SHORT COMMUNICATIONS

Translocation (1;7) in a Case of Secondary Chronic Myelomonocytic Leukemia

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ABSTRACT *A case of secondary chronic myelomonocytic leukemia with t(1;7)(p11;p11) is reported. This patient had been treated without interruption with chlorambucil for multiple sclerosis since 1970. The t(1;7), to our knowledge, is the first described in secondary chronic myelomonocytic leukemia.*

INTRODUCTION

Recently, 22 patients with myelodysplastic syndromes (MDS) and a t(1;7) have been reported [1–5]. The great majority of these patients had a history of exposure to toxic substances or radiation (usually therapeutic, rarely occupational). In a few cases, however, no such history could be obtained [2–5]; t(1;7) has also been encountered in a few cases of de novo acute non lymphocytic leukemia.

We report herein a case of chronic myelomonocytic leukemia (CMML) classified according to the FAB group, occurring after prolonged chemotherapy and exhibiting a t(1;7).

CASE REPORT

A 41-year-old man was referred to our institution in April 1984 because leukemia was suspected. He had a history of multiple sclerosis diagnosed in 1970 and had been treated since then with chlorambucil (6 mg daily) without interruption, in association with weekly injections of tetracosactide. On admission, he had mild purpura and no organomegaly was found.

The blood count showed moderate aregenerative anemia (Hb, 10 g/dl) with macrocytosis (VGM : 105μ^3), leukocytes 15 $\times$ 10^9/L with a differential of 9% neutrophils, 15% lymphocytes, 75% monocytes, and platelets 60 $\times$ 10^9/L.

The bone marrow smears were cellular with 13% blasts and 30% monocytes, which (as well as the blood monocytes) displayed only mild morphologic abnormalities. Conspicious myelodysplastic features were present in the granulocytes and erythroblasts. A bone marrow biopsy showed increased cellularity with a mod-

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erate excess of blasts but no fibrosis. Serum lysozyme was increased to 75 mg/L (Normal 5–15 mg/L); a polyclonal hypergammaglobulinemia was found.

A diagnosis of CMML was made based on FAB criteria. No chemotherapy was instituted. The patient's course was relatively stable until October 1984, when the anemia worsened, requiring repeated transfusions. Bone marrow blasts increased in percentage. A course of low-dose cytosine arabinoside (10 mg/m^2 twice daily) was started but the patient died soon thereafter.

CYTOGENETIC FINDINGS

Bone marrow chromosome analysis was performed on the first day of admission, after a 24-hour culture. Forty-seven mitoses were analyzed and all exhibited a t(1;7). Chromosome identification using R- (RHG), G- (GTG), and C- (CB) banding showed that the translocation was t(1;7)(p11;p11) with monosomy 7q and trisomy 1q (Fig. 1). The constitutional karyotype determined on a culture of stimulated lymphocytes was normal.

DISCUSSION

Our patient had typical features of CMML [6, 7] possibly induced by prolonged treatment with chlorambucil, an alkylating agent. In fact, the term subacute myelomonocytic leukemia would be more appropriate, when the course of the disease is considered.

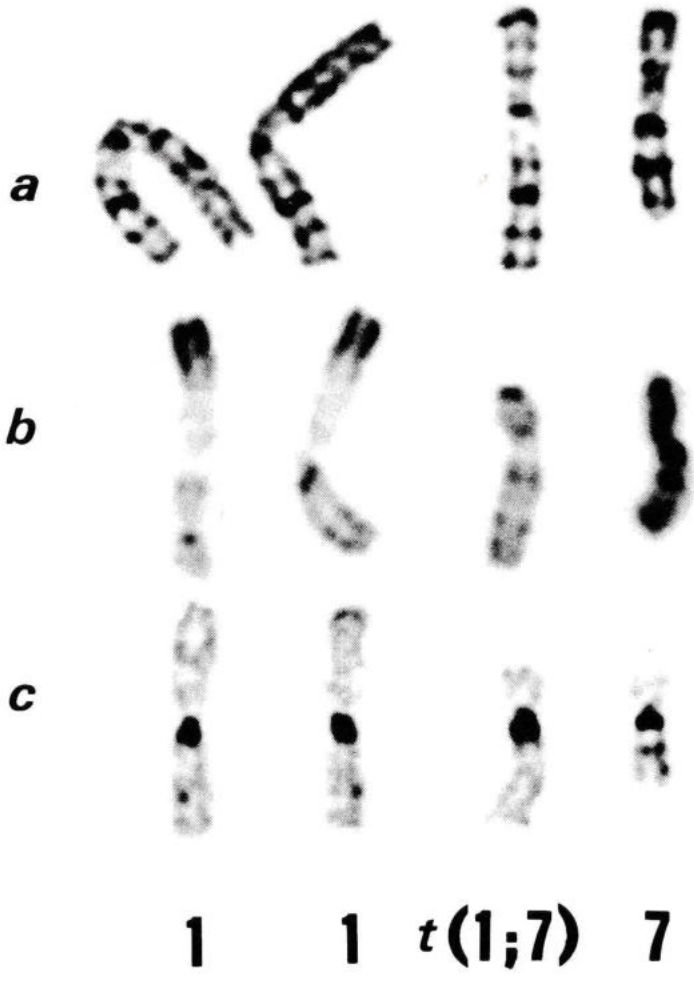

Figure 1 Partial karyotype showing t(1;7(p11;p11). (a) GTG banding; (b) RHG banding; (c) CB banding.

Few cases of secondary CMML have been reported [8–10], although involvement of monocytes in secondary MDS and ANLL has often been noted [11, 12]. Among cases of secondary CMML those with cytogenetic studies did not show t(1;7). This chromosome anomaly was not found in a large series of karyotypes performed on patients with de novo CMML [13].

CMML is now included by most investigators in MDS, particularly by the FAB group [14]. It is not surprising, therefore, to find a t(1;7) in secondary CMML as in other subtypes of secondary MDS. Accordingly, t(1;7) seems to be a marker of the secondary character of MDS, rather than of a particular subtype of MDS.

At the molecular level, a correlation between the breakpoints (1p11 and 7p11) and the location of cellular oncogenes appears to exist: The N-*ras* oncogene is situated in the p11→p13 region of chromosome #1 [15] and c-*erb*-B near the centromeric region of chromosome #7 [16].

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