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Pediatric Acute Promyelocytic Leukemia (AML-M3): A Case Report

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Abstract:

Introduction: Acute promyelocytic leukemia (APL) is a subtype of AML characterized by a translocation between chromosomes 15 and 17 [t(15;17)]. In children, APL represents 4% to 8% of pediatric AML cases and about 10% of all APL cases. This case report evaluate the management of a female child with Acute Promyelocytic Leukemia at Ngoerah Hospital, Denpasar.

Case Presentation: A 1-year and 9-month-old female was admitted with weakness, paleness, bleeding gums, and lip sores. Examination revealed normochromic normocytic anemia, thrombocytopenia, and fever. Laboratory tests confirmed Acute Myeloblastic Leukemia (AML-M3) and a urinary tract infection, leading to comprehensive therapeutic management.

Conclusion: This case report describes Acute Promyelocytic Leukemia in a child with bleeding gums, bruising, severe thrombocytopenia, and Auer rod bundles, diagnosed through medical history, physical examination, and laboratory investigations.

Keywords: acute promyelocytic leukemia, acute myeloblastic leukemia, management

Introduction

Acute myeloid leukemia (AML) is a rapidly developing myeloid neoplasm characterized by the clonal expansion of immature myeloid cells, known as blasts, in the peripheral blood and bone marrow. This expansion results in ineffective erythropoiesis and megakaryopoiesis, which clinically manifests as relatively rapid bone marrow failure compared to chronic leukemia.¹

Acute promyelocytic leukemia (APL) is a subtype of AML characterized by a translocation between chromosomes 15 and 17 [t(15;17)]. The disease is classified according to the French-American-British (FAB) classification system as AML-M3. Acute promyelocytic leukemia is a relatively rare type of leukemia, accounting for approximately 7% to 8% of AML cases in adults. In children, APL represents 4% to 8% of pediatric AML cases and about 10% of all APL cases. The disease is typically found in adults with an average age of 47 years and is extremely rare in those under 20 years of age.^{2,3}

Acute promyelocytic leukemia is considered an oncological emergency. In APL, most deaths occur within the first month after diagnosis. Death is often caused by bleeding complications associated with coagulopathy at the time of diagnosis.³⁻⁵ This case report evaluate the management of a female child with Acute Promyelocytic Leukemia at Ngoerah Hospital, Denpasar.

Case Presentation

A 1-year and 9-month-old female was admitted to Ngoerah Hospital with the primary complaint of weakness lasting four days. The patient was referred to the Emergency Department due to weakness and paleness, which were preceded by bleeding gums and lip sores that began two weeks before admission. The initial symptoms involved a lip wound with clotted blood, followed by spontaneous gum bleeding without any history of trauma. The bleeding was minor and stopped on its own. Her parents also reported frequent unexplained bruising on her body. Additionally, the child had a fever from June 24 to June 28, 2024, with a peak temperature of 38.6°C. She experienced difficulty with bowel movements, though there

was no mucus or blood. There were no complaints of cough, runny nose, or difficulty breathing, and no issues with urination.

Upon physical examination, the patient appeared weak with a Glasgow Coma Scale (GCS) score of E4V5M6. Her vital signs included a pulse rate of 120 beats per minute, respiratory rate of 20 breaths per minute, temperature of 36.6°C, and oxygen saturation of 98% on room air. She measured 76 cm in height and weighed 8.5 kg. Notable findings included pale conjunctivae, gum hypertrophy, and a non-painful, non-inflamed lymph node in the left submandibular region measuring 2.5 cm. Cardiac examination revealed normal heart sounds with no murmurs or cardiomegaly, and lung examination showed normal breath sounds without any abnormal sounds. The abdomen was not distended, with normal bowel sounds and no tenderness; the liver was palpable 1 cm below the costal margin, and the spleen was palpable (Schuffner 1). Her extremities were warm to the touch.

The patient underwent various supporting examinations. The results of the laboratory results are showed as follows:

1. Laboratory Tests

a. Hematology Examination

The complete blood count results are shown in Table 2.1.

Table 2.1 Complete Blood Count Results at Ngoerah Hospital

Parameter	09/07	10/07	13/07	14/07	16/07	Reference Value
WBC ($10^3/\mu\text{L}$)	11.09	13.30	14.72	18.06	16.58	6.0 - 14.0
% Neu	27.60	30.90	24.30	55.80	33.20	18.30 - 47.10
% Lym	47.00	41.40	29.60	21.70	18.40	30.00 - 64.30
% Mono	23.80	19.50	23.30	21.80	30.60	0.0 - 7.10
% Eos	1.10	7.80	22.40	0.40	16.90	0.0 - 5.0
% Baso	0.50	0.40	0.40	0.30	0.90	0.0 - 0.70
# Neu ($10^3/\mu\text{L}$)	3.06	4.12	3.59	10.07	5.51	1.10 - 6.60
# Lym ($10^3/\mu\text{L}$)	5.21	5.50	4.35	3.92	3.05	1.80 - 9.00
#Mono ($10^3/\mu\text{L}$)	2.64	2.59	3.43	3.94	5.07	0.00 - 1.00
#Eos ($10^3/\mu\text{L}$)	1.10	1.04	3.29	0.08	2.80	0.00 - 0.70
#Baso ($10^3/\mu\text{L}$)	0.06	0.05	0.06	0.05	0.15	0.0 - 0.10
RBC ($106/\mu\text{L}$)	3.71	3.35	3.07	2.61	4.11	4.10 - 5.3

HGB (g/dL)	10.60	9.50	8.60	7.30	11.10	12.0 - 16.0
HCT (%)	32.00	28.90	26.50	22.00	34.20	36.0 - 49.0
MCV (fL)	86.30	86.30	86.30	84.30	83.20	78.0 - 102.0
MCH (pg)	28.60	28.40	28.00	28.00	27.00	25.0 - 35.0
MCHC (g/dL)	33.10	32.90	32.50	33.20	32.50	31 - 36
RDW (%)	14.90	14.60	14.60	14.30	14.00	11.6 - 18.7
PLT (10³/μL)	9.00	140.00	23.00	46.00	30.00	140 - 440
NLR	0.59	0.75	0.82	2.57	1.80	<= 3.13

From the complete blood count results, the patient shows a decrease in red blood cells, hemoglobin decrease manifesting as normochromic normocytic anemia, an increase in white blood cells, and a decrease in platelets (thrombocytopenia).

b. Clinical Chemistry

Examination The results of the clinical chemistry tests are shown in Table 2.2.

Table 2.2 Clinical Chemistry Test Results at Ngoerah Hospital:

Parameter	13/07	18/07	27/07	03/08	Reference Value
AST/SGOT (U/L)	26.00		32.00		< 34
ALT/SGPT (U/L)	13.00		19.00		< 55
BUN (mg/dL)	13.0		10.9		8.4 – 25.7
Serum Creatinine (mg/dL)	0.32		0.41		0.72 – 1.25
Procalcitonin (ng/mL)		0.10		2.53	< 0.05
CRP (mg/L)				116.40	< 5

The clinical chemistry tests reveal a decrease in serum creatinine, an increase in procalcitonin, and elevated C-reactive protein (CRP).

c. Urinalysis

The urinalysis results are shown in Table 2.3.

Table 2.3 Urinalysis Results at Ngoerah Hospital:

Parameter	15/07	24/07	25/07	26/07	27/07	28/07	29/07	Reference Value
Specific Gravity	1.019	1.009	1.003	1.014	1.008	1.003	1.004	1.003–1.035
Clarity	Clear	Clear	Clear	Clear	Unclear (+)	Clear	Clear	
pH	7.00	7.00	7.00	7.00	8.00	7.00	6.50	4.5 – 8
Leukocytes	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Nitrites	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Protein	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Glucose	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Ketones	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Blood	(3+)	(2+)	(2+)	(2+)	(2+)	(2+)	(1+)	Negative
Urobilinogen	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Bilirubin	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Color	Yellow	Yellow	Yellow	Yellow	Yellow	Yellow	Yellow	P yellow – yellow
Leukocytes (sediment)	2	0	0	1	1	0	0	< 7
Erythrocytes (sediment)	<u>52</u>	<u>18</u>	<u>9</u>	<u>53</u>	<u>22</u>	<u>6</u>	<u>7</u>	< 5
Epithelial Cells (sediment)	1	0	0	0	1	0	0	< 8
Bacteria	<u>219.2</u> <u>0</u>	<u>361.1</u> <u>0</u>	18.40	<u>302.1</u> <u>0</u>	99.20	80.60	92.20	< 130.7

Urinalysis tests showed results of hematuria and bacteriuria.

d. Peripheral Blood Smear Examination

The results of the peripheral blood smear examination can be seen in Table 2.4 and Figure 2.1.

Table 2.4 Peripheral Blood Smear Results at Ngoerah Hospital

Peripheral Blood Smear	07/09/2024
Erythrocytes	Normochromic normocytic, poikilocytosis (positive tear drop cells, positive fragmented cells), positive polychromatic erythrocytes, negative normoblast
Leukocytes	Appears normal in count, relatively increased monocytes on differential count, positive immature granulocytes, negative toxic granules, negative vacuolization, approximately 5% large blast-like cells found
Platelets	Appears significantly reduced in count, negative giant platelets, negative platelet clumping
Impression	Normochromic normocytic anemia and thrombocytopenia
Recommendation	Bone marrow aspiration examination

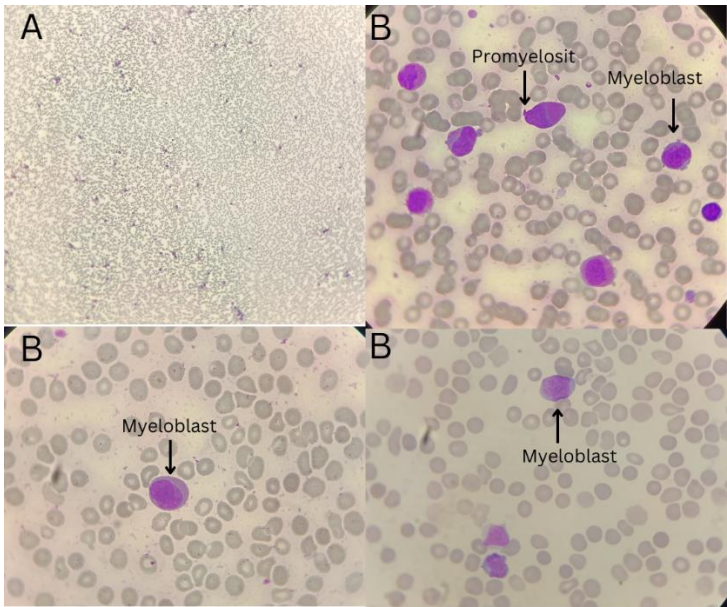


Figure 2.1 Peripheral Blood Smear Image; A. 10X Objective Magnification, B. 100X Objective Magnification

2. Bone Marrow Aspiration Examination

The results of the bone marrow aspiration examination can be seen in Table 2.5 and Figure 2.2.

Table 2.5 Bone Marrow Aspiration Results at Ngoerah Hospital

Bone Marrow Aspiration	07/11/2024
Cellularity	Slightly hypocellular
Erythroid System	Decreased activity
Myeloid System	Increased activity, 10% myeloblasts, 30% promyelocytes, positive Auer rod bundle
Megakaryocyte System	Decreased activity
Other Cells	No infiltration of non-hematopoietic cells into the bone marrow
Conclusion	Bone marrow findings consistent with Acute Myeloblastic Leukemia (AML-M3) / Acute Promyelocytic Leukemia (APL)

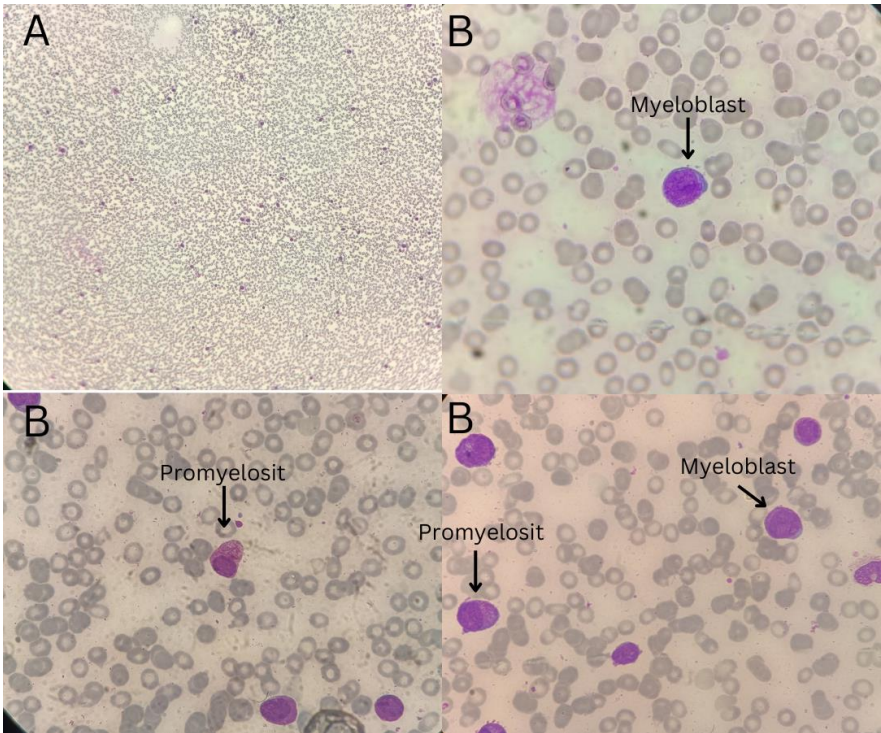


Figure 2.2 Bone Marrow Smear Image; A. 10X Objective Magnification, B. 100X Objective Magnification

3. Microbiology Examination

The microbiology examination results revealed *Acinetobacter junii* in urine culture and coagulase-negative *Staphylococcus* in blood culture from two sites.

Table 2.6 Microbiology Examination Results Culture and Antibiotic Sensitivity Test (07/15/2024)

Specimen	Urine
Organism	Acinetobacter junii
Comment	25,000 CFU/mL
Leukocytes	<5/hpf
Antibiotics	
Ampicillin/Sulbactam	S
Gentamicin	S
Ceftazidime	R
Ciprofloxacin	I
Ceftriaxone	R
Levofloxacin	S
Cefepime	S
Tigecycline	S
Meropenem	S
Trimethoprim/Sulfamethoxazole	R
Cefoperazone/Sulbactam	S
Amikacin	S
Comment	- Acinetobacter junii isolated from the patient's specimen. - Significance as an infectious agent depends on the clinical condition and infection markers of the patient. -If considered significant, Ampicillin/Sulbactam may be considered as an antibiotic therapy option. - Replace catheter if in use
Culture and Antibiotic Sensitivity Test (07/17/2024)	
Specimen	Blood
Organism	Staphylococcus, coagulase-negative
Comment	Coagulase-negative Staphylococcus isolated, considered as normal skin flora (contaminant) because it was only found in one blood draw site. Antibiotics are not recommended.

The working diagnosis for this case is Acute Myeloblastic Leukemia / Acute Promyelocytic Leukemia (AML-M3) with Urinary tract infection (UTI). The therapeutic management plan for the patient includes intravenous fluid therapy with D5 1/4 NS administered at a rate of 10 ml per hour, which is approximately 3 drops per minute using a macro drip set. The patient is also receiving cefotaxime at a dosage of 150 mg per kg per day, with 450 mg given intravenously every 8 hours. Maintenance

therapy with gentamicin is administered at 6 mg per kg per dose, resulting in a 55 mg dose given intravenously every 24 hours. Paracetamol syrup is prescribed at 85 mg orally every 8 hours when the patient's temperature reaches or exceeds 38°C. Cyclophosphamide is included in the treatment at a dosage of 200 mg per square meter, which equates to 84 mg diluted in 200 ml of normal saline, administered intravenously over 2 hours.

In addition to these treatments, the patient undergoes packed red cell (PRC) transfusion, aiming for a hemoglobin level of 10-11 g/dL. The transfusion volume is between 91-125 ml, with approximately 100 ml delivered over 3 hours. Furthermore, the patient receives a thrombocyte concentrate (TC) transfusion at a dosage of 5-10 ml per kg, which amounts to 42.5-85 ml. A single unit of TC, approximately 50 ml, is infused intravenously in less than 20 minutes.

Discussion

Acute promyelocytic leukemia (APL) is a specific subtype of AML characterized by the accumulation of abnormal promyelocytes in the bone marrow and peripheral blood. Patients with APL may present symptoms similar to other AML subtypes, such as fatigue, weakness, fever, and/or bleeding complications. By the time symptoms appear, the patient's bone marrow is usually filled with abnormal promyelocytes. This condition not only causes abnormalities in blood cell counts, such as leukopenia or leukocytosis, anemia, and thrombocytopenia, but can also trigger severe coagulation disorders, involving elements of disseminated intravascular coagulation (DIC) and primary fibrinolysis. These disorders can lead to life-threatening bleeding, including intracranial hemorrhage. As a result, APL has a high early mortality rate and is considered a serious medical emergency.⁶ In this case, the patient presented with complaints of weakness, pallor, bleeding gums, bruises on the body, and fever.

Common physical examination findings in AML include pallor, bruising, and hepatosplenomegaly, while lymphadenopathy is rare. The primary pathogenesis of AML involves a maturation blockade, which halts the differentiation of myeloid cells

at an immature stage, leading to an accumulation of blasts in the bone marrow. This accumulation disrupts normal hematopoiesis, resulting in bone marrow failure syndrome, characterized by cytopenia (anemia, leukopenia, and thrombocytopenia). Anemia causes fatigue and anemic conjunctiva, while thrombocytopenia leads to gum bleeding and hematomas. Gingival hypertrophy occurs due to extramedullary infiltration of leukemia blast cells in the oral cavity. Extramedullary involvement is associated with CD56 expression on blast cells and karyotypic abnormalities involving chromosome 11.^{1,4} In this patient's physical examination, findings included anemic conjunctiva, gingival hypertrophy, and palpable lymph nodes in the left submandibular region.

Laboratory tests revealed leukocytosis, normochromic normocytic anemia, severe thrombocytopenia, decreased serum creatinine, elevated procalcitonin, and increased CRP. Urinalysis showed hematuria and bacteriuria. Peripheral blood smear showed normochromic normocytic anemia and thrombocytopenia (positive for immature granulocytes, large blast-like cells around 5%), with bone marrow showing 10% myeloblasts and 30% promyelocytes, as well as positive Auer rod bundles. Microbiological examination revealed urine culture with growth of *Acinetobacter junii*.

Leukocytosis, characterized by an increased white blood cell count, is a common clinical finding with various potential etiologies, including infection, inflammation, malignancy, and hereditary disorders. The threshold for leukocytosis varies by age and pregnancy status, and significant deviations from normal values require further evaluation. Identifying changes in white blood cell distribution helps direct the underlying etiology. Neutrophils play a well-established role in fungal and bacterial infections, being the first cells deployed to the site of infection and acting through phagocytosis, production of reactive oxygen and nitrogen species (ROS/RNS), formation of neutrophil extracellular traps (NETs), and synthesis of proinflammatory cytokines.^{7,8}

A complete blood count is one of the initial laboratory tests performed and almost always shows abnormal results in APL. Common findings include cytopenia in all three blood cell lines (erythrocytes, leukocytes, and platelets).⁹ Anemia in this

patient could be due to decreased bone marrow function in producing erythrocytes, indicated by a reduction in erythroid precursors. This loss of bone marrow function occurs due to progressive leukocyte infiltration into the bone marrow, disrupting the red blood cell production process.¹⁰

Thrombocytopenia can result in clinical manifestations such as mucosal bleeding, easy bruising, petechiae or purpura, epistaxis, bleeding gums, and heavy menstrual bleeding. Hematuria and gastrointestinal bleeding are rare. Patients may also experience spontaneous bleeding, such as intracranial or intra-abdominal hematomas.¹⁰

Acute promyelocytic leukemia (APL) is a subtype of AML with a disease progression pattern and biological characteristics distinct from other forms of AML. Morphologically, the most common form of APL can be recognized by the dominance of hypergranular cells in the bone marrow, where the nuclei show folded and twisted appearances.⁹

Peripheral blood often contains heavily granulated cells, a hallmark of this disease, although these cells are more frequently found in the bone marrow. A peripheral blood smear is useful for verifying abnormalities and avoiding inaccurate values such as secondary thrombocytopenia due to platelet clumping. Myeloblast morphology may show more than two nucleoli with positive Auer rods. A peripheral blood smear can detect and differentiate various types of immature white blood cells in the peripheral blood.⁹

Bone marrow smears in APL patients typically show increased cell density (hypercellularity) with abnormal promyelocytes $\geq 30\%$. These promyelocytes are larger than normal promyelocytes, with round, oval, and lobulated nuclei. Auer rods are positive, with 1-10% forming multiple Auer rods (faggot cells). Erythropoiesis and thrombopoiesis activities are decreased.⁹ The following table shows the morphological subtypes of APL.

Table 3.1 Morphological subtypes of APL (Lancet et al., 2014).

Hypergranular (Classic M3)	Microgranular (M3v)	Hyperbasophilic	Promyelocytic Leukemia Zinc Finger/Retinoic
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Acid Receptor-a (M3r)			
Indented, lobulated nuclei	Irregular, indented nuclei	High nucleus-to-cytoplasm ratio	Regular, dense chromatin, Pelger cell-like
Prominent azurophilic granules	Small, dark granules	Sparse granules, very basophilic, cytoplasmic budding	Granules between classic M3 and M2
Frequent Auer rods	Rarely seen	Not visible	Few or no faggot cells

Promyelocytes, or normal progranulocytes, are the final form of myeloblasts before developing into myelocytes with granules in the cytoplasm. These azurophilic granules are dark blue and appear coarser than granules in myeloblasts. Promyelocyte cell size ranges from 12-20 μm and is often larger than myeloblasts. In a normal peripheral blood smear, promyelocytes are not found, while in bone marrow smears, promyelocytes usually make up about 1-5%.¹¹

In classic hypergranular APL, bone marrow aspiration shows a hypergranular condition dominated by abnormal promyelocytes. Malignant promyelocytes may be larger than normal promyelocytes. Their granules often obscure the nucleus, making the boundary between the nucleus and cytoplasm less clear. The nuclei are indented, and the cytoplasm often shows vacuoles and Auer rods. Auer rods are primary granules that have fused and can be present in large numbers. Multiple Auer rods grouped in one cell are known as faggot cells or bundles of sticks.⁹

Creatinine is a byproduct of creatine phosphate in muscles and is produced by the body at a constant rate. Most creatinine is cleared from the blood by the kidneys. Decreased creatinine clearance by the kidneys results in elevated blood creatinine levels. The amount of creatinine produced per day depends on muscle mass, so there is a range of creatinine levels between men and women, with lower values in children and individuals with reduced muscle mass. Diet also affects creatinine levels; creatinine can increase by up to 30% after consuming red meat.¹²

Procalcitonin is the prohormone of calcitonin, secreted by various types of cells from different organs in response to proinflammatory stimuli, particularly bacterial

stimuli. Procalcitonin (PCT) is a biomarker that can be detected in the blood during a systemic inflammatory reaction. PCT can help diagnose and assess the severity of sepsis. C-reactive protein (CRP) is an acute-phase reactant that functions as a biomarker and can increase in the presence of infection or sepsis. CRP levels can rise within hours and peak within 48 hours of infection. C-reactive protein is considered an important indicator of acute infection and can be used to detect sepsis. PCT and CRP can predict sepsis.^{13,14}

Hematuria, defined as the presence of blood in the urine, is classified into two main categories: macroscopic hematuria and microscopic hematuria. Macroscopic hematuria refers to visible blood in the urine, while microscopic hematuria indicates red blood cells detected through urinalysis or urine microscopy without visually apparent blood. Bleeding can often be an early sign of leukemia in children, usually caused by secondary thrombocytopenia due to bone marrow infiltration or triggered by disseminated intravascular coagulation (DIC). DIC is particularly common in children with acute promyelocytic leukemia (APL).¹⁰

Bacteriuria refers to the presence of bacteria in the urine. There are various ways bacteria can contaminate or infect the urinary tract. Women frequently experience urinary tract infections (UTIs) due to the urethra's proximity to the vagina and rectum, which can lead to bacteria ascending from the urethra to the bladder. *Acinetobacter* is a nonmotile, gram-negative coccobacillus commonly found in soil and water and is known for its ability to contaminate intravenous fluids. *Acinetobacter junii* is an opportunistic pathogen primarily affecting patients who have undergone previous antimicrobial therapy, invasive procedures, or suffer from malignancies. Cultures are often obtained from urine, saliva, respiratory secretions, and open wounds.^{15,16} The patient also experienced a UTI, confirmed by routine urinalysis showing bacteriuria and a urine culture isolating *Acinetobacter junii*.

According to the World Health Organization (WHO) classification, acute myeloid leukemia (AML) is divided into several groups, one of which is APL. The most commonly used AML classification is the French-American-British (FAB)

classification. Below is a table showing the AML classification according to WHO and the FAB classification.^{17,18}

Table 3.2: FAB Classification

Lineage	Marker
M0	Acute myeloid leukemia, minimally differentiated
M1	Acute myeloid leukemia without maturation
M2	Acute myeloid leukemia with maturation
M3	Acute promyelocytic leukemia
M4	Acute myelomonocytic leukemia
M4eo	Acute myelomonocytic leukemia with eosinophilia
M5a	Acute monocytic leukemia, poorly differentiated
M5b	Acute monocytic leukemia, well differentiated
M6	Acute erythroleukemia
M7	Acute megakaryocytic leukemia

Table 3.3: WHO Classification

Classification of AML	
I.	AML with recurrent cytogenetic translocations
	AML with t(8;21)(q22;q22), AML1 (CBF α)/ETO
	APL with t(15;17)(q22;q11-12) and its variants, PML/RAR α
	AML with abnormal bone marrow eosinophils with inv(16)(p13q22) or t(16;16)(p13;q11), CBF β /MHY11
	AML with 11q23 abnormalities (MLL)
II.	AML with multilineage dysplasia
	With myelodysplastic syndrome
	Without myelodysplastic syndrome
III.	Therapy-related AML and myelodysplastic syndromes due to alkylating agents or epipodophyllotoxins (some involve lymphoid abnormalities)
IV.	Unclassified AML
	AML with minimal differentiation
	AML without maturation
	AML with maturation
	AML with monocytic differentiation
	Acute monocytic leukemia
	Acute erythroid leukemia
	Acute megakaryocytic leukemia
	Acute basophilic leukemia
	Acute panmyelosis with myelofibrosis

Criteria for diagnosing APL involve a combination of clinical symptoms, physical examination, laboratory findings, and molecular genetics. Current diagnostic criteria for APL are based on the presence of at least 10% blasts in bone marrow or blood, along with specific genetic abnormalities. Although more than 20% blasts/promyelocytes are found in most cases of APL, the diagnosis can still be made if $t(15;17)(q24.1;q21.2)$ or $PML::RARA$ is present, even if the blast/promyelocyte count is less than 10%. This genetic abnormality is directly associated with disrupted myeloid maturation at the promyelocyte stage, often leading to the accumulation of blasts with abnormal promyelocytes in the bone marrow and/or peripheral blood. This condition is also accompanied by characteristic cytopenias and coagulopathy, which pose a high risk of bleeding, requiring rapid diagnosis and treatment to prevent early mortality.¹⁹ Based on the diagnostic criteria, the patient was diagnosed with APL, with 10% myeloblasts, 30% promyelocytes, and bundles of Auer rods on bone marrow smear examination. The patient also had bicytopenia and gum bleeding.

Although therapy for APL is highly effective, early mortality remains a significant issue, accounting for at least 50% of total deaths in the first three years. The primary life-threatening complication in the early stages of APL is bleeding. Therefore, patient management requires early recognition of the disorder and anticipation of known complications. Intensive monitoring during the first 3 to 5 weeks is critical to achieving remission.⁹

The majority of APL patients, around 80%, present with coagulopathy and thrombocytopenia. Laboratory tests such as platelet count, PT, APTT, and fibrinogen levels are used to assess coagulopathy, while initial therapy focuses on the administration of blood products and platelets.⁹ In this case, the patient received blood products, including packed red cells and platelets.

Anthracyclines are the main cytotoxic agents in the treatment of all AML subtypes, including APL, with proven sensitivity of APL to this class of agents. Optimizing AML induction therapy can increase remission rates and reduce the risk of relapse. Cyclophosphamide (CTX) has shown benefits in treating relapsed and refractory AML, although its use in first-line induction regimens has not been reported.

Further research is needed to assess the efficacy of CTX in induction chemotherapy for AML, especially as an alternative to non-anthracycline drugs.²⁰ In this case, the patient received cyclophosphamide therapy. Unfortunately, the patient passed away on day 28 of treatment.

Conclusion

This case report describes Acute Promyelocytic Leukemia in a 1 year and 9-month-old female, characterized by bleeding gums, gingival hypertrophy, bruising on the body, normochromic normocytic anemia, severe thrombocytopenia, the presence of myeloblasts and promyelocytes, as well as Auer rod bundles. The diagnosis was made based on medical history, physical examination, and supporting investigations.

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