weekly. One month after completion of treatment with the anti-
CD20 and 2 months after thalidomide, he had Hb concentration of
8.6 g/dL, WBC count of 3.6 × 10^9/L, PLT count of 650 × 10^9/L,
and IgM count of 1180 mg/dL. Two months later, he presented to
our department with an Hb concentration of 8.1 g/dL, WBC count of
2.4 × 10^9/L (neutrophils 60%, lymphocytes 31%, monocytes 9%),
and PLT count of 1000 × 10^9/L. Bone marrow smear and trephine
biopsy revealed an hyperplastic marrow with marked dysplasia of
the erythrocyte progenitors, highly increased number of markedly
dysplastic megakaryocytes, left shifted myeloid series with 20%
blasts, and 30% lymphocytic infiltration by small B-lymphoid cells
and lymphoplasmacytes (cIgκ). Immunophenotype of the bone
marrow, by flow cytometry, confirmed the presence of an immature
blast, cell population that was CD13^+, CD34^+, and CD38^+, and a
lymphocytic component that was CD20^+ and CD5^- with κ
light-chain restriction; karyotype was normal. The patient is
currently being given hydroxyurea with a PLT count reduction to
740 × 10^9/L after 2 weeks' administration.

It seems that the administration of thalidomide and the anti-
CD20 monoclonal antibody was ineffective in controlling the
IgM-MGUS problem that subsequently evolved into Waldenstrom
macroglobulinemia; on the other hand, it caused a myeloprolifera-
tive reaction leading to a myeloproliferative disorder not precisely
classifiable, which, however, is currently the major hematologic
problem of this patient. This case is another example of myelopro-
liferative reaction with marked thrombocytosis after thalidomide
administration as reported by Tefferi and Eliott.1 These observa-
tions indicate that, until thalidomide’s mode of action and its
complications are better known, this drug should be used with
great caution.

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To the editor:

Expression of CD10 by human T cells that undergo apoptosis both in vitro and in vivo

We are glad to hear that Drs Bladon and Taylor1 confirmed our data
that CD10 can be found on the surface of T cells induced into
apoptosis, although they used a somewhat different system. As for
the differences reported by Drs Bladon and Taylor, we wish to point
out that there are several variables to be taken into account. First,
the anti-CD10 mAb employed may greatly influence the results
obtained, and the definition of “weak” or “strong” staining varies
depending upon the CD10 mAb used. In addition, we believe that
staining cannot be defined as percentage of weakly or strongly
stained cells because we have better methods of determining
immunofluorescence intensity, such as flow-cytometry profiles, as
we reported.2 With flow cytometry analyses of peripheral blood
from HIV-seropositive individuals, it is possible to segregate
populations of CD10^+CD3^- T cells with various degrees of CD10
positivity that probably represent cells as different stages of the
apoptotic process. These CD10^+ T cells are not necrotic, as
demonstrated by triple-staining tests capable of detecting necrotic
cells. Perhaps in connection with this, we should mention some
of our recent data that show down-regulation of CD10 by
necrotic cells.

Second, the methods used to induce apoptosis both in vivo or in
vitro may influence the mode and quantity of CD10 expression.
This possibility may partly explain some of the differences between
the Bladon and Taylor’s data and ours. We have evidence in favor
of this hypothesis in preliminary studies in which T cells have been
induced into apoptosis by different signals or by the same signal
with different intensity (ie, different concentrations of CD95 mAb).
We believe that CD10 may have a role in apoptosis, as
suggested by the finding that there is considerable CD10 synthesis
upon the induction of apoptosis. But it is difficult to accept that the
only role of molecules newly synthesized during apoptosis is that
of facilitating the clearance of apoptotic cells from circulation.
Apoptosis of T (and also B) cells primarily takes place in the
peripheral lymphoid organs. The environment is likely to partici-
pate in the regulation of apoptosis. Therefore, it is possible that the
newly synthesized molecules may play a role in this regulatory
process. These considerations may open up new avenues of
research on T-cell physiology besides offering the opportunity of
discovering new markers which facilitate the in vivo identifications
of apoptotic T cells.

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Expression of CD10 by human T cells that undergo apoptosis both in vitro and in vivo

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