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Case Report

LEUKEMIA CUTIS AND ALL-TRANS RETINOIC ACID–INDUCED MYOCARDITIS IN ACUTE PROMYELOCYTIC LEUKEMIA

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Acute promyelocytic leukemia (APL) is frequently curable in the modern era using the chemotherapy-free regimen of all-trans retinoic acid (ATRA) and arsenic trioxide (ATO). However, rare disease manifestations and treatment complications may threaten these outcomes by requiring intensification or abbreviation of therapy. We present a unique case of a 36-year-old male with newly diagnosed low-risk APL with biopsy-confirmed leukemia cutis and isolated ATRA-associated myocarditis during induction therapy. Both APL leukemia cutis and ATRA-associated myocarditis are exceedingly rare, with each having less than 50 published cases to date. This report offers a comprehensive review of the literature, underscoring the importance of a comprehensive diagnostic evaluation and individualized care to ensure outstanding long-term outcomes for patients with APL.

INTRODUCTION

Acute promyelocytic leukemia (APL) represents up to 10% of all acute myeloid leukemia (AML) cases, predominantly affecting young adults, with a median age at diagnosis of 40. Remarkable advances in treatment have made APL highly treatable and often curable with a chemotherapy-free approach.[1] Unlike other AML subtypes, risk-stratification of APL relies solely on the total white blood cell count (WBC) at diagnosis. Imaging or cerebrospinal fluid evaluation is guided by patients' symptoms, as extramedullary disease (EMD) in newly diagnosed APL is exceedingly rare. For low-risk APL (WBC < $10x10^9$/L), treatment with all-trans retinoic acid (ATRA) and arsenic trioxide (ATO) yields excellent long-term outcomes. Differentiation syndrome (DS) is a common adverse event associated with ATRA and can be characterized by leukocytosis, fevers, hypotension, volume overload, pulmonary infiltrates, pleuropericardial effusions, and renal failure.[2] If recognized early, DS can be managed effectively with glucocorticoids and cytoreductive agents such as hydroxyurea if concurrent leukocytosis is present.[2] While other ATRA-ATO complications are rare, treatment-emergent adverse events may be misattributed to other causes. Herein, we present a case of a patient with newly diagnosed APL and biopsy-confirmed leukemia cutis who was successfully treated with ATRA-ATO despite developing isolated ATRA-associated myocarditis.

CASE

A 36-year-old male presented for evaluation of progressive fatigue, bruising, and mucocutaneous bleeding. His physical exam was notable for a small violaceous plaque with central eschar on the dorsum of the right hand. Interestingly, his social history included training special forces' canines using live rabbits. He was found to have pancytopenia (WBC $4.9x10^9$/L, absolute neutrophils $0.6x10^9$/L, hemoglobin 8.2 g/dL, platelets $5x10^9$/L), with 4.3% promyelocytes and 33.6% circulating blasts, concerning for APL. The patient was promptly started on ATRA.

Bone marrow biopsy showed hypercellularity (>95%) with 49% abnormal promyelocytes (bright CD33+, partial dim CD34+, dim CD45+, CD117+, HLA-DR-). t(15;17)/*PML*::*RARA* FISH was positive in 33.5% cells, confirming low-risk APL. Due to the patient's history of rabbit handling and concern for a zoonotic skin infection, a punch biopsy of the right-hand lesion was performed, revealing an infiltrate of large, myeloperoxidase-positive cells morpho-

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b Justin T. Kelley contributed clinical images for the generation of Figure 1

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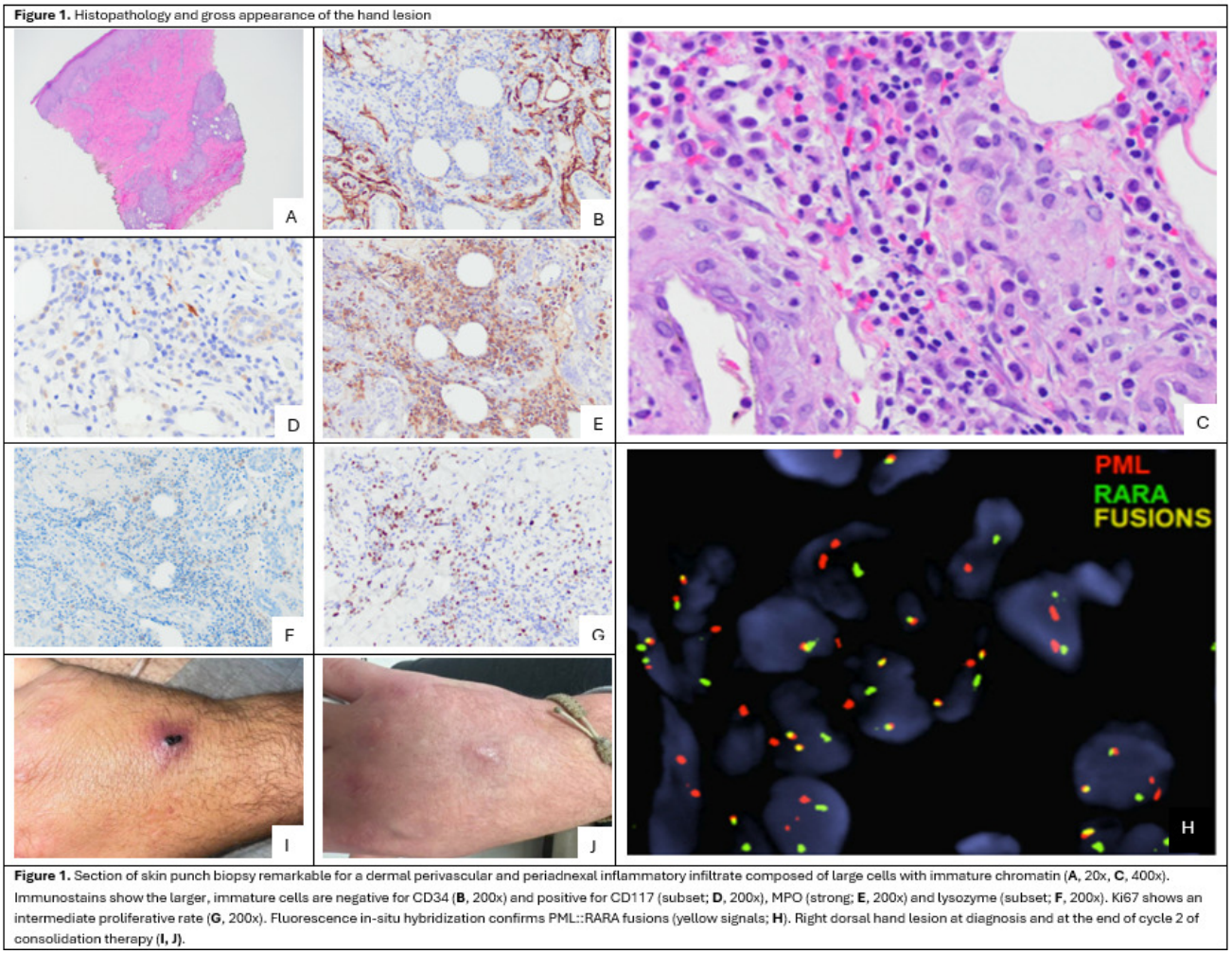

Figure 1. Section of skin punch biopsy remarkable for a dermal perivascular and periadnexal inflammatory infiltrate composed of large cells with immature chromatin (**A**, 20x, **C**, 400x). Immunostains show the larger, immature cells are negative for CD34 (**B**, 200x) and positive for CD117 (subset; **D**, 200x), MPO (strong; **E**, 200x) and lysozyme (subset; **F**, 200x). Ki67 shows an intermediate proliferative rate (**G**, 200x). Fluorescence in-situ hybridization confirms PML::RARA fusions (yellow signals; **H**). Right dorsal hand lesion at diagnosis and at the end of cycle 2 of consolidation therapy (**I, J**).

logically identified as promyelocytes. Positive t(15;17)/*PML*::*RARA* FISH on this tissue sample confirmed cutaneous APL (Figure 1).

The patient was started on ATRA and ATO induction, along with prednisone 50mg (0.5 mg/kg) daily for DS prophylaxis. The patient developed leukocytosis to $12.9x10^9$/L on day 6, so hydroxyurea was added for further prophylaxis against DS from days 6-15 . A prednisone taper was initiated on day 24.

Due to the presence of EMD at diagnosis, a lumbar puncture was performed on day 26 and was negative for CNS involvement. Also on day 26 of induction, the patient developed chest tightness and pain, nonspecific T-wave inversions on electrocardiogram, and a troponin-I elevation to 2.46 ng/dL. Given concern for therapy-induced myocarditis, ATRA was withheld, and prednisone was escalated back to 40 mg daily, resulting in resolution of chest pain. Transthoracic echocardiogram showed normal ejection fraction, chamber size, and wall motion. Regadenoson stress MRI showed no vasodilator-induced ischemia but did reveal a large area of subepicardial gadolinium enhancement consistent with active inflammation. Cardiac PET/CT revealed diffusely increased uptake in the left ventricular myocardium, highest in the lateral wall, consistent with myocarditis.

A bone marrow biopsy performed on day 28 showed no morphologic evidence of leukemia, and t(15;17)/*PML*::*RARA* FISH was negative. The right-hand skin lesion was nearly healed, leaving behind a depressed pink plaque with central heme crust consistent with healing scar. Due to laboratory normalization and continued symptom resolution, the patient was discharged on an extended prednisone taper and off ATRA on day 35.

Upon discharge, the patient abruptly discontinued prednisone due to insomnia, without recurrence of chest pains, despite not receiving any additional myocarditis-directed therapy. ATRA-ATO consolidation began on day 60, 34 days after chest pain onset. The patient has since received three cycles of ATRA-ATO consolidation at standard doses without chest pain recurrence. The hand lesion has fully healed. At the time of writing, the patient remains in MRD-negative remission by reverse transcription quantitative real-time polymerase chain reaction (RT-qPCR) at 16 months from diagnosis, and he remains without any new skin lesions.

DISCUSSION

While APL has become a largely curable disease, with its non-intensive induction regimen allowing successful treat-

Table 1. Reported cases of Acute Promyelocytic leukemia cutis

Publication	Age/Sex	Timing of LC dx	Time from dx	Therapy prior to LC dx	Therapy after LC dx	Other sites of EMD?
Collinge, 2018 [3]	49F	Diagnosis	n/a	1L: ATRA-ATO	n/a	n/a
Balasubramanian, 2018 [4]	30F	Relapse	9 mo	1L: ATRA+ATO	DNR+ATO+ATRA, autoSCT	
Shvartzbeyn, 2012 [5]	46M	Diagnosis	n/a	1L: ATRA+IDA+dexamethasone	n/a	n/a
Markowski, 2007 [7]	63F	Diagnosis	n/a	1L: DNR+ATRA	n/a	n/a
Wrede, 2005 [6]	46M	Relapse	7 yr	1L: ATRA+chemotherapy	RT, ATO	CNS, soft tissues, lymph nodes
	37M	Relapse	1 mo	1L: ATRA+chemotherapy	n/a	n/a
	24F	Relapse	4.5 yr	1L: ATRA+chemotherapy	IT chemo, ATO, RT, autoSCT	CNS, breast
Sanz, 2002 [2]*						
Liso, 1998	48M	Relapse	13 mo	1L: IDA; 2L: MTZ+Ara-C	DNR+Ara-C	n/a
	22F	Relapse	20 mo	1L: IDA+Ara-C	ATRA+Ara-C+MTZ, alloSCT	n/a
	25F	Relapse	1 yr	1L: DNT+Ara-C	DNR+Ara-C	n/a
	44M	Relapse	11 mo	1L: ATRA+IDA	MTZ+etoposide+Ara-C	n/a
	45M	Diagnosis	n/a	1L: ATRA+IDA	n/a	n/a
Kumar, 1997	22F	Relapse	5 mo	n/a	n/a	n/a
Del Rio, 1997	28F	Relapse	7 mo	n/a	n/a	n/a
Ueda, 1997	66M	Relapse	8 mo	1L: DNR+Ara-C+6-MP+ATRA; 2L: DNR+MTX+vidarabine+ATRA	n/a	n/a
Selleri, 1996	31F	Relapse	1.5 yr	1L: IDA+Ara-C	ATRA, autoSCT	n/a
Wiernik, 1996	25F	Relapse	4 mo	1L: DNR+Ara-C; 2L: paclitaxel	n/a	n/a
Nagao, 1996	30F	Relapse	>3 yr	Multiple: DNR, Ara-C, MTX, 6-MP, prednisone, ATRA	ATRA: LDAC, VP-16	n/a
Lederman, 1995	46F	Relapse	6 mo	1L: MTZ, Ara-C, etoposide	Corticosteroids, ATRA, RT	CNS
Bekassy, 1995	24F	n/a	n/a	n/a	n/a	n/a
Thomas, 1994	68F	Relapse	10 mo	n/a	n/a	n/a
Weiss, 1994	31M	Relapse	1 yr	1L: ATRA, Ara-C	ATRA	Lymph nodes
	33M	Relapse	13 mo	1L: ARTA, DNR+ Ara-C; 2L: ATRA	Ara-C, etoposide, MTZ	
Giralt, 1994	23M	Relapse	1 yr	1L IDA+ Ara-C, 2L: IDA+ ATRA	chemotherapy, autoSCT	CNS
	35M	Relapse	3 mo	1L: DNR, Ara-C; 2L: ATRA+IDA+alloSCT	ATRA+RT	n/a
	47F	Relapse	2 mo	1L: IDA 2L: DNR+Ara-C, 3L: ATRA	n/a	n/a
Longacre, 1993	19M	Relapse	n/a	n/a	chemotherapy	n/a
Niazi, 1991	26M	Relapse	11 mo	1L: Ara-C+amsacrine; alloSCT	RT	CNS
	44M	Relapse	n/a	1L: Ara-C+amsacrine; alloSCT	RT; amsacrine+Ara-C, alloSCT	n/a
Baer, 1989	59F	Relapse	>3 yr	n/a	RT	Liver, spleen
Bernengo, 1975	82M	Diagnosis	n/a	n/a	none	n/a
Matsumoto, 1978	21F	n/a	n/a	n/a	n/a	n/a
Uematsu, 1970	34M	Diagnosis	n/a	n/a	n/a	n/a

*Includes all below listed publications. Allo/autoSCT=allogeneic/autologous stem cell transplant; ATRA-ATO=all-trans retinoic and acid arsenic trioxide; Ara-C=cytarabine; CNS=central nervous system; DNR=daunorubicin; dx = diagnosis, F=Female; IDA=idarubicin; IT=intrathecal; L=line of therapy; LC=leukemic cutis; M=male; mo=months; MTX=methotrexate; MTZ=mitoxantrone; n/a = not applicable; RT=radiation therapy; 6-MP=6-mercaptopurine

ment outside of specialized academic centers,[3] unique disease characteristics and rare complications can still compromise long-term outcomes. APL cutis and ATRA-induced myocarditis highlight such challenges, presenting a complex dilemma when encountered in the same patient.

Extramedullary involvement of APL is rare, typically affecting the CNS and skin. The true incidence of APL cutis remains poorly defined, with fewer than 50 reported cases (Table 1).[4-9] The appearance of skin lesions can vary dramatically from small, isolated macules, papules, or nodules to larger infiltrated plaques.[6] Although APL cutis has been documented at diagnosis, most cases previously described have occurred in patients with relapsed or refractory disease and have been associated with poor prognosis. Theories suggest APL cutis may arise at sites of vascular disruption, such as bone marrow biopsy sites and intravenous access sites, though this is not typically seen in patients with APL-related hemorrhages.[7] Interestingly, our patient believed his hand lesion resulted from trauma while handling a rabbit.

With the introduction of ATRA in the early 1990s, concerns have emerged whether prior ATRA therapy increases the risk of developing EMD. ATRA is associated with myeloid differentiation and has been shown to induce expression of adhesion molecules such as CD11c, CD13, and CD56, facilitating infiltration of extramedullary sites.[9] However, retrospective analysis of several large studies of patients treated with chemotherapy alone and ATRA-containing regimens did not reveal increased incidence of EMD after ATRA therapy.[9]

The optimal therapy for APL cutis remains unclear. While incorporating cytotoxic chemotherapy into an ATRA-ATO induction backbone is appealing, the diagnosis is often confirmed after ATRA-ATO initiation, beyond the point when anthracyclines are typically administered in high-risk APL protocols. Additionally, there are no data to support the use of maintenance therapy following successful induction and consolidation for low-risk APL.

Given the excellent remission rate of 100% and outstanding long-term survival of 80% at 5 years with ATRA-ATO therapy, every effort is made to adhere to the protocol and minimize treatment disruptions.[10] Nevertheless, hematologic and non-hematologic toxicities often require dose reductions and treatment interruptions. Abbreviated induction therapy is especially concerning for patients with high-risk features, such as extramedullary disease and leukemia cutis.

Myocarditis is a rare complication of ATRA, with its exact etiology still unclear. Some clinicians believe that it represents DS and should be treated as such. In the literature, myocarditis symptoms appear an average of 19 days after therapy initiation (Table 2).[11-16] Notably, none of the reported patients had DS prior to onset of myocarditis, although nearly half exhibited concurrent DS symptoms at the time of myocarditis diagnosis. The typical onset of classic DS with ATRA is 7-12 days, making the delayed onset

Table 2. Reported cases of ATRA associated myocarditis

Publication	Age/Sex	Therapy	Timing of myocarditis	Prior DS	Concurrent DS	WBC *10^3/uL	Fever	Associated pericarditis or pericardial effusion	Decrease in EF?	ATRA held?	Day of tx ATRA held	Therapy for myocarditis	Steroid type/dose
Nunes, 2021 [12]	57M	IDA+ATRA	D17	No	Yes	<10	Yes	No	Yes	Yes	n/a	BB, diuresis, steroids	Dex 10mg QD
Shenoy, 2021 [13]	62F	ATRA+ATO	D18	No	No	>10	No	Yes	No	Yes	n/a	Colchicine	Dex 10mg BID
Karakulak, 2020 [14]*	23F	IDA+ATRA	D18	No	No	<10	No	No	Yes	Yes	D21	BB, ARB	n/a
Makki, 2019	27M	DNR+ATRA	D10	No	No	<10	No	Yes	Yes	Yes	n/a	BB, diuresis	n/a
Carcelero, 2018	25M	IDA+ATRA	D24	No	Yes	n/a	Yes	Yes	Yes	No	n/a	Spironolactone, ACEi	n/a
Choi, 2011	39F	IDA+ATRA	D18	No	Yes	<10	Yes	No	Yes	Yes	n/a	Diuresis	Dex
Oehler, 2014 [11]	24F	IDA+ATRA	D26	No	No	<10	No	Yes	Yes	Yes	n/a	BB, hydralazine, isosorbide, diuresis	Pred 80mg QD
De Santis, 2012[12]	76F	Ara-C+ATRA	D14	No	No	n/a	No	No	Yes	Yes	D14	Nitrates	Dex 20mg QD
Vissilakopoulos, 2017*	17M	IDA+ATRA	D22	No	No	n/a	No	Yes	Yes	n/a	n/a	BB, ACEi	Dex 24 mg QD
Rijssel, 2010	58M	IDA+ATRA	D21	No	Yes	<10	Yes	Yes	n/a	n/a	n/a	n/a	n/a
Manna, 2008	23M	IDA+ATRA	D6	No	Yes	>10	Yes	No	Yes	Yes	n/a	n/a	Prednisone
Klein, 2007	34F	IDA+ATRA	D19	No	Yes	<10	Yes	Yes	n/a	Yes	n/a	n/a	n/a
	46M	IDA+ATRA	D23	No	Yes	<10	Yes	No	n/a	Yes	n/a	n/a	n/a
Fabbiano, 2005	45M	IDA+ATRA	D23	No	Yes	<10	No	Yes	n/a	Yes	D26	n/a	Pred 50mg QD

*Includes all below listed publications. ATRA=all-trans retinoic acid; ACEi= angiotensin-converting enzyme inhibitors; ARB=angiotensin receptor blocker; BB=beta blockers; BID=twice a day; Dex=dexamethasone; DNR=daunorubicin; DS=differentiation syndrome; EF=ejection fraction; F=female; IDA=idarubicin; M=male; n/a = not applicable; Pred=prednisone; QD=daily; tx=treatment; WBC=white blood cells

of DS and myocarditis at day 19 unusual.[2] A new decline in ejection fraction has been documented in 9 of 10 reported cases, with all patients regaining normal cardiac function without long-term sequelae. Distinguishing ATRA-associated myocarditis from DS is challenging, due to common features of pleuropericardial effusion, pulmonary infiltrates, hypotension and volume overload. While fever and leukocytosis may suggest the underlying etiology, these signs and symptoms are also non-specific, with only 7 of 13 cases reporting fever, and only 2 of 11 cases reporting leukocytosis at symptom onset.

There are no established guidelines for treatment of ATRA-associated myocarditis. In most published cases, ATRA therapy was withheld, and corticosteroids were added, as myocarditis was attributed to severe DS. Treatment for myocarditis itself varied widely in the literature, with patients primarily receiving guideline-directed medical therapy for heart failure with reduced ejection fraction and diuresis.[11,13,14,16] Lastly, recommendations on when to resume ATRA therapy are lacking. According to our literature review, 10 of 13 patients were able to successfully resume ATRA therapy, which suggests ATRA re-challenge following myocarditis may be feasible.[11-13,15,16]

This case underscores the value of maintaining a high clinical suspicion for diagnosing rare APL manifestations and reinforces that ATRA-induced myocarditis can be reversible with therapy interruption and corticosteroids, without long-term sequelae or need for permanent treatment discontinuation.

AUTHORSHIP CONTRIBUTIONS

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Data curation: Kateryna Fedorov, Kian J. Rahbari, Parth C. Patel

Project administration: Kateryna Fedorov, Kian Rahbari

Resources: Justin T. Kelley and Katie O'Connell contributed clinical images for the generation of Figure 1.

Visualization: Kateryna Fedorov, Kian Rahbari, Justin T. Kelley, Katie O'Connell

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Writing (review and editing): all authors

DATA SHARING STATEMENT

Not applicable, as this is a case report with no underlying datasets.

COMPETING INTERESTS

No external funding or support was received for this work. The authors declare no competing interests.

CONFLICTS OF INTEREST

SP discloses consultancies at Genentech Inc., Rigel Pharmaceuticals Inc., and Eli Lilly & Company outside of this work. AK discloses personal fees from Servier Pharmaceuticals, Incyte, Rigel Pharmaceuticals, Sobi, MorphoSys/Novartis, Geron, and Syndax outside the submitted work.

CONSENT TO PUBLISH DECLARATION

Written informed consent for publication was obtained from the patient.

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