CASE STUDY

72-YEAR-OLD MAN WITH CHRONIC LYMPHOCYTIC LEUKEMIA
Kathryn G. Keegan, ARNP

BACKGROUND

A 72-year-old man with chronic lymphocytic leukemia (CLL) presented to the cancer care clinic for a follow-up appointment 10 days after his first cycle of chemotherapy with fludarabine, cyclophosphamide, and rituximab (FCR). He complained of mild chills and a low-grade fever of 99.9°F. He did not seek medical attention before his appointment because he attributed the fever to CLL. Other than a temperature of 101°F, his vital signs were normal. Laboratory work was obtained, including blood cultures and urine analysis and culture. He was noted to be neutropenic with an absolute neutrophil count of 120 cells/mm³. He was sent for chest X-ray before admission for neutropenic fever.

MEDICAL HISTORY

The patient was initially diagnosed in January 2003 with CLL, Rai stage I, when he underwent magnetic resonance imaging for chronic low back pain, and there was an incidental finding of lymphadenopathy involving multiple periaortic and iliac lymph nodes. Computed tomography scan demonstrated innumerable enlarged lymph nodes seen at all major lymph node stations. The patient had a leukocytosis (white blood cell count, 18 500 cells/mm³; 65% lymphs) with atypical lymphs but no anemia or thrombocytopenia. Peripheral blood sent for flow cytometry had atypical lymphoid cells that were CD5 positive, CD38 low.

By May 2004, the disease had progressed to Rai stage IV with increased lymphadenopathy, lymphocyte doubling time of less than 1 year, mild anemia and thrombocytopenia. The patient had no known family history of malignancies. His mother died at age 37 of pneumonia at age 52. The patient had a leukocytosis (white blood cell count, 18 500 cells/mm³; 65% lymphs) with atypical lymphs but no anemia or thrombocytopenia. Peripheral blood sent for flow cytometry had atypical lymphoid cells that were CD5 positive, CD38 low.

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FAMILY HISTORY

The patient had a known family history of malignancies. His mother died at age 37 of a pulmonary hemorrhage secondary to tuberculosis. His father died at age 55 of a myocardial infarction with “black lung”. His brother had diabetes mellitus and died of pneumonia at age 52.

SOCIAL HISTORY

The patient is a retired Air Force jet mechanic and retired postal worker. He is married and has 3 adult children and 10 grandchildren. He was exposed to Agent Orange during his military service. He smoked from age 12 through 28, but not since. He quit drinking at age 28 and denies recreational drug use.

PHYSICAL EXAMINATION

On presentation, the patient had a temperature of 101°F and appeared pale and weak, but nontoxic. He was alert and oriented to person, place, and time. He described feeling as if he had the flu. Eye examination was normal. Examination of ears, lips, teeth, and oropharynx was normal. Cardiac examination demonstrated a tachycardia with an accelerated pulse of 110 beats/minute without murmurs, rubs, or gallops; blood pressure, 111/63 mm Hg; respiration, 20 breaths/minute, unlabored with clear breath sounds throughout all lung fields. Abdominal examination demonstrated a soft abdomen with the liver edge palpated 2 cm below the right costal margin; the spleen tip was palpable on inspiration at left costal margin. Lymph node examination revealed enlarged, mobile nodes bilaterally in the anterior cervical chain (approximately 2 cm), left submandibular nodes (approximately 4 cm), supraclavicular nodes (left > right, 1–2 cm); bilateral axillary nodes (2–3 cm), and increased inguinal nodes (2–4 cm) bilaterally. His extremities were warm without edema or clubbing. Neurologic examination was grossly nonfocal.

TREATMENT COURSE

Laboratory work demonstrated an absolute neutrophil count (ANC) of 120 cells/mm³, a platelet count of 80 × 10⁹/mL, and a hemoglobin (Hgb) of 8.6 g/dL. Blood chemistries were normal including lactate dehydrogenase. Blood cultures were obtained from the peripherally inserted central catheter line and his left arm. A chest X-ray, urine analysis, and urine culture also were obtained. He was admitted to the inpatient oncology unit for intravenous antibiotics and closer monitoring for sepsis. The patient was started on broad-spectrum antibiotics. His fever resolved 4 days into the hospital admission, and cultures demonstrated no evidence of growth. The chest X-ray was negative for infiltrates. The ANC recovered to 700 cells/mm³ by hospital day 6, and he was discharged on the oral broad-spectrum antibiotic gatifloxaxin.

The patient was seen in follow-up post-hospitalization by his oncologist. His next cycle of chemotherapy was delayed by 10 days because of an inadequate ANC for chemotherapy. Pegfilgrastim was implemented on the second and all subsequent cycles of FCR. This facilitated receipt of the remaining cycles of chemotherapy without delay or further incidents of febrile neutropenia.

DISCUSSION

Neutropenic fever can cause life-threatening complications and has a tremendous impact on patients, including increased risk for sepsis, delay in further chemotherapy treatment, and potential chemotherapy dose reduction. This patient had a low-grade fever that he attributed to CLL. When he presented in the clinic for follow-up, the patient was febrile and neutropenic, but his vital signs remained stable. Fortunately, the neu-
tropic fever did not evolve into sepsis; however, the patient required prolonged hospitalization for neutropenic fever and coverage with broad-spectrum antibiotics, which increased his risk for nosocomial infection. The patient’s prolonged period of neutropenia also led to a delay in the subsequent cycle of chemotherapy.

The National Comprehensive Cancer Network clinical practice guidelines for myeloid growth factors (MGF) can be used by healthcare providers before chemotherapy treatment to assess a patient’s risk for neutropenic fever. Risk factors for neutropenia include treatment regimen, dose intensity, prior chemotherapy, history of neutropenia, concurrent and/or prior radiation to bone marrow, patient age, gender, performance status, nutritional status, immune function, type of cancer, bone marrow involvement with cancer, comorbidities, open wounds, and active tissue infection. This patient had several risk factors for neutropenic fever, including CLL, a myelosuppressive regimen, and advanced age. The delay in the patient’s chemotherapy treatment could have been prevented if a pretreatment risk assessment had been performed, which would have indicated the need for the implementation of MGFs with the first cycle of chemotherapy. The implementation of MGFs would have reduced the patient’s risk for neutropenic fever, potentially preventing hospitalization and treatment delay.

REFERENCE