was referred to a retinal specialist, who diagnosed hemorrhagic and leukemic retinopathy resulting in limited vision. A complete blood count was drawn during the visit with his retinal specialist. The results were abnormal, revealing a white blood cell count of 30,000/mm³, platelet count of 70,000/mm³, and a hemoglobin of 12.2 gm/dl. The peripheral smear showed peripheral blasts. A.B. was referred to a hematologist, who performed a bone marrow aspiration, confirming the diagnosis of pre-B-cell acute lymphocytic leukemia (ALL). Cytogenetic studies revealed positivity for the Philadelphia chromosome and a translocation of genes 4 and 11.

Acute Lymphocytic Leukemia

ALL accounts for 20% of all adult leukemias, which in themselves constitute about 3% of adult malignancies. The clinical signs and symptoms of ALL usually occur suddenly and derive from expansion of the leukemic cell in the marrow and from involvement of the peripheral blood and extramedullary sites such as the lymph nodes, liver, spleen, and central nervous system. Common symptoms include fatigue, fever, night sweats, weight loss, easy bruising or obvious bleeding, dyspnea, dizziness, and infections. Extremity and joint pain also may be present, sometimes leading to a misdiagnosis of arthritis (Faderl & Kantarjian, 2006).

Comorbid conditions such as diabetes mellitus with high-dose steroid administration leading to hyperglycemia often occur with treatment for ALL. Evidence suggests that hyperglycemia in patients with ALL contributes to increased infec-

tions, higher rates of recurrence, and mortality (Weiser et al., 2004).

Evaluating A.B.'s Diabetes

A.B. had been on insulin for 12 years. He had no history of diabetic ketoacidosis but did have mild peripheral neuropathy in his feet and hands. His blood glucose levels usually ran in the 200s, but he rarely checked his levels at home; he admitted that he did not follow a limited-carbohydrate diet. He typically received Humulin® 70/30 (isophane insulin [NPH]/regular, Eli Lilly and Company) twice a day: 45 units in the morning and 25 units in the evening. Upon evaluation of his diabetes mellitus, laboratory values revealed a hemoglobin A1C of 10%, C-peptide of 0.9 ng/ml, and negative urine

ketones. When A.B. was given a dose of dexamethasone 40 mg IV as part of his chemotherapy regimen, his blood glucose rose to 400 mg/dl.

Pathophysiology of Diabetes Mellitus

A.B. originally was diagnosed as having diabetes mellitus type I. However, the C-peptide result, along with a lack of episodes of diabetic ketoacidosis, defined him as insulin deficient. C-peptide is a useful marker of insulin secretion and allows discrimination of endogenous and exogenous sources of insulin in evaluation of hyper and hypoglycemia (Powers, 2006). Diabetes type I is considered an autoimmune destructive process of beta cells that occurs over months to years (see Figure 1).

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