To the Editor:

In their interesting report published in Blood, Bastard et al described three new recurrent chromosome translocations involving band 3q27 and Ig gene regions. Usually the aberrant clones contained additional chromosome aberrations, but two patients showed only t(3;14)(q27;q32) and t(2;3)(p12;q27), respectively. We found t(2;3)(p12;q27) in 11 of 39 metaphases from an unstimulated lymph node culture of a 30-year-old man (Fig 1). Translocation t(2;3) was the only aberration in nine metaphases. In two metaphases, two copies of the derivative chromosome 2 were seen. In contrast to the non-Hodgkin's lymphomas (NHLs) reported by Bastard et al, all of which were of B-cell phenotype, the histopathologic diagnosis in our case was Hodgkin's disease (HD), mixed cellularity. Typical Reed-Sternberg cells were present. It cannot be ruled out that one part of the lymph node studied histologically was infiltrated by HD whereas
another part of the lymph node studied cytogenetically was infiltrated by malignant B-cell lymphoma. However, it seems probable that this case of HD developed from an activated B lymphocyte. Thus, HD and B-cell lymphomas with t(2;3) may be related disorders. This case of HD developed from an activated B lymphocyte. Thus, HD breakpoints like 14q32 are involved both in HD and in B-cell lymphomas.5-8

Most interesting, our patient was a hemophiliac infected by human immunodeficiency virus (HIV) 3 years before he presented with enlarged lymph nodes. By computed tomography, enlarged paraaortal lymph nodes suspicious of NHL were detected. There was no serologic evidence of Epstein-Barr virus infection. Because of progressive acquired immunodeficiency syndrome no specific treatment was given. The patient died 4 months after the diagnosis of HD was made. Patient 370 in the series of Bastard et al,1 who also had t(2;3) as the aberrant clone’s only abnormality, was immunosuppressed because of kidney transplantation. These findings indicate that in addition to t(8;14) and t(8;22), which are known to be frequent in lymphomas of immunosuppressed patients,5-7 t(2;3) may also be considered to define a type of lymphoma not uncommon under immunosuppression.

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REFERENCES

RESPONSE

Drs Schlegelberger et al raise two interesting issues in relation to 3q27 translocations. The first one is the possible relationship between these translocations and immunosuppression. In our series of 20 patients with non-Hodgkin’s lymphoma (NHL) and 3q27 translocations involving Ig genes,1 apart from patient 370 who had received a kidney transplantation, only one patient (unique patient number [UPN] 240) was considered to be immunosuppressed. This patient was a 34-year-old African woman, with progressive acquired immunodeficiency syndrome (AIDS) related to human immunodeficiency virus 2 (HIV2) infection. She had a complex karyotype with a t(3;14)(q27; q32).

During the period of this study, we examined a 30-year-old homosexual man who had HIV1 infection, Kapoii’s sarcoma, and chronic lymphadenopathy. Cervical lymph node biopsy showed a benign lymphoid hyperplasia but the karyotype disclosed a t(3;22). The complete karyotype was: (12) 46,XY/t(3;22)(q27; q11)/t(2;3)(p12;q27) 47,XY,+12, t(3;22)(q27; q11)/t(2;3)(p12;q27; q11)/(2) 46,XY.der(15)t(1;15)(q21;p11). Eighteen months later, there is no evidence of development of NHL in this patient. This case could be compared with those of Offit et al2 and Takeuchi et al3 in HIV-positive patients who had lymphoid hyperplasia and, respectively, t(2;3) and t(3;22). Thus, as t(8;14) and t(8;22), all three translocations involving 3q27 and Ig genes can be considered to characterize a type of lymphoma or of clonal lymphoid proliferation not uncommon in immunocompromised patients.

The second point is the occurrence of 3q27 translocations in Hodgkin’s disease (HD). We have described the involvement of this region in 7 of 33 patients with HD and an abnormal karyotype.4 t(2;3) and t(3;22) are characteristic and they were not present in these
33 patients, even as a part of a marker chromosome. However, in two of these patients (cases 32 and 41), the aspect of chromosome 3, described as a deletion, could be relevant of a t(3;14). Molecular cloning of the 3q27 breakpoint is in progress and will probably allow to detect these translocations in the near future.

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REFERENCES
t(2;3)(p12;q27) in Hodgkin’s disease of a human immunodeficiency virus- positive patient with hemophilia [letter; comment]

B Schlegelberger, W Grote, HH Wacker and H Bartels