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CASE REPORT

Peripartum Cardiomyopathy in a Patient with Undiagnosed Persistent Patent Ductus Arteriosus (PDA): A Rare Clinical Association

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Abstract:

Peripartum cardiomyopathy (PPCM) is a rare form of heart failure occurring in late pregnancy or early puerperium, typically in women without pre-existing heart disease. This report details the rare co-existence of PPCM and an uncorrected Patent Ductus Arteriosus (PDA). A 19-year-old woman, diagnosed with PPCM (EF-43%) during her late pregnancy, presented with heart failure symptoms (NYHA Class II dyspnea). Physical examination revealed a classic Grade 4/6 continuous machinery murmur. Echocardiography confirmed a moderate PDA (H+4mm) with a significant left-to-right shunt (Qp/Qs-2.1), after conservative treatment her heart failure symptoms were improved with a preserved Left Ventricular Ejection Fraction (EF-62%). The symptoms were primarily driven by PDA-induced volume overload. The patient underwent successful transcatheter PDA closure. This case emphasizes the critical need for heightened clinical vigilance to evaluate cardiopulmonary symptoms in pregnant women, as their presentation can be confused with normal pregnancy changes, delaying diagnosis causes serious underlying cardiac issues.

Keywords: Peripartum cardiomyopathy, Patent Ductus Arteriosus

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Bullet Point for Learnings:

- Uncover how an untreated PDA and overlapping PPCM can mimic typical pregnancy symptoms and how neglecting to be vigilant might postpone a potentially life-saving diagnosis.
- Recognize how PDA-driven volume overload during pregnancy affects hemodynamics and how it may overshadow PPCM in clinical presentation.

Introduction:

Peripartum cardiomyopathy (PPCM) refers to a relatively rare form of heart failure. It occurs because of new onset left ventricular dysfunction during late pregnancy or early puerperium. Despite an exact pathophysiology being yet unclear, it has been realized that peripartum cardiomyopathy represents an important source of serious morbidity and mortality among pregnant women. The onset of peripartum cardiomyopathy normally occurs in previously healthy women. Also, it usually occurs without pre-existing heart disease.

A congenital heart defect caused by an abnormal persistence of the ductus arteriosus, known as patent ductus arteriosus (PDA), is commonly detected and treated in childhood. A silent PDA might exist for several years before, potentially lead to conditions like volume overload, pulmonary hypertension, arrhythmias, and heart failure, and might be unmasked with the hemodynamic changes associated with pregnancies. However, there have been no more than a few reports on co-occurrences of PPCM and congenital heart conditions like PDA.

The concurrent features of PPCM and uncorrected congenital heart disease add complexity. Symptoms like shortness of breath and palpitations might be erroneously blamed on the physiological changes associated with pregnancy. The case report adds to a very short list of literature on PPCM presenting in a pregnant woman with an uncorrected PDA. A heightened index of suspicion needs to be maintained, and a search initiated for cardiopulmonary complaints presenting in pregnant women.

Case Report:

A 19-year-old woman presented with 4 days of worsening dyspnea, palpitations, and leg swelling, progressing to

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NYHA Class II. She had a similar cardiorespiratory episode seven months later, three days postpartum after her first cesarean delivery, when she was diagnosed with peripartum cardiomyopathy and PDA on the basis of Echo report, (Figure-5) and placed on standard heart failure therapy.

On admission, she was stable (pulse 88 bpm, BP 100/60 mmHg, SpO, 96%). A displaced apex beat and a palpable continuous thrill were noted at the left upper parasternal border, with a classic grade 4/6 continuous machinery murmur. Lungs were clear. Laboratory tests were normal, ECG showed sinus rhythm, and chest X-ray revealed cardiomegaly with increased pulmonary vascularity.

Echocardiography demonstrated a moderate PDA (H+4 mm) with significant left-to-right shunt (Qp/Qs 2.1), dilated left heart chambers, moderate aortic regurgitation, mild mitral regurgitation, mild pulmonary hypertension (PASP 40 mmHg), and preserved LV systolic function EF 62% (Figure-3).

A diagnosis of PPCM coexisting with moderate PDA was made. She underwent transcatheter PDA closure, and post-procedural imaging confirmed complete occlusion with preserved biventricular function.

Table-1

Name of Investigation	Result
Hb	11.5gm/dl
S. Creatinine	0.64mg/dl
S. Electrolytes	Na: 138, K: 4.1, Cl: 103
SGPT	11.7u/l
BT	3min 50sec
CT	5min 50sec
S. TSH	3.48
PT	10.9sec

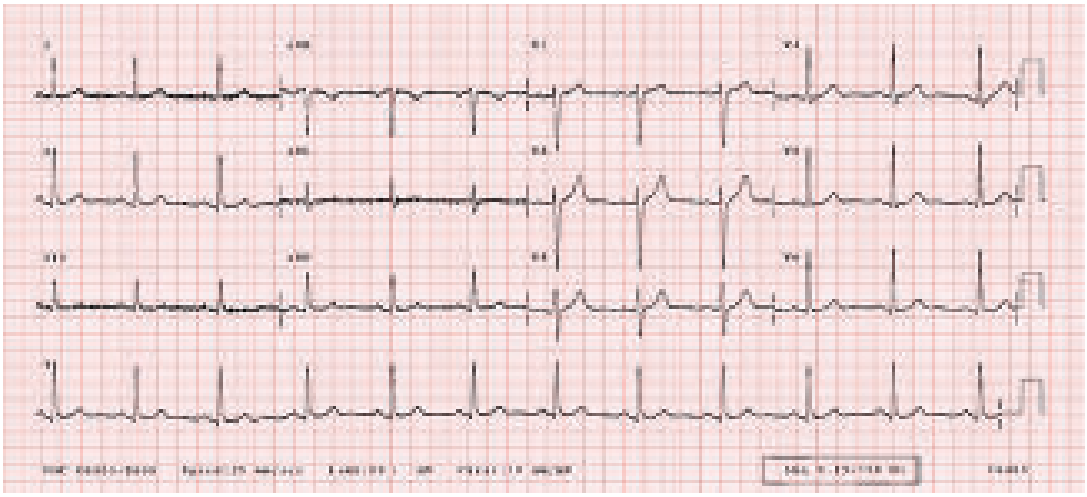

Figure 1: *ECG is normal*

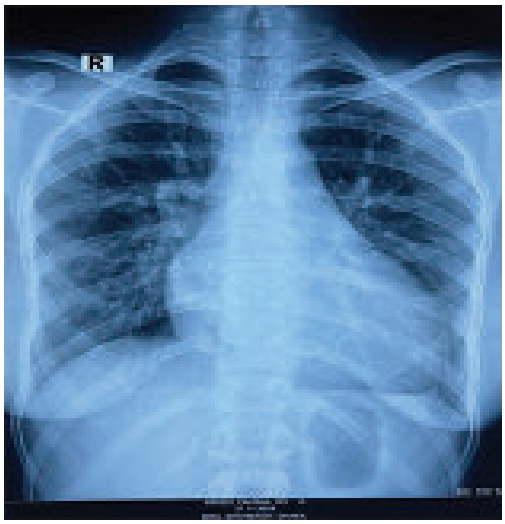

Figure 2: *Chest X-ray P/A view shows cardiomegaly with LV type apex.*

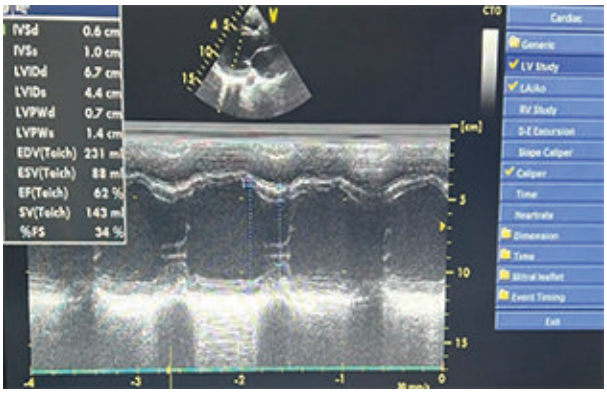

Figure 3: *Echocardiography after treatment of PPCM showing EF-62%*

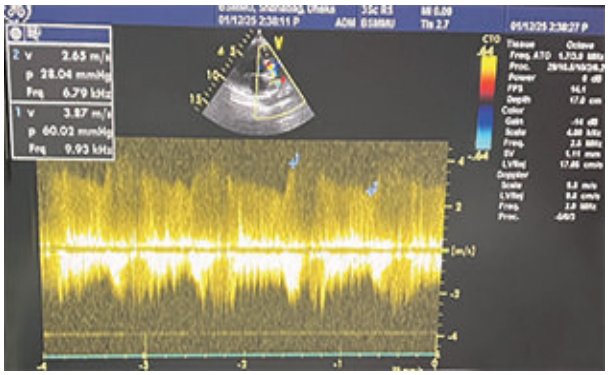

Figure 4: *Showing picket French appearance which indicate PDA*

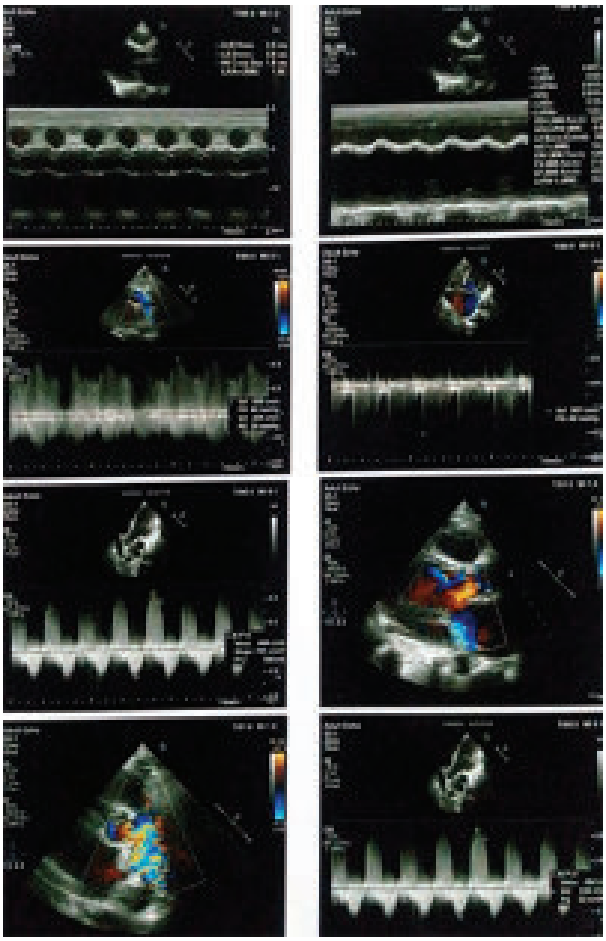

Figure 5: *Echocardiography during 7th pod of LUCS showing EF-43%.Congenital heart disease-PDA with left to right shunt(PPG-84 mmHg).Dilated Cardiomyopathy. Global hypokinesia of LV. Mild LV and RV systolic dysfunction (TAPSE-14mm).Severe Pulmonary HTN(PASP-70mmHg).*

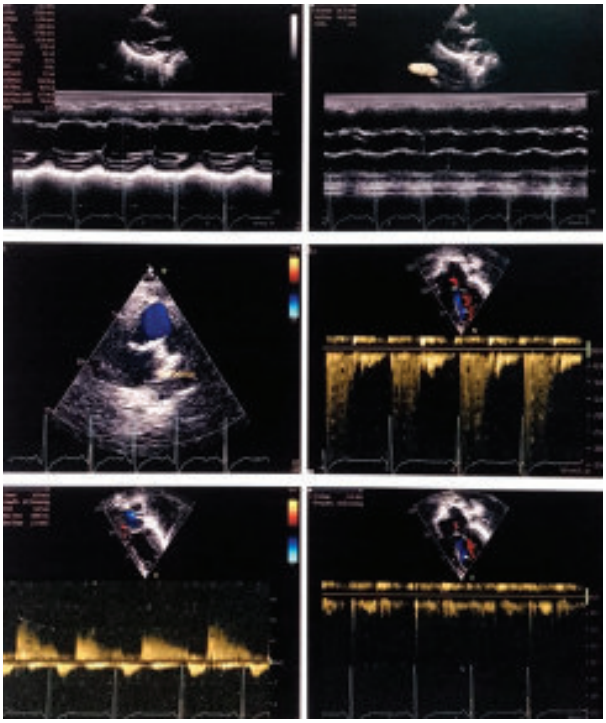

Figure 6: *Echocardiography after PDA device closure. PDA device in situ. No residual flow. Good flow through LPA and descending aorta. Good bi-ventricular function.*

Discussion

The current case presents a rare and significant diagnostic challenge involving the co-existence of Peripartum Cardiomyopathy (PPCM) and an uncorrected Patent Ductus Arteriosus (PDA) in a pregnant woman. PPCM is defined as new-onset left ventricular systolic dysfunction in late pregnancy or the early postpartum period, typically arising in previously healthy women without underlying structural heart disease. Our patient, however, presented with two overlapping cardiac conditions.

The patient's presentation—progressive shortness of breath, palpitations, and bilateral leg swelling, advancing to NYHA Class II dyspnea—is highly characteristic of heart failure[3333]. Notably, she had a similar episode seven months prior following a Cesarean delivery of her first child, where she was conservatively managed for presumed PPCM based on an echocardiogram showing reduced Ejection Fraction (EF-43%)[4]. This previous episode, occurring in the *postpartum* window, aligns perfectly with the definition of PPCM[5].

The clinical diagnosis was complicated by the discovery of a PDA on physical examination, evidenced by a collapsing pulse and the pathognomonic Grade 4/6

continuous "machinery" murmur heard best at the left upper parasternal area during end-expiration. Transthoracic echocardiography confirmed a moderate-sized PDA (approximately 4 mm) with a significant left-to-right shunt (Qp/Qs ratio of 2.1). This structural lesion, when uncorrected, predisposes adults to heart failure, particularly when unmasked by the severe hemodynamic stress of pregnancy. The key diagnostic dilemma lies in separating the effects of the PDA from PPCM. While the patient was diagnosed with PPCM during her first pregnancy due to systolic dysfunction. However, the large left-to-right shunt from the PDA caused severe volume overload, leading to dilation of the left atrium and left ventricle, moderate aortic regurgitation, and mild pulmonary hypertension. Therefore, the patient's symptoms of heart failure were primarily driven by the volume overload imposed by the PDA shunt, a burden that was amplified by the hyperdynamic circulation of pregnancy.

The standard approach to managing PPCM involves guideline-directed medical therapy. However, the presence of a significant PDA fundamentally alters the management strategy. The ultimate therapeutic intervention was cardiac catheterization with device closure of the PDA. The successful closure eliminated the shunt, which was confirmed by a post-procedure echocardiogram showing no residual shunt and stable biventricular systolic function. This intervention was crucial, as closing the shunt effectively removed the primary trigger and perpetuator of the heart failure symptoms in this patient.

This case reinforces the observation that common cardiopulmonary symptoms like dyspnea and palpitations are often falsely attributed to normal pregnancy physiology, delaying the recognition of serious underlying cardiac issues. Clinicians, particularly in resource-limited settings where congenital heart disease screening may be less common, must maintain heightened clinical vigilance to rapidly evaluate these symptoms, especially when a continuous murmur or signs of volume overload are detected. The timely, definitive correction of the PDA was essential and prevented potential morbidity and mortality associated with both the large shunt and the superimposed PPCM risk. PPCM collective treated with standard therapy for heart failure, dopamine D2 receptor agonists, and anticoagulation displays a high and stable long-term recovery rate with low mortality at 5-year follow-up. (Moulig et al., 2019). Women who develop peripartum cardiomyopathy are at high risk of developing the same problem with future pregnancies. The rate of recurrence is about 30%. (Pennmedicine.org, 2025). The variation in recovery is seen even regionally; a case series of PPCM patients in Bangladesh indicated that while 45% of patients were able to recover normal cardiac function, 30% ended up developing chronic cardiomyopathy (Salim et al., 2020). These figures emphasize the necessity for comprehensive long-term follow-ups, particularly when PPCM occurs alongside congenital heart defects.

Conclusion:

Peripartum cardiomyopathy occurs in last trimester of pregnancy or following delivery within 5 months of postpartum period without any structural heart diseases. congenital heart disease in patient with PDA may remain asymptomatic since childhood and become symptomatic during pregnancy due to hyperdynamic circulation with volume overload and patient can present with heart failure. Appropriate heart failure management followed by successful closure of PDA is a mainstay of treatment.

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Author Contributions:

Case data collection, case reporting, manuscript drafting, and manuscript writing: FA (Fatema Akter). Conception, clinical guidance, critical revision, and supervision of the manuscript: MFIK (Md. Fakhrul Islam Khaled). Expert review, academic guidance, and final approval of the manuscript: KA (Khurshed Ahmed).

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Conflict of Interest:

The authors declare no conflict of interest.

Ethical Approval:

We did not ask for ethical approval but obtained written consent from the patients for this publication.

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