Richter syndrome in CLL – an aspiration cytologic diagnosis

Prashant Sharma · Namrata Setia · Sonu Nigam · Shyama Jain · Manorama Gaiha · Tejinder Singh

Received: 30 December 2007 / Accepted: 22 February 2008

Dear Editor,

We read with interest the case report by Izhar et al [1] and would like to share a similar case at our institution.

A 50-year-old male diagnosed with chronic lymphocytic leukaemia (CLL), stage A (Binet) in October 2003 was put on long-term follow-up. In May 2004, he developed extreme fatigue, rapid weight loss and fever. Examination revealed, massive axillary lymphadenopathy along with inguinal and cervical lymph node enlargement, splenomegaly (6 cm below costal margin) and hepatomegaly (2 cm below costal margin). Blood counts, peripheral smear, trephine biopsy and touch preparation were all consistent with CLL. The clinical differential diagnosis included clonal evolution/disease transformation versus overwhelming infection, possibly tubercular.

Fine needle aspirates from the multiple enlarged nodes revealed sheets of large, pleomorphic, round to polygonal plasmacytoid cells with unevenly distributed nuclear chromatin, prominent, usually single central nucleoli; occasional multiple nucleoli; a perinuclear clear zone, frequent mitoses, and abundant pale blue cytoplasm. An occasional tingible body macrophage was seen. On immunochemistry, these cells were positive with anti CD-20 antibodies and

negative with anti CD-3, CD-15 and CD-30 antibodies. A diagnosis of B-cell non-Hodgkin lymphoma (NHL), morphologically suggestive of immunoblastic differentiation was made. The patient had a rapidly downhill course and died within a week of diagnosis before definitive therapy could be instituted.

Lymphadenopathy is common in patients with CLL and these in uncomplicated disease are small, discrete, freely mobile and non-tender. On morphologic evaluation, such nodes usually reveal infiltration by small leukaemic lymphocytes. Painful enlarged nodes are a sign of infection – patients commonly have a hypogammaglobulinemia as well as more subtle defects in T cell, complement and polymorphonuclear leukocyte function. Rapidly progressive or massive enlargement is ominously suggestive of a transformation to a more aggressive and distinct hematolymphoid malignancy. Prolymphocytic leukaemia, acute lymphoblastic leukaemia, Hodgkin lymphoma, multiple myeloma and large cell NHL with immunoblastic features (as in our case) have all been described [2].

In conclusion therefore, both our case and the previous report by Izhar et al [1] emphasize the importance of recording and repeatedly assessing lymph node size in patients with CLL. They also bring out the role of pathological sampling of lymphadenopathy in evaluating these patients. We propose fine needle aspiration cytology as a rapid, convenient, minimally-invasive and cost-effective first-line technique towards this end.

P. Sharma¹ · N. Setia² · S. Nigam³ · S. Jain⁴ · M. Gaiha⁵ · T. Singh⁶

¹Former Senior Resident

²Senior Resident, Department of Pathology

³Ex-Professor

⁴Professor

⁵Former Director-Professor, Department of Internal Medicine

⁶Director-Professor and Head

Departments of Pathology and Internal Medicine,
Maulana Azad Medical College and Lok Nayak Hospital,
BSZ Marg
New Delhi - 110 002, India.

P. Sharma (✉)

e-mail: Prashant.sh@gmail.com

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