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Case Report

Myelodysplastic Syndrome with CD34$^+$ Micromegakaryocytes and Giant Platelets in Peripheral Blood: A Case Report

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Abstract

Background: Myelodysplastic syndromes (MDS) are a group of hematopoietic disorders characterized by ineffective hematopoiesis, with manifestations of cytopenias in one, two, or three lineages. CD34$^+$ micromegakaryocytes and giant platelets are very rarely seen in MDS patients but may lead to unnecessary treatments. Therefore, we report and follow up on an MDS case with such an unusual finding. ***Case presentation:*** A 57-year-old male veteran with a history of MDS, alcoholic cirrhosis, and portal hypertension presented to the Emergency Department in 2020 for evaluation after a blackout, at which time peripheral blood samples and bone marrow biopsies were obtained. Flow cytometry analysis of his peripheral blood detected 8% CD34$^+$ cells. This finding raised the possibility of acute leukemic transformation from MDS. Further studies revealed that these CD34$^+$ cells represented dysplastic micromegakaryocytes and giant platelets rather than blasts. During his 4-year follow-up, the patient was alive and complained only of easy fatigability, lasting several weeks. His laboratory results showed pancytopenia and persistent iron-deficiency anemia. ***Conclusions:*** The distinction between micromegakaryocytes and giant platelets versus megakaryoblasts is extremely important in patients with MDS. This distinction may prevent misdiagnosis of acute leukemia and unnecessary treatments such as chemotherapy.

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Introduction

Myelodysplastic syndromes (MDS) are a group of hematopoietic disorders characterized by ineffective hematopoiesis, leading to blood cytopenias and an increased risk of transformation to acute myeloid leukemia (AML). In MDS, various cell lineages in the bone marrow can be affected, including the megakaryocytic lineage. The differentiation and maturation of megakaryocytes are abnormal, leading to dysplastic megakaryocytes. One peripheral blood finding in MDS can be the presence of giant platelets. Giant platelets are abnormally large platelets that typically have a diameter greater than 3.5 μm. Normal mature platelets are usually between 1.5 and 3 μm in diameter. Giant platelets often display irregular shapes, with increased granularity, and may exhibit differences in the distribution and density of granules compared to normal platelets. Examination of a peripheral blood smear under a microscope is a standard method for identifying and characterizing giant platelets. Giant platelets may be as large as or larger than standard red blood cells. Giant platelets are seen in various clinical conditions, including MDS, myeloproliferative neoplasms (e.g., essential thrombocythemia), immune thrombocytopenia, and certain inherited platelet disorders (e.g., Bernard-Soulier syndrome). Due to their abnormal size and morphology, giant platelets often exhibit impaired function. This can include altered adhesion and aggregation responses, as well as an increased risk of bleeding or thrombotic events. In MDS patients, the presence of giant platelets in the blood is indicative of dysplastic megakaryopoiesis and disruption of the normal platelet production process.[1,2] Micromegakaryocytes are abnormal, small-sized megakaryocytes. Unlike the multilobulated nuclei of normal megakaryocytes, micromegakaryocytes often possess a single round or bilobed nucleus. The cytoplasm of micromegakaryocytes may still retain granular characteristics typical of the megakaryocytic lineage, but the overall cellular morphology is less distinct compared to normal large megakaryocytes. Micromegakaryocytes are commonly associated with MDS and myeloproliferative neoplasms. Their presence often indicates a disruption in normal megakaryocyte maturation and platelet production processes.[1,2]

The concept of CD34$^+$ cells typically refers to hematopoietic stem and progenitor cells found in bone marrow and peripheral blood. This marker is used mainly for identifying stem cells and progenitor cells in the hematopoietic hierarchy. Detection of CD34$^+$ cells in peripheral blood and bone marrow samples is frequently used as a key finding in the clinical diagnosis of acute leukemia.

Here, we report a case in which CD34 expression was detected on giant platelets and micromegakaryocytes in a patient with long-standing MDS. However, the patient had a stable clinical course without evidence of disease progression. These findings highlight a potential pitfall in interpret-

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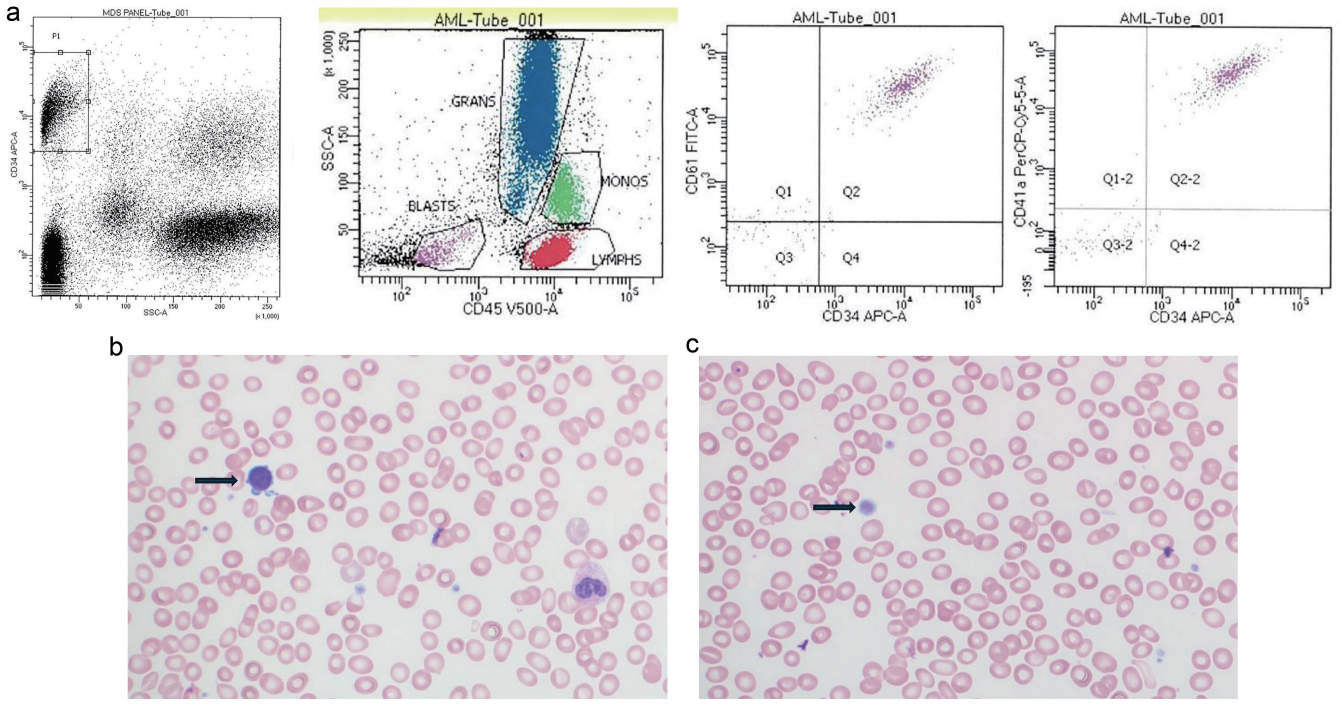

Fig. 1. Flow cytometry analysis of peripheral blood (a) in 2020 showed that the events with characteristics of blasts were positive for CD34, CD61, and CD41a. Peripheral blood smear showed circulating micromegakaryocytes (designated by arrow in b) and giant platelets (designated by arrow in c). (b and c: Wright-Giemsa, 600×). AML, acute myeloid leukemia; MDS, myelodysplastic syndrome.

ing flow cytometry results alone, which could result in an erroneous diagnosis of acute leukemia. It is extremely important to correlate flow cytometric findings with morphologic features to make a comprehensive assessment and reach the correct diagnosis.

Case presentation

The patient is a 57-year-old male veteran with a history of alcoholic cirrhosis and portal hypertension. He was admitted to the hospital in 2020 with syncope, hepatosplenomegaly, depression, and post-traumatic stress disorder. He was in his usual state of health in December 2020 when he suddenly blacked out. In the Emergency Department, he was found to have pancytopenia with hemoglobin of 9 g/dL, white blood cell count of 3.1 K/μL, and platelet count of 38 K/μL. Physical examination showed the following: General appearance: obese, no acute distress; HEENT: sclera anicteric; Neck: jugular venous pressure at mid-neck level, no lymphadenopathy; Chest: normal effort and breath sounds; no respiratory distress; Heart: normal rate, regular rhythm, and normal heart sounds; Abdomen: protuberant; normal bowel sounds; soft, non-tender, non-distended; no mass; hepatomegaly present; Extremities: 2+ edema to the knees; Neurological: alert; motor and sensory function grossly intact and symmetric; no tremor or asterixis. Flow cytometry analysis of peripheral blood revealed 8% $CD34^+$ cells. These cells were positive for CD34, CD41a, CD61, CD56 (subset), CD36, and HLA-DR (subset), but negative for CD117, CD33, CD15, CD14, CD64, CD11b, CD13, CD10, CD19, glycophorin A, and CD3. The increased $CD34^+$ cells exhibited a megakaryocytic immunophenotype, including positivity for CD61 and CD41a (Fig. 1a). Review of peripheral blood smears showed scattered cells, mostly at the feathered edge of the slide, most consistent with micromegakaryocytes with non-lobated nuclei, scant cytoplasm, and cytoplasmic pseudopods (Fig. 1b). Giant platelets were also present (Fig. 1c). No blasts were identified on the peripheral blood smears. Therefore, the $CD34^+$ cells detected by flow cytometry most likely represented dysplastic micromegakaryocytes and giant platelets rather than blasts. In addition, $CD34^+$ dysplastic micromegakaryocytes/giant platelets can be distinguished by lower FSC/SSC compared with $CD34^+$ megakaryocytic blasts.

$CD34^+$ cells seen on peripheral blood flow cytometry can represent either micromegakaryocytes or giant platelets. Comparing CD34 intensity on flow cytometry between peripheral blood giant platelets and bone marrow myeloblasts, the CD34 signal appears very strong in the peripheral blood sample. Although this raises the possibility of platelet aggregation with nonspecific binding, the presence of micromegakaryocytes on the peripheral blood smear makes the latter explanation more likely. Comparing $CD34^+$ myeloblasts versus $CD34^+$ megakaryocytes on flow cytometry, CD34 signal is usually weaker in dysplastic megakaryocytes than in myeloblasts on immunohistochemical staining. Evidence of $CD34^+$ megakaryocytes on bone marrow biopsy also supports release of dysplastic megakaryocytes into the peripheral blood.

Review of his medical records shows that he was first seen in the Hematology Clinic in March 2015 for bicytopenia, i.e., persistent anemia and transient thrombocytopenia. Flow cytometry analysis of peripheral blood at that time showed a similar population of $CD34^+$ cells with positivity for CD61 (Fig. 2a). Bone marrow biopsy showed dysplastic megakaryocytes (highlighted by CD61) (Fig. 2c), which were also positive for CD34 (Fig. 2d). The patient was diagnosed with MDS, unclassifiable in 2015, which would be best classified as MDS with multilineage dysplasia under the current WHO 2022/ICC classifications. He also had iron-deficiency anemia treated with intravenous iron therapy.

Bone marrow studies were repeated in 2020 for further

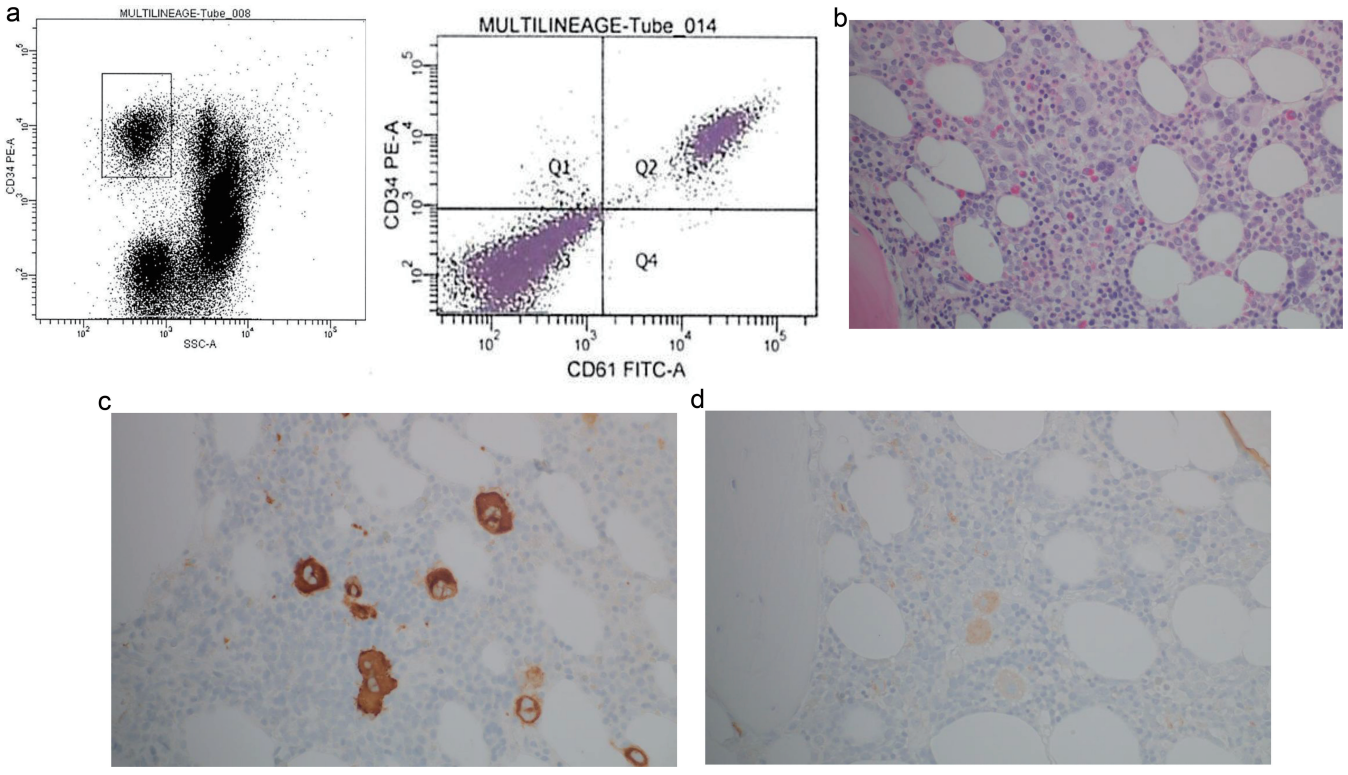

Fig. 2. Flow cytometry analysis of peripheral blood (a) in 2015 showed that CD34⁺ cells were only positive for CD61. Bone marrow biopsy showed dysplastic megakaryocytes (b), which were highlighted by CD61 (c). Some megakaryocytes were also positive for CD34 (d). (b: H&E, 400×; c and d: Immunostaining, 400×). H&E, hematoxylin and eosin.

evaluation. The bone marrow aspirate was cellular, spicular, and exhibited trilineage hematopoiesis. Megakaryocytes were dysplastic and included small hypolobated forms and forms with widely separated nuclei (Fig. 3a). Erythroid elements were dysplastic, including marked nuclear irregularity and nuclear budding (Fig. 3b). Myeloid elements showed normal maturation. Myeloblasts were not increased. Iron staining of the aspirate smear showed scant iron stores with no ring sideroblasts identified. The findings were consistent with persistent involvement by the patient's known MDS, best classified as MDS with multilineage dysplasia. Concurrent flow cytometry analysis of the bone marrow aspirate detected only 1.5% CD34⁺ myeloblasts, which were positive for CD34, CD117, HLA-DR, CD13, CD33, and CD36, but negative for CD41a, CD61, CD56, CD11b, CD16, CD14, CD7, CD10, CD3, CD19, glycophorin A, MPO, and TdT. No CD34⁺ CD41a⁺ CD61⁺ cells were identified. These findings differ immunophenotypically from the CD34⁺ populations seen in peripheral blood. In addition, CD34⁺ cells in the bone marrow aspirate showed high FSC, indicative of larger cell size. The bone marrow biopsy was small, subcortical, and approximately 30% cellular. Megakaryocytes were adequate in number but dysplastic, including small hypolobated forms and forms with widely separated nuclei (Fig. 3c). The biopsy showed relative erythroid hyperplasia, resulting in a decreased myeloid-to-erythroid (M:E) ratio. CD34 and CD117 immunostains showed that blasts comprised <5% of total cells. Megakaryocytes were highlighted by CD61 (Fig. 3d). A subset of megakaryocytes was also positive for CD34 (Fig. 3e).

The patient was seen at follow-up in 2024 and complained of easy fatigability lasting several weeks. Laboratory results showed pancytopenia with a white blood cell count of 3.02 K/μL, worsening anemia with hemoglobin of 7.6 g/dL, and a platelet count of 30 K/μL. He had persistent iron-deficiency anemia with a transferrin saturation of 5%. He required blood transfusions, but there was no evidence of leukemic transformation. Peripheral blood flow cytometry showed CD34⁺ cells comprising 7.4% of total cells, with a similar immunophenotype: positive for CD34, CD41a, CD61, CD36, and CD64 (subset), but negative for CD117, CD33, CD56, CD15, CD14, CD11b, CD13, glycophorin A, and CD71 (Fig. 4). Next-generation sequencing was performed on the patient's peripheral blood sample. The assay included a total of 88 genes using an Illumina NextSeq 550Dx with 150-bp paired-end reads. Analytical sensitivity varied by locus but was estimated to be 3.0% at 325× consensus coverage. There were no pathogenic single-nucleotide variants or small insertions/deletions. Read count analysis showed no significant copy number alterations in the regions tested, including IKZF1 deletion, ERG deletion, and KMT2A (MLL) partial tandem duplication. The assay did not detect FLT3 internal tandem duplications. It revealed JAK3 c.3157C>G (p.L1053V) with 51.3% variant allele frequency (343× consensus coverage) and SETD2 c.1256A>G (p.Y419C) with 44.9% variant allele frequency (773× consensus coverage). Both variants have been reported at very low frequency in population SNP databases. However, since both reported variants show VAFs close to 50% and matched germline testing was not performed, these variants of uncertain significance cannot be definitively classified as somatic MDS-related mutations based on the current data.

Discussion

Here we report the highly unusual presence of CD34⁺ gi-

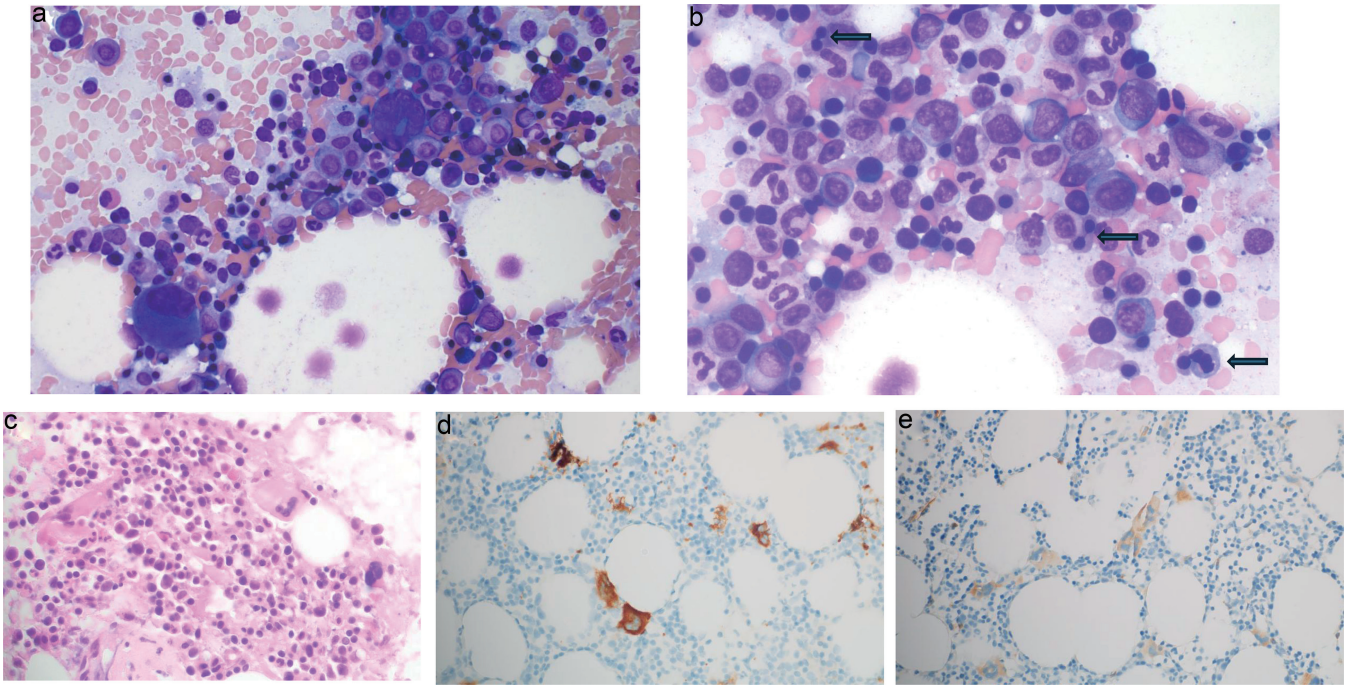

Fig. 3. Bone marrow findings in 2020. Bone marrow aspirate smear showed that megakaryocytes were small, hypolobated, and dysplastic with abnormal lobation. Myeloid precursors showed normal maturation with no increased myelo blasts. Erythroid precursors showed dysplasia with nuclear irregularity and budding (arrows) (a and b: Wright-Giemsa, 600×). Bone marrow core biopsy findings: (c) Megakaryocytes were increased many small and hypolobated forms (H&E, 400×). (d) Immunohistochemistry for CD61 highlighted the increased in number with clusters of megakaryocytes (400×). (e) Immunohistochemistry for CD34 highlights a subset of megakaryocytes and rare blasts (400×). H&E, hematoxylin and eosin.

ant platelets in an MDS patient's peripheral blood. Dysplastic giant platelets are often functionally abnormal, but these mature platelets do not usually express CD34. The findings in our case are interesting in that the dysplastic giant platelets not only showed aberrant expression of CD34 but also showed CD45 expression and side scatter characteristics very similar to those of blasts. Such findings could lead to confusion and misdiagnosis of acute leukemia, particularly in laboratories where flow cytometric findings are reported separately from morphology. In our case, CD34 expression, as well as CD61 and CD41a positivity of the events within the blast gate, raised the possibility of acute megakaryoblastic leukemia. However, there was a clear discrepancy between the flow cytometric findings and the percentage of blasts identified morphologically. This prompted further investigation, which identified $CD34^+$ dysplastic giant platelets as the source of discrepancy. This case emphasizes the need to always correlate flow cytometric findings with morphology.

While many studies have investigated the immunophenotypic abnormalities seen in myeloid, erythroid, and megakaryocytic elements in MDS patients, only two studies have explored potential immunophenotypic alterations that may be present in the platelets of MDS patients.[3,4] In one study, dysplastic platelets showed aberrant CD34 expression in one case (2% of all studied cases).[3] The other study was a case report showing aberrant CD34 expression in platelets.[4] Are $CD34^+$ giant platelets derived from $CD34^+$ megakaryocytes in MDS patients? We know that in patients with MDS, the differentiation and maturation of megakaryocytic progenitors can be disrupted, leading to dysplastic megakaryocytes. Dysplastic megakaryocytes, which can be $CD34^+$, can indeed give rise to abnormal giant platelets seen in peripheral blood. These platelets are often functionally abnormal and can contribute to clinical manifestations such as bleeding or thrombotic events. While $CD34^+$ progenitors are involved in the production of megakaryocytes, giant platelets themselves do

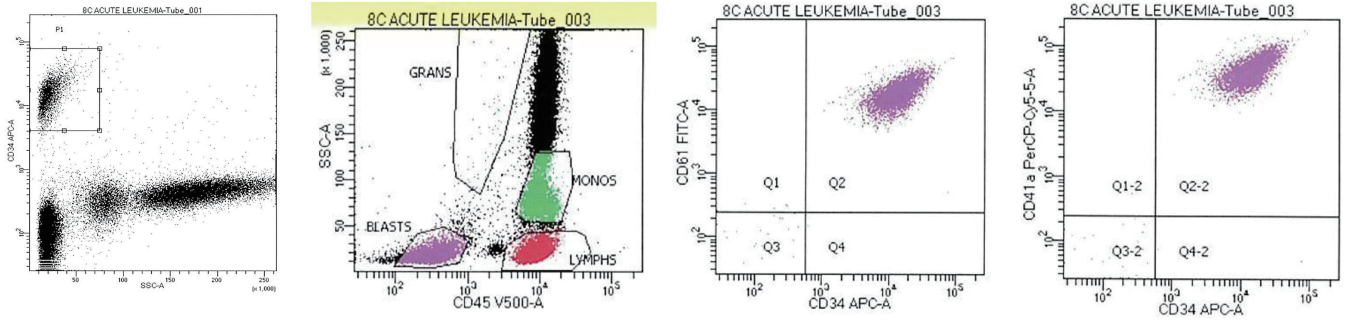

Fig. 4. Flow cytometry analysis of peripheral blood in 2024 showed the same population of $CD34^+$ cells, also positive for CD61 and CD41a.

not usually retain CD34 expression. Literature commonly discusses abnormal megakaryocyte and platelet production in MDS, emphasizing the role of genetic and molecular abnormalities in CD34+ progenitors leading to these dysfunctions, but not the presence of CD34 on mature platelets.[5,6]

There are key studies and reviews on genetic changes in MDS that affect megakaryopoiesis and lead to abnormalities in CD34+ megakaryocytes. These genetic alterations play a crucial role in the pathogenesis of MDS and the resulting hematopoietic dysregulation. Bejar et al. discusses the wide variety of genetic mutations seen in MDS and their impact on hematopoietic progenitor cells, including CD34+ cells.[7] It also details how these mutations lead to various dysplastic features. Key mutations (e.g., in TET2, DNMT3A, ASXL1, and others) have been shown to drive the disease process in MDS and affect hematopoietic stem and progenitor cells, including CD34+ megakaryocytic progenitors.[8] A comprehensive study maps the genetic mutations found in a large cohort of MDS patients, detailing how these mutations contribute to the aberrant proliferation and differentiation of progenitor cells.[9] This work discusses the genetic evolution from clonal hematopoiesis to MDS, emphasizing the importance of genetic mutations in CD34+ progenitors.[10] It also provides insights into how these mutations affect the megakaryocytic lineage. Clonal evolution and genetic architecture in secondary AML, which can originate from MDS, have also been analyzed, and this work discusses implications for CD34+ progenitor cells and resultant dysplasia, including abnormalities in megakaryocyte development.[11] Yoshida et al. focuses on frequent mutations in spliceosome machinery genes in MDS and how these contribute to abnormal development of hematopoietic progenitors, including megakaryocytes.[12] Mutations involving genes such as TET2, RUNX1, and TP53 are implicated in the molecular pathogenesis and progression of MDS and may contribute to dysregulated hematopoiesis.[13] These references and studies provide a comprehensive understanding of the genetic landscape in MDS and how these genetic alterations affect hematopoietic progenitors and megakaryopoiesis, leading to dysplastic megakaryocytes and abnormal platelet formation.

The presence and characteristics of CD34+ megakaryocytes in MDS have significant clinical implications, including prognostic value. High levels of CD34+ cells, including CD34+ megakaryocytes, in the bone marrow may correlate with more severe disease and a higher risk of progression to AML.[14] The increased presence of CD34+ cells often reflects a higher degree of bone marrow involvement and clonal expansion of abnormal progenitor cells, which are hallmarks of advanced MDS. An elevated CD34+ blast count, including early megakaryocytic progenitors, is a crucial factor in the International Prognostic Scoring System and its revised version used for risk stratification in MDS. Co-expression of markers such as CD34, CD117, and various myeloid antigens in dysplastic progenitor cells, including megakaryocytes, can provide additional prognostic information. Studies have suggested that distinct immunophenotypic profiles of CD34+ cells, including aberrant expression patterns, can aid in differentiating between lower-risk and higher-risk MDS subtypes. The genetic landscape of CD34+ progenitors, including those in the megakaryocytic lineage, can also affect prognosis. For example, mutations in genes such as TP53, RUNX1, ASXL1, and SF3B1, often occurring in CD34+ cells, have prognostic implications. Specific mutations can influence disease course, response to treatment, and overall survival, contributing to a stratified prognosis based on molecular and cytogenetic profiles. Treatment response, including hypomethylating agents, lenalidomide, and allogeneic stem cell transplantation, can be influenced by the burden and characteristics of CD34+ cells. Monitoring CD34+ cell populations, including megakaryocytic progenitors, can help assess treatment effectiveness and minimal residual disease.[15]

Investigating the presence of CD34+ giant platelets in peripheral blood through advanced genomic and transcriptomic approaches such as single-cell RNA sequencing can provide valuable insights into the characteristics and origin of these unusual cells. Single-cell RNA sequencing allows for detailed characterization of gene expression profiles at the single-cell level. This can help determine whether these CD34+ giant platelets are truly platelets or represent another cell type with aberrant marker expression. One possible approach is to isolate CD34+ giant platelets from peripheral blood and compare their transcriptomes with those of typical peripheral blood platelets from the same patient. This may help identify genes and pathways that are dysregulated. Additional investigation of potential genetic mutations or epigenetic modifications could further explain gain-of-function changes or abnormal characteristics of these giant platelets. This strategy would provide a comprehensive understanding of these atypical cells, their clinical significance, and their potential implications for the patient's condition. It could also offer novel insights into the pathology of rare hematologic presentations and contribute to a broader understanding of the field.

Limitations

The limitation of this study is that the findings are from a single patient. There is limited data on the patient's treatment, survival, and performance status. Therefore, these findings may not be representative. In addition, additional genetic analyses such as single-cell RNA sequencing, platelet-specific molecular studies, or paired germline/somatic testing, would be helpful to further understand the mechanism, but these were not performed due to technical limitations.

Conclusions

The finding of CD34+ dysplastic giant platelets in our case is unusual. Such findings could lead to misdiagnosis of acute leukemia. It is important for pathologists to integrate morphologic and flow cytometric findings for overall diagnosis. This will help prevent overdiagnosis of acute leukemia in MDS patients. Furthermore, studying the population and behavior of CD34+ dysplastic platelets in MDS is valuable and may improve understanding of disease pathology.

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None.

Conflict of interest

Dr. Hongbo Yu is an editorial board member of *Journal of Clinical and Translational Pathology*. The author declares no other conflicts of interest.

Author contributions

HY is the sole author of this article.

Ethical statement

This study was performed in accordance with the Declaration of Helsinki (as revised in 2024). This case report does not include any identifiable patient information. According to institutional policy, this case report is exempt from Institutional Review Board approval and informed consent.

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