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Case Reports

Acute Bilateral Limb Ischemia in Peripartum Cardiomyopathy: An Often Overlooked Complication

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Abstract

Background: Peripartum Cardiomyopathy (PPCM) is a specific subset of systolic heart failure with potentially devastating complications. Thromboembolism, of complication, requires careful evaluation to assess risk and guide management. This case report of acute limb ischemia complicating peripartum cardiomyopathy is an example of how to deal with thromboembolism in PPCM.

Case Illustration: A 42-year-old woman presented with clinically decompensated heart failure, which started three weeks after her second childbirth and had been worsening over the last four months. At presentation, pulmonary and extremity congestion was evident. Echocardiography revealed a dilated heart and reduced LVEF of 23%. She was diagnosed with PPCM and treated accordingly. On the first night in the ward, she felt sudden pain and paresthesia in her right foot. The distal pulses were weakly palpable, and there was hypoesthesia of the toes. Duplex ultrasound found fresh thrombi in the bilateral popliteal arteries. Diagnosis of acute limb ischemia was confirmed, warranting the use of anticoagulants aside from her existing heart failure medications. Symptoms continued to improve until discharge.

Conclusions: A case of a 42-year-old woman diagnosed with PPCM suffering from an acute thromboembolic episode was reported. Risk assessment is essential for predicting the risk of future thromboembolism and implementing preventive measures. Different anticoagulants are indicated for different PPCM patient profiles, and careful consideration of their safety profiles in this population is needed.

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Keywords: PPCM, thromboembolism, risk assessment, anticoagulant.

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Introduction

Peripartum Cardiomyopathy (PPCM) is a specific form of systolic heart failure with potentially life-threatening complications, one of which is thromboembolism. Peripartum women are in a hypercoagulable state, predisposing them to stasis of blood, which ultimately leads to thromboembolic events that may present in various forms, from acute arterial thrombosis to cardioembolic stroke. In a cohort of the global PPCM registry by the European Society of Cardiology (ESC) EURObservational Research Programme, thromboembolism occurred in 6.8% of women, whereas the US IPAC Cohort reported a 6.6% of thromboembolism. At our center, there were 37 cases of PPCM from 2017-2022; 3 of them had manifestations of thromboembolism in various forms.

Case Illustration

A 42-year-old woman came to our Emergency Room with the chief complaint of breathlessness that worsened over the last week. She had difficulty sleeping at night due to positional dyspnea, nausea, and decreased tolerance of activity. Both of her legs were markedly swollen, although she had no complaints of foot ache or paresthesia at that time.

Her Heart Rate (HR) at admission was 112 bpm, blood pressures were 147/96 mmHg, respiratory rate was 26 x/minute, and peripheral oxygen saturation was 96% on room air. On physical examination, there was elevated jugular venous pressure, rales on lung auscultation, and slight hepatomegaly. Pitting edema was also found on both legs.

Her Electrocardiogram (ECG) at presentation revealed sinus tachycardia (HR 121 bpm), Left

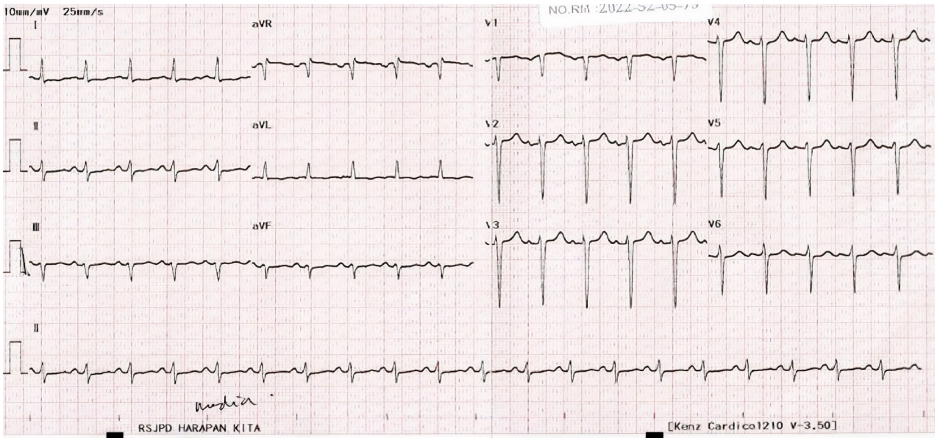

Figure 1. ECG at presentation showed sinus tachycardia and LVH with poor R wave progression.

Ventricular Hypertrophy (LVH), and poor R-wave progression. Bedside echocardiography revealed a dilated heart with Left Ventricular End-Diastolic Diameter (LVEDD) of 56 mm and markedly reduced ejection fraction (LVEF) of 29%. Right Ventricular (RV) function was normal with a Tricuspid Annular Plane Systolic Excursion (TAPSE) of 17 mm. No intracardiac thrombus was seen on bedside echocardiography examination. Routine laboratory results were within normal limits; however, NT-ProBNP was elevated at 3033.

On further history-taking, these symptoms started three weeks after delivering her second child, which was four months before. The pregnancy and delivery itself were uncomplicated. She was already on heart failure medications (bisoprolol 1.25 mg, spironolactone 25 mg, ramipril 5 mg, and furosemide 40 mg), although not routinely, because she was not keen on the diuretic effect. She also received a monthly injection of birth control therapy. Recently, she was hospitalized due to the worsening of her complaints and was discharged with some improvement of symptoms.

Initial diagnosis at admission was Acute Decompensated Heart Failure (ADHF) due to PPCM with moderate severity, and she was admitted to the general ward. IV furosemide was given at the rate of 5mg/hour, and she resumed her previous medications with the addition of warfarin 2 mg and prescription of bromocriptine 2.5 mg bid.

The next day, she underwent comprehensive echocardiography, which revealed a markedly dilated heart with an LVEDD of 62.9mm, a Left Ventricular End-Systolic Diameter (LVESD) of 56.2mm, and a Left Ventricular Ejection Fraction (LVEF) of 23%. RV function was normal, and still no intracardiac thrombus nor Spontaneous Echo Contrast (SEC) was found.

On her first night in the ward, she experienced sudden pain in her right foot with a Visual Analog

Scale (VAS) score of 8/10, accompanied by paresthesia and hypoesthesia of the medial toes. She had cold sweat, and the distal pulsation was weak. However, motoric function was normal, and neurologic examination confirmed hypoesthesia on the affected foot. Laboratory examination revealed a hypercoagulable state with D-dimer level at 4090, fibrinogen of 419, INR of 1.11, and A1C of 6.8. Liver functions were within normal limits. Doppler ultrasound showed fresh thrombi on both her popliteal arteries (left and right) with still some flow to the distal legs, and a diagnosis of acute bilateral limb ischemia was confirmed.

She was then transferred to the intermediate ward for observation and started on heparinization with Low Molecular Weight Heparin (LMWH) (enoxaparin at 0.6mg subcutaneous bid injection). Paracetamol as a pain reliever was administered, along with medications with anti-inflammatory and antioxidant properties (pentoxifylline 1200 mg, allopurinol 300 mg tid, bicarbonate 500 mg tid, and vitamin E 400 mg bid). She showed improvement of symptoms over the first day of diagnosis. She was immediately consulted for surgery for surgical revascularization. On further assessment by the surgeon, she did not need surgery and was advised to continue heparinization.

She continued to show improvements of both ischemic leg symptoms and heart failure symptoms. Her leg examination on the seventh day revealed improvement of flow in lower leg arteries, warm legs, and no neurological deficits on both legs. There were no more rales in both lungs, pitting edema of the legs had resolved, and the liver had returned to standard size. She achieved a 300 m distance on a 6-minute walk test, confirming complete decongestion. She was discharged on the seventh day on the following medications: furosemide 40 mg, bisoprolol 5 mg, candesartan 16 mg, spironolactone 50 mg, aspirin 80 mg, atorvastatin 80 mg, warfarin 2 mg, and metformin 500 mg.

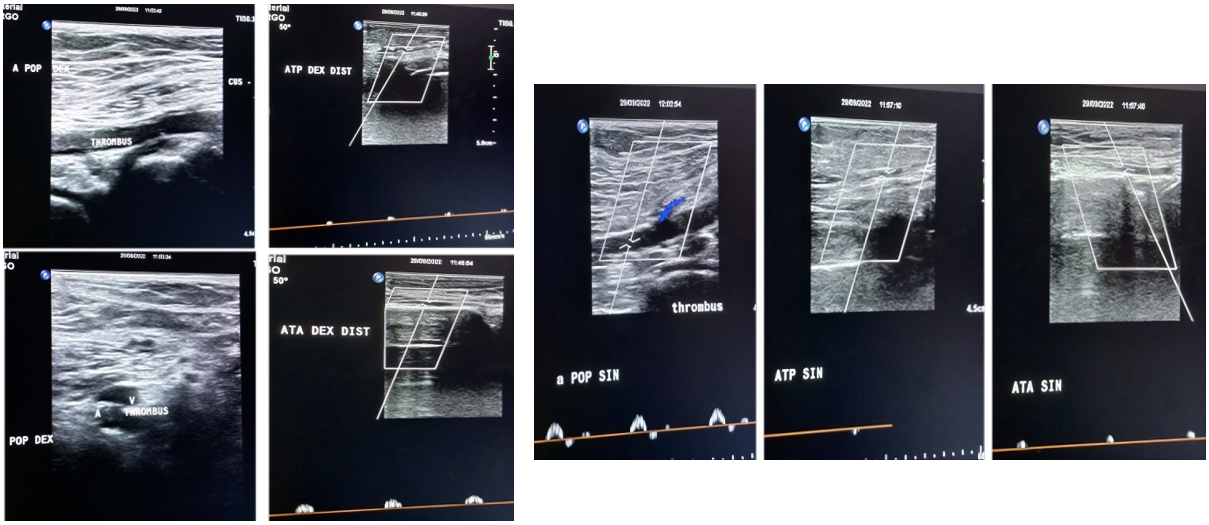

Figure 2. Duplex ultrasound of the lower limb showed fresh thrombus in the bilateral popliteal arteries. The flow is diminished in the distal artery in both legs.

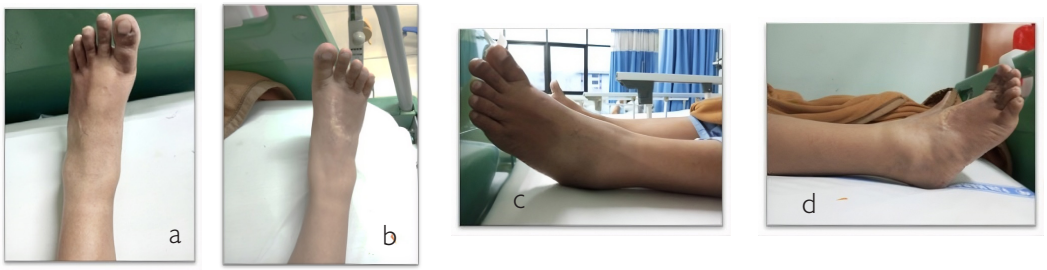

Figure 3. Clinical pictures of the patient's foot. (b) and (d) right foot, the affected side, appeared paler than the left foot (a) and (c).

Discussion

Our patient was initially diagnosed with PPCM. During the course of her disease, she suffered from an acute thromboembolism event manifesting as acute limb ischemia. Various factors are at play in the pathophysiology of thromboembolism in this patient, such as the peripartum period, acute heart failure due to PPCM, and the use of hormonal contraception.

Pathophysiology of Thrombosis in PPCM

PPCM is a specific subset of left heart failure occurring in women in their late pregnancy to the peripartum period. Currently, there is no comprehensive registry of PPCM in Indonesia. However, an observational study by Prameswari et al in 2016 revealed that 26% out of 305 women with pregnancy and cardiovascular complications were diagnosed with PPCM in Bandung, Indonesia.[1] At our center alone, there were 37 cases of PPCM during 2017-2021.

Our patient presented with an acute heart failure presentation, respiratory insufficiency, elevated NT-ProBNP, and LVEF at 23%, all of which pointed to her symptoms being categorized as "moderate" according to the 2019 ESC Position Statement on PPCM by Bauersach J et al.[3] This position statement also recommended that the mainstay treatment of PPCM was promoted under the tag "BOARD" therapy, consisting of bromocriptine, oral heart failure drugs, anticoagulants, vasorelaxants, and diuretics.

On the first night in the hospital, she experienced sudden pain and paresthesia in her right foot. Duplex ultrasound revealed fresh thrombosis in her right and left popliteal arteries, despite an asymptomatic left leg. She was diagnosed with acute limb ischemia and was promptly started on LMWH enoxaparin 0,6 mg subcutaneously. Bromocriptine was not given yet at this point and was withheld.

Thromboembolism is one of the most debilitating complications of PPCM. Women in general were at higher risk of thromboembolism compared to men, due to the pro-thrombotic nature of the hormone estrogen. This risk increases tenfold during pregnancy and up to twenty-fivefold in the postpartum period. Various mechanisms have been proposed to explain this predisposition, all of which contribute to Virchow's Triad of Pregnancy. Although traditionally used in the context of Venous Thromboembolism (VTE), this concept can be applied to arterial thromboembolism in the PPCM population, as the risk of both is similar in this population.[4]

The three elements of Virchow's Triad are all present during pregnancy and the postpartum period. Blood stasis, which begins in the first trimester and reaches its peak on the 36th week, is caused by progesterone-induced vasodilation and compression of blood vessels by the gravid uterus. Vascular damage is caused by delivery, both vaginal and assisted C-section. A hypercoagulable state during pregnancy is progressively activated to prepare for the challenge of delivery. Procoagulant activity is increased by heightening concentrations of fibrinogen and factors V, VIII, IX, and X, enhancing production of thrombin. Thrombus dissolution is reduced by decreasing the activity of tissue plasminogen activator (tPA) and increasing the activation of its inhibitors, types 1 and 2. All these resulted in increased risk of thromboembolism in pregnant and postpartum women.

A change in coagulable status of postpartum women increases the risk of thromboembolism. This hypercoagulable state was caused by changes of various thrombotic factor parameters, which take different times to return to baseline levels after delivery. Some parameters, such as factor VIII and von Willebrand factor, return to baseline within 72 hours postpartum, whereas others, such as the reduction in protein S, take several weeks to recover. In general, it is assumed that the hypercoagulability state is resolved after 6 weeks postpartum.[13] However, the risk of thrombosis may persist beyond 6 weeks. A study by Kamel H et al. in 2014 found that thromboembolic risk is highest during the first 6 weeks, remains significantly elevated through 15 weeks, and then declines. This is consistent with findings of laboratory markers, most of which return to baseline by the sixth week, whereas some remain abnormal through at least 12-15 weeks postpartum.

Our patient presented at four months postpartum (16 weeks), yet she suffered from an acute thromboembolic event. As her coagulable state is likely to return to baseline by this time, other factors may contribute to the development of arterial thrombosis.

Echocardiography of our patient revealed a low LVEF of 23% and a dilated heart, predisposing her to a condition of arterial stasis and increased risk of thromboembolism, as stated by Agarwal et al in 2019. In PPCM, there is also endothelial injury and immobilization due to heart failure symptoms. All these contribute to a condition of increased thromboembolism risk.

Risk Assessment of Thromboembolism in PPCM

The well-known Wells Score, widely used to assess thromboembolic risk, cannot be applied in the pregnant population.[5] To address these risks, the Royal College of Obstetricians and Gynecologists (RCOG) issued a guideline in 2015 stating that all women should undergo a documented assessment of risk factors for thromboembolism in early pregnancy or pre-pregnancy.[6] The guideline included a risk-scoring system to guide risk assessment and management in this vulnerable population, as depicted in Table 1.

Our patient admittedly did not undergo such a risk assessment in her previous pregnancy. Although she stated that the pregnancy and delivery itself were uneventful and uncomplicated, she should still be assessed accordingly. When this RCOG risk-scoring system was applied to our patient, she scored 5, indicating a "high risk". Admittedly, this is because she had a medical comorbidity of heart failure, which alone contributed 3 points to her score. But excluding the comorbidity, she still scored two due to her age (>35) and obesity, with a Body Mass Index (BMI) of 29.1. This rendered her at "intermediate risk," which, according to the guideline, should be managed with prophylaxis LMWH during pregnancy and the peripartum period for at least 10 days postpartum. The recommended dosing of LMWH is depicted in Table 2.

Table 1. Risk Scoring of Thromboembolism in pregnant and peripartum women by RCOG, 2018.

Pre-existing risk factors	Tick	Score
Previous VTE (except a single event related to major surgery)	☐	4
Previous VTE provoked by major surgery	☐	3
Known high-risk thrombophilia	☐	3
Medical comorbidities (e.g., cancer, heart failure, active SLE, inflammatory polyarthropathy or inflammatory bowel disease, current IV drug user)	☐	3
Family history of unprovoked or estrogen-related VTE in a first-degree relative	☐	1
Known low-risk thrombophilia (no VTE)	☐	1
Age >35 years	☐	1
Obesity	☐	1 or 2
Parity >= 3	☐	1
Smoker	☐	1
Gross varicose veins	☐	1
Obstetric risk factors		
Pre-eclampsia in the current pregnancy	☐	1
ART/IVF (antenatal only)	☐	1
Multiple pregnancy	☐	1
Caesarean section in labour	☐	2
Elective caesarean section	☐	1
Mid-cavity or rotational operative delivery	☐	1
Prolonged labour (>24 hours)	☐	1
PPH (>1 litre or transfusion)	☐	1
Preterm birth <37 weeks in current pregnancy	☐	1
Stillbirth in the current pregnancy	☐	1
Transient risk factors		
Any surgical procedure in pregnancy or puerperium except immediate repair of the perineum, e.g., appendectomy	☐	3
Hyperemesis	☐	3
OHSS (first trimester only)	☐	4
Current systemic infection	☐	1
Immobility, dehydration	☐	1

VTE: Venous Thromboembolism ; SLE: Systemic Lupus Erythematosus; ART/IVF: Assisted Reproduction Therapy/In Vitro Fertilization; PPH: Post-Partum Haemorrhage; OHSS: Ovarian Hyperstimulation Syndrome

Table 2. LMWH doses by RCOG Guideline.

Weight	Enoxaparin Doses
<50 kg	20 mg daily
50-90 kg	40 mg daily
91-130 kg	60 mg daily (may be divided into two doses)
131-170 kg	80 mg daily (may be divided into two doses)
>170 kg	0.6 mg/kg/day (may be divided into two doses)
High prophylactic dose for women 50-90 kg	40 mg 12-hourly

LMWH is the agent of choice for thrombosis prophylaxis in the pregnant and postpartum population. A 2011 study by Lindqvist et al. found a 88% relative risk reduction of 88% in obstetric patients with prior VTE who were given LMWH.[7] It does not cross the placenta (except fondaparinux, which crosses the placental barrier in small amounts) and is mainly eliminated by renal clearance. Doses should be adjusted in patients with renal failure; alternatively, unfractionated heparin (UFH) can be considered. Vitamin K Antagonist (VKA) is not safe for pregnant women because of fetal toxicity. Women on long-term VKA should be counseled to stop the drug before the 6th week of gestation to eliminate the risk of warfarin embryopathy.[8]

The Role of Bromocriptine in PPCM

Bromocriptine, a dopamine antagonist, has been widely studied as a new disease-specific therapy for PPCM. It works by inhibiting prolactin release, which is believed to have an essential role in PPCM pathophysiology. Under conditions of elevated reactive oxygen species (ROS) in late pregnancy and the peripartum period, the peptidase Cathepsin D is released, cleaving the hormone prolactin into its of kDa antiangiogenic fragment.[9] A study by Hilfiker-Kleiner et al. in 2017 reported a higher rate of LVEF recovery at 6 months in patients treated with bromocriptine than in those who were not.[10-11] However, this drug is pro-thrombotic in nature and has been reported to be linked to cerebrovascular ischemia and acute myocardial infarction in previous case reports.[12-13] Therefore, bromocriptine use in PPCM should always be accompanied by anticoagulation, at least at a prophylactic dose.

Our patient was prescribed bromocriptine 2,5 mg bid, planned to be given for two weeks, along with warfarin 2 mg as a prophylactic anticoagulant. However, before the administration of its first dose, the patient experienced an acute thromboembolic event in both her legs. Needless to say, bromocriptine was promptly stopped, and LMWH was administered.

Acute Limb Ischemia in PPCM

Thromboembolism in PPCM may present in multiple forms, the most common being thrombi in the left and right cardiac chambers. A study in Senegal reported that up to 30% of PPCM cases had LV thrombi.[15] There were also reported cases of multiple intra-abdominal thromboemboli[16], pulmonary embolism[17-18], cardioembolic stroke[19], and lower extremity arterial thromboembolism, which, in a case report by Carlson KM et al, was a presenting complaint of PPCM instead of heart failure.[20] At our center, there were three cases of thromboembolism complicating PPCM over the past 5 years. The presentations were acute cerebral ischemia caused by suspected cardiac emboli, acute limb ischemia, and acute pulmonary embolism.

The pathophysiology of lower-limb thromboembolism in PPCM, as highlighted by Isezuo et al., is linked to the hypercoagulable state of the peripartum period, ventricular dilatation, and hypokinesis observed in this subset of patients. Intracardiac thrombosis due to low ventricular ejection fraction was often thought to be the culprit, as reported in a case report.[21]

Our patient had an episode of acute thrombosis in the legs, manifesting as sudden foot pain and paresthesia. Duplex ultrasound of the legs revealed fresh thrombi in the bilateral popliteal arteries. She was diagnosed with acute limb ischemia stage I based on Rutherford classification, promptly given LMWH, and referred for surgery for urgent surgical revascularization. According to the 2017 ESC Guideline of Peripheral Arterial Disease, revascularization with endovascular therapy is preferred to surgical thrombectomy/bypass, owing to reduced morbidity and mortality. However, there are many considerations before deciding which method works best for each case, some of

them being the presence of a neurological deficit, ischaemia duration, its localization, comorbidities, type of conduit (artery or graft), and therapy-related risks and outcomes.[22]

Management of Acute Limb Ischemia

TASC-II Consensus on Management of Peripheral Arterial Disease recommends endovascular treatment for infrainguinal lesions.[23] However, Percutaneous Transluminal Angioplasty (PTA) was not readily available in our center, so surgery was the method of choice. The patient demonstrated clinical improvement on the surgical team assessment, and the surgery was ultimately deferred.

Her symptoms improved rapidly after analgesics and heparinization. Pulsation was palpated more strongly on the right foot, paresthesia and hypoesthesia were gone, and the VAS Score went down to 2/10. Surgery was withheld, and she continued to receive LMWH for 5 days, during which heart failure medications were optimized.

Choosing LMWH as an anticoagulant in this patient is based on multiple considerations. Zhu et al. proposed different anticoagulant regimens for different cardiomyopathy profiles of.[24] In the PPCM population, LVEF below 45% warrants administration of VKA, provided that the patient is in the late trimester of pregnancy or after delivery. Patients with other comorbidities such as atrial fibrillation, previous thromboembolism, or cardiac emboli were recommended to be given LMWH, as was the case with our patient. Novel Oral Anticoagulants (NOAC) such as rivaroxaban, apixaban, or dabigatran are generally avoided during pregnancy and the lactation period due to a lack of available safety data.

On the seventh day of admission, she had completed decongestion, as evidenced by a six-minute walk test distance >300 m. Pain in the leg had also resolved, and duplex evaluation demonstrated

Table 3. Anticoagulation of choice for patients with cardiomyopathy. From Zhu et al, 2021.

	Concomitant AF	Enlarged LAD	Previous TE/ evident intracavitary thrombus	Ventricular dilatation/ dysfunction, LVAA	Using bromocriptine
HCM	OAC recommended	OAC considered in obstructive HCM with LAD >=48 mm	-	OAC suggested in HCM with LVAA	-
DCM	OAC recommended as CHA2DS2-VASc score >=1	-	Anticoagulant therapy suggested	-	-
RCM	OAC suggested	-	OAC suggested	-	-
CM	VKA or direct thrombin inhibitors are recommended	-	VKA or direct thrombin inhibitors are recommended	-	-
HES	-	-	VKA recommended	-	-
ARVC	OAC considered	-	OAC recommended	-	-
LVNC	VKA preferred	-	VKA preferred	VKA preferred as LVEF <40%	-
TTS	-	-	Heparin (IV/SC)/ VKA/NOACs recommended	Heparin/VKA/NOACs considered if LVEF <= 30%, or large LVD involving apex	-
PPCM	LMWH recommended, VKA might be considered during lactation or during the 2nd/3rd trimester	-	LMWH recommended, VKA might be considered during lactation or during the 2nd/3rd trimester	LMWH suggested if LVEF <45%, VKA might be considered during lactation or during 2nd/3rd trimester	Heparin recommended

AF: Atrial Fibrillation; LAD: Left Atrial Diameter; TE: Thromboembolism; LVAA: Left Ventricular Apical Aneurysm; HCM: Hypertrophic Cardiomyopathy OAC: Oral Anticoagulation/Oral Anticoagulant; DCM: Dilated Cardiomyopathy; CHA_2DS_2-VASc: Congestive heart failure, Hypertension, Age ≥75 (2 points), Diabetes mellitus, Stroke/TIA/systemic embolism (2 points), Vascular disease, Age 65–74, Sex category; RCM: Restrictive Cardiomyopathy; CM: Cardiomyopathy; VKA: Vitamin K Antagonist; HES: Hypereosinophilic Syndrome; ARVC: Arrhythmogenic Right Ventricular Cardiomyopathy; LVNC: Left Ventricular Noncompaction; LVEF: Left Ventricular Ejection Fraction; TTS: Takotsubo Syndrome; IV: Intravenous; SC: Subcutaneous; NOACs: Non–Vitamin K Oral Anticoagulants; LVD: Left Ventricular Dysfunction; PPCM: Peripartum Cardiomyopathy; LMWH: Low-Molecular-Weight Heparin.

improved flow in the distal leg arteries. She was discharged on optimal heart failure medications, atorvastatin, warfarin as an anticoagulant, and aspirin as an antiplatelet.

Contraceptive Measures in PPCM and Risk of Thromboembolism

On discharge, she was counseled on prognosis and subsequent pregnancy. Previously, she had been on a monthly injection of birth control, containing a combination of estrogen and progestin. The hormone estrogen is strongly associated with prothrombotic effects, affecting various factors in the coagulation and fibrinolysis cascade.[25]

A review by Abou-Ismail et al in 2020 found that women on combined oral contraception had an increased absolute risk of venous and arterial thromboembolism. There is currently little evidence evaluating the risk of thromboembolism in women using combined injectable contraceptives, like our patient. However, since any form of estrogen is contraindicated in patients with cardiac disease, it is reasonable to advise patients on other methods of birth control. A survey by Rosman L, et al revealed the three most common contraceptives used by women with PPCM are tubal ligation, condoms, and Intrauterine Device (IUD).[26] These long-acting reversible contraceptives are the most effective, and since they have little risk of thromboembolism, they should be the first choice for women with PPCM. Our patient was advised and agreed to the placement of an IUD device as her future contraceptive.

Conclusion

A case of acute limb ischemia complicating peripartum cardiomyopathy has been presented. There is an increased risk of thromboembolism in the PPCM population, which may manifest in various presentations. Risk assessment is essential for predicting the occurrence of future thromboembolism and, therefore, implementing necessary preventive measures before they occur. Different anticoagulants are indicated for different PPCM patient profiles, and careful consideration of their safety profiles in this population is needed.

List of Abbreviations

ADHF	Acute Decompensated Heart Failure
AF	Atrial Fibrillation
ARVC	Arrhythmogenic Right Ventricular Cardiomyopathy
ART/IVF	Assisted Reproduction Therapy/ In Vitro Fertilization
BMI	Body Mass Index
Bpm	Beat per minute
DCM	Dilated Cardiomyopathy
ECG	Electrocardiogram
ESC	European Society of Cardiology
HCM	Hypertrophic Cardiomyopathy
HES	Hypereosinophilic Syndrome
HR	Heart Rate
LAD	Left Atrium Diameter
LMWH	Low Molecular Weight Heparin
LVAA	Left Ventricular Apical Aneurysm
LVEDD	Left Ventricular End-Diastolic Diameter
LVEF	Left Ventricular Ejection Fraction
LVESD	Left Ventricular End-Systolic Diameter
LVH	Left Ventricular Hypertrophy
LVNC	Left Ventricular Noncompaction
LVD	Left Ventricular Dysfunction
NOAC	Novel Oral Anticoagulants
OAC	Oral Anticoagulants
OHSS	Ovarian Hyperstimulation Syndrome
PPH	Post-Partum Haemorrhage
PPCM	Peripartum Cardiomyopathy
PTA	Percutaneous Transluminal Angioplasty
RCM	Restrictive Cardiomyopathy
RCOG	Royal College of Obstetricians and Gynecologists
ROS	Reactive Oxygen Species
RV	Right Ventricle
SEC	Spontaneous Echo Contrast
SLE	Systemic Lupus Erythematosus
TAPSE	Tricuspid Annular Plane Systolic Excursion
TTS	Takotsubo Syndrome

UFH	Unfractioned Heparin
VKA	Vitamin K Antagonist
VTE	Venous Thromboembolism

Ethical Clearance

Not Applicable.

Publication Approval

All authors are consent to the publication of this manuscript.

Authors Contributions

All authors contributed to the case report writing. Material preparation, data collection and analysis were performed by Muthia Syarifa Yani and Hary Sakti Muliawan. The first draft of the manuscript was written by Muthia Syarifa Yani, and Hary Sakti Muliawan commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Conflict of Interest

None.

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Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work, the authors did not use Generative AI and AI-Assisted Technologies. The authors reviewed and revised the materials and take full responsibility for the content.

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