CORRESPONDENCE

ACUTE MEGAKARYOBLASTIC LEUKEMIA IN CHILDHOOD

To the Editor:

The recent paper by Chan et al. (Blood 62:92, 1983), in which one of the two children described with megakaryoblastic leukemia displayed trisomy and quadrasyomy 21, underlines the striking representation of patients with abnormalities of chromosome 21 among the reported cases of young children (<3 yr of age) with megakaryoblastic leukemia.1

Unfortunately, when the authors state that “only the second case reported by Evans and those by Hamazaki and Hillman have features of megakaryoblastic leukemia,” they ignore six probable cases of acute megakaryoblastic leukemia in nonneonatal cases of Down’s syndrome who exhibited increased marrow fibrosis.2,3

Further, platelet peroxidase has been studied in Down’s syndrome associated with megakaryoblastic leukemia. Thus, Bevan et al. did report a positive platelet peroxidase reaction.2 In our case,1 the platelet peroxidase was also performed but was negative, and the blasts were shown to be megakaryoblasts by positivity with the AN51 reagent. In both cases, the blasts showed the typical cytochemical profile of the megakaryoblast. A neonatal case of Down’s syndrome,4 who showed features of megakaryoblastic leukemia, was also studied using the platelet peroxidase reaction, although bone marrow reticulin was not reported on.

There are several other neonatal7–9 cases of Down’s syndrome that have displayed features of megakaryoblastic leukemia, and at least two of these have shown increased bone marrow reticulin.5,10

It has been postulated that children with Down’s syndrome can display prenatally a myeloproliferative process affecting particularly the thrombopoietic system, which can progress into a florid megakaryoblastic leukemia usually within 3 yr of birth.11 This can be associated with cytogenetic evolution.

REFERENCES

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To the Editor:

Our literature search on childhood myelofibrosis, acute (malignant) myelofibrosis, and acute megakaryoblastic leukemia unfortunately failed to show some of the relevant cases cited by Dr. Lewis. We regret these omissions.

We would like to make two comments regarding Down’s syndrome and acute megakaryoblastic leukemia:

1. Our second patient does not have any clinical features of Down’s syndrome, although abnormalities involving chromosome 21 were demonstrated in many of her leukemic cells. Chromosomal abnormalities such as these may be acquired or may represent a congenital trisomy 21 mosaicism affecting hematopoietic cells.1

2. In Down’s syndrome, a leukemic blood picture with a prominent megakaryoblastic component may be observed. This proliferation, however, may have an unusual and unpredictable course. In the case reported by Bevan et al.,2 the megakaryoblasts spontaneously disappeared from the peripheral blood followed by a striking normoblastosis. We have observed a female child with Down’s syndrome and marked megakaryoblastic proliferation who spontaneously went into complete remission. Spontaneous remission of “acute myeloid leukemias” in children with Down’s syndrome is well documented1,3 and appears to apply to megakaryoblastic proliferations as well. Trisomy 21 may predispose to a variety of acute leukemias whose full expression, perhaps, requires additional factors.

REFERENCES


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Acute megakaryoblastic leukemia in childhood [letter]

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