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From Urologic Symptoms to Promyelocytic Leukemia-Retinoic Acid Receptor Alpha (PML::RARA): Acute Promyelocytic Leukemia Recognized Through Mucocutaneous Bleeding and Pancytopenia

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Abstract

Acute promyelocytic leukemia (APL) is a distinct subtype of acute myeloid leukemia characterized by the promyelocytic leukemia-retinoic acid receptor alpha (PML::RARA) fusion, most commonly resulting from t(15;17). Despite substantial improvements in treatment outcomes, early hemorrhagic mortality remains a major challenge, particularly when the recognition and initiation of all-trans retinoic acid (ATRA) are delayed. Atypical presentations involving urinary symptoms may complicate early diagnosis when potentially serious hematologic findings coexist with more common clinical complaints.

We report a 54-year-old man with Parkinson's disease transferred for the evaluation of left-sided flank and back pain associated with urination. He also reported lower-extremity bruising, gingival bleeding, oral discomfort, and subjective fever. Dipstick urinalysis demonstrated positive nitrites and moderate blood, raising concern for urinary tract pathology, although computed tomography showed no nephrolithiasis, hydronephrosis, or acute intra-abdominal inflammatory process. Complete blood count (CBC) revealed profound pancytopenia: white blood cell (WBC) count, 2.0 × 10^3/µL; hemoglobin, 7.2 g/dL; and platelets, 7,000/µL. Peripheral blood smear demonstrated circulating blasts. Bone marrow examination showed greater than 95% cellularity with abnormal promyelocytic proliferation; fluorescence in situ hybridization (FISH) confirmed PML::RARA t(15;17). Despite fibrinogen of 528 mg/dL, severe thrombocytopenia and mucocutaneous bleeding signaled substantial hemorrhagic risk. ATRA and arsenic trioxide (ATO) induction therapy began on hospital day 3, with transfusion support and intensive monitoring. By day 6, hemoglobin increased to 9.3 g/dL and platelets to 37,000/µL during supportive and leukemia-directed treatment, although neutropenia persisted.

This case emphasizes the complete blood count and peripheral blood smear as diagnostic pivots when urinary complaints coexist with unexplained bleeding and multilineage cytopenias. The encouraging early course highlights coordinated management while reinforcing guideline recommendations to initiate ATRA immediately once APL is suspected, before molecular confirmation.

Categories: Hematology, Oncology, Internal Medicine
Keywords: acute promyelocytic leukemia, atra, coagulopathy, diagnostic delay, hematuria, pancytopenia, pml::rara, urologic symptoms

Introduction

Acute promyelocytic leukemia (APL) is a genetically defined subtype of acute myeloid leukemia characterized by the accumulation of abnormal promyelocytes and the promyelocytic leukemia-retinoic acid receptor alpha (PML::RARA) fusion, usually arising from t(15;17)(q24.1;q21.2). The fusion protein disrupts retinoic acid receptor signaling and arrests differentiation at the promyelocyte stage [1,2]. Although APL is highly responsive to differentiation treatment with all-trans retinoic acid (ATRA) and arsenic trioxide (ATO), it remains a hematologic emergency because patients can develop life-threatening hemorrhage and coagulopathy at or soon after presentation [3].

Despite improvements in long-term survival, early mortality remains substantial, and delays in both recognition and treatment contribute to it. Park et al. documented persistent early deaths despite access to ATRA, with hemorrhage a major contributor [4]. In a multicenter study of 204 patients, Altman et al. reported 11% early mortality, with hemorrhage responsible for 61% of these deaths. Only 31% had ATRA ordered on the same day APL was suspected, underscoring the gap between recommendations and practice. In the same cohort, the median time from the presentation to suspicion of APL was one day but

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extended to 37 days in some patients. In addition, 39% of patients did not receive ATRA until the diagnosis was confirmed by marrow, cytogenetic, or molecular testing [5].

In a more recent multicenter cohort of 313 patients, Kim et al. reported 13.1% early mortality and identified ATRA administration more than 24 hours after presentation as an independent predictor of early death [6]. These data emphasize the importance of immediate ATRA at clinical suspicion, without awaiting confirmatory molecular testing, alongside urgent transfusion and serial coagulation monitoring [3].

APL commonly presents with bruising, gingival bleeding, petechiae, infection, fatigue, or cytopenias. Initial assessment may nevertheless focus on more familiar conditions when an individual reports dysuria, flank pain, or abnormal urinalysis. Yapor et al. described an APL presentation involving hematuria, gingival bleeding, fever, and urinary findings suggesting infection; competing infectious diagnoses initially complicated evaluation [7]. Patel et al. described a patient with acute myeloid leukemia presenting predominantly with gross hematuria, demonstrating how urinary bleeding may dominate the first clinical encounter even outside APL [8].

The educational gap addressed by this report is how first-contact clinicians should interpret severe multilineage cytopenias and spontaneous bleeding when an apparently plausible urinary problem is also present. This case combines dysuria-associated flank pain, abnormal urinalysis, profound pancytopenia, circulating blasts, and initially elevated fibrinogen, with a documented timeline to diagnosis and treatment. Reports of APL presenting through the urinary tract are few and heterogeneous, and this combination of findings has rarely been described [7,9-11]. The central lesson is to avoid attributing discordant hematologic findings to uncomplicated urinary disease.

Case Presentation

A 54-year-old man with Parkinson's disease diagnosed approximately four years earlier was transferred from an outside hospital because of left-sided flank and back pain associated with urination. His Parkinson's medications included selegiline, pramipexole, and carbidopa-levodopa. One week earlier, he developed flank and back pain while urinating and noticed spontaneous bruises on his lower extremities and gingival bleeding with oral discomfort during toothbrushing. He reported subjective fever one day before presentation and denied recent trauma.

The patient worked in construction and reported poor water intake, stating that he generally consumed soda throughout the day. He denied chest pain, dyspnea, nausea, vomiting, abdominal pain, melena, hematochezia, gross hematuria, or urinary symptoms other than flank pain associated with urination. His initial presentation suggested urinary pathology or dehydration but did not explain the spontaneous mucocutaneous bleeding.

At presentation, temperature was 99°F, heart rate 94 beats/minute, respiratory rate 19 breaths/minute, blood pressure 124/72 mmHg, and oxygen saturation 96%. He was alert and in no acute distress. There was no documented active oral lesion despite his history of gingival bleeding. Lungs were clear and heart rhythm regular; the abdomen was soft and nontender with costovertebral angle tenderness. Examination demonstrated purpura over the right leg and thigh. Gross neurologic examination was nonfocal.

Urine dipstick showed slightly cloudy yellow urine, positive nitrites, moderate blood, trace protein, and elevated urobilinogen, suggesting possible urinary infection or another urinary source. However, urine culture results and microscopic red blood cell quantification were not available. Computed tomography of the abdomen and pelvis without contrast showed no nephrolithiasis, hydronephrosis, or acute intra-abdominal inflammatory process; prostatomegaly was the only notable finding.

Given the patient's dysuria-associated flank pain, costovertebral angle tenderness, and nitrite-positive urinalysis, empiric piperacillin-tazobactam was initiated at a dose of 3.375 g every eight hours for suspected urinary tract infection. Although these findings supported empiric antimicrobial coverage, the urinary infection was not microbiologically confirmed. The concurrent presence of profound pancytopenia, spontaneous bruising, and gingival bleeding prompted additional hematologic investigation, ultimately establishing the diagnosis of acute promyelocytic leukemia.

The complete blood count (CBC) redirected clinical assessment toward a hematologic emergency. White blood cell (WBC) count was 2.0 × 10^3/µL, absolute neutrophil count 700/µL, hemoglobin 7.2 g/dL with normocytic indices, and platelet count 7,000/µL. The reticulocyte count was 2.10%, an inadequate response relative to severe anemia. Prothrombin time (PT) was 11.9 seconds, international normalized ratio (INR) 1.1, and fibrinogen 528 mg/dL (Table *1*).

Test/study	Result	Reference interval	Interpretation
WBC	2.0 × 10^3/μL	5.0-11.0 × 10^3/μL	Leukopenia
ANC	700/μL	>1,500/μL	Neutropenia
Hemoglobin	7.2 g/dL	14.0-18.0 g/dL	Severe normocytic anemia
Platelets	7,000/μL	130,000-450,000/μL	Critical thrombocytopenia
Reticulocytes	2.10%	0.5%-2.5%	Inadequate relative response to anemia
PT	11.9 seconds	11-13.5 seconds	Within reference interval
INR	1.1	0.8-1.2	Within reference interval
Fibrinogen	528 mg/dL	200-400 mg/dL	Elevated; may be acute phase; does not exclude APL coagulopathy
Urine nitrites	Positive	Negative	Possible urinary infection; no confirming culture
Urine dipstick blood	Moderate	Negative	Positive dipstick; gross hematuria denied
CT of the abdomen/pelvis	No stones, hydronephrosis, or inflammatory process; prostatomegaly	No acute pathology	No structural urinary tract cause

TABLE 1: Initial hematologic, coagulation, urinary, and imaging findings

Urine microscopy, urine culture, activated partial thromboplastin time, D-dimer, C-reactive protein, procalcitonin, exact mean corpuscular volume, and mean corpuscular hemoglobin values were not available. Reference intervals vary by laboratory

WBC, white blood cells; ANC, absolute neutrophil count; PT, prothrombin time; INR, international normalized ratio; CT, computed tomography; APL, acute promyelocytic leukemia

An elevated fibrinogen concentration does not exclude APL-associated coagulopathy. Fibrinogen is an acute-phase reactant, and inflammation or infection can elevate its measured concentration even when coagulation activation or consumption is occurring. In the setting of severe thrombocytopenia and bleeding, a single elevated value cannot provide reassurance; serial fibrinogen, platelet, PT/INR, activated partial thromboplastin time (aPTT), and D-dimer assessment is necessary [3,12].

Hematology was consulted because of profound pancytopenia with bruising and gingival bleeding. Neutropenic precautions, smear review, transfusion support, serial laboratory monitoring, and marrow evaluation were recommended.

Peripheral smear showed few circulating blasts against a background of thrombocytopenia and normocytic anemia, raising concern for acute leukemia. Differential diagnoses included acute leukemia, aplastic anemia, marrow infiltration, infection-associated suppression, drug or toxin effects, immune disorders, and nutritional deficiency. Circulating blasts, profound cytopenias across multiple cell lines, and mucocutaneous bleeding prioritized marrow examination.

Bone marrow aspirate, clot, and core biopsy were obtained. Aspirate cytology identified abnormal immature myeloid cells compatible with promyelocytes, including bilobed hypogranular forms and rare Auer rod-like structures (Figure *1*). The core biopsy demonstrated >95% cellularity with the near-complete replacement of normal hematopoietic tissue by abnormal myeloid cells (Figure *2*).

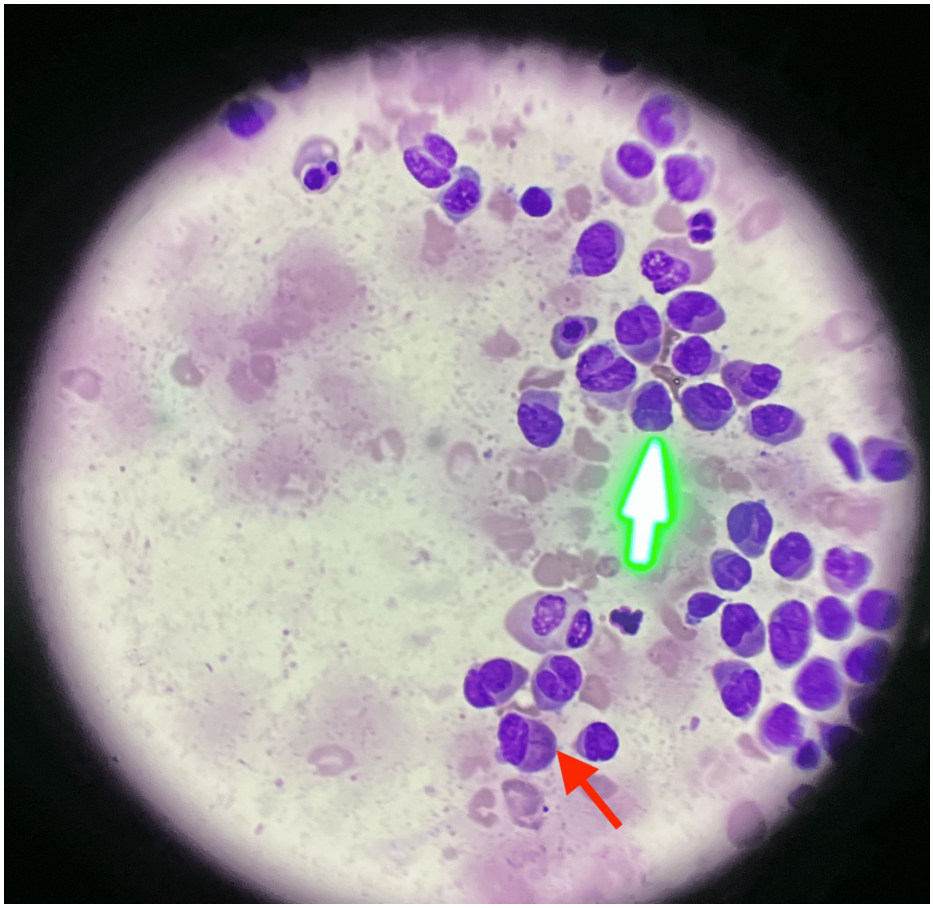


FIGURE 1: Bone marrow aspirate smear demonstrating abnormal promyelocytic cells

Wright-Giemsa stain; original magnification ×1000. Green arrow: bilobed abnormal promyelocytic morphology. Red arrow: Auer rod-like structure. Findings support APL, with confirmatory PML::RARA testing

APL, acute promyelocytic leukemia; PML::RARA, promyelocytic leukemia-retinoic acid receptor alpha

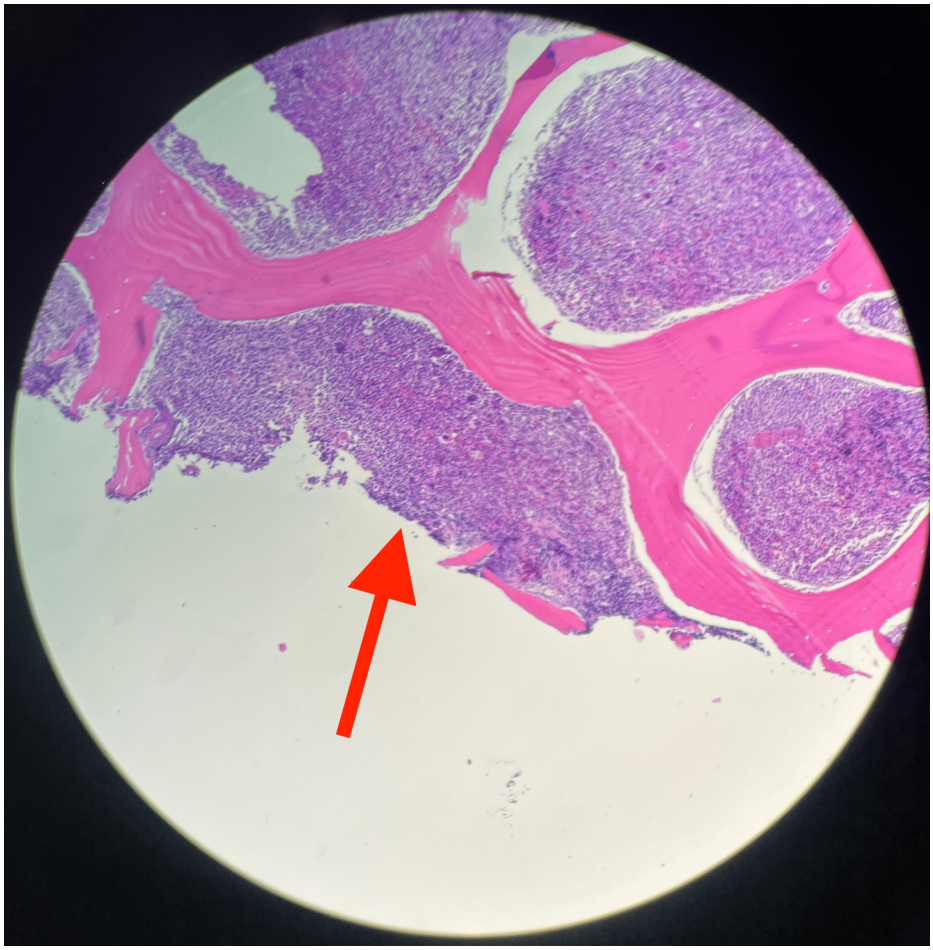


FIGURE 2: Bone marrow core biopsy showing extensive leukemic marrow involvement

Hematoxylin and eosin stain; original magnification ×100. Red arrow: dense abnormal myeloid cellular infiltration replacing normal marrow. Final report documented >95% cellularity

Integrated marrow examination confirmed APL with PML::RARA t(15;17) detected by fluorescence in situ hybridization (FISH). Flow cytometry demonstrated an abnormal immature myeloid population comprising approximately 68% of cells, with CD117 positivity, a CD34-positive subset, and HLA-DR negativity. Specimen viability was described as low. A preliminary morphologic estimate of approximately 80% blasts and the 68% flow-cytometric abnormal population represent different methods and should not be directly equated.

Reticulin staining showed mild fibrosis (MF grade 1 of 3), and iron staining demonstrated moderately increased stainable iron without ring sideroblasts (Table *2*).

Component	Finding	Significance
Peripheral blood smear	Few circulating blasts; anemia and thrombocytopenia	Prompted acute leukemia evaluation
Marrow aspirate	Bilobed hypogranular promyelocytes and rare Auer rod-like forms	Morphologically supported APL
Marrow core	>95% cellularity and near-complete abnormal myeloid replacement	Extensive marrow involvement
Flow cytometry	~68% abnormal immature myeloid cells; CD117+, CD34+ subset, and HLA-DR-	Supportive immunophenotype
PML::RARA FISH	t(15;17) detected	Confirmed APL
Reticulin	MF grade 1 of 3	Mild reticulin fibrosis
Iron stain	Moderately increased iron; no ring sideroblasts	Ancillary marrow finding

TABLE 2: Diagnostic hematopathology findings

APL, acute promyelocytic leukemia; FISH, fluorescence in situ hybridization; PML::RARA, promyelocytic leukemia-retinoic acid receptor alpha; MF, mild fibrosis

Induction began on hospital day 3 with ATRA 45 mg/m² daily in two divided doses and intravenous ATO 0.15 mg/kg daily, with a 35-day course planned. With a presenting WBC of 2.0 × 10^3/µL, the patient was non-high-risk by European LeukemiaNet criteria, the group for which chemotherapy-free ATRA/ATO is standard [3,13]. Allopurinol was provided for tumor lysis prophylaxis, along with the monitoring of uric acid, electrolytes, renal function, and lactate dehydrogenase. The clinical team monitored for differentiation syndrome, including new fever, edema, dyspnea, weight gain, and pulmonary infiltrates, with dexamethasone planned if suspected. Complete blood counts and coagulation tests were followed, and platelet and red blood cell transfusions were provided according to clinical status and laboratory thresholds. Corrected QT (QTc), potassium, magnesium, hepatic function, and renal function were monitored during ATO administration; potassium >4 mEq/L and magnesium >2 mg/dL were targeted.

By day 6, hemoglobin increased to 9.3 g/dL and platelets to 37,000/µL; the white blood cell count increased to 19.9 × 10^3/µL, while manual neutrophils remained at approximately 0.5 × 10^3/µL (Table 3). The early improvement in hemoglobin and platelets was encouraging during transfusion-supported treatment but did not establish leukemia remission or spontaneous marrow recovery. The leukocytosis required ongoing observation for treatment-associated myeloid differentiation and differentiation syndrome.

Time	Event
Day -7	Flank/back pain during urination; bruises and gingival bleeding appeared
Day -1	Subjective fever
Day 0	Urinary evaluation, antibiotic initiation, CT without stones or obstruction, and CBC showing severe pancytopenia
Days 0-1	Peripheral smear with blasts; hematology consultation and marrow assessment
Day 2	Marrow findings supported APL; PML::RARA t(15;17) confirmed
Day 3	ATRA and ATO induction initiated; transfusion and supportive monitoring
Day 6	Hemoglobin, 9.3 g/dL; platelets, 37,000/µL; WBC, 19.9 × 10^3/µL; neutropenia, persisted

TABLE 3: Timeline from urologic symptoms to APL diagnosis and treatment

ATRA, all-trans retinoic acid; ATO, arsenic trioxide; APL, acute promyelocytic leukemia; CT, computed tomography; PML::RARA, promyelocytic leukemia-retinoic acid receptor alpha; CBC, complete blood count; WBC, white blood cell

Discussion

This case illustrates how a plausible urinary complaint can coexist with a life-threatening hematologic emergency. Dysuria-associated flank pain and positive urine nitrites warranted the consideration of urinary

disease, but spontaneous purpura, gingival bleeding, severe pancytopenia, and circulating blasts were discordant with uncomplicated urinary infection. The complete blood count and peripheral smear were the decisive pivot leading to marrow evaluation and PML::RARA confirmation.

APL has unusually high early hemorrhagic risk despite curability with modern treatment. Park et al. demonstrated persistent early deaths in population-based analyses [4]. Altman et al. reported 11% early mortality in 204 patients and that only 31% received an ATRA order on the day APL was first suspected [5]. Kim et al. reported 13.1% early mortality among 313 patients and identified ATRA later than 24 hours after presentation as an independent risk factor [6]. These results reinforce why suspected APL warrants immediate ATRA and transfusion-based coagulation support before waiting for molecular confirmation [3].

In the present case, marrow confirmation was obtained by day 2, and ATRA/ATO began on day 3. This sequence demonstrates appropriate escalation from a urinary complaint to urgent hematologic evaluation and disease-specific therapy while also prompting reflection on the evidence favoring treatment at the moment of clinical suspicion. The exact time when APL was first suspected was not established; consequently, an avoidable ATRA delay cannot be inferred solely from the day-3 start.

Published APL cases with urologic features follow two patterns (Table *4*). In the first, hematuria or an abnormal urinalysis accompanies the bleeding diathesis and is initially attributed to a urinary cause. Yapor et al. described a 57-year-old woman with prior renal calculi whose hematuria and infection-suggestive urinalysis prompted ceftriaxone before APL was recognized [7]. Reyhanoglu et al. described a man with previously irradiated prostate cancer whose hematuria and easy bruising preceded diagnosis [9]. In the second, rarer pattern, APL involves the urinary tract directly. Examples include myeloid sarcoma of a kidney allograft that presented with gross hematuria and was initially read as rejection [10] and ureteral myeloid sarcoma causing allograft hydronephrosis [11]. In each report, a plausible urologic explanation was available, and the diagnosis depended on recognizing cytopenias, coagulopathy, or atypical cells. Our patient resembles the first pattern but, unlike the patient described by Yapor et al., had leukopenia rather than leukocytosis, elevated rather than low fibrinogen, and no gross hematuria [7].

Report	Patient	Urologic presentation	Confounding context	Hematologic clue
Yapor et al. [7]	57 F	Hematuria; urinalysis positive for bacteria and leukocyte esterase	Treated as UTI (IV ceftriaxone); recurrent renal calculi; dengue exposure	WBC, 16.3 × 10³/μL; platelets. 45,000/μL; fibrinogen, 85 mg/dL with DIC
Reyhanoglu et al. [9]	67 M	Hematuria with easy bruising	Prior prostate cancer treated with radiation	Marrow with 80% blasts/promyelocytes; FISH-confirmed APL
Wong et al. [10]	65 M, kidney transplant recipient	Intermittent gross hematuria, pyuria, and rising creatinine	Allograft biopsy initially read as acute cellular rejection	Allograft myeloid sarcoma with rare Auer rods; donor-derived APL
Ying et al. [11]	26 M, kidney transplant recipient	Rising creatinine and allograft hydronephrosis	Treated as acute rejection; ureteral stenosis	Ureteral myeloid sarcoma; bone marrow showed APL
Present case	54 M	Flank and back pain with urination; CVA tenderness; nitrite-positive dipstick with moderate blood	Treated as UTI (piperacillin-tazobactam); CT without stones or hydronephrosis	WBC, 2.0 × 10³/μL; platelets, 7,000/μL; fibrinogen, 528 mg/dL; circulating blasts

TABLE 4: Published APL cases with urologic presentations compared with the present case

APL, acute promyelocytic leukemia; F, female; M, male; UTI, urinary tract infection; WBC, white blood cells; DIC, disseminated intravascular coagulation; FISH, fluorescence in situ hybridization; CVA, costovertebral angle; CT, computed tomography

Patel et al. reported acute myeloid leukemia presenting chiefly as gross hematuria [8]. Although that diagnosis was not APL, it demonstrates that urinary bleeding can be the most prominent clinical symptom of a hematologic malignancy. Our patient denied gross hematuria, and dipstick blood was not quantified by microscopy, so urinary bleeding cannot be presumed the primary manifestation of APL. Together, the reports show that urologic symptoms and hematologic malignancy may coexist through different mechanisms. The key clinical distinction is the recognition of profound cytopenias and bleeding symptoms disproportionate to uncomplicated urinary pathology.

APL-associated hemorrhage is multifactorial, reflecting severe thrombocytopenia, disseminated intravascular coagulation, and enhanced fibrinolysis. Although hypofibrinogenemia may be present, coagulation abnormalities are dynamic and need not appear simultaneously. Fibrinogen is also an acute-phase reactant and may be elevated during inflammation, infection, or other systemic stress. Thus, an elevated initial value of 528 mg/dL cannot rule out evolving consumption or clinically significant APL-associated coagulopathy [3,12].

A possible urinary infection might have contributed to acute-phase fibrinogen elevation, but without a culture or inflammatory-marker series, this cannot be established. In a patient with 7,000 platelets/µL and mucocutaneous bleeding, serial PT/INR, aPTT, fibrinogen, D-dimer, platelet count, and bleeding surveillance remain necessary regardless of the initial fibrinogen value.

The major positive learning points are threefold. First, the CBC immediately identified severe multilineage cytopenias and prevented attributing all symptoms to urinary disease. Second, the blood smear identification of blasts motivated urgent marrow investigation and integrated confirmation by morphology, flow cytometry, and PML::RARA FISH. Third, the patient received ATRA/ATO induction with transfusion support, tumor lysis prophylaxis, and appropriate monitoring for coagulation and treatment complications.

ATRA and ATO target the differentiation defect produced by PML::RARA, and randomized data demonstrate excellent outcomes in selected patients [13]. Hemoglobin and platelet increases by day 6 were encouraging, but they occurred during transfusion and early treatment and should not be equated with remission. The concurrent increase in WBC reinforces the need to monitor for leukocytosis and differentiation syndrome.

For primary care, internal medicine, and emergency specialists, the lesson is not that routine dysuria warrants leukemia screening. Rather, unexplained bruising, mucosal bleeding, profound cytopenias, or circulating blasts require an urgent systemic evaluation regardless of the apparent source of pain. Literature on early death underscores the importance of initiating ATRA immediately at clinical suspicion while blood product and coagulopathy support proceed [3-6,14].

The available record did not include urine culture results, urine microscopy, aPTT or D-dimer, or long-term marrow and molecular response. These facts constrain conclusions about the urinary findings, complete coagulation evolution, and durable outcome. They do not undermine the established molecular diagnosis or the central diagnostic transition documented in this case.

Conclusions

Acute promyelocytic leukemia is a highly treatable but immediately dangerous hematologic malignancy in which failure to recognize mucocutaneous bleeding and severe cytopenias can result in preventable early death. In this patient, dysuria-associated flank pain and abnormal urinalysis initially suggested urinary disease, but spontaneous bruising, gingival bleeding, profound pancytopenia, and circulating blasts prompted urgent hematologic evaluation. Peripheral smear and marrow investigation confirmed PML::RARA-positive APL. Although fibrinogen was initially elevated, the profound thrombocytopenia and clinical bleeding required ongoing coagulopathy surveillance. ATRA/ATO induction began on day 3 with encouraging early laboratory changes during supportive care. The key lesson for first-contact clinicians is to recognize hematologic red flags that are discordant with a common presenting diagnosis and initiate ATRA promptly whenever APL is suspected.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Daniela Prado Escobar, Judith Sanchez, Kian Memari

Supervision: Daniela Prado Escobar, Judith Sanchez, Kian Memari, Joren Manuel, Peter Cohen, Shane Williams, Lissette P. Lazo

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