

Peripartum cardiomyopathy with intrauterine fetal death complicated by multi-organ failure and a leukoerythroblastic blood picture: A case report

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare but life-threatening form of heart failure developing in the last month of pregnancy or within 5 months postpartum, in the absence of pre-existing cardiac disease. We report a 22-year-old primigravida at 34.6 weeks who presented with progressive breathlessness and a severely impaired left ventricular ejection fraction of 15% on echocardiography, consistent with PPCM. Her course was further complicated by intrauterine fetal death, breech presentation, cardiogenic and septic shock, acute kidney injury, disseminated intravascular coagulation (DIC), and refractory metabolic acidosis. Peripheral blood smear examination, performed in the setting of a rising total leucocyte count, revealed a leukoerythroblastic picture with 33 nucleated red blood cells (nRBCs) per 100 white blood cells – a finding attributable to the convergence of profound hypoxia, DIC, multi-organ stress, and sepsis rather than primary bone marrow disease. This case highlights the catastrophic potential of unrecognized PPCM, the multifactorial etiology of reactive leukoerythroblastosis in critical obstetric illness, the imperative to correct the automated leucocyte count for nRBCs before interpretation, and the critical importance of culture-directed urgent antibiotic therapy.

Key words: Leukoerythroblastic reaction, Multi-organ failure, Peripartum cardiomyopathy, Sepsis

Peripartum cardiomyopathy (PPCM) is defined as idiopathic cardiac failure presenting in the last month of pregnancy or within 5 months of delivery, with a left ventricular ejection fraction (LVEF) below 45% on echocardiography, in the absence of any identifiable pre-existing heart disease [1]. Its incidence varies from 1 in 300 pregnancies in certain high-burden regions to approximately 1 in 3,000–4,000 in Western countries, with South Asian data remaining sparse [2]. PPCM is frequently unrecognized until advanced hemodynamic compromise has occurred, particularly in young primigravidae who attribute breathlessness to the pregnancy itself. When PPCM is compounded by intrauterine fetal death (IUFD), sepsis, and multi-organ dysfunction, the clinical picture becomes extraordinarily complex. Among the hematological derangements encountered in such patients, the leukoerythroblastic blood picture, defined as the simultaneous appearance of nucleated red blood cells (nRBCs) and immature myeloid precursors on peripheral smear – demands careful interpretation. In the non-neonatal adult, this

finding has traditionally been associated with bone marrow infiltration by malignancy; however, in critical illness it may arise as a purely reactive phenomenon driven by severe hypoxia, hemolysis, DIC, and extreme physiological stress [3]. Distinguishing between these two scenarios prevents both diagnostic overreach and therapeutic delay.

We present this case because it encapsulates, in a single young woman, the confluence of PPCM, IUFD, cardiogenic-septic shock, acute kidney injury (AKI), disseminated intravascular coagulation (DIC), and a striking leukoerythroblastic blood picture. The hematological findings and their correct interpretation form the central pathological thread of this report.

CASE REPORT

A 22-year-old primigravida (LMP: October 08, 2025; EDD by early ultrasound [USG]: July 17, 2026; gestational age (GA) at presentation: 34.6 weeks) presented to an outside cardiac center on June 08, 2026 with a 2–3-day history of progressive breathlessness and frontal headache. She reported no chest pain or palpitations; fetal movements had not been felt for

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approximately 24 h. Antenatal care had been irregular at a cottage facility; two doses of tetanus toxoid had been received. History was notable for bronchial asthma managed with inhalers and tablets throughout the pregnancy. There was no history of hypertension, diabetes, or prior cardiac disease. The pregnancy was a spontaneous conception; married life of 1½ years.

At the outside center, pulse was 140/min, blood pressure (BP) 110/94 mmHg, Oxygen saturation (SpO₂) 98% on face mask oxygen. Cardiovascular examination revealed an S3 gallop. Electrocardiogram showed sinus tachycardia with non-specific ST–T wave changes. A 2D echocardiogram demonstrated severe global left ventricular hypokinesia, LVEF 15%, Grade II diastolic dysfunction, mild mitral and pulmonary artery systolic pressure tricuspid regurgitation (jet 36 mmHg), and a thin rim of pericardial effusion, with no structural cardiac anomaly, clot, or vegetation – consistent with PPCM. Intravenous furosemide 20 mg was administered, and the patient was referred urgently to our tertiary care center.

On arrival at 01:31 AM on June 09, 2026, the patient was conscious and oriented but febrile and agitated, on 5 L oxygen via face mask. Pulse 124/min, BP 120/80 mmHg, RR 36/min, SpO₂ 99%. General examination: Moderate pallor, mild bilateral pedal edema, no icterus. Cardiovascular: S1–S2 heard, increased breathing rate; respiratory: Bilateral air entry, no adventitious sounds. Per-abdomen: Uterus 36-week size, breech presentation; fetal heart sounds absent on auscultation and Doppler.

Emergency obstetric USG confirmed a single intrauterine fetus in breech presentation at 33–34 weeks (average GA 33.2±3 weeks; estimated fetal weight

2200±321 g), with absent fetal cardiac activity, absent fetal middle cerebral artery (MCA) flow on color Doppler, and Spalding's sign – diagnostic of IUFD. Amniotic fluid index was 13–14 cm.

Key investigations are summarized in Table 1. The patient was shifted immediately to the critical care unit. A multi-speciality team encompassing Obstetrics and Gynecology, Cardiology, Critical Care Medicine, and Nephrology was mobilized. Intubation and mechanical ventilation (BiPAP, subsequently invasive) were required for worsening respiratory failure. Three vasopressors were commenced concurrently – Noradrenaline, Adrenaline, and Dobutamine – targeting systolic BP above 90 mmHg. Strict fluid restriction (maximum 1 L/day) was enforced.

For the IUFD, induction of labor was initiated with intracervical Dinoprostone gel followed by titrated Pitocin drips. Per-vaginal examination revealed os 1 finger loose, cervix 1.8–2 cm effaced, soft, posterior, with breech presentation and intact membranes. Labor progressed slowly given maternal hemodynamic instability. AKI progressed rapidly to the point where continuous renal replacement therapy (CRRT)/sustained low-efficiency dialysis (SLED) was initiated on Day 2, with a triple-lumen HD port inserted into the right internal jugular vein under USG guidance. Despite dialysis and repeated sodium bicarbonate boluses, metabolic acidosis remained refractory.

Antibiotic coverage was instituted with Piperacillin-Tazobactam, then escalated to Meropenem with Metronidazole. Urine culture (Day 2) grew *Staphylococcus haemolyticus* resistant to all beta-lactams, ciprofloxacin,

Table 1: Key laboratory and investigative findings

Parameter	Result	Comment
Hemoglobin	11.2 g/dL	Mild anemia
TLC (automated)	20,000→25,600/cumm (over 4 days)	Rising; spuriously elevated by nRBCs
Corrected WBC	~19,248/cumm	(25,600×100) ÷ (100+33)
nRBCs on smear	33 per 100 WBCs	Leukoerythroblastic picture
Platelet count	2.83→4.6 lakhs/cumm (falling)	DIC risk
NT-proBNP	>35,000 pg/mL	Severely elevated (normal <300 pg/mL)
Troponin I (hs)	465.76 pg/mL	Elevated (normal female <15.9 pg/mL)
Fibrinogen	149 mg/dL	Low; DIC (normal 238–498 mg/dL)
Serum LDH	682 IU/L	Elevated (normal 200–400 IU/L)
PT/INR	12/1; APTT 33 s	Borderline coagulopathy
Serum creatinine	2.1 → 4.0 mg/dL (rising)	Acute kidney injury
Serum sodium	116–130 mmol/L	Hyponatremia
Serum potassium	5.2–6.6 mmol/L	Hyperkalemia
ABG – pH (worst)	7.058–7.076	Severe metabolic acidosis
Lactate (worst)	12.32–13.55 mmol/L	Critical tissue hypoperfusion
HCO ₃ (worst)	9.2–14.1 mmol/L	Severely low
Urine C/S	<i>Staphylococcus haemolyticus</i>	Resistant to oxacillin, ciprofloxacin, clindamycin; sensitive to linezolid, vancomycin
LVEF (2D Echo)	15%	Peripartum cardiomyopathy
POCUS	Bilateral B-profile, IVC dilated, TAPSE 16 mm	Pulmonary edema, right heart strain

TLC: Total leukocyte count, WBC: White blood cells, nRBCs: Nucleated red blood cells, DIC: Disseminated intravascular coagulation, LDH: Lactate dehydrogenase, PT/INR: Prothrombin time/international normalized ratio ABG: Arterial blood gas, LVEF: Left ventricular ejection fraction, IVC: Inferior vena cava, APTT: Activated partial thromboplastin time

clindamycin, and erythromycin but sensitive to Linezolid, Vancomycin, and Teicoplanin – a critical coverage gap that was flagged for urgent correction. Blood glucose was monitored hourly; sliding-scale Actrapid insulin was administered. Multiple units of Fresh Frozen Plasma (>40 FFP) and Packed Red Blood Cells were transfused for coagulopathy and anemia, which increased reticulocyte count from 2% (on June 09, 2026) to 4.5% (June 11, 2026).

Given the rising total leukocyte count (TLC) and critical illness, a peripheral blood smear was examined (Fig. 1). It revealed a leukoerythroblastic picture: nRBCs at 33/100 white blood cells (WBCs) alongside immature myeloid forms (band forms, occasional myelocytes), toxic granulation, and Dohle bodies in neutrophils – hallmarks of extreme bone marrow stress response. Red cell morphology showed hypochromic normocytic anemia (Hemoglobin 11.2 g/dL). A technically critical point: Automated analyzers cannot distinguish nRBCs from leucocytes, spuriously inflating the TLC. The automated TLC of 25,600/cumm is, in fact, a ghost count – inflated by 33 nRBCs per 100 WBCs that the analyzer cannot distinguish from true leucocytes. Once corrected ($\text{Corrected WBC} = [\text{Reported WBC} \times 100] \div [100 + \text{nRBC count}]$), the genuine leucocyte count falls to approximately 19,248/cumm, stripping away the false alarm of a leukemoid reaction and revealing a more modest reactive leucocytosis entirely consistent with sepsis and physiological stress.

Notably, the peripheral blood smear findings did not herald clinical improvement. In the days that followed the demonstration of a reactive leukoerythroblastic picture, the patient's multi-organ status remained grave: cardiogenic-septic shock persisted despite triple vasopressor support, AKI required ongoing CRRT/SLED, and metabolic acidosis remained refractory (final arterial blood gas pH 7.076, lactate 12.32 mmol/L, on Day 5). The fetus, already confirmed dead in utero at presentation, was delivered stillborn following induction of labor. The family was counseled regarding the grave prognosis, and the patient remained critically ill on

maximal organ support at the time of drafting this case report. This trajectory underscores that the reactive nature of the leukoerythroblastic reaction, while reassuring from a hematological standpoint, did not mitigate the severity of the underlying cardio-obstetric-septic illness. The timeline of events is shown in Table 2.

DISCUSSION

PPCM is defined by the Heart Failure Association of the European Society of Cardiology as an idiopathic cardiomyopathy presenting with heart failure secondary to LV systolic dysfunction (LVEF <45%) toward the end of pregnancy or in the months following delivery, where no other cause is found [1]. In this patient, the LVEF of 15% represents a degree of systolic impairment associated with very high risk of hemodynamic collapse, arrhythmia, and death. The constellation of a critically low LVEF, pericardial effusion, Grade II diastolic dysfunction, and markedly elevated filling pressures (NT-proBNP >35,000 pg/mL; Troponin I 465 pg/mL) indicated severe myocardial injury that had likely been evolving silently through the third trimester, masked by the breathlessness attributed to late pregnancy and bronchial asthma.

PPCM itself is rare, with a global incidence estimated at 1 in 1,000–1,500 pregnancies, though this figure rises to 1 in 100–300 in high-burden regions such as Nigeria and Haiti [2]. IUFD complicates only a small minority of published PPCM cases, most of which are isolated case reports rather than cohort data [4–6]. The additional finding of a reactive leukoerythroblastic blood picture in this setting is, to the best of our knowledge and following a review of the available literature, without precedent: no previously published report has described the co-occurrence of PPCM, IUFD, and a leukoerythroblastic reaction in a single patient. This case is therefore exceptionally rare, both for the severity of the multi-organ collapse and for the unusual convergence of cardiac, obstetric, septic, and hematological derangements it illustrates.

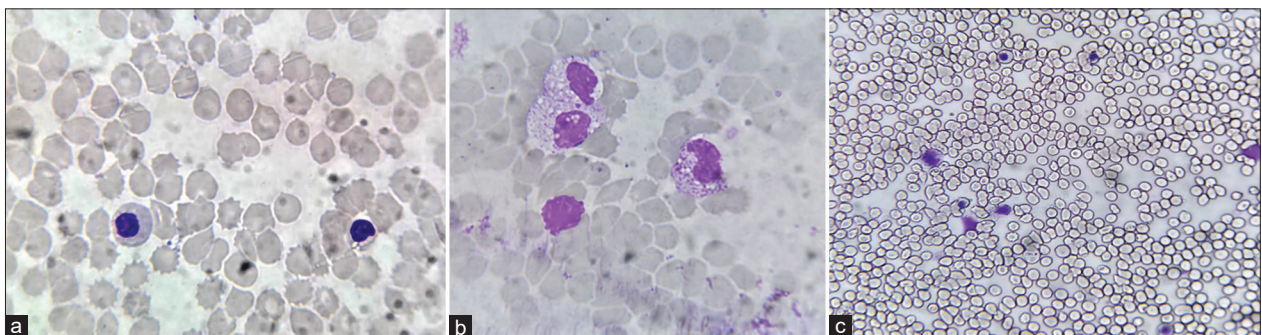


Figure 1: (a) Peripheral blood smear ($\times 100$, oil immersion, Leishman stain): two orthochromatic normoblasts (nucleated red blood cells) with dense, pyknotic, deeply basophilic nuclei and a thin rim of hemoglobinized cytoplasm (arrows). The surrounding red cells are hypochromic and normocytic, consistent with the anemia of critical illness; (b) Peripheral blood smear ($\times 100$, oil immersion, Leishman stain): Upper left shows a segmented neutrophil with coarse dark-purple toxic granulation and cytoplasmic vacuolization alongside mononuclear myeloid precursors; right shows a neutrophil with prominent toxic granulation; (c) Peripheral blood smear ($\times 40$, high power, Leishman stain): Scattered deeply basophilic nucleated red cells (purple dots) are seen dispersed among a sea of mature red cells and large myeloid precursors, illustrating the frequency with which nucleated red blood cells are encountered across the smear. It confirms a reactive leukoerythroblastic picture

Table 2: Timeline of key events

Date/Hospital day	Key events
Jun 08, 2026 (Day 0)	Presented to outside cardiac center with breathlessness. ECG: Sinus tachycardia. 2D Echo: LVEF 15%, peripartum cardiomyopathy. Referred to Krishna Charitable Hospital, Karad.
Jun 09, 2026 (Day 1)	Admitted to our tertiary care center at 01:31 AM. Obstetric USG confirmed IUFD, breech at 34.6 weeks. Shifted to CCU. NT-proBNP >35,000 pg/mL; Troponin I 465.76 pg/mL. Intubation, vasopressors commenced. Cardiogenic + septic shock recognised.
10 Jun 2026 (Day 2)	Peripheral blood smear: nRBCs 33/100 WBCs; rising TLC 20,000→25,600/cumm. Severe metabolic acidosis on ABG. HD port (right IJV, triple lumen) inserted. CRRT initiated. Urine culture: <i>Staphylococcus haemolyticus</i> .
Jun 11–12, 2026 (Days 3–4)	Induction of labor for IUFD. Pitocin drips titrated. Patient intubated on BiPAP; serial ABGs showed persistent severe metabolic acidosis. Multiple FFP and PRBC transfusions. Antibiotic escalation to Meropenem; glycopeptide gap identified.
Jun 13, 2026 (Day 5)	Final ABG pH 7.076, lactate 12.32 mmol/L. POCUS: Bilateral B-profile, IVC dilated, LVEF ~15–20%. Grave prognosis communicated to family. Patient on maximal support.

LVEF: Left ventricular ejection fraction, CCU: Critical care unit, ABG: Arterial blood gas, TLC: Total leukocyte count, CRRT: Continuous renal replacement therapy, IUFD: Intrauterine fetal death, FFP: Fresh frozen plasma, ECG: Electrocardiogram, IVC: Inferior vena cava, USG: Ultrasound

The co-occurrence of IUFD illustrates the bidirectional relationship between maternal cardiac output and fetal wellbeing. Prolonged maternal hypoxia and reduced placental perfusion, consequent to compromised cardiac function, are plausible contributors to fetal demise in PPCM [7]. The ultrasound USG findings of absent fetal cardiac activity, absent MCA flow, and Spalding's sign pointed to fetal death having occurred at least 24–48 h before imaging [8,9].

A small number of previous case reports have described PPCM complicated by IUFD, though none combined this with a leukoerythroblastic blood picture. Hakata *et al.* reported a 36-year-old woman with preeclampsia and IUFD after 24 weeks of gestation who developed PPCM following cesarean section, with cardiac function restored using inotropic support and bromocriptine [10]. Akbar *et al.* described a 25-year-old woman with antepartum PPCM and preeclampsia complicated by AKI, whose fetus was found dead on antenatal USG and was spontaneously expelled during admission [11]. As in the present case, both reports illustrate the bidirectional maternal-fetal compromise that characterizes PPCM presenting with concurrent multi-organ dysfunction, though neither patient reached the degree of hematological derangement, cardiogenic-septic shock, or metabolic collapse seen here.

The leukoerythroblastic blood picture in this patient demands careful interpretation. In the classical teaching, leukoerythroblastosis signals bone marrow infiltration by malignancy, myelofibrosis, or granulomatous disease [3]. However, a well-recognised reactive form also exists in settings of extreme physiological stress: severe hemolytic anemia, DIC, massive hemorrhage, overwhelming sepsis, and profound hypoxia [3,5]. In this patient, all these stressors were simultaneously present: hypoxia (pH 7.07, lactate 12–13 mmol/L), DIC (fibrinogen 149 mg/dL, falling platelets), hemolysis (lactate dehydrogenase 682 IU/L), massive blood product transfusion (>40 FFP units, donor blood itself may contain nRBCs), and sepsis (*S. haemolyticus*). Together, these explain the nRBC count of 33/100 WBCs without invoking marrow infiltration.

This reactive mechanism is increasingly well documented outside the obstetric setting. In a two-center cohort of critically ill adults, the presence of nRBCs was an independent predictor of post-discharge mortality, rising in a dose-dependent fashion with nRBC count [12] – supporting the interpretation of nRBCs as a marker of illness severity rather than a primary marrow disorder. Similarly, a prospective study of COVID-19 patients found a leukoerythroblastic picture in 12% of smears reviewed, again driven by hypoxia and systemic inflammation rather than marrow infiltration, and associated with poor outcome [13]. Lee AC in 2021 has emphasised that a broad range of physiological stressors – most sharing hypoxia, cytokine release, or marrow stimulation as a common pathway – can produce a leukoerythroblastic film in the absence of malignancy, reinforcing the need for clinical correlation before invasive marrow evaluation is pursued [14].

The automated TLC of 25,600/cumm is, in fact, a ghost count – inflated by 33 nRBCs per 100 WBCs that the analyzer cannot distinguish from true leucocytes. Once corrected, the genuine leucocyte count falls to approximately 19,248/cumm, stripping away the false alarm of a leukemoid reaction and revealing a more modest reactive leucocytosis entirely consistent with sepsis and physiological stress. This distinction is clinically important: a TLC of 25,600/cumm may prompt premature diagnosis of leukemoid reaction (defined as WBC >50,000/cumm with left shift), whereas the corrected count is unambiguously reactive [5,6]. A Leucocyte Alkaline Phosphatase score would be high in reactive leucocytosis and low in chronic myeloid leukemia – the principal hematological mimic – though the clinical context here made bone marrow workup inappropriate.

From a microbiological standpoint, *S. haemolyticus* in urine – resistant to all beta-lactams, ciprofloxacin, clindamycin, and erythromycin but sensitive to Vancomycin and Linezolid – represents a significant gap in the initial regimen of Piperacillin-Tazobactam and Meropenem. Prompt rationalization to a glycopeptide

Table 3: Differential diagnosis of the leukoerythroblastic picture in this patient

Condition	nRBCs	Immature WBCs	Likelihood in this case
Reactive leukoerythroblastosis (hypoxia, DIC, sepsis)	Present	Toxic forms, band forms	HIGH – fully supported by clinical context
Bone marrow infiltration (malignancy)	Present	Blasts	LOW – no prior hematological history
Leukemoid reaction	Variable	Marked left shift; WBC >50,000	UNLIKELY – corrected WBC ~19,248
Chronic myeloid leukemia	Variable	Basophilia, low LAP score	VERY UNLIKELY – acute presentation, no splenomegaly
Massive transfusion effect	Possible	Absent	CONTRIBUTORY – >40 FFP units given

WBCs: White blood cells, nRBCs: Nucleated red blood cells, FFP: Fresh frozen plasma, DIC: Disseminated intravascular coagulation

or Linezolid is essential, as undertreated Staphylococcal sepsis in a hemodynamically compromised host carries exceedingly high mortality [7]. Differential diagnosis of the leukoerythroblastic picture in this patient is mentioned in Table 3.

The management of PPCM concurrent with IUFD poses unique dilemmas. Standard heart failure therapy – angiotensin-converting enzyme inhibitors, beta-blockers, aldosterone antagonists – is contraindicated or dose-limited in hemodynamically unstable patients. Dobutamine was the mainstay of inotropic support alongside vasopressors. The decision to proceed with induction of labor rather than Cesarean section was guided by IUFD (removing the fetal urgency), the need to avoid general anesthesia in the setting of cardiogenic shock and coagulopathy, and the recognition that uterine evacuation would reduce the afterload on the failing heart [8].

CONCLUSION

This case distills four practical multi-disciplinary lessons. First, PPCM can hide behind ordinary pregnancy symptoms – if breathlessness keeps getting worse or doesn't add up, check the heart right away; don't wait until after delivery. Second, when a critically ill patient shows immature blood cells on a smear, assume it's the body's stress response first – not bone marrow disease – since low oxygen, clotting problems, and infection can fully explain it; a bone marrow aspirate/biopsy can wait. Third, always double-check the white cell count when many nucleated red cells are present on peripheral blood smear – the CBC machine's raw number is falsely high, and trusting it can send you down an unnecessary and costly workup. Fourth, don't guess with antibiotics – common first-line drugs may not cover the actual organism (as with this patient's *S. haemolyticus*), so the antibiotic plan must be corrected the moment culture results come back.

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