Dear Editor,

The association of CLL with hematologic malignancies such as AML is relatively rare [1, 2], with mostly associated with prior cytotoxic chemotherapy [3]. Few reports of untreated CLL manifesting with or followed by AML suggested that each tumor probably evolved from simultaneous expansion of two independent clones, not a common clone [1]. Concomitant AML and CLL having a common clonal origin is exceptional, and to our knowledge, only two cases were reported previously [4, 5]. We present a case of de novo AML concurrent with untreated CLL, with two karyotypic abnormalities in the same clone.

A 76-yr-old man was presented with fever and chills in August 2015, with a history of hypertension, but no cancer, chemotherapy, or irradiation. Physical examination indicated mild pallor and a skin lesion with redness and swelling on the left thigh, without lymphadenopathy or gingival lesions. Laboratory examinations showed moderate leukopenia (white blood cells [WBC], 2.8×10^9/L), comprising 1% myeloblasts and 60% mature lymphocytes, macrocytic anemia (Hb, 92 g/L; mean corpuscular volume, 104.0 fL), and thrombocytopenia (platelets [PLT], 31×10^9/L). Bone marrow (BM) examination indicated a normocellular marrow for his age (cellularity, 20%), including 21.6% myeloblasts and 16.6% mature lymphocytes (Fig. 1). The clot section showed multiple irregularly shaped medium-to-large lymphoid nodules, comprising small mature lymphocytes. Immunohistochemical stains of the BM clot section showed that the myeloblasts were positive for CD34 and CD117, and the small lymphoid cells in the lymphoid nodules were positive for CD5, CD19, and CD23.

Flow cytometric immunophenotyping of BM aspirate showed two populations: one positive for myeloblast markers (CD34, CD13, CD33, CD117, and HLA-DR) and another for B lymphoid markers (CD5, CD19, CD20, and CD23) along with co-expression of CD19 and CD5 (Fig. 2A). Chromosome analysis showed an abnormal karyotype (46,XY,del(13)(q14),add(14)(q32)[3]/46,XY[17]) (Fig. 2B). FISH analysis performed by using the probes D13S319/13qter Dual Color Probe (Cytocell, Cambridge, UK) and LSI IGH Dual Color Probe (Vysis, Downers Grove, IL, USA) showed nucish (D13S319x2,13qterx1)[18/200] and nucish (IGHx1)[30/200], respectively (Fig. 2C). Skin biopsy of the left thigh suggested pyoderma.

On the basis of the WHO 2008 criteria, the patient was diagnosed as having AML, NOS (AML with maturation). However, he did not receive induction chemotherapy for AML because of his advanced age and comorbidities such as hypertension and worsening left thigh cellulitis. He was treated with a hypomethyl-
A case of AML concurrent with CLL

Fig. 1. Coexistence of myeloblasts and neoplastic lymphoid cells on bone marrow aspirate smear (Wright stain, ×1,000).

Fig. 2. Flow cytometric immunophenotyping and chromosomal analysis with bone marrow aspirate. (A-a) Gating for the two populations of neoplastic cells (myeloblasts of AML and mature lymphoid cells of CLL); (A-b) Myeloblasts with co-expression of CD34 and CD117; (A-c) Mature B-lymphoid cells with co-expression of CD19 and CD5; (A-d) Mature B-lymphoid cells with co-expression of CD19 and CD23. (B) Abnormal karyotype with del(13q) and add(14q), were observed in the same clones on karyotyping. Del(13q) could be found in myeloid and lymphoid malignancies. While del(14q) and 14q32-related translocation are associated with B lineage malignancies, the significance of additional material on 14q32 such as add(14q) in tumor cytogenetics is unclear. Moreover, 14q32/IGH rearrange-
ment of B-lymphoid malignancies was not detected, suggesting that both myeloid and lymphoid cells originated from the same clone. Hou et al [8] reported a human B-cell/myeloid common progenitor, which matured into a lineage according to the culture environment; thus, the pathways for myeloid and lymphoid development may not be very strict. In the current case, we believe that a common progenitor clone expanded and differentiated into two separate lineages. The flow cytometric sorting of the two populations into myeloid or lymphoid cells, followed by karyotyping of each population, is required to confirm the clonality. Thus, we report a case of concomitant occurrence of AML and CLL. On the basis of the chromosomal abnormalities in this patient, we suspect that the myeloid and lymphoid lineages were evolved from common progenitor cells.

Authors’ Disclosures of Potential Conflicts of Interest

No potential conflicts of interest relevant to this article were reported.

REFERENCES