The Coexistence of Chronic Lymphocytic Leukemia and Multiple Myeloma

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Multiple myeloma (MM) and chronic lymphocytic leukemia (CLL) are neoplastic diseases originating from different stages of B-cell maturation. Coexistence of MM and CLL in the same patient is quite rare\cite{1} We report a case with CLL who later developed kappa light chain MM.

A 67-year-old male was diagnosed as CLL with CD5+, CD19+, CD23+ B lymphocytes detected by flow cytometry. Karyotype analysis revealed 45,X,Y[4]/46,XY[10] and deletion of 11q22.3 was found in 55% as sole anomaly by FISH (Fluorescence in situ hybridisation). He was treated with chlorambucil-dexamethasone.

Five years later, he admitted with fatigue, back pain, hypercalcemia and acute renal failure. There was no palpable lymphadenopathy or hepatosplenomegaly. Serum and urine immune fixation electrophoresis revealed kappa light chain monoclonal protein and bone marrow (BM) aspiration at least 30% atypical plasma cells. Similarly, BM immunophenotyping revealed 30% of clonal plasma cells (CD38+, CD138+) and approximately 1% of residual CLL cells. Complex karyotype was found as 46,XY,der(6)t(1;6)(q11;q23),t(11;14)(q13;q32), dup(17)(q23q25)[17] /46,XY[2] (Fig 1).

FISH analysis revealed t(11;14) and deletion 6q23, but not prior 11q22.3 deletion. He was diagnosed as MM. Intravenous hydration, plasmapheresis, furosemide, pamidronate and dexamethasone were started. Following discharging the hospital he was lost to follow up.

Coexistence of CLL and MM is quite rare and and there is a controversy whether two diseases arise from the same clone or distinct clones. Fermandet al showed that these malignancies come from same clone by the identification of Ig idiotypes.\cite{2} After

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CLL cells were exposed to mitogens and allogenic T cells, a class switch from IgG to IgA observed, showing CLL cells can be precursors of plasma cells. Brouet et al, Novak et al and Kaufmann et al showed Ig molecules synthesizing different light chains revealing the coexistence of two distinct clones. But Ig subtyping can not always eliminate clonality. In some cases CLL and MM were diagnosed together whereas in others MM is diagnosed 1-15 years after CLL. Barlogie et al indicates difference in pathophysiology; in CLL most tumour B cells are inert and arrested in G0/G1 phase whereas in MM there is an increase in proliferation with stromal cell cytokines like IL-6. In our case, there is ATM deletion at CLL diagnosis, but not at MM diagnosis; indicating multiclonality.

Compatible with our case, there are reported cases diagnosed as MM after CLL who have chromosome 11 anomaly at time of CLL diagnosis raising a question: “Could some of the chromosome 11 anomalies be related to transformation from CLL to MM, and used as a predictor? To clarify the role of these genetic, epigenetic or microenvironmental factors for the coexistence two diseases, more case reports are needed.

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References:


Figure 1. Karyotyping analysis showing 46, XY, der(6) t(1;6) (q11;q23), t(11;14) (q13;q32), dup (17) (q23q25) [17]/46, XY [2]

94x64mm (72 x 72 DPI)