Multiple Myeloma and Its Treatment

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Case Study

In 1995, Mr. F visited an emergency room with symptoms of an upper respiratory infection and severe rib pain induced by coughing. He was diagnosed initially with pneumonia and two fractured ribs; however, further workup revealed elevated serum creatinine, calcium, and protein levels and severe anemia (hemoglobin = 4.1 mg/dl). Malignancy was suspected, and additional diagnostic test results, including a serum immunoglobulin A (IgA) level higher than 7,000 mg/dl, led to the diagnosis of stage IIIIB multiple myeloma (MM).

Mr. F was treated with vincristine, doxorubicin, and dexamethasone (VAD) for one year and had a partial response to the chemotherapy. His bone pain resolved, renal function recovered, IgA level dropped to within normal limits, and hemoglobin rose to and remained greater than 10 mg/dl. He then received interferon followed by cyclophosphamide, but his disease was refractory to these agents. Mr. F underwent high-dose chemotherapy with tandem autologous stem cell transplants in March and September 1997. Disease progression was noted in January 1998 when his IgA was 3,775 mg/dl; as a result, he started on cyclophosphamide, dexamethasone, etoposide, and cisplatin. Mr. F’s disease continued to progress, and he was started again on the VAD regimen in March 1998. Prior to restarting the VAD regimen, Mr. F’s cardiac ejection fraction was 65%. After three cycles of VAD, his IgA level dropped to 1,700 mg/dl and his ejection fraction dropped to 57%. In July 1998, dexrazoxane was added to the VAD regimen and he received nine dexrazoxane and VAD treatments at his home. Doxorubicin was administered as a 96-hour continuous infusion, and a homecare nurse administered 100 mg of dexrazoxane over 30 minutes prior to initiation of the VAD regimen and every 12 hours during the doxorubicin infusion.

In January 1999, thalidomide (200 mg daily, increased to 800 mg daily) was added to the dexrazoxane and VAD regimen. Mr. F’s IgA levels dropped from 2,680 mg/dl in September 1998 to 221 mg/dl in August 1999. His ejection fraction rose to 62% in October 1998, and in January 2000, it was 66%.

Mr. F received maintenance therapy consisting of thalidomide and dexamethasone from August 1999 to May 2000. At the end of May 2000, his IgA level rose to 3,572 mg/dl and a high-grade anaplastic plasmacytoma of his right masseter muscle originating from the same cell line as his MM was diagnosed. Another cycle of dexrazoxane and VAD was administered, but Mr. F’s IgA level rose to 5,960 mg/dl. He received topotecan in July and August 2000, and despite frequent transfusions, his hemoglobin level remained low. His serum creatinine level remained high, and he chose not to receive dialysis. His disease rapidly progressed, and he died in September 2000.

1. Which of the following statements best reflects current statistics regarding the incidence and death rate of MM in the United States?
   A. The number of new cases of MM in 2002 approximated the number of people who died from the disease.
   B. The number of people diagnosed with MM in 2002 was three times greater than the number who died from the disease.
   C. The number of people who died from MM was three times greater than the number diagnosed with the disease in 2002.
   D. The incidence and death rate statistics for MM are similar to rates for breast cancer, colon cancer, and lymphoma.

2. Clinical manifestations of MM result from
   A. Paraproteins that block small blood vessels when the weather is cold.
   B. Monoclonal protein production and its accumulation in the serum or urine.
   C. Light chain plasma cells and serum proteins that combine to form amyloid protein.
   D. High plasma viscosity, which causes the blood to become thick and sticky.

Discussion

Question 1: The correct answer is choice A, the number of new cases of MM in 2002 approximated the number of people who died from the disease. Approximately 14,400 new cases of MM were diagnosed in 2002, and 11,200 people died of the disease (Jemal, Thomas, Taylor, & Thun, 2002). According to the MM Research Fund (2002), about 40,000 people in the United States in 2002 are living with MM.

MM is a clonal B cell disorder arising from plasma cells. The disease affects older adults,