Journal of Cardiology Cases 10 (2014) 200–203

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Myopericarditis in a pregnant woman with acute promyelocytic leukemia

Andrew Oehler (MD)[a,*], Shimoli Shah (MD)[b]

[a] OHSU Internal Medicine, Portland, OR, USA
[b] OHSU Knight Cardiovascular Institute, Portland, OR, USA

ARTICLE INFO

Article history:
Received 8 April 2014
Received in revised form 16 June 2014
Accepted 15 July 2014

Keywords:
Pregnancy
Acute promyelocytic leukemia
Differentiation syndrome
Myopericarditis

ABSTRACT

Acute promyelocytic leukemia (APL) is a form of acute leukemia with a characteristic translocation, t(15;17), and is considered a hematologic emergency, typically treated with all-trans retinoic acid and an anthracycline. We present the case of a young, gravid woman who was diagnosed with APL in the third trimester, initiated typical treatment, and suffered uncommon cardiac complications.

<**Learning objective:** Myopericarditis is not a side effect often encountered in the management of acute promyelocytic leukemia with alpha t-retinoic acid, and its mechanism is incompletely understood but possibly related to the differentiation syndrome. This complication can be effectively treated with systemic glucocorticoids, supportive care, and withdrawal of the offending agent, even in the pregnant population.>

© 2014 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Introduction

Acute promyelocytic leukemia (APL) is an M3 form of acute leukemia with a characteristic translocation t(15;17) and is considered a hematologic emergency. Leukemia generally affects 1 in 70,000 pregnancies, a subset at higher risk of morbidity and mortality by virtue of underlying physiologic stress and potential placental transfer of chemotherapeutics. APL presents more commonly relative to other myeloid leukemias in pregnancy due to its higher prevalence in younger populations [1,2]. Among treated, non-pregnant APL patients in the general population, survival rates exceed 70% [3]. Similar outcome data in pregnant patients with treated APL are not available, but case reports of successful therapy have been published. Management of APL in pregnancy varies by trimester at diagnosis [1,4]. The most commonly used regimens involve high-dose all-trans retinoic acid (ATRA) along with an anthracycline both of which can have significant cardiotoxic effects. The case that follows is of a young, gravid woman who was diagnosed with APL in the third trimester with ensuing complications.

Case report

A 24-year-old obese woman with history of a prior pregnancy complicated by preeclampsia was admitted to an outside hospital for easy bruising and headaches at 27 and 1/7 weeks pregnancy. On presentation, she was found to be pancytopenic, with an initial white blood cell (WBC) count of 1.0×10^9/L. Bone marrow biopsy revealed an abnormally elevated number of myeloid blasts. Cytogenetic studies indicated t(15;17) consistent with APL. ATRA was initiated on day 4 of admission at a dose of 45 mg/m^2/day, with dexamethasone to induce fetal lung maturity. Cell counts were supported with intermittent packed red blood cell and platelet transfusions. At day 7 she was transferred to a tertiary center for further management of this complicated pregnancy.

ATRA therapy was continued after transfer. Idarubicin at a dose of 12 mg/m^2 and prednisone were also initiated (for four total doses on days 21, 23, 25, and 27). On the evening of day 26, the 22nd day of ATRA therapy, she developed substernal, sharp chest pain radiating to the back, worse with deep inspiration, and positional changes. She had been tachycardic throughout with a heart rate ranging from 120 to 140 bpm, but she was afebrile. Systolic BP had ranged from 110 to 140 mmHg over the previous 24 h. Physical examination revealed tachycardia with a regular rhythm. S1 and S2 were normal. A ventricular gallop was present. There was a Grade I pericardial rub, with the patient leaning forward at end expiration. Hemogram was remarkable for WBC count of 9.8×10^9/L with a neutrophilic predominance. Serum

* Corresponding author at: OHSU Division of General Internal Medicine, 3181 SW Sam Jackson Park Road L475, Portland, OR 97239-3098, USA. Tel.: +1 503 494 6551; fax: +1 503 494 0979.
E-mail addresses: oehler@ohsu.edu, oehlerd@gmail.com (A. Oehler).

http://dx.doi.org/10.1016/j.jccase.2014.07.010
1878-5409/© 2014 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Table 1 Timeline of laboratory abnormalities.

Day	0	8	26	33
Event	Day of admission	Day of transfer	Onset of symptoms	Resolution of symptoms
WBC ($\times 10^9$/L)	1.2	2.2	9.7	0.26
Hct (%)	24.2	23.2	25.3	29.8
Plt ($\times 10^3$/mm^3)	8	26	41	11
Sodium (mmol/L)	136	139	135	133
Potassium (mmol/L)	3.8	3.8	4.1	3.7
Chloride (mmol/L)	105	109	105	101
Bicarbonate (mmol/L)	23	22	22	24
BUN (mg/dL)	6	8	8	7
Cr (mg/dL)	0.5	0.5	0.4	0.51
Troponin (ng/mL)			3.66	1.26[a]
NT-proBNP (pg/mL)			2877	

WBC, white blood cells; Hct, hematocrit; Plt, platelets; BUN, blood urea nitrogen; Cr, creatinine; BNP, B-type natriuretic peptide.
[a] Specimen collected on day 27 and was the last known troponin prior to resolution of symptoms.

chemistry revealed normal electrolytes and baseline creatinine of 0.56 mg/dL. B-type natriuretic peptide was elevated at 2877 pg/mL. Troponin was elevated at 3.66 ng/mL. See Table 1 for timeline of laboratory abnormalities. An electrocardiogram (ECG) revealed ST elevations with associated PR depressions (Fig. 1). A chest X-ray was significant for hydrostatic pulmonary edema and normal heart size. Computed tomography angiography of the chest did not reveal evidence of pulmonary embolism or consolidation. A transthoracic echocardiogram showed a small, circumferential pericardial effusion (Fig. 2) with multiple segmental wall motion abnormalities in a non-coronary distribution. The estimated ejection fraction was mildly reduced, at 45%. The constellation of findings was felt to be consistent with acute myopericarditis in the setting of ATRA and idarubicin therapy.

ATRA and idarubicin were immediately discontinued due to concern for acute cardiotoxic effects. Prednisone 60 mg daily was initiated to treat the myopericarditis. Chest pain improved only after an increase in prednisone dosage to 80 mg daily. Metoprolol was added for arrhythmia prophylaxis and treatment of cardiomyopathy. Hydralazine and isosorbide dinitrate were used for afterload reduction. Low-dose furosemide was given for diuresis as needed. The patient developed a brief, symptomatic episode of atrial fibrillation with rapid ventricular response on hospital day 31 with spontaneous conversion to sinus rhythm. The metoprolol dose was subsequently increased. No further atrial or ventricular arrhythmias occurred.

Chest pain resolved completely 7 days after initiation of prednisone. Troponin down trended. A repeat transthoracic echocardiogram at admission day 40 showed normalization of left ventricular function and segmental wall motion abnormalities. The pericardial effusion was no longer present. There was no evidence of pericardial constriction.

The patient's course was further complicated by Gram-negative bacteremia and hyperglycemia secondary to steroid administration. Labor was induced, and she delivered vaginally at 34 weeks with an estimated blood loss of 200 mL. The baby was healthy with only a brief hospital stay for hyperbilirubinemia. The patient was discharged shortly after delivery with plans for outpatient consolidation chemotherapy. A slow prednisone taper of 10 mg per week was recommended. Metoprolol was continued. Therapy

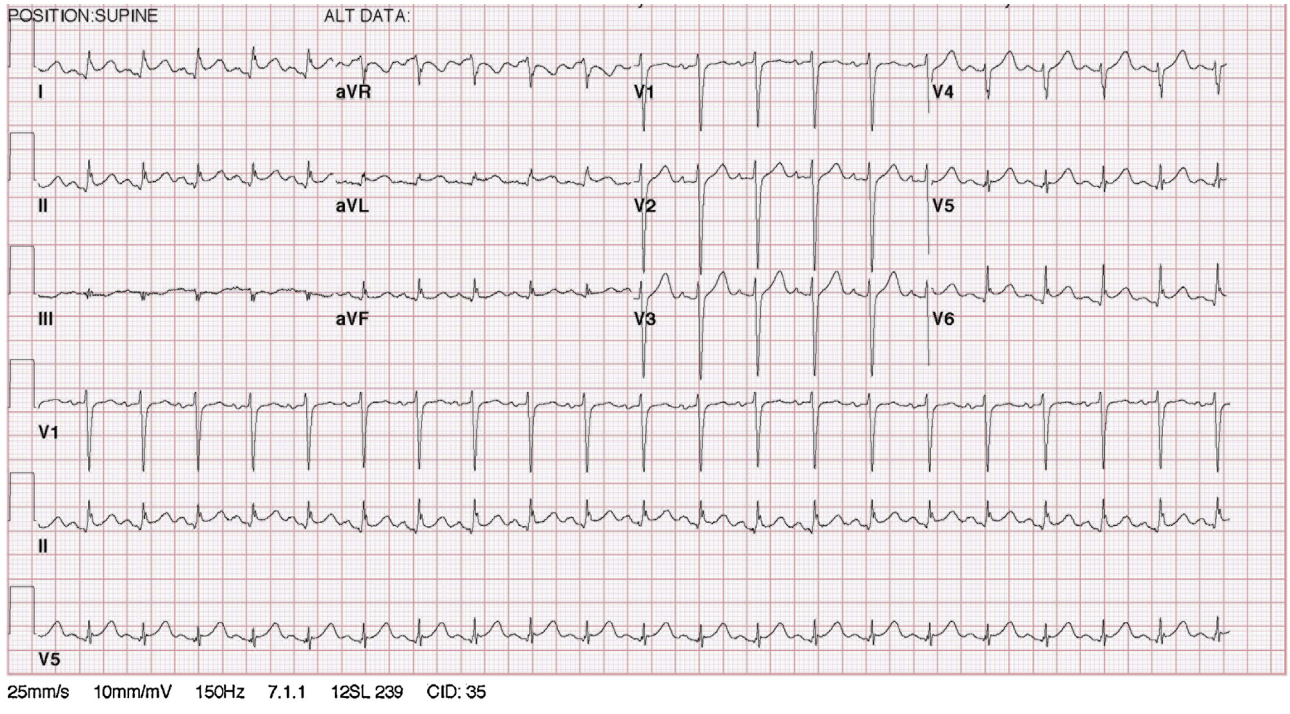

Fig. 1. 12-lead electrocardiogram from day 26 with chest pain. Notice ST segment elevations in leads I, II, aVL, and V3–V6 with PR depressions in the associated leads indicative of both ventricular and atrial epicardial inflammation.

Download English Version:

https://daneshyari.com/en/article/5984572

Download Persian Version:

https://daneshyari.com/article/5984572

Daneshyari.com