Incidental diagnosis of CML in a patient with anemia and IgG lamda monoclonal protein in blood

Dana Shani · Afshan Malik

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Dear Editor,

We would like to report a case of an incidental diagnosis of CML in a patient with anemia and IgG lamda monoclonal protein in blood.

A 74-year-old African American male with a history of hypertension, diabetes mellitus, and mild chronic kidney disease was found to have low hemoglobin of 8.25 g/dl with normal WBC and platelet count. On further work up, he was found to have IgG lamda monoclonal protein in blood. Pt was referred to the hematologist to rule out multiple myeloma.

Serum immunofixation showed an IgG lambda-monoclonal gammopathy with IgG level of 1,300 mg/dl (upper limit ~ 1,618 mg/dl), WBC count of 3.0K, platelet count of 165K, and beta-2 microglobulin of 4.00 µg/ml (normal <2.51 µg/ml). Physical exam was within normal limits. No lymphadenopathy or hepatosplenomegaly was appreciated. Review of systems was significant for unintentional 10-lb weight loss in the last 3 months.

Bone marrow biopsy was performed at this point to rule out multiple myeloma. It revealed normocellular marrow with maturing trilineage hematopoiesis and polytypic plasma cells less than 5%. Flow cytometric analysis did not show evidence for a clonal plasma cell population; however, cytogenetic analysis unexpectedly revealed a Philadelphia (Ph) chromosome resulting from a translocation between the long arms of chromosome 9 and 22 in three metaphases, and FISH was positive for the presence of BCR and ABL gene. BCR-ABL gene analysis done by PCR on the patient's blood performed later at a different laboratory was positive at 0.119.

The diagnosis of CML was made, and patient will receive therapy with Imatinib.

Others have noted that the lambda light chain gene locus and BCR translocation breakpoint are both located on the 22q11 band [1]. Also, cases of multiple myeloma together with CML have been described [2–5]. But, to our knowledge, this is the first reported case of incidental finding of Philadelphia chromosome in a patient with anemia and IgG lamda monoclonal protein in blood, without the expected findings of marrow hyperplasia, represented by leukocytosis or thrombocytosis. This most likely represent very early CML, which is now an easily treated disease, and brings up the question of checking FISH for BCR-ABL gene for certain patients with a monoclonal Lambda chain protein in blood, even without the typical manifestations of CML. We hope this observation will lead to a prospective study before recommendation can be made to check bcr-abl gene mutation in patients with a monoclonal gammopathy.

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D. Shani · A. Malik (✉)
Lenox Hill Hospital,
100 East 77th Street,
New York, NY 10075, USA
e-mail: afshantmalik@gmail.com

D. Shani
e-mail: docshani@aol.com