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<th>A der(13)t(7;13)(p13;q14) with monoallelic loss of RB1 and D13S319 in myelodysplastic syndrome</th>
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A novel translocation der(13)t(7;13)(p13;q14) involving 13q14 with monoallelic loss of *RB1* and *D13S319* in myelodysplastic syndrome

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Abstract

Deletions or translocations of chromosome band 13q14, the locus of the retinoblastoma (RB1) gene, have been observed in a variety of hematological malignancies including myelodysplastic syndrome (MDS). We describe here a novel unbalanced translocation der(13)t(7;13)(p13;q14) involving 13q14 in a patient with MDS. A 66-year-old woman was admitted for precise examination of progressive anemia. She was diagnosed as MDS, refractory anemia with excess of blasts (RAEB-I) because of 7.4% blasts and trilineage dysplasia in the bone marrow cells. The blasts were positive for CD4, CD10, CD16 and CD41a as well as CD13, CD14 and CD33. Chromosome analysis showed complex karyotypes as follows; 46,XX,del(6)(q?),-7,der(13)t(7;13)(p13;q14),+mar1. Spectral karyotyping revealed that these cytogenetic aberrations were derived from complex translocations involving chromosomes 6, 7 and 13. As a result, the revised karyotype was 46,XX,der(6)t(6;7)(q11;?),der(7)del(7)(p13?)t(6;7)(q?;q11)t(6;13)(q?;q?),der(13)t(7;13)(p13;q14). Fluorescence in situ hybridization (FISH) analyses demonstrated that one allele of the RB1 gene and the microsatellite locus D13S319, located at 13q14 and telomeric to the RB1 gene, was deleted. Considering other reported cases, our results indicate that submicroscopic deletions accompanying 13q14 translocations are recurrent cytogenetic aberrations in MDS. The RB1 gene and/or another tumor suppressor gene in the vicinity of D13S319 may be involved in the pathogenesis of MDS with 13q14 translocations by monoallelic deletion.
1. Introduction

Myelodysplastic syndrome (MDS) is a clonal hematopoietic stem cell disorder characterized by ineffective hematopoiesis that leads to bone marrow failure, leukemic transformation, and clonal cytogenetic aberrations [1]. Total or partial chromosome losses and chromosome gains, such as 5q-, -7 and +8, are predominantly observed, whereas balanced translocations are relatively rare in MDS [1, 2]. Unbalanced translocations are also frequently found, and they are usually detected as a part of complex karyotypes, associated with loss of chromosomal material, and related to disease progression [1, 3].

Deletions or translocations of chromosome band 13q14, the locus of the retinoblastoma (RB1) gene, are observed in a variety of hematological malignancies including myelofibrosis (MF), MDS, acute myeloblastic leukemia (AML), chronic myelogenous leukemia (CML) and chronic lymphocytic leukemia (CLL) [4-6]. Recently, it has been shown that deletions of 13q14 are detected at a high frequency (more than 40%) in cases of CLL and multiple myeloma (MM) by fluorescence in situ hybridization (FISH) analyses [7, 8]. The microsatellite locus D13S319, which is located at 13q14.3 and telomeric to the RB1 gene, is the most commonly deleted marker in CLL and MM [7, 8]. It is suggested that the deletion of a candidate tumor suppressor gene in the vicinity of D13S319 is a significant event in the tumorigenesis of these lymphoid malignancies. On the other hand, in myeloid malignancies including MDS, deletions or translocations of 13q14 have been also analyzed in association with RB1 and D13S319 [9-13], but the pathological roles of these abnormalities, especially of 13q14 translocations, remain to be completely elucidated.

We describe here a novel translocation der(13)t(7;13)(p13;q14) involving 13q14 in MDS. The results suggest that the RB1 gene and/or another
tumor suppressor gene near \textit{D13S319} may be involved in the pathogenesis of MDS with 13q14 translocations by monoallelic deletion.
2. Materials and methods

2.1. Case History

A 66-year-old woman was admitted to our hospital for precise examination of progressive anemia in December 2003. As of 1986, she had been already diagnosed as mild macrocytic anemia in another hospital, but the definite diagnosis could not be obtained. In June 2002, her hemoglobin level decreased to below 7.0 g/dl and the treatment with transfusion of red blood cells was started once a month. Chromosome analyses have not been performed during the clinical course before admission to our hospital.

Peripheral blood on admission showed hemoglobin 6.4 g/dl, platelets 189 x 10^9/l and white blood cells 2.6 x 10^9/l with 1% blasts, 1% bands, 35% segmented neutrophils, 4% monocytes, 5% eosinophils, 2% basophils and 52% lymphocytes. Bone marrow was hypercellular with 7.4% blasts. Dysplastic changes including cytoplasmic hypogranulation of neutrophils, megaloblastic morphology of erythroblasts and micromegakaryocytes, were observed in the bone marrow cells. Surface marker analysis by three-color flow cytometry with CD45 gating revealed that the blasts were positive (more than 20%) for CD4 (73.7%), CD10 (63.1%), CD16 (59.0%), CD13 (96.2%), CD14 (56.2%), CD33 (97.4%), CD41a (64.4%) and HLA-DR (26.1%), but negative for CD34 (9.1%). She was diagnosed as MDS, refractory anemia with excess of blasts (RAEB) in the French-American-British (FAB) classification, or RAEB-I in the World Health Organization (WHO) classification. She received non-myeloablative cord blood transplantation in March 2004, but the disease relapsed soon after. She died of disease progression and pneumonia in May 2004.

2.2. Chromosome analysis and Spectral Karyotyping (SKY)

Chromosome analyses were performed on short-term culture of the cells
obtained from bone marrow on admission (December 2003) and before transplantation (February 2004) by the G-banding technique. Karyotypes were described according to the International System for Human Cytogenetic Nomenclature (ISCN 1995) [14]. Spectral karyotyping (SKY) was carried out with SkyPaint™ kit (Applied Spectral Imaging, Migdal Ha’Emek, Israel) according to the manufacturer’s instructions. A total of 5 metaphase spreads obtained on admission were analyzed for spectral karyotyping.

2.3. Fluorescence in situ hybridization (FISH) analysis

Probes used in FISH analyses were LSI 13 (RB1) SpectrumGreen Probe and LSI D13S319 (13q14.3) SpectrumOrange Probe (Vysis, Downers Grove, IL, USA). The LSI 13 DNA probe hybridizes to band 13q14, spanning the RB1 region of human chromosome 13. In a normal cell hybridized with the RB1 probe, the expected signal pattern is two distinct signals. In a cell with a RB1 deletion, one signal will be observed. The LSI D13S319 DNA probe hybridizes to the band 13q14.3, locus of D13S319 located telomeric to the RB1 gene, of human chromosome 13. In a normal cell, the expected pattern for the D13S319 probe is the two distinct signals. In a hybridized abnormal cell containing the deletion, one signal pattern will be observed. FISH analyses were performed on 20 metaphase spreads and 100 interphase nuclei obtained on admission.
3. Results

Chromosome analysis on admission showed the complex karyotypes as follows (Fig. 1): 46,XX,del(6)(q?),-7,der(13)t(7;13)(p13;q14),+mar1 [20]. The same karyotype was also detected just before cord blood transplantation. To identify the origin of deleted and marker chromosomes, we applied SKY analysis on metaphase spreads of the same sample preparation. SKY analysis revealed that the marker chromosome was derived from chromosome 7 with unbalanced translocations involving chromosome 6 and 13. Deletion of 6q was found to be resulting from unbalanced translocation with chromosome 7. Furthermore, the structure of der(13)t(7;13)(p13;q14) was confirmed. The der(13)t(7;13)(p13;q14) was generated by the translocation of the segment 7p13-7pter onto 13q14, whereas the chromosome 13q material was translocated onto the 6q fragment of the der(7)t(6;7)(q?;q11), but not 7p13. As a result, the karyotype shown by G-banding was revised as follows (Fig. 2):

46,XX,der(6)t(6;7)(q11;?),der(7)del(7)(p13?)t(6;7)(q?;q11)t(6;13)(q?;q?),der(13)t(7;13)(p13;q14)[5].

G-banding and SKY analyses identified the der(13)t(7;13)(p13;q14), suggesting that the *RB1* gene and *D13S319* at 13q14 may be involved in the translocation. Hence, we performed FISH analyses with these probes on metaphase spreads and interphase nuclei. FISH with *RB1* showed that only a single signal was observed on normal chromosomes 13 in all 20 metaphase spreads analyzed (Fig. 3A). On the other hand, FISH on interphase nuclei revealed that 59% and 41% of 100 non-dividing cells exhibited one and two *RB1* signals, respectively (Fig. 3B). FISH with *D13S319* also showed similar results. Only one *D13S319* signal was detected on normal chromosome 13 in all 20 metaphase spreads examined (Fig. 3C), whereas 65% and 35% of 100 cells demonstrated one and two *D13S319* signals by
interphase FISH, respectively (Fig. 3D). These results indicated that one allele of \textit{RB1} and \textit{D13S319} was deleted following the translocation between 13q14 and 7p13.
4. Discussion

On the basis of G-banding and SKY analyses, we defined an unbalanced translocation der(13)t(7;13)(p13;q14) in a case of MDS. Furthermore, FISH analyses revealed monoallelic loss of RB1 and D13S319 in all metaphase spreads and in approximately 60% of interphase cells. This discrepancy in FISH analyses may be due to the higher ability of cells with RB1/D13S319 deletions to divide in culture than cells without deletions. Translocations between 7p13 and 13q14 have never been described in human malignancies [15], then this is the first case with the der(13)t(7;13)(p13;q14). This translocation appeared as a part of three-way translocations involving chromosomes 6, 7 and 13, but we could not completely identify the breakpoints on the der(6)t(6;7) and the der(7)t(6;7)t(6;13) because of the complexity of the translocations. In view of the structures of three derivative chromosomes, it is supposed that the translocations evolved from a primary translocation involving chromosomes 6 and 7, followed by the second exchange between the long arm of chromosome 13 and the der(7)t(6;7)(q?;q11). Alternatively, it is possible that these complex translocations occurred simultaneously. However, considering that the translocations were identified at the initial examination and in all metaphase spreads analyzed, it is difficult at present to elucidate whether the der(13)t(7;13)(p13;q14) is a primary genetic event or an additional change during disease progression of MDS.

In myeloid malignancies including MDS, interstitial deletions involving 13q14 are recurrently found [4-6], and it is naturally expected that the RB1 gene at 13q14 may be deleted. In fact, it has been shown that RB1, D13S319 and D13S25, which is located more downstream of D13S319, are deleted in almost all cases of myeloid malignancies with 13q14 deletions examined, especially in cases of agnogenic myeloid metaplasia (AMM)/MF,
AML and MDS [9-12]. Furthermore, Nagamura et al. [16] revealed by colony-forming assay and interphase FISH that the majority of hematopoietic progenitor cells of MDS with del(13)(q12q14) lacked one allele of the RB1 gene and suggested an important role of the RB1 gene in abnormal hematopoiesis.

On the other hand, myeloid malignancies with 13q14 translocations have been less evaluated about RB1 deletions. Juneau et al. [9] reported loss of the RB1 gene in 2 cases of AMM with 13q14 translocations. La Starza et al. [10] delineated a commonly deleted region covering RB1, D13S319 and D13S25 loci in 3 cases of AML/MDS with translocations involving 13q13-q14. Sinclair et al. [12] reported that 2 MF patients with balanced translocations involving 13q14 exhibited loss of RB1, D13S319 and D13S25. Tanaka et al. [11] investigated 7 cases of myeloid malignancies with 13q14 translocations and showed allelic loss of these 3 loci in 3 cases of AML/MDS and loss of the RB1 gene only in 2 cases of AML. However, these 3 loci were intact in 2 cases of AML/CML. Coignet et al. [13] also reported that 6 cases of AML/CML with t(12;13)(p12;q14) retained the RB1 gene and their breakpoints at 13q14 were clustered centromeric of the RB1 gene. As a result, breakpoints and deleted regions were heterogeneous in myeloid malignancies with 13q14 translocations, although several cases showed monoallelic loss of RB1 and D13S319.

As shown in Table 1, a total of 14 cases of MDS with translocations involving 13q14 have been described in the literature to date [10-11, 17-25]. Almost all cases had complex karyotypes. Balanced and unbalanced translocations were observed in 7 cases and 7 cases, respectively. In 3 cases (No. 3, 7, 8), the 13q14 translocations were not detected in the stem line, suggesting that these aberrations might be acquired during disease progression and have different roles from others. Subtypes of MDS were various and common clinical features in these cases could not be found, because only
limited information was available. Three cases (No. 3, 9, 10) exhibited the same translocation t(12;13)(p13;q14), suggesting that t(12;13)(p13;q14) is one of the non-random aberrations in MDS. Deletions of RB1 and D13S319 were examined in 5 and 4 cases, respectively and loss of these 2 loci was detected in 4 cases (No. 10-12, 14). Only one case with t(12;13)(p13;q14) (No. 9) retained the RB1 gene, whereas it was deleted in another case (No. 10). That is, molecular mechanisms of t(12;13)(p13;q14) in MDS appear to be not equivalent. Other three cases (No. 10-12) lacked D13S25 as well as RB1 and D13S319, although we could not perform FISH with D13S25 in the present case. These findings indicate that submicroscopic deletions accompanying balanced or unbalanced 13q14 translocations are recurrent cytogenetic aberrations in MDS and that commonly deleted regions at 13q14 extend from RB1 to at least D13S319 and may prolong to D13S25 loci. It is possible that unidentified tumor suppressor gene(s) located in the vicinity of D13S319 as well as RB1 may be implicated in the pathogenesis of MDS having 13q14 translocations associated with cryptic deletions.

The RB protein acts as a cell cycle regulator which blocks the transition of normal cells from G0/G1 into S phase of the cycle. Mice with homozygous disruption of the RB1 alleles resulted in an overall normal development but had lethal anemia, suggesting a critical role of the RB1 gene in erythropoiesis [26]. In the present case, anemia was predominantly observed during the clinical course, while white blood cells and platelets counts were relatively preserved. This impaired erythropoiesis might be related to monoallelic loss of the RB1 gene. In several solid tumors carrying 13q14 deletions of one allele, the RB1 gene is usually inactivated by point mutations and/or gross rearrangements of the other allele. Unfortunately, we could not examine the mutation of the RB1 gene in the remaining allele of the present case. Tanaka et al. [11] tested for RB1 gene mutations in 2 MDS patients with 13q14 translocations (No. 11 and 12 in Table 1) and 4
AML patients with 13q14 deletions. However, no detectable alterations of five exons (exons 12 to 16) of the \textit{RB1} gene encoding pocket A in RB protein was demonstrated. In general, the abnormalities of the \textit{RB1} gene and its expression appear to be a rare event in MDS [27]. These findings may support the possibility that deletions or translocations of 13q14 affect other tumor suppressor gene(s) distinct from the \textit{RB1}, for instance the \textit{RFP2} gene near \textit{D13S319}, as suggested in CLL and MM [28].

Chromosome rearrangements at 7p are often detected in hematological malignancies including MDS and AML, but recurrent aberrations involving 7p13 have not been identified [15, 29]. It is unlikely that chromosome region 7p13 preferentially contains well-known oncogenes or genes involved in tumorigenesis. Recently, the \textit{PURB} gene was identified at 7p13 with \textit{PURA} gene at 5q31 and deletions of these genes were detected in MDS and AML [30]. The Purβ protein functions in a complex with Purα, which is an RB-binding protein and implicated in cell cycle control. It is possible that the \textit{PURB} gene at 7p13 might be involved in the der(13)t(7;13)(p13;q14) in association with \textit{RB1}. Further cytogenetic and molecular analyses for more cases are needed to elucidate the pathogenesis of der(13)t(7;13)(p13;q14) and the pathological roles of \textit{RB1} and \textit{D13S319} in MDS.
References


[9] Juneau AL, Kaehler M, Christensen ER, Schad CR, Zinsmeister AR,


[16] Nagamura F, Takabe T, Takahashi S, Ohno N, Uchimaru K, Ogami K,


Figure legends

Fig. 1. G-banded karyotype of the bone marrow cells. The karyotype is 46,XX,del(6)(q?),-7,der(13)t(7;13)(p13;q14),+mar1. Arrows indicate rearranged chromosomes.

Fig. 2. Spectral karyotypes of the metaphase spread after spectra based classification (left side, reverse DAPI; right side, SKY). Chromosomes were assigned a pseudocolor according to the measured spectrum. The revised karyotype is 46,XX,der(6)t(6;7)(q11;?),der(7)del(7)(p13?)t(6;7)(q?;q11)t(6;13)(q?;q?),der(13)t(7;13)(p13;q14). Arrows indicate rearranged chromosomes.

Fig. 3. FISH analyses with the \textit{RB1} probe (A, B) and the \textit{D13S319} probe (C, D) on metaphase spreads (A, C) and interphase nuclei (B, D). The arrow indicates (A) one \textit{RB1} signal on normal chromosome 13, (B) single \textit{RB1} signal, (C) one \textit{D13S319} signal on normal chromosome 13, and (D) single \textit{D13S319} signal.
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<th>D13S319 FISH</th>
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Abbreviations: M, male; F, female; ND, not done; Del, deletion; sMDS, secondary myelodysplastic syndrome; RA, refractory anemia; RARS, refractory anemia with ringed sideroblasts; RAEB, refractory anemia with excess of blasts; RAEB-t, refractory anemia with excess of blasts in transformation; CMML, chronic myelomonocytic leukemia; AML, acute myeloblastic leukemia. Bold letters show 13q14 chromosomal translocations.