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Co-occurrence of AIDS and acute promyelocytic leukemia: a case report and review of the literature

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Acute promyelocytic leukemia (APL) is a rare and aggressive subtype of acute myelogenous leukemia (AML), characterized by the PML-RARA fusion gene. When APL presents concurrently with acquired immunodeficiency syndrome (AIDS), it creates unique challenges in diagnosis and treatment due to the immunocompromised state of the patient. This case report describes a 46-year-old male patient with long-standing AIDS who developed APL. Initial treatment involved all-trans retinoic acid (ATRA) monotherapy due to the patient's severe lung infection and liver dysfunction, followed by the addition of arsenic trioxide (ATO) once infection and liver function improved. The patient achieved complete remission (CR) after combined ATRA and ATO therapy, with successful molecular remission of the PML-RARA fusion gene. We discuss the complexity of managing APL in the context of HIV infection, including the challenges posed by infections, liver dysfunction, and the impact of chemotherapy on antiretroviral therapy (ART). This case highlights the need for immediate initiation of ATRA in APL patients, even before genetic confirmation, and the potential therapeutic role of ATO in both leukemia treatment and HIV reservoir management. Further studies are needed to optimize treatment protocols for patients with concurrent AIDS and APL, focusing on personalized approaches to maximize efficacy while minimizing complications.

KEYWORDS

acute promyelocytic leukemia (APL), acquired immunodeficiency syndrome (AIDS), all-trans retinoic acid (ATRA), arsenic trioxide (ATO), PML-RARA fusion gene

1 Introduction

Acquired immunodeficiency syndrome (AIDS) is a severe immunodeficiency disease caused by human immunodeficiency virus (HIV) infection, which predisposes patients to various malignancies (1). Acute promyelocytic leukemia (APL) is a specific subtype of acute myelogenous leukemia (AML), characterized by a chromosomal translocation t(15;17)(q22;q12), resulting in the formation of the PML-RARA fusion gene. This fusion protein blocks cell differentiation and inhibits apoptosis, representing the primary molecular mechanism underlying APL (2, 3). APL typically affects young and middle-aged individuals, with a mean onset age of 44 years. It accounts for 10% to 15% of AML cases, with an incidence rate of approximately 0.23 per 100,000 (2). As a distinct AML subtype, APL presents unique clinical features and treatment responses. When AIDS and APL occur simultaneously, the complexity of diagnosis and treatment increases significantly, posing significant clinical challenges.

2 Case report

A 46-year-old male patient, living with HIV infection for over a decade, had not received treatment for four consecutive years after his diagnosis (Figure 1). The patient self-reported that in 2017, diagnostic findings confirmed progression to the AIDS stage, prompting initiation of antiretroviral therapy (ART). During this period, the patient underwent viral load and $CD4^+$ T-cell count monitoring at the local CDC in their hometown; however, specific reports are unavailable. The patient self-reported a $CD4^+$ T-cell count of approximately 300 cells/μL during ART, still indicating incomplete immune reconstitution. The current treatment regimen comprises tenofovir (TDF), lopinavir/ritonavir (LPV/r), and lamivudine (3TC). In December 2023, the patient presented with dizziness lasting over two months, accompanied by fever, cough, and sputum for two days. A routine blood test revealed a 28% blast cell population in peripheral blood, leading to his immediate hospitalization. Complete bone marrow analysis indicated an increase in promyelocytes, with visible *azurophilic granules* and Auer rods, suggestive of APL (Table 1) (Figures 2A, B). The diagnosis was confirmed by flow cytometry and positive *PML-RARA* fusion gene results (Figures 2C, D).

Upon admission, the patient's $CD4^+$ T-cell count was measured at 329.6 cells/μL (indicating moderate immunosuppression), accompanied by severe pulmonary infection, along with prolonged prothrombin time and activated partial thromboplastin time due to APL, elevated D-dimer levels, granulocytopenia, thrombocytopenia, anemia, and other signs suggestive of disseminated intravascular coagulation (4). Given that the patient was stratified as low-risk for APL prognosis, Treatment was initiated with all-trans retinoic acid (ATRA) ($25mg{\cdot}m^{-2}{\cdot}d^{-1}$) to induce differentiation (while deferring arsenic trioxide (ATO) ($0.16mg{\cdot}kg^{-1}{\cdot}d^{-1}$) due to the severe lung infection). Simultaneously, supportive therapies were administered, including anti-infective measures, bleeding prevention, multiple plasma and platelet transfusions, and anti-HIV treatment. The patient subsequently developed liver dysfunction and leukocytosis (white blood cell count exceeding 10×10^9/L), prompting adjustments in treatment strategy. Concurrently, the patient received comprehensive supportive care comprising: cefotaxime 2.0g IV q12h, piperacillin-sulbactam 4.5g IV q8h (escalated from cefotaxime based on clinical suspicion of extended-spectrum beta-lactamase (ESBL)-producing pathogens), and micafungin 100mg IV qd for antimicrobial prophylaxis; bleeding prevention measures; multiple fresh frozen plasma and platelet transfusions; and antiretroviral therapy for HIV comanagement. During the treatment course, the patient developed hepatotoxicity accompanied by leukocytosis (white blood cell count $>10\times10^9$/L). Therapeutic interventions were escalated with hydroxyurea 0.5g PO bid (days 8–10 post-diagnosis) and idarubicin $8mg/m^2$ IV daily (days 9–11 post-diagnosis) for leukocyte cytoreduction, supplemented by hepatoprotective agents including ursodeoxycholic acid and glutathione infusion. Hydroxyurea (0.5g bid 8–10 days in the course of the disease) and idarubicin ($8mg{\cdot}m^{-2}{\cdot}d^{-1}$ Days 9–11 of the course of disease) were used to reduce leukocytosis, and liver protection measures were implemented. Follow-up exams showed improvement in the lung infection and normalization of liver function, with bone marrow tests performed on day 20 indicating partial remission.

The patient then began ATO ($0.16mg{\cdot}kg^{-1}{\cdot}d^{-1}$) intravenous chemotherapy combined with oral ATRA ($25mg{\cdot}m^{-2}{\cdot}d^{-1}$), continuing with the first treatment course for induction therapy. During this phase, he experienced fever again, and chest CT revealed patchy and nodular density shadows in both lungs. A regimen of meropenem and micafungin was administered, improving the infection, with serial monitoring of $CD4^+$ T-cell counts revealing a nadir of 148.4 cells/μL on day 26 of the disease course. On day 33 of the disease course, a follow-up evaluation was performed. Continued induction therapy led to morphological remission in bone marrow. After another course of ATO ($0.16mg{\cdot}kg^{-1}{\cdot}d^{-1}$) and ATRA ($25mg{\cdot}m^{-2}{\cdot}d^{-1}$), the PML-RARA fusion gene turned negative, and the patient achieved complete remission (CR). At the end of the second treatment course, follow-up testing revealed a $CD4^+$ T-cell count of 369.9 cells/μL. Currently, the patient is undergoing consolidation therapy, showing an increased $CD4^+$ T-cell count, persistent fusion gene negativity, and only mild anemia.

Abbreviations: AIDS, acquired immunodeficiency syndrome; AML, acute myeloid leukemia; APL, acute promyelocytic leukemia; ART, antiretroviral therapy; ATO, arsenic trioxide; ATRA, all-trans retinoic acid; CR, complete remission; ESBL, extended-spectrum beta-lactamase; HIV, human immunodeficiency virus; LPV/r, lopinavir and ritonavir; PLT, platelets; SIV, simian immunodeficiency virus; 6-MP, 6-Mercaptopurine; TDF, tenofovir disoproxil; 3TC, lamivudine; WBC, white blood cell.

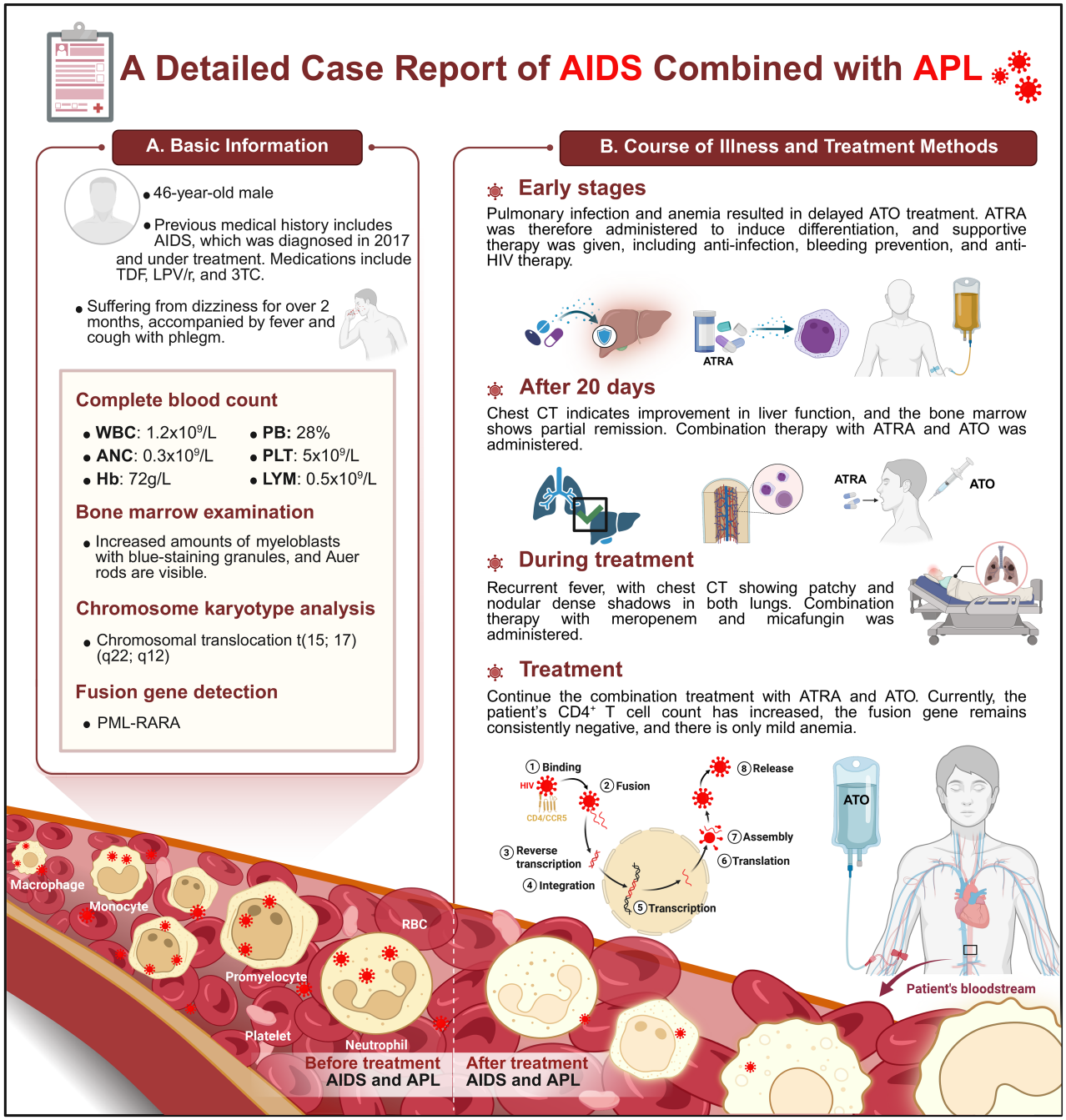

FIGURE 1
Management of acute promyelocytic leukemia in a patient with AIDS: a case study. **(A)** Preliminary diagnosis and basic information. A 46-year-old male with HIV infection for over a decade, untreated for four consecutive years post-diagnosis, progressed to AIDS in 2017. In December 2023, the patient presented with dizziness lasting over two months and fever with cough for two days. After comprehensive testing, including routine blood and bone marrow analysis and genetic examination, acute promyelocytic leukemia (APL) was diagnosed, confirmed by a specific chromosomal translocation t(15;17)(q22;q12) resulting in the PML-RARA fusion gene. **(B)** Disease evolution and treatment process. Upon admission, the patient's severe lung infection and anemia delayed the initiation of ATO intravenous chemotherapy. As his condition improved, ATO chemotherapy was started in combination with oral ATRA induction therapy. However, the patient soon developed a fever, and chest CT revealed multiple patchy and nodular densities in both lungs, prompting the discontinuation of ATO and the introduction of anti-infection treatment with meropenem and micafungin. After induction therapy, bone marrow examination confirmed morphological remission. ATO and ATRA therapy was resumed, and subsequent tests showed a negative PML-RARA fusion gene, achieving complete remission. The patient remains in good condition under ongoing treatment. AIDS, acquired immunodeficiency syndrome; APL, acute promyelocytic leukemia; ATO, arsenic trioxide; ATRA, all-trans retinoic acid; LPV/r, lopinavir and ritonavir; TDF, tenofovir disoproxil; 3TC, lamivudine.

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TABLE 1 Dynamic changes of laboratory indexes in AIDS patients with APL.

Indicators	December 28th	January 6	January 25	February 4
White blood cells ($\times 10^9$/L)	1.2	10.1	1.2	6.1
Neutrophil granulocyte ($\times 10^9$/L)	0.3	3.6	0.9	4
Lymphocyte ($\times 10^9$/L)	0.5	0.9	0.3	1.3
Blood platelet ($\times 10^9$/L)	5	42	51	348
Hemoglobin (g/L)	72	74	71	96
Procalcitonin (ng/ml)	0.17	0.06	–	–
Alanine Aminotransferase (U/L)	20	139	35	28
Aspartate Aminotransferase (U/L)	37	104	25	30
Partial prothrombin activity time (s)	1.13	18.5	30.3	29
Fibrinogen (g/L)	3.78	1.61	4.47	3.6
D-Dimer (μg/ml)	>20000	1663	585	–

3 Discussion

APL presents with severe clinical manifestations, with high risks of bleeding and embolism during onset and induction therapy, potentially leading to fatal outcomes. Over the past three decades, the standardized clinical use of ATRA and ATO has transformed APL into a curable leukemia without the need for hematopoietic stem cell transplantation (5, 6).

A review of 11 previously reported AIDS-related APL cases (7–16) highlights that most patients achieved remission with ATRA alone or in combination with chemotherapy (anthracyclines or arsenic) (Table 2). Only one high-risk patient succumbed to severe disseminated intravascular coagulation. Long-term remission was achieved in most cases through consolidation and maintenance therapy. One patient who discontinued ATRA due to nausea relapsed one year later but achieved a second CR following salvage therapy with ATO (12).

Given the high mortality associated with APL, immediate ATRA treatment is recommended based on cytological and clinical criteria, while awaiting genetic confirmation (3, 4). In this case, the patient's severe lung infection and liver dysfunction posed risks for initial ATO combination therapy. Since 1986, Shanghai Ruijin Hospital has successfully utilized ATRA-induced differentiation therapy for APL, with a CR rate of 75% to 85% for ATRA alone (17). Therefore, we opted for ATRA monotherapy during the early treatment phase. After the infection and liver function improved, ATO was added, and ATRA was continued for one week, achieving morphological remission, as observed in two other cases treated with ATRA alone (7, 8).

In the consolidation phase, we continued ATRA combined with ATO. Given the patient's history of HIV and long-term ART, we carefully evaluated whether this chemotherapy regimen might interfere with his HIV treatment. Previous studies indicate that while ATRA upregulates HIV mRNA transcription in infected human HL-60 cell lines, it does not increase viral replication due to blocked HIV mRNA translation and replication. This mechanism ultimately leads to the apoptosis of HIV-infected cells, reducing the proviral DNA load in a dose-dependent manner (18). Furthermore, ATRA significantly decreased viral replication in primary lymphocytes from HIV-infected patients, suggesting it may also serve as a therapeutic agent against HIV infection (19).

Research from the Chinese Academy of Sciences and Sun Yat-sen University showed that ATO specifically activates latent viral reservoirs without inducing excessive inflammation. In a simian immunodeficiency virus (SIV) model, ATO combined with ART significantly delayed viral rebound post-drug withdrawal, with some monkeys showing no viral rebound after 80 days (compared to 22 days for ART alone). ATO reduced viral reservoirs and enhanced virus-specific immune responses. Moreover, ATO downregulated the expression of CD4 and CCR5 receptors on $CD4^+$ T-cells, essential for HIV entry, thus inhibiting new rounds of viral infection (20).

In conclusion, initiating ATRA treatment based on clinical and morphological criteria can significantly improve outcomes in APL patients, even in complex cases like this. Individualized treatment tailored to the patient's condition may lead to better efficacy. For consolidation and maintenance therapy, further case studies and

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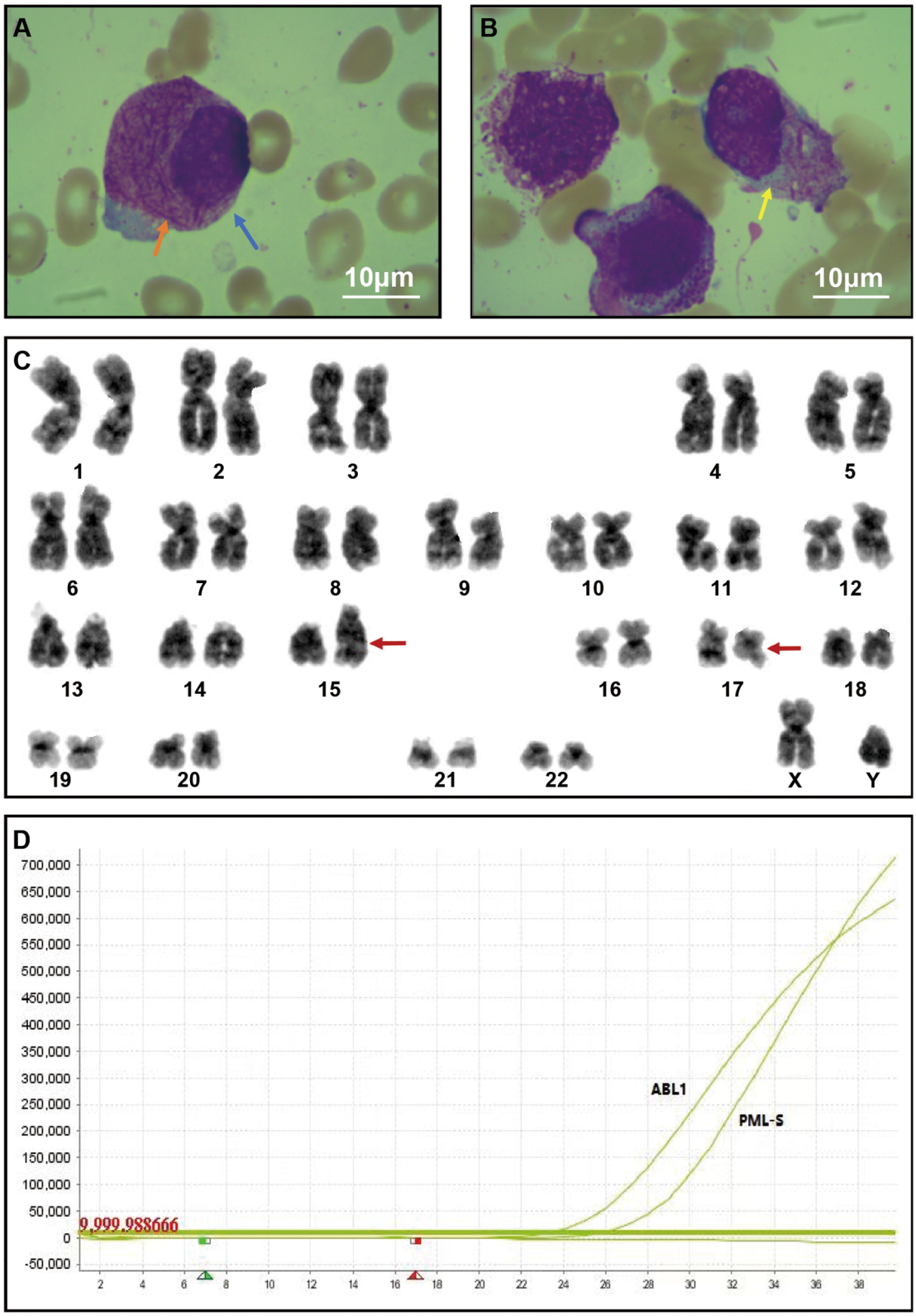

FIGURE 2
Diagnostic findings in acute promyelocytic leukemia: a case report. **(A, B)** Bone marrow examination revealed an increase in promyelocytes (blue arrow in **A**), with some hyper granularity with azurophilic granules (yellow arrow in **B**) with Auer rods visualized (orange arrow in **(A)**). **(C)** Cytogenetic analysis by G-banding showed the karyotype: 46, XY, t(15;17)(q22;q21), wherein chromosomal translocations are visually marked by red arrows. **(D)** The presence of the mutant PML-RARA fusion gene was confirmed through fluorescent PCR analysis.

TABLE 2 Review of previous cases and a detailed case report on APL combined with AIDS.

Case	Age/sex	Time from diagnosis of AIDS to discovery of APL	Initial WBC/PLT count ($\times 10^9$/L)	APL risk groups	AIDS treatment regimen at the time of diagnosis of APL	Induction therapy regimen	Response to induction therapy	Consolidation treatment	Maintenance treatment	Survival outcome/duration of follow-up	Ref
1	30/M	2 years	4.8/2	Moderate risk	NA	ATRA alone	Morphological CR was achieved on day 29	Daunorubicin, cytarabine/mitoxantrone, and cytarabine	NA	Survival/8 months	(7)
2	36/M	coordinate expression	4/NA	Low risk	NA	ATRA alone	Morphological remission was achieved, but relapse occurred 303 days later	NA	Mitoxantrone/6-MP	Death/350 days	(8)
3	27/M	8 years	8/19	Moderate risk	Indinavir was replaced by nelfinavir, 3TC, and zidovudine was replaced by stavudine	ATRA, idarubicin, and cytarabine	Morphological CR was achieved at 30 days. Cytogenetic and molecular CR was achieved at week 9	High-dose cytarabine	ATRA/mitoxantrone/6-MP	Survival/40 months	(9)
4	46/F	2 years	5.1/1	Moderate risk	Efavirenz, TDF, 3TC	ATRA and idarubicin	Molecular CR	ATRA, idarubicin/ATRA, and mitoxantrone	ATRA/mitoxantrone/6-MP	Survival/21 months	(10)
5	35/M	10 years. The patient has a history of primary central nervous system lymphoma and achieved CR after whole brain radiation therapy.	1.6/28	Moderate risk	Stavudine and LPV/r	ATRA and idarubicin	Morphological and molecular CR	ATRA	NA	Survival/14 months	(11)
6	37/M	7 years	1.6/112	Low risk	3TC, nevirapine, and desanosine	ATRA and idarubicin	Morphological and cytogenetic CR was achieved on day 77	NA	ATRA (poor adherence due to nausea)	Relapsed 1 year later and was treated with ATO as salvage therapy, achieving a second CR. Survival/17 months	(12)
7	43/F	coordinate expression	40.7/15	High risk	Azanavir was replaced with fosanavir, TDF/emtricitabine, and lategravir	ATRA and idarubicin	Day 29 Morphological and molecular CR	ATRA, idarubicin/ATRA, and mitoxantrone	ATRA/mitoxantrone/6-MP	Survival/8 months	(13)

(Continued)

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TABLE 2 Continued

Case	Age/sex	Time from diagnosis of AIDS to discovery of APL	Initial WBC/PLT count ($\times 10^9$/L)	APL risk groups	AIDS treatment regimen at the time of diagnosis of APL	Induction therapy regimen	Response to induction therapy	Consolidation treatment	Maintenance treatment	Survival outcome/duration of follow-up	Ref
8	32/M	5 months	4/22	Moderate risk	Abacavir/3TC, darunavir, r	ATRA, idarubicin, and cytarabine	CR was achieved on day 40	ATRA, idarubicin/ ATRA, and mitoxantrone	ATRA/ mitoxantrone/6-MP	Survival/38 months	(14)
9	46/M	5 months	10/19	Moderate risk	Raltegravir, emtricitabine, TDF	ATRA and idarubicin	Morphological remission by day 31	ATRA, idarubicin/ ATRA, and mitoxantrone	Maintenance therapy was not performed because of liver dysfunction	Survival/30 months	(14)
10	49/M	18 years	89/55	High risk	3TC, TDF, LPV/ r, atazanavir	ATRA, cytarabine, and daunorubicin (not available for idarubicin)	non-remission	NA	NA	Died on the 10th day after admission due to refractory DIC with severe pulmonary hemorrhage	(15)
11	67/M	5 days	1.2/45	Low risk	Biktarvy	ATRA and ATO	The specific duration of remission was not specified	ATR and ATO	ATRA and ATO	Survival/NA	(16)
12	46/M	10years	1.2/5	Low risk		After 20 days of ATRA treatment alone (when liver function improved), ATO was added	Morphological remission was achieved on day 32	ATR and ATO	ATRA and ATO	Survival/more than 7 months	In this example

AIDS, acquired immunodeficiency syndrome; APL, acute promyelocytic leukemia; ATO, arsenic trioxide; ATRA, all-trans retinoic acid; CR, complete remission; LPV/r, lopinavir and ritonavir; PLT, platelets; 6-MP, 6-Mercaptopurine; TDF, tenofovir disoproxil; 3TC, lamivudine; WBC, white blood cell.
NA, Not Applicable.

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multicenter clinical trials are needed to confirm whether ATRA combined with ATO is the optimal approach. Continuous optimization of treatment protocols is essential to improve patient outcomes and quality of life.

Data availability statement

The original contributions presented in the study are included in the article/supplementary material. Further inquiries can be directed to the corresponding authors.

Ethics statement

Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

SD: Funding acquisition, Writing – review & editing. CA: Conceptualization, Writing – original draft. JS: Conceptualization, Visualization, Writing – original draft. HX: Visualization, Writing – review & editing. YX: Funding acquisition, Writing – review & editing.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Generative AI statement

The author(s) declare that no Generative AI was used in the creation of this manuscript.

Publisher's note

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