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Case Report

Acute promyelocytic leukemia with secondary myelofibrosis and positive CD34 expression

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Abstract: Acute promyelocytic leukemia (APL) is characterized by a translocation t(15;17), which leads to the fusion of the promyelocytic leukemia (PML) gene with the retinoic acid receptor alpha (RARA) gene. This entity represents a true medical emergency due to the high risk of coagulopathy and disseminated intravascular coagulation (DIC); thus, early diagnosis is crucial to initiate treatment and achieve a better outcome. Here, we present a challenging case of APL in a 25-year-old male that mimicked classic acute myeloid leukemia (AML) based on morphologic and immunophenotypic features. Furthermore, marked myelofibrosis was identified in our patient - an unusual finding for APL. By sharing this observation, we emphasize the importance of a comprehensive approach in achieving an accurate and timely diagnosis of APL, including the integration of morphologic assessment, immunohistochemistry, flow cytometry, and molecular techniques.

Keywords: Acute promyelocytic leukemia, myelofibrosis, Auer rods, CD34 expression, PML-RARA rearrangement

Introduction

Acute promyelocytic leukemia (APL) accounts for 10-15% of adult acute myeloid leukemia (AML) cases. Targeted treatment for the PML:: RARA fusion gene has significantly improved outcomes. However, diagnosing APL can be challenging due to the variety of cytomorphologic variants, including hypergranular (classic), microgranular (hypogranular), and rare variants such as hyperbasophilic or those with a promyelocytic leukemia zinc finger/RARα fusion product [1]. Occasionally, APL may present with a nonspecific morphologic picture that blends features from multiple variants, further complicating recognition.

Typically, the expression of CD13, CD33, CD64, CD117, and myeloperoxidase (MPO), with low or absent CD34 and HLA-DR, raises the possibility of APL. Aberrant immunophenotypes in APL can mimic other entities and these have been reported [1, 2]. One of them is CD34 expression, which is thought to result from leukemogenic mutations occurring in at least a subset of primitive hematopoietic progenitor cells [3]. It is more commonly observed in the microgranular variant [4] and has been associated with an unfavorable prognosis [5, 6].

Another particularly infrequent morphologic feature in APL is secondary myelofibrosis, which may pose an additional diagnostic challenge. Myelofibrosis, characterized by increased reticulin fibers in the bone marrow, is common and well described in inflammatory, infectious, or neoplastic disorders, including acute megakaryocytic leukemia (AMKL) [7], chronic myeloid leukemia (CML) [8, 9], myelodysplastic syndrome (MDS) [10], and diffuse large B-cell lymphoma [11]. While myelofibrosis generally predicts poor outcomes in these conditions [12], neither its mechanism nor its correlation with prognosis in APL have been thoroughly studied and remain largely unknown. To the best of our knowledge, only 15 cases have been reported to date [13].

Our report highlights the coexistence of these two uncommon features in APL, emphasizing that this typically straightforward entity can sometimes present significant diagnostic challenges. We conducted a review of the literature to compare previously reported cases, providing context for this unusual association. By documenting this presentation, we aim to raise awareness of atypical manifestations, diagnostic pitfalls, and the clinical importance of early recognition to guide timely therapy.

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Table 1. Summary of reagents used in the study

Immunohistochemical/histochemical stain	Vendor/Manufacturer	Origin	Catalog #
CD34	Roche	Tucson, AZ	790-2937
CD117	Cell Marque	Rocklin, CA	117R-18
MPO	Agilent	Santa Clara, CA	A0398
E-cadherin	Cell Marque	Rocklin, CA	246R-18
Reticulin	Roche	Tucson, AZ	860-024

Clinical case

A 25-year-old male presented to the emergency department with complaints of fatigue, weight loss, and fever. Diagnostic workup revealed severe pancytopenia (RBC: 2.39 million/mm^3, Hgb: 7.6 g/dL, MCV: 88.4 fL, WBC: 0.6 × 10^9/L, platelets: 18 × 10^9/L), elevated prothrombin time (16.8 seconds), and an increased International Normalized Ratio (INR: 1.40).

Materials and methods

Immunohistochemistry was performed including CD34, CD117, MPO, and E-cadherin. Histochemical staining was performed using reticulin. Detailed information for all reagents is provided in **Table 1**. Immunophenotyping was conducted using a 10-color Navios EX flow cytometer (Beckman Coulter, Miami, FL) and included the following antibodies: CD2, CD3, CD4, CD5, CD7, CD8, CD10, CD11b, CD13, CD14, CD15, CD16, CD19, CD20, CD22, CD23, CD25, CD33, CD34, CD38, CD45, CD56, CD57, CD61, CD64, CD117, CD123, cMPO, cTdT, and HLA-DR.

Cytogenetic analysis: Chromosome preparation and G-banding analysis were performed following standard procedures. Twenty metaphase cells were analyzed using the MetaSystems/Ikaros auto-scan, imaging, and analysis system.

Fluorescence *in situ* hybridization (FISH) was performed using the MetaSystems/Ikaros auto-scan, imaging, and analysis system. Two hundred nuclei were analyzed for each probe set.

Next-generation sequencing (NGS): DNA was fragmented, ends were repaired, and universal adapters were ligated. Unidirectional gene-specific primers were used to create target-enriched DNA libraries (Variantplex library preparation). RNA samples underwent reverse transcription (Fusionplex library preparation) and were then processed according to the Variantplex library preparation protocol. Pooled libraries were sequenced using the Illumina NextSeq 550 platform. DNA and RNA sequencing data were analyzed using Archer® Analysis, and variant and QC output files were uploaded to the Clinical Genomics Workspace (CGW) software platform (PierianDx) for variant annotation.

Results

Bone marrow biopsy demonstrated markedly hypercellular marrow (>90%) with reduced trilineage hematopoiesis and increased reticulin fibrosis (MF-2). Approximately 60-70% of the marrow was involved by immature myeloid precursors, which expressed MPO, CD34 (large subset), and CD117 (subset) (**Figure 1**). No aspirate smears or clot sections were available for review due to a dry tap. Peripheral blood smear showed a few atypical cells with a high nuclear-to-cytoplasmic ratio, occasional bilobed nuclei, and no definitive intracytoplasmic inclusions (**Figure 2**).

Flow cytometry identified 58% immature myeloid precursors expressing CD34 (major subset), CD117, CD33, CD13, CD11b (subset), CD123 (subset), CD38 (dim), and MPO, with aberrant expression of CD7 (subset), while negative for HLA-DR. PML::RARA fusions [t(15;17)] were detected by FISH in 57.5% (115/200) of nuclei (**Figure 3**), and chromosome analysis revealed an abnormal karyotype: 46,XY,t(15;17)(q24;q21)[19]/46,XY[1], establishing the diagnosis of APL.

Pan-Heme NGS findings are summarized in **Table 2**. The patient was started on all-trans retinoic acid (ATRA) and arsenic trioxide (ATO) therapy.

Discussion

APL is a distinct variant of AML caused by the PML::RARA rearrangement resulting from the

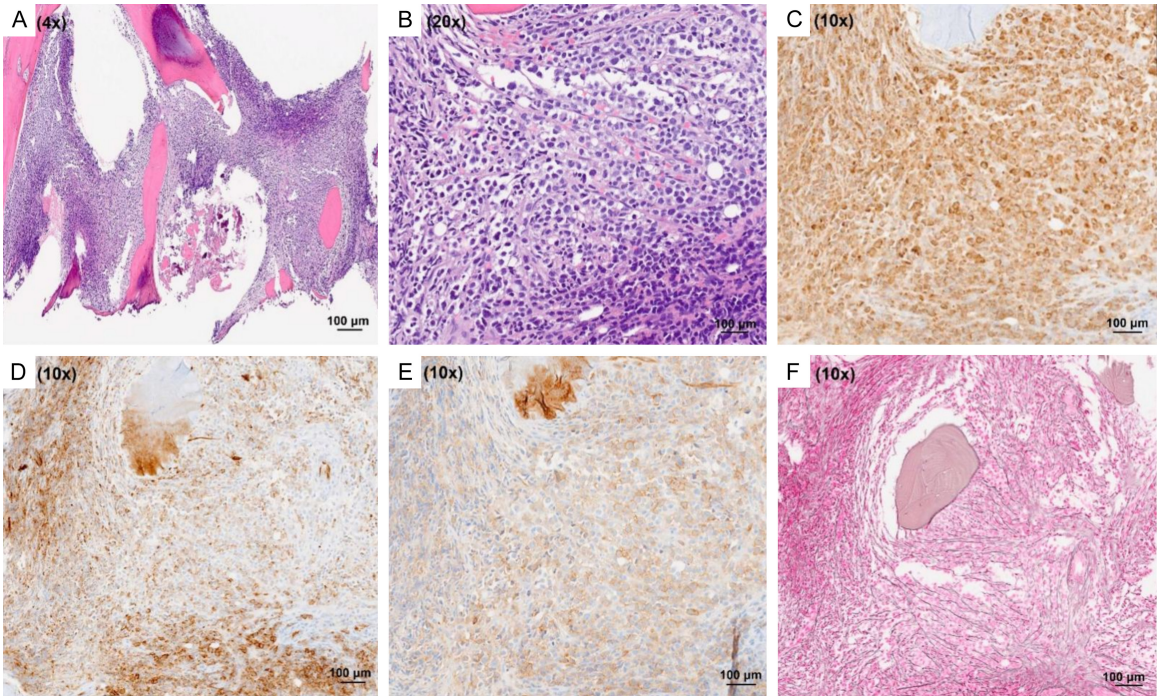

Figure 1. A composite picture of morphologic and immunohistochemical findings of bone marrow biopsy. A. H&E stain: low-power magnification (4×) shows markedly hypercellular bone marrow (>90%) with extensive involvement by immature cells. B. H&E stain: high-power magnification (20×) showing reduced trilineage hematopoiesis and numerous cells with irregular nuclear contours and variable amount of cytoplasm. C. MPO expression by leukemic cells (10×). D. CD34 expression (10×). E. CD117 expression (10×). F. Reticulin stain shows moderately increased bone marrow fibrosis (2/3+ MF scale) (10×).

t(15;17) translocation, which blocks myeloid differentiation and leads to the accumulation of abnormal promyelocytes. Morphologic evaluation, together with immunophenotyping, should provide a first indication of the disease, prior to confirmation by molecular and cytogenetic studies.

Clinically, APL typically presents with pancytopenia, fatigue, weakness, and bleeding. Leukocytosis can be observed in the microgranular variant and in cases lacking RARA rearrangements [14]. WBC and platelet counts are considered useful prognostic factors [15]. Bone marrow biopsy typically shows a hypercellular marrow with sheets of abnormal promyelocytes. In the classic APL variant (hypergranular), cells have lobulated or folded nuclei with large pink-to-purple, needle-like cytoplasmic granules (Auer rods). Auer rods are considered a morphologic hallmark of myeloid differentiation in blasts but can also be found in other hematologic malignancies [16-19]. Microgranular APL is characterized by cells with irregular bilobed nuclei and hypogranular cytoplasm. The classic variant is more common, comprising up to 70% of APL cases [20]. The hyperbasophilic type features promyelocytes with hyperbasophilic cytoplasm, cytoplasmic projections, and scattered granules. Round nuclei with no cytoplasmic granules have been associated with the zinc finger/RARα fusion product [2, 21, 22]. CD34 is usually negative in the hypergranular variant, though less differentiated promyelocytes can express it, particularly in the hypogranular variant [4]. HLA-DR is negative in nearly all APL cases; however, its expression does not exclude the diagnosis of APL [23]. However, if HLA-DR is expressed in APL, it is more often partial rather than uniform [24]. Overall, CD2, CD13, CD34, CD117, CD56, CD64, and MPO were identified as key markers to differentiate APL from other AML subtypes [24, 25]. Expression of T-cell markers (cCD3, CD2, and CD7) is extremely unusual in APL and raises the possibility of mixed-phenotype acute leukemia [26].

Despite the common observation of fibrosis in AML, fibrotic marrows are rare in APL, and

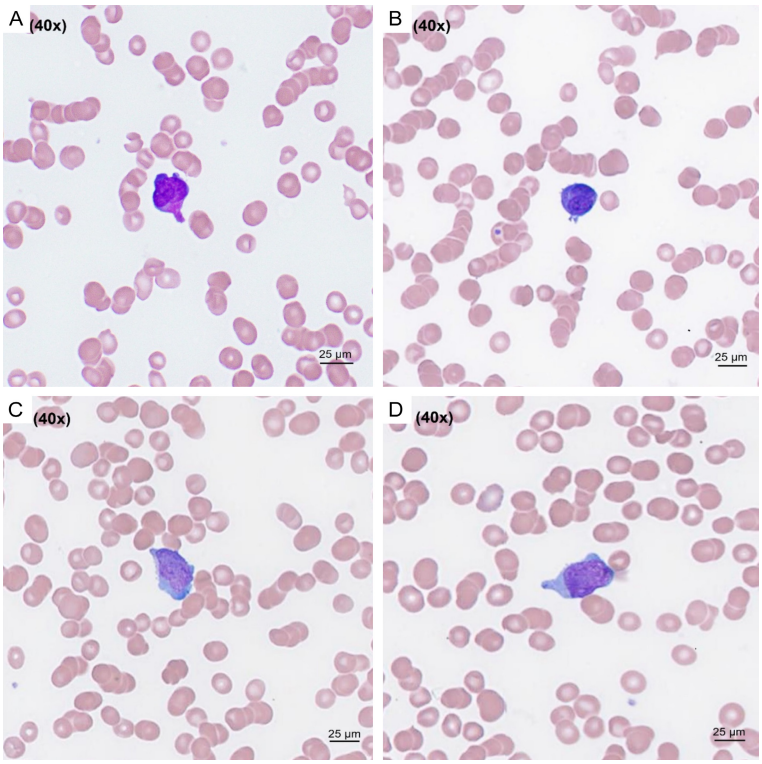

Figure 2. A composite picture of circulating immature cells from the peripheral blood smear. A-D. Atypical cells with high nuclear to cytoplasmic ratio, occasional bilobed nucleus and no definitive intracytoplasmic inclusions (40×).

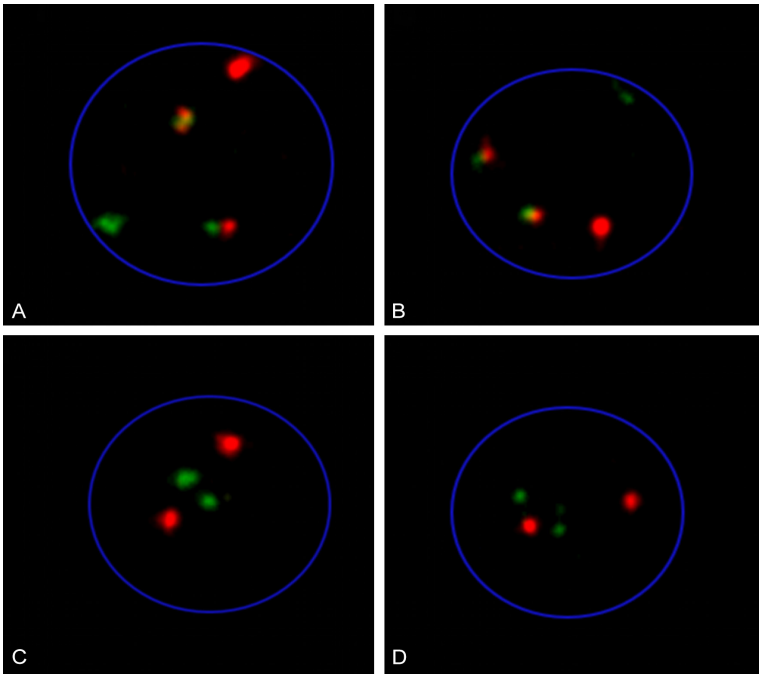

Figure 3. A composite picture of FISH study using a PML::RARA dual-color, dual-fusion translocation probe (MetaSystem). A, B. The cells show one red (PML), one green (RARA), two fusion (PML::RARA) signal patterns indicative of the t(15;17). C, D. Two separate green (*RARA* gene) and red signals (*PML* gene) are noted in normal interphase cells.

the underlying mechanism remains under investigation. Mori et al. studied the role of TGF-β1 in marrow fibrosis in a 26-year-old patient with APL. The leukemic cells showed overexpression of TGF-β1 at presentation, accompanied by diffuse fibrosis, but no increase or persistent fibrosis was observed at relapse [27]. Marrow fibrosis is more commonly seen in acute megakaryoblastic leukemia and primary myelofibrosis, typically due to cytokine release from megakaryocytes and platelets. In APL, promyelocytes with CD34 and HLA-DR expression may contribute to reticulin synthesis [28, 29]. Some studies suggest no correlation between fibrosis and treatment response or prognosis in APL [13, 30], in contrast to AML patients with marrow fibrosis, who generally have poor outcomes [12]. Bueche et al. also reported that bone marrow fibrosis may contribute to imatinib treatment failure in CML [9]. Interestingly, arsenic trioxide (ATO) and all-trans retinoic acid (ATRA) therapy may induce myelofibrosis [31, 32].

The definitive diagnosis of APL can be established only through cytogenetic analysis or molecular studies. The t(15;17)(q24.1;q21.2) translocation, resulting in the PML::RARA fusion, is the hallmark of the disease and is present in more than 95% of cases [33]. Myelofibrosis is commonly associated with JAK2, CALR, and MPL mutations [34]. Nadiminti et al. described a presentation of primary myelofibrosis with t(15; 17) and a CALR mutation but without the PML::RARA fusion [35]. The association of APL with the JAK2 V617F mutation has also been document-

Table 2. Summary of genomic profile by NGS

Gene Mutation	Tier	Variant Allele Fraction (VAF)
PML, RARA: Fusion Transcript	IA	16.9%
FLT3: Tandem Duplication	IA	13.4%
ATRX: p.I1003M c.3009T>G, NM_000489.3	III	52.9%
ETV6: p.R418G c.1252A>G, NM_001987.4	III	27.4%

ed [36-38] and may have contributed to the presence of myelofibrosis in described cases [12], however, was not detected in our patient.

We initially suspected classic AML based on morphologic and immunophenotypic findings. Immature cells in the peripheral blood were large, with occasional bilobed irregular nuclei, prominent nucleoli, and no definitive Auer rods, thereby mimicking myeloblasts. The majority of cells (-80%) expressed CD34 by flow cytometry. CD34 positivity is observed in 20-30% of APL, typically associated with leukocytosis at presentation and microgranular morphology [2]. Given the patient's marked leukopenia at presentation, we favored the classic variant of APL. The pronounced CD34 expression in our patient may be related to the development of myelofibrosis, similarly to prior cases [12, 29]. According to Xiao et al., myelofibrosis in APL is most often detected in middle-aged and elderly patients, with a median age of 34 years, whereas our patient was 25 years old [13]. It remains unclear whether secondary myelofibrosis is associated with any specific morphologic variant of APL, although previous reports more commonly describe hypergranular morphology.

Conclusion

This case highlights the heterogeneity of APL, including nonspecific promyelocyte morphology, CD34 expression in the majority of cells, and fibrotic bone marrow. It is still unknown whether increased collagen deposition could serve as an additional diagnostic clue in APL. Although deceptive morphology and aberrant immunophenotype can lead to misdiagnosis, rapid ancillary molecular testing enables the correct diagnosis.

Disclosure of conflict of interest

None.

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