Splenic marginal zone lymphoma with t(8;14)(q24.1;q32)/MYC rearrangement

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Introduction

Splenic marginal zone lymphoma (SMZL) is a distinctive low-grade B-cell lymphoma that was first termed by Schmid and colleagues [1] in 1992. The 2008 World Health Organization (WHO) classification of tumors of haematopoietic and lymphoid tissues defines SMZL as a “B-cell neoplasm comprising of small lymphocytes that surround and replace the splenic white pulp germinal centers, efface the follicle mantle, and merge with a peripheral zone of larger cells, including scattered transformed blasts; both small and larger cells infiltrate the red pulp” [2]. According to the WHO, this rare low-grade B-cell lymphoma is considered to be a distinct clinical and pathological entity. SMZL is characterized by massive splenomegaly with very frequent involvement of the bone marrow and peripheral blood [2]. The splenic hilar lymph nodes and bone marrow are often involved, and the lymphoma cells are often found in the peripheral blood as villous lymphocytes [2]. Therefore, the terminology “splenic lymphoma with circulating villous lymphocytes” has been used in the past literature [3].

SMZL is a diagnosis of exclusion, given that the spleen may present with marginal-zone hyperplasia or, most importantly, be secondarily involved by a malignant small B-cell lymphoma such as follicular lymphoma, mantle cell lymphoma, hairy cell leukemia, or lymphoplasmacytic lymphoma [2, 4]. Therefore, these mimickers can make the diagnosis of SMZL challenging. SMZL cells consistently display, albeit

not specifically, the phenotypic profile of pan-B antigens (CD20, CD79a, and PAX-5)⁺/BCL-2⁺/CD5[−]/CD10[−]/CD23[−]/CD43[−]/CD103[−]/BCL-6[−]/cyclin D1[−]/surface IgM⁺/surface IgD^{+/−}. This profile helps to distinguish SMZL from other small B-cell lymphomas secondarily involving the spleen.

In addition, approximately 70% to 80% of SMZL cases have been reported to exhibit cytogenetic and/or molecular genetic abnormalities. However, no consistent or unique genetic alteration for SMZL has been detected. The most frequent cytogenetic abnormalities reported are deletions at the 7q31–32 band [5]. The potential diagnostic value of this frequent deletion in SMZL is unknown [6]. Other cytogenetic abnormalities involve chromosomes 1, 3, 6, 8, 12, and 14 [7]. Since SMZL is biologically a heterogeneous disease, it has a variable clinical course with 13%–30% of reported cases being aggressive [6, 8]. Therefore, it has been stated that there is a need for prognostic indicators in determining the patient's clinical outcome [6].

We present the first case report of a de novo SMZL with t(8;14)(q24;q32) as the only cytogenetic aberration by standard cytogenetic analysis. We used fluorescence in situ hybridization (FISH) to confirm the MYC gene rearrangement and array comparative genomic hybridization (cGH) to confirm no other genomic alterations. The c-MYC gene, located at 8q24, is involved in three translocations, most commonly t(8;14)(q24;q32), less often t(8;22)(q24;q11), and infrequently t(2;8)(p12;q24). This gene results in the overexpression of c-MYC, which induces cell proliferation by up-regulating other genes involved in cell growth. The MYC gene (8q24) rearrangement with immunoglobulin heavy chain [t(8;14)] is most commonly associated with Burkitt lymphoma (BL) [9]. A secondary c-MYC gene rearrangement has been recognized occasionally in the transformation of follicular lymphoma with t(14;18)(q32;q21) to diffuse large B-cell lymphoma (DLBCL), BL, and various subtypes of acute lymphoblastic leukemia. This

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case report shows that this particular translocation can also present in SMZL. The current case exhibits typical clinical, morphologic, and immunophenotypic features of SMZL. Therefore, the t(8;14) translocation revealed in this SMZL may be representative of a progressing form of disease.

Case report

The patient is a 42-year-old woman with a history of chronic microcytic anemia who presented with left upper-quadrant abdominal pain for 10 days. The pain was not associated with fever, chills, or weight loss. An ultrasound and computed tomography of the abdomen revealed a markedly enlarged spleen (Fig. 1A). No other organomegaly or systemic lymphadenopathy was noted. The serology studies for hepatitis B virus, hepatitis C virus (HCV), and human immunodeficiency virus (HIV) were all negative. A serum protein electrophoresis showed no evidence of a monoclonal protein.

The peripheral blood showed a mild atypical lymphocytosis with a white blood cell count of $19.0 \times 10^9/L$ and a differential consisting of an increased absolute neutrophil count of 15.20 K/mm^3 , normal absolute lymphocyte count of 1.3 K/mm^3 , increased absolute monocyte count of 0.8 K/mm^3 , and an increased absolute “other” cell count of 1.7 K/mm^3 . The “other” cells consisted of atypical lymphocytes that were medium sized with round eccentric nuclei, dense chromatin, inconspicuous nucleoli, and a moderate amount of cytoplasm with rare polar villi (Fig. 1B). The peripheral blood also showed a microcytic anemia with a decreased red cell count of 3.04 M/mm^3 , hemoglobin of 7.1 g/dL , hematocrit of

21.8% , and a mean corpuscular volume (MCV) of $71.7 \mu\text{m}^3$. The platelets were decreased at 90 K/mm^3 .

The bone marrow biopsy consisted of atypical lymphoid infiltrates in a nodular and interstitial pattern (Fig. 1C). No intrasinusoidal pattern was identified. The lymphocytes in the marrow were predominantly small- to medium-sized mature forms with round eccentric nuclei, dense chromatin, and a moderate amount of cytoplasm. Reticulin fibrosis was focally increased within the lymphoid aggregates. Immunophenotyping by flow cytometry on peripheral blood and bone marrow revealed a lambda light-chain restricted monotypic B-cell population. They were CD19^+ , CD20^+ , cCD22^+ , cCD79a^+ , CD38^+ , FMC-7^+ , CD5^- , CD10^- , CD11c^- , CD23^- , CD25^- , and CD103^- , consistent with a marginal zone lymphoma. Cytogenetics of the bone marrow revealed solely t(8;14)(q24.1;q32). In order to further characterize the B-cell lymphoproliferative disorder, a laparoscopic splenectomy was performed.

The enlarged spleen weighed 987 g. Gross examination of the spleen revealed numerous small, round, white pulp nodules measuring approximately $1.0 \times 1.0 \text{ mm}$ throughout the specimen (Fig. 1D). Multiple slightly enlarged lymph nodes were detected. The hematoxylin and eosin (H&E)-stained sections of the spleen showed prominent nodular atypical lymphoid proliferation in the white pulp with expansion into the red pulp (Fig. 2A, B). On high magnification, the white pulp nodules were almost exclusively composed of medium-sized lymphocytes with eccentric round nuclei and a moderate amount of pale cytoplasm, rendering monocytoïd or plasmacytoïd appearances (Fig. 2C). Rare larger transformed blasts were present. Mitotic figures were rare (0–1/50 high-power fields). No

Fig. 1 (A) Computed tomographic scan of abdomen showing massive splenomegaly. (B) Atypical lymphocytes with polar villi (villous lymphocytes) in the peripheral blood. (C) Nodular and interstitial atypical lymphoid infiltrate in the bone marrow. (D) Gross photograph of a section of the spleen showing numerous expanded white pulp nodules (B: Giemsa stain, original magnification $\times 1,000$; C: H&E stain, original magnification $\times 200$)

Fig. 2 (A) Low-magnification examination of the spleen showing a micronodular homogeneous pattern. The prominent white pulp nodules infiltrate into the red pulp sinuses. (B) High-magnification examination of spleen showing monophasic marginal zone-like white pulp nodules. (C) High-magnification showing medium-sized lymphocytes with round nuclei and pale cytoplasm intermingled with rare larger transformed blast cells. (D) Splenic hilar lymph node showing a micronodular infiltrate by splenic marginal zone lymphoma (H&E, original magnifications $\times 200$ [A], $\times 100$ [B and D], and $\times 400$ [C])

areas of high-grade transformation or areas resembling DLBCL were seen upon thorough histological examination. The splenic hilar lymph nodes were positive for lymphoma, showing micronodular infiltrates of SMZL cells (Fig. 2D).

Immunohistochemistry of the spleen demonstrated that atypical nodules were diffusely positive for B-cell markers (CD20 and CD79a) with coexpression of BCL-2. The tumor cells were negative for BCL-6, CD3, CD5, CD10, CD30, CD43, and cyclin D1 (Fig. 3). Morphologically, the

tumor cells showed uniformly low-grade cytological features and a low cell proliferation index (Ki-67 $<10\%$). The immunostains for p53 and p16 were negative. No evidence of Epstein–Barr virus infection was detected by Epstein–Barr encoded RNA in situ hybridization. Overall, the combined clinical, morphologic, and immunophenotypic features were classical for the diagnosis of SMZL.

Molecular analysis of the splenic tissue included classical cytogenetics, interphase FISH, and array cGH.

Fig. 3 By immunohistochemistry, the white pulp nodules are diffusely positive for B-cell marker (CD20) with aberrant coexpression of Bcl-2 but are negative for CD3, CD5, and CD10. The lymphoma cells show low cell proliferation index of Ki-67 ($<10\%$) (immunohistochemistry with hematoxylin counterstain, original magnification $\times 200$)

The standard karyotyping analysis of the bone marrow and spleen demonstrated an identical finding with t(8;14)(q24.1;q32) as the only cytogenetic aberration (Fig. 4). FISH confirmed the MYC (8q24.1) and IgH (14q32) gene rearrangement (Fig. 4). Array cGH confirmed the cytogenetic results. There were no other genomic alterations including duplication or deletions events at the translocation break points at 8q24 and 14q32. No other genomic alterations were identified (46,XY arr cgh) confirming that the translocation was the primary alteration, albeit there could be smaller alterations that were not detected (< 9 KB).

Up to 6 months postsplenectomy, the patient did not receive any further medical treatment and remained free of disease. On physical examination, no organomegaly or lymphadenopathy was identified. Her complete blood count normalized, except for the persistent microcytic anemia. No atypical lymphocytes were detected in the peripheral blood smear. The repeat bone marrow biopsy showed adequate trilineage hematopoiesis. No atypical lymphoid aggregate was detected.

Materials and methods

The H&E and Wright–Giemsa-stained slides were reviewed. Immunohistochemical studies were performed on formalin-fixed, paraffin-embedded tissue using an automated immunostainer (Ventana Medical Systems, Tucson, AZ) with antibodies to CD20, CD79a, CD3, CD5, CD43, CD10, BCL-2, BCL-6, Ki-67, and cyclin D1 (Ventana Medical Systems) and p16, p53, and CD138 (DAKO, Carpinteria, CA) according to the manufacturer's instructions. Epstein–Barr encoded RNA in situ hybridization was performed at a reference laboratory (US Labs, Irvine, CA). Flow cytometry studies were performed as part of the clinical workup of the cases. The medical records were reviewed for clinical presentation, staging, laboratory data, radiological studies, treatment, and outcome.

Conventional chromosome analysis of the splenic lymphoma tissue was performed by the standard cytogenetics protocols for neoplastic studies. Eight cells were available for analysis, and the karyotype was 46,XX,t(8;14)

Fig. 4 (A) Conventional cytogenetic analysis performed on G-banded metaphases demonstrates 46, XY, t(8;14)(q24.1;q32). (B) Interphase FISH revealed one fusion (intact MYC), one red (*c-myc* centromeric probe), and one green (*c-myc* telomeric probe) signals confirming the rearrangement of MYC

(q24.1;q32)[7],46,XX[1]. Interphase FISH analysis was performed on cell suspensions or paraffin blocks using a commercially available dual-color break-apart FISH probe set for *c-MYC* gene rearrangement (Vysis, Downers Grove, IL). Two hundred interphase nuclei were examined for the probe and revealed a rearrangement of the *MYC* gene in 58% of the nuclei. Based on the performance characteristics established on this assay, the specimen was considered positive for *MYC* gene rearrangement.

Array cGH was performed utilizing the Agilent Human Genome CGH 244 K (p/n G4411B) array. This array has 8.9 KB overall median probe spacing (7.4 KB in Refseq genes). Genomic DNA was extracted from H&E-stained slides with 10-μm of splenic lymphoma tissue sections. Reference genomic DNA was collected from normal male (46, XY). Two micrograms of genomic DNA was chemically labeled with Universal Linkage System™ (ULS™) Cy5- or Cy3- dyes (Agilent's Oligo aCGH Hybridization Kit). Hybridization was carried out for 40 h at 65°C. Slides were scanned on an Agilent 2565BA microarray scanner. The images were analyzed using Feature Extraction software (version 9.5; Agilent Technologies, Santa Clara, CA).

Discussion

Splenic marginal zone lymphoma (SMZL) is a rare low-grade B-cell lymphoma considered to be a distinct clinical and pathological entity according to the WHO classification. SMZL is characterized by massive splenomegaly with very frequent involvement of the bone marrow and peripheral blood. The pathogenetic mechanism underlying the development of SMZL remains unexplained. Immuno-

globulin variable heavy-chain (IgVH) mutations have been described and are consistent with their postfollicular marginal zone B-cell origin, whereas the occurrence of ongoing mutations could be acquired from the surrounding splenic environment [10]. SMZL has been suggested to be clinically, biologically [3], and molecularly heterogeneous [11]. This supports the hypothesis that besides the proportion of cases derived from marginal zone cells showing BCL-6 hypermutation, another significant proportion of cases could originate from an unmutated naive precursor that is possibly located in the mantle zone [3].

Analysis of the mutation status in IgVH genes in SMZL reveals a distinct and biased use of selected VH1 genes (VH1.2 segment) [10]. This suggests the role of antigen stimulation in the initiation and stimulation of the disease process. Interestingly, HCV infection is more common in SMZL, and the persistent HCV antigenic stimulus might be involved in the lymphomagenesis process [3]. So far, no pathognomonic genetic alteration characterizes SMZL. However, about 40% of cases have been reported to have allelic loss in the region of 7q21 to 7q36 [5]. Watkins et al. [6] reported that the most recurrent genomic gains are 3q, 5, 8q, 9q, 12q, and 18 and that the most recurrent losses are 7q, 6q, 8p and 17p. In a series of 330 SMZL cases, the most frequently involved chromosomes in order of frequency were 7, 3, 1, 8, 6, 12, and 14 [7]. By cDNA microarray analysis, a gene expression signature was identified, comprising three genes (namely, ILF1, SENATAXIN, and CD40) whose expression may distinguish SMZL from other small B-cell lymphomas [12].

To the best of our knowledge, the sole cytogenetic t(8;14)(q24.1;q32) translocation in SMZL has not previously been reported. The significance of this finding is currently

Table 1 Current literature review of SMZL genetics

	Transformation	Aggressive/worse prognosis
<i>Most frequent</i>		
Deletion of 7q 31–32	1q32 alterations	Deletion 17p
Gains of 3 or 3q	p53 mutation/overexpression	Deletion 14q
Gains of 13q11	t(8;14)/MYC–IgH?	Concurrent deletions 8p and 17p
Deletion of 8p and gains of 8q		Absence/lack of IgVH gene somatic mutation
Structural abnormality of 1q		p53 mutation/overexpression/deletion
Gains of 5, 9q, 12q, 18		Concurrent t(2;8) and t(14;18)
Losses of 6q, 8p, 17p		Loss of acquired gain of 3q
<i>Others</i>		
14q32 abnormalities		
BCL-6 or IgVH gene somatic mutations		
<i>Rare</i>		
t(11;14)		
t(14;18)		

IgH, immunoglobulin heavy locus; t, translocation

unknown, but it may represent an aggressive variant of SMZL or a possible genetic signature indicating progressive disease. The SMZL histological high-grade transformation into large B-cell lymphoma occurs with a frequency of approximately 13%–20% [8, 13]. Chromosomal defects including 1q32 alterations [13] and P53 mutation/overexpression [7, 8] were found to be usually associated with switching into high-grade histology for SMZL and, therefore, resulting in a more aggressive clinical course. Deletion of 14q also inferred an inferior prognosis [7]. In addition, Rinaldi et al. [14] reported that the presence of both deletion 8p and 17p indicated an intermediate to high risk clinical outcome in SMZL patients (Table 1). The genetic or molecular markers that characterize other B-cell lymphomas, such as t(11;14) (q13;q32), or t(14;18)(q21;q32) translocations, are extremely rare in SMZL.

We report a unique case of SMZL associated with t(8;14)(q24.1;q32). FISH study performed on interphase cells using the break-apart probes demonstrates MYC gene rearrangement, suggesting that dysregulation of the c-myc gene is one of the major oncogenetic processes of the lymphoma. This case provides additional evidence that MYC translocation, originally described in BL, can also be observed in other aggressive high-grade B-cell lymphomas such as DLBCL and lymphoblastic lymphoma [9, 15] in addition to B-cell lymphomas, unclassifiable, with features intermediate between DLBCL and BL. Recent reports demonstrated the presence of MYC gene rearrangement in 20%–40% of cases of plasmablastic lymphoma [16]. Other previous reports have noted the presence of MYC gene translocation in follicular lymphoma [17] and mantle cell lymphoma [18]. This finding has been associated with tumor progression to high-grade morphology with an aggressive clinical course. Important distinctions in the role of MYC in these lymphomas likely reflect whether it is a primary or secondary genetic event [9]. A chromosomal break point affecting the MYC/8q24 locus was considered as the second hit of B-cell lymphoma pathogenesis [15]. Therefore, these findings suggest a common histogenesis for both the low-grade SMZL and its potential link to high-grade lymphoma.

The translocation t(8;14) (q24;q32) is the characteristic chromosomal aberration of BL [9]. On the molecular level, the t(8;14) juxtaposes the c-MYC oncogene in 8q24 next to the IgH promoter locus in 14q32, resulting in overexpression of the transcription factor c-MYC [19]. Overexpression of *c-Myc* gene affects transcriptional regulation in many downstream target genes that can play key roles in signaling, cell cycle regulation, DNA replication, protein biosynthesis, apoptosis, and energy metabolism [20]. It was reported that Myc-mediated repression of microRNA-34a promotes high-grade transformation of B-cell lymphoma by dysregulation of FoxP1 [21]. It is expected that the

dramatic changes in multiple processes of cellular functions will point toward malignant transformation [19]. Thus, this novel translocation may be associated with a distinct clinicopathological subset of SMZL with a potentially aggressive clinical course [8, 13, 15, 22].

The patient did not show evidence of residual or progression of disease at 6 months postsplenectomy. Therefore, further long-term clinical follow-up is required. Matsuhashi et al. [22] reported a case of recurrent SMZL with leukemic transformation at 30 months postsplenectomy that was subsequently followed by death. A cell line, designated UCH1, was established from the patient's original SMZL lymphoma. The tumor cell line derived from SMZL carried a very complex numerical and structural chromosomal aberrations involving t(8;14)(q24;q32) and chromosome 3 [22]. Furthermore, the c-MYC gene rearrangement was confirmed in UCH1 cells. Our current case presented a classical SMZL carrying t(8;14) as the sole genetic abnormality; therefore, it should prove more useful to study the pathophysiology of the c-MYC rearrangement in the oncogenesis of this rare disorder. The long-term clinical follow-up is critical to ascertain the clinical significance.

In summary, we present the first case of SMZL with t(8;14)/MYC–IgH gene rearrangement as the sole genetic aberration in a patient with a typical disease presentation of SMZL. The underlying mechanism of t(8;14)/MYC gene rearrangement in the development of SMZL remains undetermined. It may be indicative of an aggressive variant of SMZL or represent a progressive form of disease. The presence of t(8;14) as the sole genetic abnormality should not be used as a criteria to exclude SMZL from the differential diagnosis of small B-cell lymphomas.

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