t(9;14)(p13;q32) Denotes a Subset of Low-Grade Non-Hodgkin’s Lymphoma With Plasmacytoid Differentiation


In this series of 426 consecutively ascertained, karyotypically abnormal non-Hodgkin’s lymphomas (NHLs) derived from 407 patients, a t(9;14)(p13;q32) was encountered in 7 cases; an additional case demonstrated t(9;14)(p173;q32). At the time of detection of t(9;14), four cases were small lymphocytic lymphomas with plasmacytoid features; in three of these the t(9;14) was the sole karyotypic abnormality. In two cases of large-cell NHL demonstrating t(9;14), retrospective review of prior lymph node biopsies showed the presence of a small lymphocytic lymphoma of the plasmacytoid subtype. The remaining two cases comprised a large-cell lymphoma of the brain and a follicular NHL. Thus, six of eight cases (75%) had an initial identical low-grade histology. Immunohistochemical analysis of six cases showed no reactivity with CD1, CD2, CD4, CD5, CD8, and CD10 and high reactivity with CD19 and CD20. All four lymphocytic lymphomas and one of the two large-cell NHLs showed cytoplasmic Ig, consistent with plasmacytoid differentiation. Of the eight cases in this series, six presented with or developed stage IV disease; all were characterized by a 6-month to 5-year clinical phase of indolent disease before treatment was instituted. All five patients with low-grade NHL at the time of cytogenetic analysis were alive with recurrent disease at 3-year median follow-up. The remaining three patients with large-cell diffuse histologies relapsed after intensive therapy and expired at a median of 3 years from diagnosis; two of these showed previous or metachronous small lymphocytic tumors. These results suggest a novel biologically distinct subset of NHL; a neoplasm of mature B lymphocytes with plasmacytoid differentiation, characterized by t(9;14); and an indolent presentation followed by gradual clinical progression of disease.

© 1992 by The American Society of Hematology.

THE IDENTIFICATION of recurring reciprocal translocations has led to a description of the molecular mechanisms underlying malignant transformation in non-Hodgkin’s lymphoma (NHL).1,2 The recurring translocations t(14;18)(q32;q21), t(8;14)(q24;q32), t(2;5)(q27;q11), t(11;14)(q13;q32), and t(2;5)(p23;q35) have been observed in greater than 1% of NHLs, and have been correlated with histologic and clinical subsets of this disease.2 Frequent as well as rare recurring translocations have served to identify novel genes that play an important role in growth and differentiation of lymphoid and other cell types.3,4 In this series of 448 karyotypically abnormal NHL, we identified a new recurring translocation, t(9;14)(p13;q32), an abnormality that previously has been reported only in two sporadic cases of NHL.5,6 We observed that the t(9;14)(p13;q32) translocation was highly correlated with small lymphocytic lymphoma of the plasmacytoid subtype, and demonstrated immunohistochemical features consistent with mature B lymphocytes with plasmacytoid differentiation.

MATERIALS AND METHODS

Between January 1984 and December 1990, 693 consecutive specimens of histologically confirmed NHL seen at Memorial Hospital were ascertained for cytogenetic analysis. Biopsy material was split for histopathologic, cytogenetic, and immunophenotypic/immunogenotypic analysis as previously described.7 Chromosome preparations were obtained and cytogenetic analysis was performed as previously described.8 Clonal chromosome changes were defined and karyotypes were described according to the International System for Human Cytogenetic Nomenclature (1991).9 Karyotypic complexity was measured by derivation of the number of marker chromosomes, as previously described.7 For routine histology, B3-fixed, paraffin-embedded (B3PE) sections were stained with hematoxylin and eosin. The lymphomas were classified according to the International Working Formulation.10 Immunohistochemical analysis was performed on 4-μm sections of frozen tissue fixed for 1 minute in acetone/methanol (3:1). A modification of the immunoperoxidase batch screening technique was used as previously described.11 The panel of reagents used comprised monoclonal antibodies (MoAbs) reactive against: CD1, CD2, CD4, CD5, CD10, CD19, CD22, λ (Becton Dickinson, Mountain View, CA), and κ (Dako Corp, Carpinteria, CA). Immunoperoxidase staining of paraffin-embedded tissue and DNA analysis of frozen tissue for detection of clonal rearrangements in Ig and T-cell receptor (TCR) genes for assignment of lineage were performed as described previously.7 Flow cytometric analysis of cell surface markers and DNA/RNA content was performed as described previously.12 Of the cases included in this report, a summary description of selected features of cases 368, 423, 794, 851, and 857 was included in the ascertainment previously reported by us.7

CLINICAL FEATURES

The histologic, clinical, and cytogenetic data pertaining to the eight cases are summarized in Table 1. The median age of the patients (6 females and 2 males) at initial diagnosis of NHL was 60.5 years (range, 37 to 80 years). Median lactate dehydrogenase (LDH) at presentation was 173 (range, 123 to 443). Six patients presented with or developed stage IV disease; in each case, an atypical clinical presentation resulted in an extended pretreatment period before diagnosis of malignant lymphoma. However, case 423 presented with focal neurologic abnormalities that led to a radiographic and surgical diagnosis of a pleomorphic large-cell lymphoma involving the parietal lobe. Bone marrow biopsy was not performed. There was no evidence of lymphomatous involvement outside of the central nervous system. The patient had a prolonged response to combined chemotherapy and radiotherapy.

From the Laboratory of Cancer Genetics, Sloan-Kettering Institute; and the Departments of Pathology (Cytogenetics and Surgical Pathology Services) and Medicine (Lymphoma Service), Memorial Hospital, New York, NY.

Submitted November 8, 1991; accepted July 24, 1992.

Supported by Grants No. CA-34775 and CA-20194 from the National Institutes of Health (Bethesda, MD), the Molin Foundation, and the Lymphoma Foundation.

Address reprint requests to Kenneth Offit, MD, Memorial Sloan-Kettering Cancer Center, Box 192, 1275 York Ave, New York, NY 10021.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. section 1734 solely to indicate this fact.

© 1992 by The American Society of Hematology.

Blood, Vol 80, No 10 (November 15), 1992: pp 2594-2599

2594
were present, but a bone marrow biopsy was nondiagnostic and the chemotherapy was initiated that resulted in partial response.

t(9;14) lymphoma containing mostly large cleaved, noncleaved, as well as plasmacytoid lymphocytes. The cytogenetic evaluation performed at Memorial Hospital showed a t(9;14) and a k(18)(q11.2;q21). The original cytogenetic analysis of this specimen revealed a t(9;14) and a k(18)(q11.2;q21), which was considered consistent with a t(9;14) lymphoma.

Case 994 had a prodrome of fever, weight loss, and serologies consistent with reactivation of infection by Epstein-Barr virus (EBV). The patient presented with pericardial and retroperitoneal adenopathy, which was stable for 2 years when new hilar and abdominal adenopathy appeared. Bone marrow as well as lymph node biopsies showed areas of fibrosis and infiltration by small lymphocytic lymphoma with plasmacytoid differentiation. Cytogenetic analysis of the lymph node showed a t(9;14). The original cytogenetic evaluation performed at Memorial Hospital showed a t(9;14) and a k(18)(q11.2;q21), which was considered consistent with a t(9;14) lymphoma.

Case 851 was diagnosed with small lymphocytic lymphoma of the plasmacytoid subtype noted as an incidental finding during large bowel resection for Dukes stage B rectal carcinoma. This specimen showed a population of plasmacytoid lymphocytes that was read by lymphocytes of various sizes, including small cells with clefted nuclei as well as large cells with multiple nucleoli. The patient died from recurrent disease 2 years after lymphoma diagnosis, without chemotherapeutic intervention.

Case 1052 presented with splenomegaly and increased atypical lymphocytes in the bone marrow and peripheral blood, which were characterized by an irregular shape, abundant cytoplasm, and nuclei containing occasional nucleoli (Fig 1). No therapy was administered for 4 years, at which time a biopsy of retroperitoneal lymphoma containing mostly large cleaved, noncleaved, as well as plasmacytoid lymphocytes. The cytogenetic evaluation performed at Memorial Hospital showed a t(9;14) and a k(18)(q11.2;q21), which was considered consistent with a t(9;14) lymphoma.

Case 994 initially presented elsewhere with symptoms of a non-A, non-B hepatitis. Percutaneous needle biopsy of the liver showed an infiltration with atypical lymphocytes and a repeat biopsy was nondiagnostic. Biopsy of an inguinal lymph node at this time also was interpreted as nondiagnostic; however, retrospective review of slides showed an infiltration by small lymphocytes with plasmacytoid features. Frozen tissue was not available for DNA analysis of this specimen. A bone marrow biopsy and radiographic examination of the abdomen were negative. Approximately 1 year later, the patient developed axillary adenopathy; biopsy and cytogenetic evaluation performed at Memorial Hospital showed a t(9;14). The original liver biopsy slides, upon rereview, showed pleomorphic large-cell lymphoma cells. Despite intensive combination chemotherapy, relapse occurred in bone marrow, which demonstrated infiltration by lymphocytes of various sizes, including small cells with clefted nuclei as well as large cells with multiple nucleoli. The patient died after an additional 6 months of therapy.

Case 1052 presented with splenomegaly and increased atypical lymphocytes in the bone marrow and peripheral blood, which were characterized by an irregular shape, abundant cytoplasm, and nuclei containing occasional nucleoli (Fig 1). No therapy was required for 6 months until increasing peripheral counts and anemia developed. A splenectomy showed a small lymphocytic lymphoma, which was considered consistent with a t(9;14) lymphoma.

Case 368 presented with asymptomatic adenopathy. A biopsy showed areas of follicular hyperplasia as well as portions of the salivary gland infiltrated by small lymphoplasmatoid cells with amphiologic cytoplasm. Peritoneal adenopathy was noted 6 months later; a biopsy of the mass showed a pleomorphic large-cell lymphoma of the plasmacytoid subtype. No further treatment was administered for 4 years, at which time a biopsy of retroperitoneal lymphoma containing mostly large cleaved, noncleaved, as well as plasmacytoid lymphocytes. The cytogenetic evaluation performed at Memorial Hospital showed a t(9;14) and a k(18)(q11.2;q21), which was considered consistent with a t(9;14) lymphoma.

Case 994 had a prodrome of fever, weight loss, and serologies consistent with reactivation of infection by Epstein-Barr virus (EBV). The patient presented with pericardial and retroperitoneal adenopathy, which was stable for 2 years when new hilar and abdominal adenopathy appeared. Bone marrow as well as lymph node biopsies showed areas of fibrosis and infiltration by small lymphocytic lymphoma with plasmacytoid differentiation. Cytogenetic analysis of the lymph node showed a t(9;14). An extended partial remission was obtained after combination chemotherapy.

Table 1. Clinical, Histologic, Immunologic, and Karyotypic Data on Patients With t(9;14)(p13;q32)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Site of Biopsy</th>
<th>Immuno- type</th>
<th>Treatment Status</th>
<th>Treatment</th>
<th>Pathology</th>
<th>Age/ Sex</th>
<th>LDH/ Stage</th>
<th>Survival (months from diagnosis)</th>
<th>Full Karyotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>368 LN</td>
<td>B Post</td>
<td>LNCC-D*</td>
<td>MACOPB</td>
<td>Cyt, Pred</td>
<td>73/F</td>
<td>179/IV</td>
<td>34e,r</td>
<td>87-90,XXX,add(1)(p32),dup(7)(q11q32)(t9;14) (p13;q32),t(14;18)(q32;q21),der(18)(t14;18)(q32; q21)x2,inc[4]/46,XX[16]</td>
<td></td>
</tr>
<tr>
<td>423 Brain</td>
<td>B Post</td>
<td>LNCC-D</td>
<td>MTX</td>
<td>Ara-C/RT</td>
<td>51/M</td>
<td>141/IE</td>
<td>43e</td>
<td>48,XX,-Y,+B;19(13q;32),t(12;22)(p13;q11), t(9;14),del[4]+mar[4]/46,XY[16]</td>
<td></td>
</tr>
<tr>
<td>851 LN</td>
<td>B Pre</td>
<td>SM-LYM (pl)</td>
<td>M2</td>
<td>69/F</td>
<td>123/IV</td>
<td>32+</td>
<td>32+</td>
<td>46,XX,t(9;14)(p13;q32)[6]/46,XX[1]</td>
<td></td>
</tr>
<tr>
<td>857 LN</td>
<td>B Pre</td>
<td>Mx-F</td>
<td>IFOSF</td>
<td>37/F</td>
<td>443/III</td>
<td>27+</td>
<td>27+</td>
<td>46,XX,add(12)(q24),del(14)(t9;14)(p13;q32)[6]/46,XX[1]</td>
<td></td>
</tr>
<tr>
<td>994 LN</td>
<td>B Post</td>
<td>LNCC-D†</td>
<td>ISOSF</td>
<td>73/F</td>
<td>173/IV</td>
<td>38e</td>
<td>38-70,XX,inv(1)(p1q25),del(6)(q21),del(7)(q12),q(19),q(21),q(22),q(23),q(24),q(25), 46,XX[X]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1052 LN</td>
<td>B Post</td>
<td>SM-LYM (pl)†</td>
<td>Chlor</td>
<td>51/F</td>
<td>203/IV</td>
<td>79+</td>
<td>79+</td>
<td>46,XX,t(9;14)(p13;q32)[6]</td>
<td></td>
</tr>
<tr>
<td>1113 Spleen</td>
<td>B Post</td>
<td>SM-LYM (pl)†</td>
<td>CVP</td>
<td>80/F</td>
<td>173/IV</td>
<td>27+</td>
<td>27+</td>
<td>47,XX,del(1)(q23q32),del(3)[p21],t(9;14)(q13;q32),del(10)(q24q26),dup(17)(q22q24)[17]/46,XX[3]</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: LN, lymph node; LNCC-D, large noncleaved cell diffuse; Mx-F, mixed follicular; pl, plasmacytoid; Pre, pretreatment; Post, cytogenetics sampled posttreatment; CMOPP, cyclophosphamide, vincristine, procarbazine, prednisone; CHOP, cyclophosphamide, daunorubicin, vincristine, prednisone; IP, ifosfamide, MTX, methotrexate; Ara-C, cytosine arabinoside; RT, radiation therapy; CVP, cyclophosphamide, vincristine, prednisone; M2, cyclophosphamide, vincristine, CNU, melphalan, prednisone; MACOPB, methotrexate, daunorubicin, cyclophosphamide, vincristine, prednisone, bleomycin; PRMOPP = same drugs as CHOP plus etoside, nitrogen mustard, vincristine, prednisone, procarbazine; +, alive at last follow-up; r, relapse; e, expired.

*Prior biopsy retrospectively reviewed demonstrated small lymphocytic lymphoma, plasmacytoid subtype (see text).

†Bone marrow at time of progression showed mixture of small cleaved and large lymphoid cells.

From www.bloodjournal.org by guest on April 13, 2017. For personal use only.
Fig 1. Circulating abnormal lymphocytes in a patient with t(9;14)(p13;q32). A polymorphonuclear leukocyte is at the bottom of the field. The pattern of nuclear chromatin and the abundance of the cytoplasm are consistent with plasmacytoid differentiation; however, the size and irregular shape of the cells differed from that of the smaller plasmacytoid cells seen in the formalin-fixed sections (Fig 2). Such morphologic variations most likely result from the greater cohesion of cells with plasmacytoid differentiation.

typical of the others, are illustrated in Fig 2. Cytogenetic analysis of the lymph node, as well as of the circulating abnormal lymphocytes, showed a t(9;14). After 9 months of alkylating agent chemotherapy, a repeat bone marrow biopsy and aspirate showed a mixture of large and small malignant lymphocytes. Repeat cytogenetic evaluation was performed on poorly banded preparations, although the t(9;14) clone was confirmed. Radiographic evaluation showed enlarged abdominal adenopathy that improved with combination chemotherapy.

Case 1113 presented with anemia and a bone marrow demonstrating 54% immature small to intermediate-size lymphocytes with abundant cytoplasm. Although peripheral adenopathy was absent, computer tomography (CT) showed retroperitoneal adenopathy. The patient was asymptomatic on no therapy for 7 months. Worsening anemia necessitated a splenectomy that showed a small lymphocytic lymphoma of the plasmacytoid subtype. Cytogenetic analysis of this specimen demonstrated a t(9;14). After 1 additional year off therapy, recurrent anemia and bone marrow infiltration by lymphoma necessitated treatment with alkylating agent chemotherapy.

RESULTS

Cytogenetic analysis. In this series of 693 specimens on which karyotypic analysis was attempted, 426 specimens derived from 407 patients were karyotypically abnormal. Among these, seven cases exhibited t(9;14)(p13;q32), while in one, because of poor banding quality, the karyotype could be designated only as t(9;14)(p13?;q32) (Table 1).

In three cases (794, 851, and 1052), all of which were ascertained before treatment and with identical histology, the t(9;14)(p13;q32) translocation was the sole karyotypic abnormality. Six of the eight cases had both reciprocal partners, while in two (794 and 857) only the der(14)t(9; 14)(p13;q32) chromosome was noted. Partial karyotypes of
three cases (423, 1052, and 1113) illustrating the t(9;14)(p13; q32) are shown in Fig 3. The karyotypes of the three cases sampled posttreatment (368, 994, and 1113) showed multiple aberrations in addition to t(9;14), including structural abnormalities of chromosomes 1 and 7, and duplication of 17q. As expected, karyotypic complexity was greater in the posttreatment samples; the mean number of marker chromosomes was 3.0 in the five pretreatment samples, compared with 7.7 in posttreatment samples (P = .005). There was no correlation between karyotypic complexity and histologic grade. Only case 368 showed evidence of another translocation affecting 14q32, namely, a t(14;18)(q32;q21) (Table 1).

**Immunohistochemical, DNA, and flow cytometric analyses.** Immunohistochemical analysis was performed on tissues frozen at the time of cytogenetic analysis in cases 794, 851, 857, 994, 1052, and 1113, whereas in cases 368 and 423 analysis was performed only on flow cytometric analysis of cell suspensions. The frozen specimens showed strong positive reactivity by the tumor cells to antibodies against CD19 and CD22, and negative reactivity to antibodies against CD2, CD4, CD5, CD8, and CD10. Cases 851 and 1113 demonstrated Ig restricted to κ chain type in the cytoplasm and at the cell membrane, whereas case 857 demonstrated κ reactivity only at the cell membrane. Flow cytometric analysis of cell suspensions derived from cases 368 and 423 also demonstrated κ light chain restiction, although this technique of analysis precluded assessment of the cellular localization of the Ig. Cases 794, 994, and 1053 demonstrated Ig restricted to λ type in the cytoplasm and at the cell membrane. DNA analysis of cases 423, 794, 851, 994, 1052, and 1113, using a JH probe, confirmed clonal rearrangement of the IgH gene, whereas case 857 showed only germine IgH (data not shown). DNA was unavailable for analysis for case 368. None of six cases studied demonstrated a monoclonal paraprotein in the serum. Flow cytometric analysis of bone marrows involved by lymphoma in three cases (851, 1052, and 1113) showed low S phase and total RNA content. In each of these, the light chain type of the circulating cells was identical to that determined by immunohistochemical analysis of lymph node biopsies.

**DISCUSSION**

In this study, we identify a new recurring translocation in NHL, t(9;14)(p13;q32), that shows strong association with low-grade plasmacytoid differentiation. Previously, associations have been established between t(8;14)(q24;q32), t(14; 18)(q32;q21), t(11;14)(q13;q32), and t(3;22)(q7;q11) and Burkitt’s, follicular, “intermediate differentiation,” and diffuse intermediate-grade NHL, respectively.2 However, these associations, as in the case of t(9;14)(p13;q32) reported here, have not been absolute.2 Thus, in one series, fully 73% of t(8;14) cases occurred in a variety of histologies other than Burkitt’s lymphoma, and 38% of t(14;18) occurred in nonfollicular lymphomas.7

Small lymphocytic lymphomas, many of which are morphologically indistinguishable from chronic lymphocytic leukemia (CLL), comprise 5% to 10% of NHLs.13 These lymphomas have been associated with t(11;14), trisomy 12, and trisomy 3, although the majority of these low-grade tumors do not share a recognized cytogenetic abnormality.2 A subset of small lymphocytic tumors with plasmacytoid features (SM-LYM [pl]) have been referred to by the Kiel classification as “LP immunocytomas.”14 These tumors comprise 5% of NHLs and have an indolent clinical course.13 The cytogenetic features of SM-LYM (pl) have not previously been described.

In this series, the t(9;14)(p13;q32) was identified at tissue diagnosis of SM-LYM (pl) in four of the eight cases; in three of these, the t(9;14) was the sole cytogenetic abnormality. In two additional cases, SM-LYM (pl) was documented on retrospective review of prior pathologic material of large-cell lymphomas that showed the t(9;14) translocation at presentation. We suggest that the t(9;14) was, in fact, present in these two tumors at the low-grade stage and was clonally retained during progression to the higher grade. In support of this was the clonal retention of t(9;14) during histologic progression in case 1052, in which the translocation was observed in both the low-grade SM-LYM (pl) as well as in the higher-grade diffuse mixed tumor detected in the marrow. Furthermore, clonal retention of t(14;18) during histologic progression of low-grade follicular tumors to high-grade diffuse tumors has been well documented.3,5 These data indicate thus that in six of the eight NHLs in this series, the t(9;14) translocation was associated with SM-LYM (pl). However, the possibility that the t(9;14) may have been acquired in association with transformation in a subset of cases, although unlikely, cannot be formally ruled out.

Similar to other low-grade to intermediate-grade NHLs, the t(9;14) cases presented in the sixth to seventh decade
and demonstrated a tendency for stage IV disease despite a low tumor bulk as measured by serum LDH. These characteristics are consistent with the predominant histology of SM-LYM NHL. The pretreatment phase of 6 months to 5 years in seven of the eight cases is consistent with the slow natural history of "LP immunocytoma," and the tendency for clinical as well as histologic progression is also characteristic of other low-grade NHLs. All three cases demonstrating diffuse large-cell lymphoma at initial presentation or progression succumbed to disease despite intensive combination chemotherapy and favorable prognostic factors (low serum LDH and low bulk of disease). One of these tumors, however, arose in the central nervous system, a known indicator of poor prognosis. The five patients with low-grade lymphoma at the time of cytogenetic analysis were alive with recurrent or residual disease at a median follow-up of 3 years from diagnosis of lymphoma. Thus, the t(9;14) low-grade to intermediate-grade NHL appears to share a prolonged survival with disease characteristic of t(14;18)- and t(11;14)-bearing tumors.2,15

Immunologic and IgH gene rearrangement studies of t(9;14) SM-LYM (pl) NHLs suggest their differentiation state as mature B cells. The detection of cytoplasmic Ig in five cases studied, including a large-cell lymphoma with documented transformation from a prior SM-LYM (pl) NHL, is consistent with the plasmacytoid features of these cases. The absence of reactivity with CD5 further distinguishes these cases from CLL and intermediate lymphoma.15

Case 857 was unusual in that it represented the only follicular tumor in the series. It lacked a t(14;18) and cytoplasmic Ig and developed in the setting of an autoimmune disease. The inability to detect clonal IgH gene rearrangement in the setting of light chain clonal restriction most likely represents a low incidence of lymphoma-derived DNA in the tissue analyzed.

A t(9;14)(p11;q32) has previously been reported in a case of T-cell leukemia/lymphoma4 and a case of α heavy chain disease/CLL.6,17 In addition, a t(9;14)(p13;q32) has been observed in a cell line derived from a patient with a Ki-1-positive large-cell lymphoma.5 Preliminary molecular characterization of two cases demonstrated junction fragments containing DNA sequences derived from the Ig heavy chain and chromosome 9.5,17

Only one of the eight cases of t(9;14) in this series (368) presented with another translocation involving the IgH gene, namely a t(14;18). The greater than 1% incidence of the t(9;14)(p13;q32) in NHL, its correlation with the histologic subset of small lymphocytic plasmacytoid NHL, and its occurrence as the sole karyotypic aberration in three tumors suggest that this translocation denotes an important clinical and histologic subset of NHL. The molecular characterization of a functional gene involved in this translocation will be necessary to establish the pathogenetic role of t(9;14)(p13;q32) in this subset of NHL tumors.

The inability of previous series to document and associated this translocation with a subset of low-grade NHLs may be attributed to the smaller sample sizes of the prior reports. Large single institution ascertainment studies, which use uniform cytogenetic and pathologic criteria, have continued to detect biologically and clinically important new recurring translocations in NHL.18 Such series are especially important in resolving cytogenetic subsets of low-grade NHL, in which up to 35% of cases may not exhibit the t(14;18) translocation.7,19

ACKNOWLEDGMENT

We thank Dr Philip H. Lieberman for his contributions toward the pathologic characterization of the tumors and Dr Carol Portlock for her comments on the manuscript. We thank Drs David Straus, James P. O'Brien, and Gwen Nichols for providing clinical data on the cases reported. We thank Suvas Desai for help with DNA analysis, Mary Ann Gangi for expert technical assistance with immunohistochemical staining, and Amelia Panico for help with illustrations.

NOTE ADDED IN PROOF

Subsequent to submission of this manuscript, we ascertained a new case of t(9;14)(p13;q32) (case no. 1178). A 56-year-old woman presented with splenomegaly and thrombocytopenia. Splenectomy showed small lymphocytic lymphoma with plasmacytoid differentiation. Flow cytometric analysis demonstrated α light chain clonal restriction, high reactivity with CD19, and low reactivity with CD5. The patient remains untreated 1 year after diagnosis.

REFERENCES

4. Neri A, Chang CC, Lombardi L, Salina M, Corradi P, Maiolo AT, Chaganti RSK, Koziner B, Clarkson BD, Lieberman PH, Chaganti RSK, Portlock for her comments on the manuscript. We thank Drs David Straus, James P. O'Brien, and Gwen Nichols for providing clinical data on the cases reported. We thank Suvas Desai for help with DNA analysis, Mary Ann Gangi for expert technical assistance with immunohistochemical staining, and Amelia Panico for help with illustrations.
10. Non-Hodgkin's Lymphoma Pathologic Classification Project:


15. Raffeld M, Jaffe ES: bcl-1, t(11;14), and mantle cell derived lymphomas. Blood 78:259, 1991


t(9;14)(p13;q32) denotes a subset of low-grade non-Hodgkin's lymphoma with plasmacytoid differentiation

K Offit, NZ Parsa, D Filippa, SC Jhanwar and RS Chaganti