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Case Reports

Acute promyelocytic leukemia in pregnancy: Balancing treatment and toxicity

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Acute promyelocytic leukemia (APL) during pregnancy is an exceptionally rare event, estimated at fewer than 1 per 100,000 pregnancies, reflecting the convergence of a hematologic emergency and the therapeutic challenges of gestation [1].

We report a 37-year-old woman at 22 weeks of gestation, referred for lower-limb edema and extensive bruising. Laboratory workup showed severe pancytopenia with early coagulopathy. Bone marrow examination confirmed APL by flow cytometry, cytogenetics, and molecular studies, which demonstrated the t(15;17) translocation and the PML-RARA fusion transcript [2].

Multidisciplinary assessment led to treatment with all-trans retinoic acid (ATRA); arsenic trioxide (ATO) was withheld for its teratogenic potential [2–4]. The course was complicated by differentiation syndrome, disseminated intravascular coagulation, and progressive obstetric deterioration, requiring corticosteroids, ICU admission, and dynamic adjustments [2,3,5,6]. Cesarean delivery occurred at 30 weeks of gestation with intensive transfusion support. The patient is now on ATRA/ATO consolidation with favorable evolution; the newborn, after prematurity-related complications, has been discharged from the neonatal ICU.

This case, together with a focused review of the available literature, illustrates the complexity of managing APL during pregnancy and highlights continuous inpatient care, multidisciplinary collaboration, and real-time decision-making as central to its management [1-3].

INTRODUCTION

Acute promyelocytic leukemia (APL) during pregnancy arises from the overlap of two low-incidence phenomena: acute leukemia in pregnancy, estimated at 1 in 75,000–100,000 pregnancies, and APL itself, which accounts for only 5–10% of acute myeloid leukemia cases. Together, these figures place the incidence of APL in pregnancy at fewer than 1 per 100,000 pregnancies.[1,2]

APL is defined by the t(15;17)(q24;q21) translocation and the resulting PML-RARA fusion gene, which confers marked sensitivity to differentiating agents such as all-trans retinoic acid (ATRA) and arsenic trioxide (ATO) and has transformed APL into one of the most curable hematologic malignancies, with cure rates approaching 90%.[2] Despite this, early mortality remains a major concern, driven chiefly by a coagulopathy that combines consumption of coagulation factors, hyperfibrinolysis, and proteolytic activity from leukemic promyelocytes, accounting for early death — predominantly intracranial hemorrhage — in 10–30% of patients.[2-4] Because of this risk, APL constitutes a hematologic emergency requiring immediate treatment initiation, even when the diagnosis is only suspected on clinical grounds.

Management becomes markedly more complex when APL arises during pregnancy. ATRA and ATO, the cornerstone agents of therapy, are both teratogenic — particularly during the first trimester — forcing clinicians to weigh maternal disease control against fetal risk at every gestational stage.[2,5,6] Treatment may additionally precipitate differentiation syndrome, a potentially fatal complication (mortality ~11%) characterized by fever, respiratory distress, pulmonary infiltrates, effusions, and hypotension, attributed in part to ATRA-induced upregulation of endothelial-permeability mediators such as cathepsin G[2,5,7-9]; in a pregnant patient, this syndrome can compound the endothelial dysfunction and coagulation disturbances already inherent to gestation.

Managing APL during pregnancy therefore requires a multidisciplinary approach spanning hematology, obstet-

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Table 1. Previous reports of APL during pregnancy

Reference (year)	Design / N	Treatment	Key maternal / fetal outcome
Hoffman et al.,[11] 1995	Case report + literature review (23 pregnancies)	ATRA / chemotherapy (late pregnancy)	Early report establishing feasibility of ATRA and chemotherapy after the first trimester; CR in 72% of treated patients.
Consoli et al.,[12] 2004	3 cases	ATRA followed by chemotherapy	Emphasized individualized balance of maternal/fetal risk according to coagulopathy severity and gestational age.
Culligan et al.,[13] 2007	3 cases	Variable (ATRA ± chemotherapy)	Illustrated divergent therapeutic decisions and outcomes depending on gestational age and disease severity.
Yang & Hladnik,[5] 2009	Review, 42 patients	ATRA, anthracyclines, antimetabolites	CR 83%; first-trimester therapy linked to malformation/ abortion; 2nd/3rd-trimester outcomes more favorable.
Ganzitti et al.,[14] 2010	Case report	ATRA-based	Favorable maternal and neonatal outcome with obstetric-focused multidisciplinary management.
Troitskaia et al.,[15] 2013	9 patients	ATRA / chemotherapy	All infants alive and healthy; maternal course more variable, with late relapse in ~33%.
Sanz et al. (PETHEMA),[16] 2015	14 patients	ATRA + idarubicin	CR 92% among evaluable patients; 8 of 9 late-pregnancy patients delivered a healthy infant.
Verma et al.,[17] 2016	Systematic analysis, 71 patients	Mixed (ATRA, chemotherapy, or both)	Preterm delivery in 46%; abortion or intrauterine fetal death in roughly one-third of pregnancies.
Li et al.,[18] 2019	2 cases	ATRA-based	New-onset APL in pregnancy; underscored the urgency of prompt ATRA initiation.
Santolaria et al.,[2] 2020	Systematic review, 96 patients	ATRA alone (32%), ATRA + chemotherapy (43%)	CR 89%; pregnancy loss concentrated in the first trimester; favorable neonatal outcomes beyond it.
Cochet et al.,[19] 2020	Case report	ATRA + ATO	First reported ATRA/ATO combination during pregnancy; suggests possible late-pregnancy use, though evidence remains very limited.
Puttirangsan et al.,[20] 2025	Case + literature review, 97 patients	ATRA + modified-dose idarubicin	Third-trimester diagnosis; uncomplicated vaginal delivery and healthy neonate; review confirmed best maternal/ fetal survival with third-trimester treatment.

CR: complete remission

rics, and critical care, with discussion of pregnancy continuation, gestational-age-adapted treatment selection, and close surveillance for both hematologic and obstetric complications. Disseminated intravascular coagulation (DIC), itself responsible for up to 25% of maternal deaths,[10] is amplified by the coexistence of APL-related coagulopathy and the hemostatic changes of pregnancy, further heightening clinical risk.[2,4,5,10] We report a case that illustrates these challenges and the value of continuous multidisciplinary inpatient management, and we review the available literature on APL during pregnancy to contextualize our findings (Tables 1 and 2).

CLINICAL CASE

A 37-year-old woman with an obstetric history of four pregnancies (two spontaneous abortions and two live births) was referred to our center at 22 weeks of gestation with a strong clinical suspicion of acute leukemia. She had initially presented to the emergency department with lower-limb edema and extensive hematomas.

Her history was notable for a 13-year chronic liver disease of undetermined etiology, with hepatomegaly, splenomegaly, and cytopenias previously attributed to portal hypertension after an unremarkable outside bone marrow aspirate.

Initial laboratory evaluation revealed macrocytic anemia (hemoglobin 9.4 g/dL; mean corpuscular volume, 99.8 fL), leukopenia with neutropenia (386 leukocytes/mm^3, 38% neutrophils), and severe thrombocytopenia (27,900/mm^3). Although the initial coagulation profile was within normal limits, fibrin degradation products were elevated at 28.6 µg/mL. Together with the clinical presentation, these findings raised a strong suspicion of APL.

Bone marrow examination confirmed the diagnosis: flow cytometry demonstrated an aberrant promyelocyte population comprising 59.65% of total cellularity, while cytogenetic and molecular studies confirmed the t(15;17) translocation and the PML-RARA fusion transcript.

In view of the hematologic emergency posed by APL and the coexistence of an ongoing pregnancy, a multidisciplinary team was convened, involving hematology, internal medicine, critical care, and obstetrics. Following comprehensive counseling on maternal and fetal risks, the patient elected to initiate treatment and continue the pregnancy. ATRA was started together with dexamethasone as prophy-

Table 2. Clinical dilemmas in the management of APL during pregnancy, and recommendations

Clinical issue	Dilemma	Recommendation (literature and present case)
Timing of diagnosis by trimester	Urgency of treatment *vs.* teratogenic risk	First trimester: individualized discussion of termination *vs.* delayed treatment given high malformation/abortion risk.[2,5] Second/third trimester: prompt ATRA-based induction, as performed in this case (22 weeks).
ATRA initiation	Teratogenic in the first trimester but essential for coagulopathy control	Initiate promptly beyond the first trimester, even on clinical suspicion alone, given the survival benefit.[2,5,11-13,16]
ATO use	Strong teratogenic/ embryotoxic potential	Avoid throughout pregnancy; defer to postpartum consolidation, as done in this case. Only isolated late-pregnancy reports exist.[2,5,6,19]
Anthracycline addition	Cardiotoxicity/fetal growth concerns *vs.* improved remission rates	Consider idarubicin beyond the first trimester when ATRA alone is insufficient, with fetal cardiac monitoring.[16,20]
Differentiation syndrome	May mimic obstetric/ infectious complications; treatment may require ATRA interruption	Corticosteroid prophylaxis from ATRA initiation; escalate dose and interrupt ATRA if the syndrome develops, with ICU-level monitoring, as occurred in this case.
DIC / coagulopathy monitoring	Balancing transfusion needs against the hemostatic changes of pregnancy	Daily coagulation monitoring; maintain platelets >30–50 × 10^9^/L and fibrinogen >100–150 mg/dL with targeted component transfusion.
Obstetric surveillance	Placental/cervical complications may be difficult to distinguish from treatment effects	Serial obstetric ultrasound and cervical assessment from diagnosis; low threshold for tocolysis and antenatal corticosteroids if preterm labor threatens.
Timing of delivery	Prematurity risk *vs.* maternal/fetal deterioration	Individualize; expedite delivery when maternal hematologic or obstetric status deteriorates despite treatment, as in this case (Caesarean at 30 weeks).
Breastfeeding	ATRA/ATO excreted in breast milk	Contraindicated during consolidation; counsel the patient accordingly postpartum.
Care model	Fragmented care across specialties risks delayed recognition of complications	Continuous inpatient, multidisciplinary care (hematology, obstetrics, critical care, neonatology) from diagnosis to delivery, as emphasized by this case.

laxis against differentiation syndrome; ATO was deferred due to its teratogenic potential.

From the time of diagnosis, the patient remained continuously hospitalized, enabling close surveillance of both disease progression and obstetric evolution. Throughout this period, the major complications of the disease and its treatment were systematically documented.

Fifteen days after initiating ATRA, the patient developed leukocytosis with monocytic predominance, interpreted as early differentiation syndrome, which prompted escalation of corticosteroid therapy. Daily monitoring of coagulation parameters and tumor lysis markers was maintained, alongside targeted transfusion support.

On day +27, obstetric assessment revealed cervical shortening (10 mm) and placental abnormalities in the setting of uterine contractions. Antenatal corticosteroids were administered to promote fetal lung maturation, together with tocolytic therapy and progesterone supplementation.

On day +30, the patient developed critical hypofibrinogenemia (fibrinogen nadir 59 mg/dL). Fibrin degradation products rose to a peak of 217 µg/mL, and coagulation factors deteriorated progressively, with factor V decreased to a minimum value of 40%. Transfusion support with irradiated and leukocyte-filtered blood products was initiated to maintain platelet counts above 30,000/mm^3 and hemoglobin above 8 g/dL; fibrinogen replacement therapy was administered to sustain plasma levels above 150 mg/dL. In this context, DIC secondary to severe differentiation syndrome was suspected.

Despite these complications, bleeding manifestations remained limited to minor events, including hematomas at varying stages of evolution, mild bleeding from venipuncture sites, gingival bleeding, and epistaxis. No major hemorrhagic episodes occurred, owing to intensive hematologic support. Management required temporary discontinuation of ATRA, initiation of high-dose dexamethasone, and ICU admission for hemodynamic monitoring and correction of the coagulopathy.

Following resolution of differentiation syndrome and stabilization of coagulation parameters, ATRA was resumed. However, the patient subsequently developed progressive obstetric complications, including worsening placental abnormalities and preeclampsia. Given the increasing maternal and fetal risk, delivery by preterm Caesarean section was performed at 30 weeks of gestation. The procedure required intensive transfusion support, administration of tranexamic acid and magnesium sulfate, and temporary interruption of ATRA.

Histopathological examination of the placenta revealed findings consistent with maternal vascular malperfusion and thrombotic pathology, including decidual vasculopathy and a massive subchorionic thrombohematoma involving the entirety of the placental disc.

Further work-up during hospitalization excluded viral and autoimmune hepatitis and demonstrated portosystemic shunting with sinusoidal obstruction on liver biopsy; hepatic synthetic function remained preserved throughout (factor V 63–68%), and this comorbidity did not significantly alter APL management.

In the immediate postpartum period, the patient continued ATRA therapy and subsequently initiated consolidation with ATO; breastfeeding was contraindicated accordingly. She is currently under outpatient follow-up, with good treatment tolerance and no new hematologic complications. Bone marrow evaluation performed 30 days after the completion of induction therapy showed no evidence of residual disease, confirming complete remission.

The newborn had been discharged from the neonatal ICU at the time of this report after complications related to prematurity, including bronchopulmonary dysplasia and a ventricular septal defect.

DISCUSSION

Acute leukemia during pregnancy is rare, occurring in an estimated 1 per 75,000–100,000 pregnancies, and APL — representing only 5–10% of acute myeloid leukemia — is correspondingly rarer still, with fewer than 1 case per 100,000 pregnancies.[1,2] The available evidence is limited to case reports, small series, and two systematic reviews (Table 1), yet several consistent themes emerge that directly informed the management of our patient.

Early reports such as Hoffman *et al.*[11] and Consoli *et al.*[12] first demonstrated that maternal remission and live birth were achievable when ATRA-based treatment was administered outside the first trimester, establishing the therapeutic template still followed today. Larger series have since refined outcome expectations: Yang and Hladnik,[5] reviewing 42 patients, reported an overall complete remission (CR) rate of 83%, with first-trimester therapy associated with malformation and pregnancy loss, while second- and third-trimester treatment yielded substantially better outcomes. The largest available synthesis, the systematic review by Santolaria *et al.*[2] of 96 cases, reported a maternal CR rate of 89%, with pregnancy loss concentrated in the first trimester and generally favorable neonatal outcomes thereafter, aside from rare early neonatal deaths.

Obstetric and fetal complications, however, remain frequent even with appropriate second- or third-trimester management. Verma *et al.*,[17] in a systematic analysis of 71 patients, found preterm delivery in 46% of pregnancies and abortion or intrauterine fetal death in roughly one-third — figures that resonate with our patient's own course of cervical shortening, placental vascular malperfusion, and eventual preterm Caesarean delivery at 30 weeks. Troitskaia *et al.*[15] similarly described favorable short-term neonatal outcomes alongside a more variable maternal course, including late relapse in about one-third of patients, a reminder that a good delivery outcome does not guarantee durable maternal remission.

Regarding treatment intensity, the PETHEMA trial experience reported by Sanz *et al.*,[16] combining ATRA with idarubicin in 14 pregnancies, achieved a 92% CR rate among evaluable patients and healthy outcomes in most infants delivered in late pregnancy, supporting anthracycline addition beyond the first trimester when clinically indicated. ATO, by contrast, remains generally avoided during pregnancy given its known embryotoxic potential; only isolated reports, such as Cochet *et al.*,[19] describe an ATRA-ATO combination in late pregnancy, and this remains an area of substantial uncertainty rather than an established recommendation.[2,5,6] Consistent with this caution, ATO was withheld throughout gestation in our patient and introduced only postpartum, during consolidation.

More recent case reports, including Li *et al.*[18] and Puttirangsan *et al.*,[20] reinforce the urgency of prompt ATRA initiation once APL is clinically suspected — even before molecular confirmation — and describe good maternal and neonatal outcomes with third-trimester diagnosis managed by immediate ATRA-based induction; the latter's accompanying literature review of 97 cases further confirmed that survival for both mother and fetus is highest when treatment is initiated in the third trimester.

Our case is consistent with this second/third-trimester consensus: continuous ATRA with corticosteroid prophylaxis, deferred ATO, and anthracycline-free induction resulted in maternal CR and a surviving, though premature, infant. Its more distinctive contribution is the patient's uninterrupted hospitalization from diagnosis to delivery, which allowed continuous, real-time documentation of the interplay between differentiation syndrome, evolving coagulopathy, and obstetric deterioration — an observational granularity rarely available in the largely retrospective literature summarized in Table 1. This continuous multidisciplinary surveillance directly informed the timing of corticosteroid escalation, temporary ATRA interruption, and ultimately the decision of preterm delivery. The clinical issues this course raised, together with the corresponding recommendations drawn from the literature and from our own experience, are summarized in Table 2.

CONCLUSIONS

APL during pregnancy represents a highly complex clinical scenario arising from the simultaneous management of a dynamic hematologic malignancy, its treatment, and the physiological demands of gestation — each of which may compound the others. This case, together with the focused literature review presented in Tables 1 and 2, underscores the importance of early treatment initiation, gestational-age-adapted therapeutic selection, and rigorous, continuous monitoring of both the hematologic disease and the pregnancy.

Continuous hospitalization ensured permanent access to a multidisciplinary team, enabling real-time decision-making in response to hematologic and obstetric complications

alike. This coordinated approach proved essential for the timely adjustment of therapeutic interventions and illustrates the value of this model of care in high-complexity clinical settings.

COMPETING INTERESTS

The authors declare that they have no competing interests relevant to this work.

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CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical information.

DATA AVAILABILITY

All relevant data supporting the findings of this study are included within the article. Additional information is available from the corresponding author upon reasonable request.

AUTHOR CONTRIBUTIONS

Conceptualization: Catalina Bosch (Equal), Eugenia Pérez=Lloveras (Equal), María de los Milagros Blanco-Ziegler (Equal), Erika-Barbara Brulc (Equal), María-Adela Aguirre (Equal). Data curation: Catalina Bosch (Equal), Eugenia Pérez=Lloveras (Equal), María de los Milagros Blanco-Ziegler (Equal), Erika-Barbara Brulc (Equal), María-Adela Aguirre (Equal). Writing – original draft: Catalina Bosch (Equal), Eugenia Pérez=Lloveras (Equal), María de los Milagros Blanco-Ziegler (Equal), Erika-Barbara Brulc (Equal), María-Adela Aguirre (Equal). Writing – review & editing: Catalina Bosch, Eugenia Pérez=Lloveras (Equal), María de los Milagros Blanco-Ziegler (Equal), Erika-Barbara Brulc (Equal), María-Adela Aguirre (Equal).

USE OF ARTIFICIAL INTELLIGENCE (AI)

To prepare this manuscript, the authors used Claude (Anthropic) as a writing assistance tool to support translation of the manuscript from its original Spanish version into English, improve language and phrasing, integrate and organize the literature review, and assist in summarizing published data into tables. The literature search, case description, interpretation of the findings, and all scientific content were performed by the authors. All AI-assisted output was carefully reviewed, edited, and verified by the authors, who take full responsibility for the accuracy and integrity of the manuscript.

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