An Unusual Cause of Eosinophilia in AML-M4 without the Inv(16) Abnormality

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Dear Editor

A 14-year-old male, residing in a filariasis-endemic region of India presented with fever, epistaxis and generalized malaise for 2 weeks. On examination he was toxic with petechiae over trunk and abdomen and gum hypertrophy. His liver and spleen were both palpable 3 cm below costal margins. A bone marrow examination was done and the diagnosis of acute myelomonocytic leukemia, FAB subtype AML-M4 was established on morphology and cytochemistry and confirmed on flow cytometric immunophenotyping (Figure 1). In addition to the neoplasm, occasional microfilaria of Wuchereria bancrofti were also seen (Figure 2). Eosinophilia (9% of all nucleated cells of the bone marrow) was present, albeit sans the abnormal eo-basophils of inversion 16 abnormality (AML-M4-Eo). An accompanying peripheral blood film showed no microfilaria. The peripheral blood absolute eosinophil count (AEC) was 960/microlitre (reference range 50-600/microlitre). A conventional cytogenetic study of the bone marrow showed a normal male karyotype in all 40 metaphases studied. No clinical stigmata of the parasitosis (chyluria, lymphedema, chronic skin changes or lymphadenopathy) were present.

He received AML induction chemotherapy (daunorubicin, 45 mg/m² for 3 days and cytosine arabinoside 100 mg/m² for 7 days) followed by two courses of high dose cytosine arabinoside along

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with supportive care and was in remission at last follow up, 6 months from diagnosis. He also received diethylcarbamazine 2 mg/kg three times a day for 12 days after the first consolidation phase followed by albendazole 400 mg. A subsequent bone marrow specimen (for the assessment of remission status) as well as repeated peripheral blood smears were negative for microfilaria. Normalization of AEC too suggested a cessation of parasitic activity.

Asymptomatic microfilaremia is relatively common in India and the larvae have long been described the bone marrow [1]. The co-existence of parasitic and neoplastic illnesses in our case is purely incidental, although the richly vascular leukemic bone marrow was an optimal site to yield the diagnostic larval forms. Filariasis in association with solid malignancies is well described in literature [2] however; this case is, to the best of our knowledge, the first with concomitant normal cytogenetics acute myelomonocytic leukemia.

In conclusion, this case highlights filariasis as an unusual cause of eosinophilia in acute myelomonocytic leukemia. Diagnostic confusion with M4-Eo may have been created if the cytogenetic study had been unavailable. And finally, the hematopathologist in the tropics must always remain alert to the possibility of unexpected infectious agents lurking in unusual situations.

References