

Impact Factor: 6.145

ISSN: 2181-0990
DOI: 10.26739/2181-0990
www.tadqiqot.uz

JRHUNR

JOURNAL OF REPRODUCTIVE HEALTH AND URO-NEPHROLOGY RESEARCH



TADQIQOT.UZ

VOLUME 7,
ISSUE 2

2026

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ
РЕСПУБЛИКИ УЗБЕКИСТАН

Журнал репродуктивного здоровья и уро-
нефрологических исследований

JOURNAL OF REPRODUCTIVE HEALTH AND URO-NEPHROLOGY RESEARCH

Главный редактор: Б.Б. НЕГМАДЖАНОВ

Учредитель:

Самаркандский государственный
медицинский университет

Tadqiqot.uz

Ежеквартальный
научно–практический
журнал

ISSN: 2181-0990
DOI: 10.26739/2181-0990

Журнал включен в перечень научных изданий, рекомендованных к публикации основных научных результатов диссертаций по медицинским наукам с 25 июня 2025 года Высшей аттестационной комиссией Республики Узбекистан (письмо № 371/6 от 2025 года).

№ 2
2026

Главный редактор: Chief Editor:

Негмаджанов Баходур Болтаевич
доктор медицинских наук, профессор
Самаркандский государственный медицинский университет

Doctor of Medical Sciences, Professor
Samarkand State Medical University

Заместитель главного редактора: Deputy Chief Editor:

Каттаходжаева Махмуда Хамдамовна
доктор медицинских наук, профессор
Ташкентский Государственный медицинский университет

Doctor of Medical Sciences, Professor
Tashkent State Medical University

ЧЛЕНЫ РЕДАКЦИОННОЙ КОЛЛЕГИИ | MEMBERS OF THE EDITORIAL BOARD:

Зуфарова Шахноза Алимджановна
Доктор медицинских наук, профессор
Ташкентский Государственный медицинский университет
Doctor of Medical Sciences, Professor
Tashkent State Medical University

Агабабян Лариса Рубеновна
Кандидат медицинских наук, профессор
Самаркандский государственный медицинский университет
Candidate of Medical Sciences, Professor,
Samarkand State Medical University

Зокирова Нодира Исламовна
Доктор медицинских наук, профессор, Самаркандский
государственный медицинский университет
Doctor of Medical Sciences, Professor,
Samarkand State Medical University

Кадыров Зиёратшо Абдуллоевич
Доктор медицинских наук, профессор,
Российский университет дружбы народов
Doctor of Medical Sciences, Professor,
Russian University of Friendship of Peoples

Пахомова Жанна Евгеньевна
доктор медицинских наук, профессор
Ташкентский Государственный медицинский университет:
Doctor of Medical Sciences, Professor
Tashkent State Medical University

Юлдашев Ботир Ахматович
доктор медицинских наук, доцент Самаркандский
государственный медицинский университет
Doctor of Medical Sciences, associate professor
Samarkand State Medical University

Надырханова Наталья Суратовна
доктор медицинских наук, Директор
ГУ «Республиканского специализированного
научно-практического медицинского
центра здоровья матери и ребёнка»
Doctor of Medical Sciences,
Director of the State Institution Republican
Specialised Scientific and Practical Medical
Centre for Maternal and Child Health

Ответственный секретарь:

Махмудова Севара Эркиновна
PhD по медицинским наукам, Самаркандский
государственный медицинский университет
PhD in Medical Sciences,
Samarkand State Medical University

Караченцова Ирина Васильевна
Кандидат медицинских наук, доцент
Российского национального исследовательского
Медицинского Университета им. Н.И.Пирогова
PhD, Associate Professor at the N.I. Pirogov
Russian National Research Medical University

Аллазов Салах Алазович
Доктор медицинских наук, профессор,
Самаркандский государственный медицинский университет
Doctor of Medical Sciences, Professor,
Samarkand State Medical University

Дильмурадова Клара Равшановна
Доктор медицинских наук, профессор,
Самаркандский государственный медицинский университет
Doctor of Medical Sciences, Professor,
Samarkand State Medical University

Негматуллаева Мастура Нуруллаевна
Доктор медицинских наук, профессор
Бухарский медицинский институт
Doctor of Medical Sciences, Professor,
Bukhara Medical Institute

Локшин Вячеслав Нотанович
Доктор медицинских наук,
профессор, член-корр. НАН РК, президент Казахстанской
ассоциации репродуктивной медицины (Казахстан)
Doctor of Medical Sciences, Professor,
Corresponding Member of the National Academy
of Sciences of the Republic of Kazakhstan,
President of the Kazakhstan Association
of Reproductive Medicine (Kazakhstan).

Аскеров Арсен Аскерович
Доктор медицинских наук., профессор Кыргызско-Российского
Славянского университета, президент Кыргызской
ассоциации акушер-гинекологов и неонатологов
Doctor of Medical Sciences, Professor, Kyrgyz - Russian
Slavic University, President of the Kyrgyz Association
of Obstetricians and Neonatologists

Азизов Маматкул Курбанович
Доктор медицинских наук, профессор, Самаркандский
государственный медицинский университет
Doctor of Medical Sciences, Professor,
Samarkand State Medical University

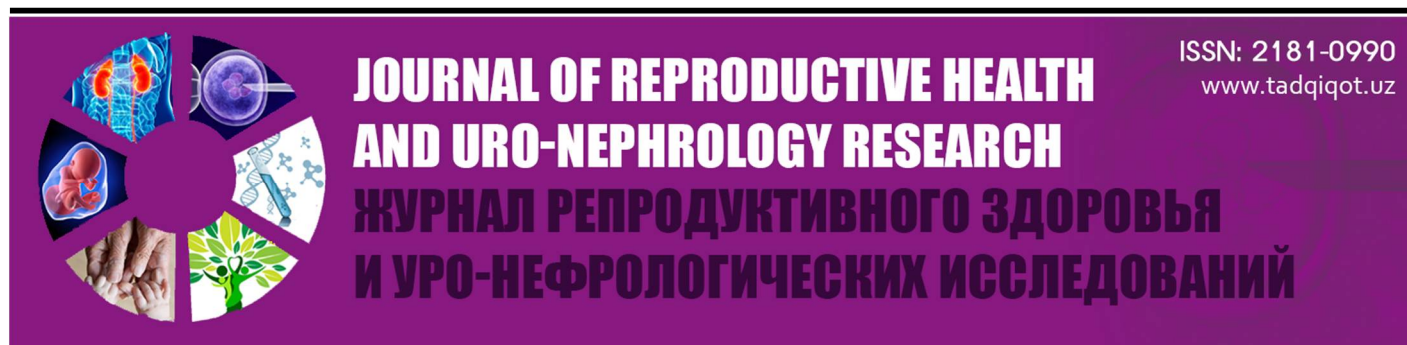
Шавази Наргиз Нуралиевна
доктор медицинских наук, доцент
Самаркандский государственный медицинский университет
DSc, Associate Professor
Samarkand State Medical University

Page Maker | Верстка: Хуршид Мирзахмедов

Контакт редакций журналов. www.tadqiqot.uz
ООО Tadqiqot город Ташкент,
улица Амира Темура пр.1, дом-2.
Web: <http://www.tadqiqot.uz/>; Email: info@tadqiqot.uz
Телефон: +998 (94) 404-0000

Editorial staff of the journals of www.tadqiqot.uz
Tadqiqot LLC the city of Tashkent,
Amir Temur Street pr.1, House 2.
Web: <http://www.tadqiqot.uz/>; Email: info@tadqiqot.uz
Phone: (+998-94) 404-0000

МЕНОПАУЗАЛЬНОГО ПЕРЕХОДА ПО ДАННЫМ АМБУЛАТОРНОГО СКРИНИНГА/STRUCTURE OF CONCOMITANT GYNECOLOGICAL AND EXTRAGENITAL PATHOLOGY IN WOMEN DURING THE MENOPAUSAL TRANSITION ACCORDING TO OUTPATIENT SCREENING DATA/MENOPAUZA DAVRIDAGI AYOLLARDA AMBULATOR SKRINING MA'LUMOTLARIGA KO'RA QO'SHMA GINEKOLOGIK VA EKSTRAGENITAL PATOLOGIYA TUZILISHI.....	54
6. Хаккулов Эркин Бекмирзаевич, Алимов Жалолиддин Усмонхон угли АНАЛИЗ ДИНАМИКИ УРОЛОГИЧЕСКОЙ СИМПТОМАТИКИ ГИПЕРАКТИВНОСТИ МОЧЕВОГО ПУЗЫРЯ ПОСЛЕ ТЕРАПИИ В ЗАВИСИМОСТИ ОТ ГРУППЫ ИССЛЕДОВАНИЯ В ЗАБОЛЕВАЕМОСТИ РАКОМ ПРОСТАТЫ /ANALYSIS OF THE DYNAMICS OF UROLOGICAL SYMPTOMS OF OVERACTIVE BLADDER AFTER THERAPY DEPENDING ON THE RESEARCH GROUP IN PROSTATE CANCER INCIDENCE/PROSTATA BEZI SARATONI INSIDENSIYASIGA QARAB TADQIQOT GURUHIGA BOG'LIQ HOLDA ORTIQCHA FAOL SIYDIK PUFAGI UROLOGIK SIMPTOMLARINING TERAPIYADAN KEYINGI DINAMIKASINI TAHLIL QILISH.....	58
7. Хасанова Дилафруз Абдухамидовна АУТОИММУННЫЙ ТИРЕОИДИТ У БЕРЕМЕННЫХ КАК ФАКТОР МЕТАБОЛИЧЕСКИХ НАРУШЕНИЙ И НЕБЛАГОПРИЯТНЫХ ПЕРИНАТАЛЬНЫХ ИСХОДОВ/HOMILADOR AYOLLARDA AUTOIMMUN TIREOIDIT METABOLIK BUZILISHLAR VA NOQULAY PERINATAL NATIJALARNING MUHIM OMILI SIFATIDA/AUTOIMMUNE THYROIDITIS IN PREGNANCY AS A FACTOR IN METABOLIC DISORDERS AND ADVERSE PERINATAL OUTCOMES.....	62
8. Файзуллаева Хилола Бахроновна, Кобулова Мадина Бахадировна КЛИНИКО-ЭПИДЕМИОЛОГИЧЕСКАЯ ХАРАКТЕРИСТИКА АНТЕНАТАЛЬНОГО И ПЕРИНАТАЛЬНОГО ПЕРИОДОВ У НОВОРОЖДЁННЫХ С ЗАДЕРЖКОЙ ВНУТРИУТРОБНОГО РАЗВИТИЯ/CLINICAL AND EPIDEMIOLOGICAL CHARACTERISTICS OF THE ANTENATAL AND PERINATAL PERIODS IN NEWBORNS WITH INTRAUTERINE GROWTH RESTRICTION/BACHADON ICHIDA O'SISHNING KECHIKISHI BILAN YANGI TUG'ILGAN CHAQAQALOQLARDA ANTENATAL VA PERINATAL DAVRLARNING KLINIK-EPIDEMIOLOGIK XUSUSIYATLARI.....	66
9. Dilmuradova Klara Ravshanovna, Rajabova Nasiba Karim qizi YANGI TU'GILGAN CHAQAQALOQLARDA D VITAMINI HOLATINI BAHOLASH/ASSESSMENT OF VITAMIN D STATUS IN NEWBORNS/ОЦЕНКА СОСТОЯНИЯ ВИТАМИНА D У НОВОРОЖДЕННЫХ.....	69
10. Xamidova Shaxlo Musinovna, Xakimova Ruhshona Abduraxmonovna MUDDATIDAN OLDINGI TUG'RUQ: XAVF OMILLARI VA PROFILAKTIKA STRATEGIYALARI/ПРЕЖДЕВРЕМЕННЫЕ РОДЫ: ФАКТОРЫ РИСКА И СТРАТЕГИИ ПРОФИЛАКТИКИ/PRETERM BIRTH: RISK FACTORS AND PREVENTION STRATEGIES.....	73
11. Xasanova Dilyafuz Abduxamidovna HOMILADOR AYOLLARDA AUTOIMMUN TIREOIDIT VA SEMIZLIKNI DAVOLASHDA MIKROELEMENTLAR KORREKSIYASINING ROLI/THE ROLE OF MICROELEMENT CORRECTION IN THE TREATMENT OF AUTOIMMUNE THYROIDITIS AND OBESITY IN PREGNANT WOMEN/РОЛЬ КОРРЕКЦИИ МИКРОЭЛЕМЕНТОВ В ЛЕЧЕНИИ АУТОИММУННОГО ТИРЕОИДИТА И ОЖИРЕНИЯ У БЕРЕМЕННЫХ.....	77
12. Kamalova Dilafruz Doniyorovna, Kamalova Nigora Bobir qizi, Kamalova Dilnoza Bokhodirovna AUTOIMMUNE THYROIDITIS AND PREGNANCY/АУТОИММУННЫЙ ТИРЕОИДИТ И БЕРЕМЕННОСТЬ/AUTOIMMUN TIREOIDIT VA HOMILADORLIK.....	82
13. Kamilova N.M., Mirzoyeva Kh.M. PROGNOSTIC SIGNIFICANCE OF CLINICAL, LABORATORY, AND ECHOCARDIOGRAPHIC MARKERS OF ADVERSE OUTCOMES IN PERIPARTUM CARDIOMYOPATHY/ПРОГНОСТИЧЕСКОЕ ЗНАЧЕНИЕ КЛИНИЧЕСКИХ, ЛАБОРАТОРНЫХ И ЭХОКАРДИОГРАФИЧЕСКИХ МАРКЕРОВ НЕБЛАГОПРИЯТНЫХ ИСХОДОВ ПРИ ПОСЛЕРОДОВОЙ КАРДИОМИОПАТИИ/TUG'RUQDAN KEYINGI KARDIYOMIYOPATIYADAGI NOJOYA OQIBATLARNING KLINIK, LABORATORIYA VA EKHOKARDIYOGRAFIK BELGILARINING BASHORATLI AHAMIYATI.....	87
14. Muratova Dilafruz Iskandar qizi THE ROLE OF AUTOIMMUNE THYROID DISEASES IN THE DEVELOPMENT OF FEMALE INFERTILITY AND TREATMENT TACTICS/РОЛЬ АУТОИММУННЫХ ЗАБОЛЕВАНИЙ ЩИТОВИДНОЙ ЖЕЛЕЗЫ В РАЗВИТИИ ЖЕНСКОГО БЕСПЛОДИЯ И ТАКТИКА ЛЕЧЕНИЯ/AYOLLAR BEPUSHTLIGINING RIVOJLANISHIDA QALQONSIMON BEZNING AUTOIMMUN KASALLIKLARINING ROLI VA DAVOLASH TAKTIKASI.....	95



UDC 616.12-008.46-036.1-073.97-07:618.3-06

Kamilova N.M.

Professor, Doctor of Medical Sciences
Department of Obstetrics and Gynecology
Azerbaijan Medical University
Baku, Azerbaijan

Mirzoyeva Kh.M.

Associate Professor, Candidate of Medical Sciences
Department of Obstetrics and Gynecology
Azerbaijan Medical University
Baku, Azerbaijan

PROGNOSTIC SIGNIFICANCE OF CLINICAL, LABORATORY, AND ECHOCARDIOGRAPHIC MARKERS OF ADVERSE OUTCOMES IN PERIPARTUM CARDIOMYOPATHY

For citation: Kamilova N.M., Mirzoyeva Kh.M., Prognostic Significance of Clinical, Laboratory, and Echocardiographic Markers of Adverse Outcomes in Peripartum Cardiomyopathy, Journal of reproductive health and uro-nephrology research 2026 vol 7 issue 2


<http://dx.doi.org/10.5281/zenodo.20923526>

ABSTRACT

Background: Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of heart failure characterized by left ventricular systolic dysfunction. Early prediction of disease course remains a major clinical challenge.

Objective: To evaluate the prognostic significance of clinical, laboratory, and echocardiographic parameters and to develop an integrated risk model for adverse outcomes in PPCM.

Methods: This comparative clinical observational study included 75 pregnant women, of whom 60 constituted the main group and 15 served as controls. The main group was stratified into three subgroups according to gestational age. All participants underwent clinical and instrumental assessment, including electrocardiography, echocardiography, and measurement of NT-proBNP, cathepsin D, prolactin, C-reactive protein, and interleukin-6. Receiver operating characteristic (ROC) analysis and logistic regression were used for statistical evaluation.

Results: Progression of PPCM was associated with increasing levels of NT-proBNP, prolactin, cathepsin D, and inflammatory markers, together with a decline in left ventricular ejection fraction (LVEF). An adverse outcome at 6 months postpartum was observed in 45% of patients. The strongest predictors of adverse outcome were elevated NT-proBNP, prolactin, cathepsin D, C-reactive protein, interleukin-6, and reduced LVEF. The integrated PPCM-PRO model demonstrated high prognostic performance (AUC = 0.93).

Conclusion: The PPCM-PRO model showed high prognostic value and may be useful for early risk stratification and optimization of clinical management in patients with PPCM.

Keywords: peripartum cardiomyopathy; pregnancy; heart failure; NT-proBNP; prolactin; cathepsin D; echocardiography; prognosis; risk model

Камилова Н.М.

Профессор, доктор медицинских наук
Кафедра акушерства и гинекологии
Азербайджанский медицинский университет
Баку, Азербайджан

Мирзоева Х.М.

Доцент, кандидат медицинских наук
Кафедра акушерства и гинекологии
Азербайджанский медицинский университет
Баку, Азербайджан

ПРОГНОСТИЧЕСКОЕ ЗНАЧЕНИЕ КЛИНИЧЕСКИХ, ЛАБОРАТОРНЫХ И ЭХОКАРДИОГРАФИЧЕСКИХ МАРКЕРОВ НЕБЛАГОПРИЯТНЫХ ИСХОДОВ ПРИ ПОСЛЕРОДОВОЙ КАРДИОМИОПАТИИ

АННОТАЦИЯ

Актуальность: Р кардиомиопатия (ППКМ) — редкая, но потенциально опасная для жизни форма сердечной недостаточности, характеризующаяся систолической дисфункцией левого желудочка. Раннее прогнозирование течения заболевания по-прежнему остается одной из основных клинических задач.

Цель: Оценить прогностическую значимость клинических, лабораторных и эхокардиографических показателей и разработать комплексную модель риска неблагоприятных исходов при ППКМ.

Методы: В это сравнительное клиническое обсервационное исследование было включено 75 беременных женщин, из которых 60 составили основную группу, а 15 — контрольную. Основная группа была стратифицирована на три подгруппы в зависимости от срока беременности. Все участницы прошли клиническое и инструментальное обследование, включая электрокардиографию, эхокардиографию, а также определение уровней NT-proBNP, катепсина D, пролактина, C-реактивного белка и интерлейкина-6. Для статистической оценки использовались анализ кривой рабочих характеристик (ROC) и логистическая регрессия.

Результаты: Прогрессирование ППКМ ассоциировалось с повышением уровней NT-proBNP, пролактина, катепсина D и маркеров воспаления, а также со снижением фракции выброса левого желудочка (ФВЛЖ). Неблагоприятный исход через 6 месяцев после родов наблюдался у 45 % пациенток. Наиболее сильными предикторами неблагоприятного исхода были повышенные уровни NT-proBNP, пролактина, катепсина D, C-реактивного белка, интерлейкина-6 и сниженная ФВЛЖ. Интегрированная модель PPCM-PRO продемонстрировала высокую прогностическую эффективность (AUC = 0,93).

Вывод: Модель PPCM-PRO продемонстрировала высокую прогностическую ценность и может быть полезна для ранней стратификации риска и оптимизации клинического ведения пациенток с перипартальной кардиомиопатией.

Ключевые слова: перипартальная кардиомиопатия; беременность; сердечная недостаточность; NT-proBNP; пролактин; катепсин D; эхокардиография; прогноз; модель риска

Kamilova N.M.

Professor, tibbiyot fanlari doktori
Akusherlik va ginekologiya kafedrası
Ozarbayjon tibbiyot universiteti
Boku, Ozarbayjon

Mirzoeva X.M.

Dotsent, tibbiyot fanlari nomzodi
Akusherlik va ginekologiya kafedrası
Ozarbayjon tibbiyot universiteti
Boku, Ozarbayjon

TUG'RUQDAN KEYINGI KARDIYOMIYOPATIYADAGI NOJOYA OQIBATLARNING KLINIK, LABORATORIYA VA EXOKARDIYOGRAFIK BELGILARINING BASHORATLI AHAMIYATI

ANNOTATSIYA

Muvofiqlik: P kardiomyopatiya (PPCM) — yurak etishmovchiligining kam uchraydigan, ammo hayot uchun xavfli shakli bo'lib, chap qorincha sistolik disfunktsiyasi bilan tavsiflanadi. Kasallikning kechishini erta bashorat qilish asosiy klinik muammo bo'lib qolmoqda.

Maqsad: Klinik, laboratoriya va ekokardiografik ko'rsatkichlarning prognostik ahamiyatini baholash va PPCMda salbiy oqibatlar xavfining kompleks modelini ishlab chiqish.

Ussullari: Ushbu qiyosiy klinik kuzatuv tadqiqoti 75 homilador ayolni o'z ichiga oldi, ulardan 60 tasi asosiy guruhni va 15 tasi nazorat guruhini tashkil etdi. Asosiy guruh homiladorlik davriga qarab uchta kichik guruhga tabaqalashtirilgan. Barcha ishtirokchilar klinik va instrumental tekshiruvdan o'tdilar, jumladan elektrokardiografiya, ekokardiografiya, shuningdek, NT-proBNP, katepsin D, prolaktin, C-reaktiv oqsil va interleykin-6 darajalari. Statistik baholash uchun ishlash egri chizig'i (ROC) tahlili va logistika regressiyasi ishlatilgan.

Natijalar: PPKM ning rivojlanishi NT-proBNP, prolaktin, katepsin D va yallig'lanish belgilarining ko'payishi, shuningdek, chap qorincha ejeksiyon fraktsiyasining (FVLF) pasayishi bilan bog'liq. Bemorlarning 45 foizida tug'ilgandan 6 oy o'tgach, salbiy natija kuzatildi. Eng kuchli salbiy bashorat qiluvchilar NT-proBNP, prolaktin, katepsin D, C-reaktiv oqsil, interleykin-6 va FVLFning pasayishi edi. Integratsiyalashgan PPCM-PRO modeli yuqori prognostik samaradorlikni namoyish etdi (AUC = 0,93).

Xulosa: PPCM-PRO modeli yuqori prognostik qiymatni ko'rsatdi va erta xavfni tabaqalash va peripartum kardiomyopatiya bilan og'rigan bemorlarni klinik boshqarishni optimallashtirish uchun foydali bo'lishi mumkin.

Kalit so'zlar: peripartum kardiomyopatiya; homiladorlik; yurak etishmovchiligi; NT-proBNP; prolaktin; katepsin D; ekokardiografiya; prognoz; xavf modeli

Introduction

Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of heart failure that develops toward the end of pregnancy or within the first months after delivery in the absence of any other identifiable cause of myocardial dysfunction. The disorder is characterized by left ventricular systolic dysfunction and clinical manifestations of heart failure occurring in previously healthy women during the peripartum period. According to current position statements of the European Society of Cardiology, the principal diagnostic criterion for PPCM is reduced left ventricular ejection fraction, usually below 45%, whereas cardiac chamber dilatation may be present but is not required for diagnosis [1,2].

Despite its relatively low prevalence, PPCM has considerable clinical importance. The disease may result in severe heart failure, cardiogenic shock, thromboembolic complications, and increased maternal mortality. In addition, a substantial proportion of patients experience persistent myocardial dysfunction requiring prolonged cardiology follow-up. Contemporary studies indicate that complete recovery of left ventricular systolic function occurs in approximately 50-70% of cases, whereas the remainder develop persistent myocardial remodeling or chronic heart failure [1,3,4].

The epidemiology of PPCM demonstrates marked geographic variability. In developed countries, its incidence is estimated at approximately 1 case per 1,000-4,000 deliveries, whereas in certain

regions of Africa and the Caribbean the condition appears to be substantially more frequent. These differences are likely attributable to a combination of genetic, vascular, metabolic, and socioeconomic factors, as well as differences in diagnostic capacity and access to medical care [3-5].

Current concepts of PPCM pathogenesis are based on multifactorial myocardial injury. Major mechanisms include oxidative stress, endothelial dysfunction, impaired angiogenic balance, inflammatory activation, microvascular injury, and pregnancy-related hormonal effects [1,3,6]. One of the best studied pathogenetic hypotheses is the prolactin-dependent mechanism. According to this model, oxidative stress promotes cleavage of full-length prolactin by cathepsin D, generating a 16-kDa fragment with pronounced antiangiogenic, proapoptotic, and cardiotoxic properties. This fragment is capable of damaging endothelial cells and cardiomyocytes, amplifying inflammatory activation, and contributing to pathological myocardial remodeling [1,6,7].

The clinical course of PPCM is highly heterogeneous. Some patients experience marked improvement and recovery of left ventricular function within the first months after delivery, whereas others continue to demonstrate signs of heart failure, chamber dilatation, and subclinical myocardial dysfunction. Adverse disease course has been associated with more severe initial myocardial dysfunction,

elevated levels of heart failure and inflammatory biomarkers, and delayed diagnosis [3,4,8].

In recent years, increasing attention has been directed toward identifying laboratory and instrumental markers capable of predicting PPCM course at an early stage. The most extensively studied biomarker is NT-proBNP, which reflects myocardial stress and hemodynamic overload. Higher NT-proBNP levels have been shown to be associated with more pronounced systolic dysfunction and a lower likelihood of myocardial recovery [8,9].

In addition to NT-proBNP, cathepsin D and prolactin are of particular interest as markers of activation of the prolactin-dependent pathogenic pathway. Furthermore, systemic inflammation, reflected by elevated C-reactive protein and interleukin-6 levels, may contribute to progressive myocardial injury and adverse prognosis [3,6,7].

Echocardiographic parameters are also of major importance in assessing disease severity, as they reflect the extent of structural and functional myocardial damage. The most informative indicators include left ventricular ejection fraction, chamber size, and indices of diastolic function. However, in most studies these parameters are analyzed in isolation, whereas their combination with laboratory markers of myocardial stress and inflammation may provide more accurate risk stratification for adverse outcomes [1,3].

In view of these considerations, the aim of the present study was to evaluate the prognostic significance of clinical, laboratory, and echocardiographic parameters and to develop an integrated risk model for adverse outcomes in PPCM.

Materials and Methods

Study design and setting

This comparative clinical observational study was conducted at the Departments of Obstetrics and Gynecology and Internal Medicine of AMU between 2019 and 2023. A total of 75 pregnant women were enrolled, including 60 in the main group and 15 in the control group.

Eligibility criteria

Pregnant women were eligible for inclusion if they met the following criteria: gestational age from the first to the third trimester; presence of clinical and anamnestic features suggestive of possible cardiovascular maladaptation during pregnancy; written informed consent to participate in the study; and availability of complete medical documentation.

Patients were excluded in the presence of previously diagnosed organic heart disease, including congenital or acquired valvular defects and cardiomyopathies of other etiologies; severe extragenital pathology likely to substantially affect pregnancy course; chronic systemic disease in the stage of decompensation; or refusal to participate in the study.

Study groups

The main group included 60 pregnant women with clinical and anamnestic signs suggestive of PPCM. According to gestational age at the time of examination, these patients were stratified into three subgroups: Group I, women examined during the first trimester (up to 13 weeks of gestation; $n = 20$); Group II, women examined during the second trimester (14-27 weeks of gestation; $n = 20$); and Group III, women examined during the third trimester (28 weeks of gestation or more; $n = 20$).

This stratification allowed comparative assessment of clinical, anamnestic, and somatic risk factors according to gestational stage.

The control group consisted of 15 apparently healthy pregnant women with physiological pregnancies and no evidence of cardiovascular pathology.

The mean age of the women was 30.8 ± 4.2 years in the main group and 27.6 ± 5.1 years in the control group.

Follow-up schedule

Assessments were performed at three time points: during pregnancy, on postpartum day 10, and 6 months after delivery.

Clinical and instrumental assessment

All participants underwent comprehensive clinical and anamnestic evaluation, including assessment of obstetric and gynecologic history, somatic status, and risk factors for PPCM. Instrumental examination included 12-lead electrocardiography using the Schiller AT-102 system (Switzerland) [10], echocardiography performed on a Philips EPIQ 7 device using standard apical views to calculate left ventricular ejection

fraction by the Simpson method, and Doppler echocardiography, when indicated, to assess diastolic function and pulmonary artery pressure [11].

Laboratory assessment

Venous blood samples were collected in the morning after an overnight fast, with the patient in a seated position, using vacuum tubes containing a clot activator [12]. Samples were centrifuged at 3000 rpm for 10 minutes at $+4^\circ\text{C}$. Serum was stored at -80°C until analysis [13].

Cathepsin D was measured by enzyme-linked immunosorbent assay using the SensoLyte 520 Cathepsin D Assay Kit (AnaSpec, USA) [14]. Prolactin was measured by immunochemiluminescent assay on the Cobas e601 analyzer (Roche Diagnostics, Switzerland) [15]. NT-proBNP was determined using Elecsys test systems (Roche Diagnostics, Switzerland) [16].

In addition, inflammatory markers, including C-reactive protein and interleukin-6, were assessed.

Outcome definition

The primary study endpoint was an adverse outcome, defined as incomplete recovery of myocardial contractile function 6 months postpartum, corresponding to $\text{LVEF} < 50\%$.

Statistical analysis

Statistical analysis was performed using SPSS Statistics 26.0 (IBM Corp., USA) and MedCalc software. Continuous variables are presented as mean $\pm$ standard deviation. Comparisons between two groups were performed using Student's t-test for independent samples. Comparisons among more than two groups were performed using one-way analysis of variance with post hoc analysis. Categorical variables were analyzed using Pearson's chi-square test. A p value < 0.05 was considered statistically significant.

To identify factors associated with adverse PPCM course, a prognostic model was developed. First, ROC analysis was performed to determine optimal threshold values for clinical, laboratory, and echocardiographic parameters associated with adverse outcome. Optimal cut-off values were defined using the Youden index. Second, variables with the highest diagnostic significance were converted into binary variables and entered into a penalized logistic regression model. Penalized regression was used to obtain more stable coefficient estimates in the setting of a relatively small sample size and marked separation between clinical groups.

The diagnostic performance of the model was assessed by ROC analysis, with calculation of the area under the curve, sensitivity, and specificity.

Results

Clinical characteristics

Analysis of the clinical data showed that the most favorable profile was observed in Group I. Although transient deterioration occurred in the early postpartum period, these patients demonstrated clear recovery of myocardial contractile function by 6 months, accompanied by fewer complaints, regression of tachycardia, and improvement in overall clinical status.

In Group II, the disease course was less favorable. During pregnancy, these patients already showed more pronounced signs of heart failure, a hypotensive hemodynamic profile, and lower LVEF values. Although improvement was observed at 6 months, the degree of recovery was less marked than in Group I.

The most severe form of PPCM was recorded in Group III. These patients were characterized by more pronounced clinical symptoms, a combination of tachycardia and arterial hypertension, more frequent hospitalizations, lower baseline LVEF values, and slower reverse myocardial remodeling.

The mean age of patients did not differ significantly across the study groups and was 31.2 ± 4.6 years in Group I, 30.7 ± 4.1 years in Group II, and 32.0 ± 4.9 years in Group III ($p = 0.42$). At initial assessment, NYHA functional class differed significantly between groups. In Group I, most patients had NYHA class I-II symptoms, with a mean value of 1.6 ± 0.5 . In Group II, the mean NYHA class was 2.2 ± 0.6 , whereas Group III exhibited the most pronounced heart failure symptoms, with a mean of 2.6 ± 0.7 ($p < 0.01$) (Tabl.1).

Heart rate also differed between groups, with significantly higher values observed in Groups II and III compared with Group I.

Table 1.

Clinical and hemodynamic characteristics of patients with PPCM

Parameter	Group I	Group II	Group III	p value
Age, years	31.2 ± 4.6	30.7 ± 4.1	32.0 ± 4.9	0.42
NYHA class	1.6 ± 0.5	2.2 ± 0.6	2.6 ± 0.7	<0.01
Heart rate, beats/min	88 ± 9	96 ± 11	101 ± 12	<0.01

Reduced arterial blood pressure was more frequently observed in Group II, consistent with a hypotensive hemodynamic pattern, whereas Group III patients showed higher systolic blood pressure values, reflecting a hypertensive disease profile.

Laboratory findings

Analysis of laboratory parameters revealed significant intergroup differences in biomarkers of myocardial stress, hormone-proteolytic activation, and inflammatory response.

NT-proBNP, reflecting myocardial hemodynamic overload, increased significantly with increasing disease severity. Mean values were 620 ± 210 pg/mL in Group I, 980 ± 340 pg/mL in Group II, and 1240 ± 420 pg/mL in Group III ($p < 0.001$). Using the ROC-derived cut-off value of ≥ 890 pg/mL, elevated levels were observed in 35%, 65%, and 85% of patients in Groups I, II, and III, respectively.

Cathepsin D also showed a pronounced intergroup gradient, with mean values of 15.4 ± 4.2 ng/mL, 19.8 ± 5.1 ng/mL, and 23.6 ± 6.4 ng/mL in Groups I, II, and III, respectively ($p < 0.01$). At a threshold of ≥ 18.6 ng/mL, elevated levels were found in 30%, 60%, and 80% of patients, respectively.

Prolactin levels likewise increased with worsening clinical profile. Mean concentrations were 2450 ± 780 mIU/L in Group I, 3080 ± 960 mIU/L in Group II, and 3520 ± 1040 mIU/L in Group III ($p < 0.01$). At the diagnostic cut-off of ≥ 2920 mIU/L, increased prolactin was present in 40%, 70%, and 90% of patients, respectively.

Inflammatory markers followed a similar pattern. C-reactive protein increased from 3.1 ± 1.5 mg/L in Group I to 5.4 ± 2.1 mg/L in Group II and 6.2 ± 2.3 mg/L in Group III ($p < 0.01$). Interleukin-6 concentrations were 2.1 ± 0.9 pg/mL, 3.4 ± 1.2 pg/mL, and 4.1 ± 1.5 pg/mL, respectively ($p < 0.01$) (tabl.2).

Table 2.

Laboratory parameters in patients with PPCM

Parameter	Group I	Group II	Group III	p value
NT-proBNP, pg/mL	620 ± 210	980 ± 340	1240 ± 420	<0.001
Cathepsin D, ng/mL	15.4 ± 4.2	19.8 ± 5.1	23.6 ± 6.4	<0.01
Prolactin, mIU/L	2450 ± 780	3080 ± 960	3520 ± 1040	<0.01
CRP, mg/L	3.1 ± 1.5	5.4 ± 2.1	6.2 ± 2.3	<0.01
IL-6, pg/mL	2.1 ± 0.9	3.4 ± 1.2	4.1 ± 1.5	<0.01

Correlation between biomarkers and left ventricular function

NT-proBNP showed the strongest inverse correlation with LVEF ($r = -0.62$; $p < 0.001$). Similar negative correlations were found for prolactin ($r = -0.55$; $p < 0.01$) and cathepsin D ($r = -0.51$; $p < 0.01$). Inflammatory markers also demonstrated significant inverse correlations with LVEF: CRP, $r = -0.43$ ($p < 0.05$), and IL-6, $r = -0.41$ ($p < 0.05$) (tabl.3).

Laboratory predictors of adverse outcome

At 6 months postpartum, incomplete recovery of myocardial contractile function (LVEF < 50%) was observed in 27 patients (45%). Baseline biomarker levels were significantly higher among patients with adverse outcome (Tabl.3).

Table 3.

Baseline laboratory markers according to 6-month outcome

Parameter	Favorable outcome	Adverse outcome	p value
NT-proBNP, pg/mL	720 ± 260	1180 ± 380	<0.001
Cathepsin D, ng/mL	16.8 ± 4.6	22.4 ± 5.8	<0.01
Prolactin, mIU/L	2580 ± 820	3350 ± 1010	<0.01
CRP, mg/L	3.8 ± 1.7	5.9 ± 2.2	<0.01
IL-6, pg/mL	2.4 ± 1.0	3.8 ± 1.3	<0.01

Thus, elevated NT-proBNP, prolactin, and cathepsin D showed the strongest association with incomplete myocardial recovery.

Echocardiographic predictors of adverse outcome

Echocardiographic analysis revealed significant differences among groups in structural and functional myocardial parameters. The most pronounced abnormalities were observed in Group III and included lower LVEF, increased left ventricular end-diastolic diameter (LVEDD) and end-systolic diameter (LVESD), and signs of diastolic dysfunction.

ROC analysis identified the following informative threshold values: LVEF $\leq 41\%$ (AUC = 0.91; sensitivity 87%; specificity 81%); LVEDD ≥ 58 mm (AUC = 0.82; sensitivity 78%; specificity 73%); LVESD ≥ 42 mm (AUC = 0.84; sensitivity 80%; specificity 75%); E/A < 0.80 (AUC = 0.79; sensitivity 74%; specificity 70%).

Development of the PPCM-PRO model

Based on ROC analysis, the most informative variables were included in a penalized logistic regression model. The model

Mean LVEF values were 46.8 ± 4.1% in Group I, 41.5 ± 5.0% in Group II, and 37.2 ± 4.8% in Group III ($p < 0.001$). LVEDD measured 54 ± 5 mm, 58 ± 6 mm, and 61 ± 7 mm, respectively ($p < 0.01$), while LVESD was 38 ± 4 mm, 42 ± 5 mm, and 45 ± 6 mm ($p < 0.01$). The E/A ratio also decreased significantly with increasing disease severity, measuring 1.02 ± 0.21 in Group I, 0.86 ± 0.19 in Group II, and 0.74 ± 0.17 in Group III ($p < 0.01$).

incorporated NT-proBNP, cathepsin D, prolactin, CRP, IL-6, left ventricular ejection fraction, and heart rate as independent predictors.

The probability of adverse outcome was calculated using a logistic regression function. Among all variables, prolactin, NT-proBNP, and cathepsin D demonstrated the strongest contribution to risk prediction.

Simplified PPCM-PRO risk score

Laboratory predictors of adverse outcome

At 6 months postpartum, incomplete recovery of myocardial contractile function (LVEF < 50%) was observed in 27 patients (45%). Baseline biomarker levels were significantly higher in patients with adverse outcome compared to those with favorable recovery (Table 3).

In particular, patients with adverse outcome demonstrated elevated levels of NT-proBNP, cathepsin D, prolactin, C-reactive protein, and interleukin-6 (all $p < 0.01$).

Among these variables, NT-proBNP, prolactin, and cathepsin D showed the most pronounced intergroup differences, suggesting their potential role as key markers associated with unfavorable myocardial recovery. These findings were further supported by multivariable analysis, where NT-proBNP, prolactin, and cathepsin D contributed most significantly to the final prognostic model (Table 4).

Table 4.

PPCM-PRO risk score	
Variable	Points
Prolactin ≥ 2920 mIU/L	3
NT-proBNP ≥ 890 pg/mL	2
Cathepsin D ≥ 18.6 ng/mL	2
CRP ≥ 5.0 mg/L	1
IL-6 ≥ 3.0 pg/mL	1
LVEF $\leq 41\%$	1
Heart rate ≥ 96 beats/min	1

Maximum total score: 11.

Based on the total PPCM-PRO score, patients were stratified into three risk categories: low risk (0–2 points), moderate risk (3–5 points), and high risk (≥ 6 points) (Table 5).

Patients classified as high risk demonstrated a substantially higher probability of persistent left ventricular systolic dysfunction at 6 months postpartum compared to those in the low- and moderate-risk groups. This stratification allows early identification of patients requiring closer monitoring and more intensive management.

Table 5.

Risk categories according to PPCM-PRO	
Total score	Risk category
0-2	Low risk
3-5	Moderate risk
≥ 6	High risk

Patients with high prognostic scores had a significantly greater likelihood of persistent systolic myocardial dysfunction at 6 months postpartum.

Diagnostic performance of the model

The area under the ROC curve for the integrated PPCM-PRO model was 0.93 (95% CI 0.86–0.98), indicating excellent discriminatory ability

for predicting adverse PPCM outcomes. The optimal threshold was ≥ 6 points, at which sensitivity reached 94% and specificity 93%.

Among the individual predictors, LVEF, prolactin, and NT-proBNP showed the highest diagnostic value (Fig. 1–3).

Specificity

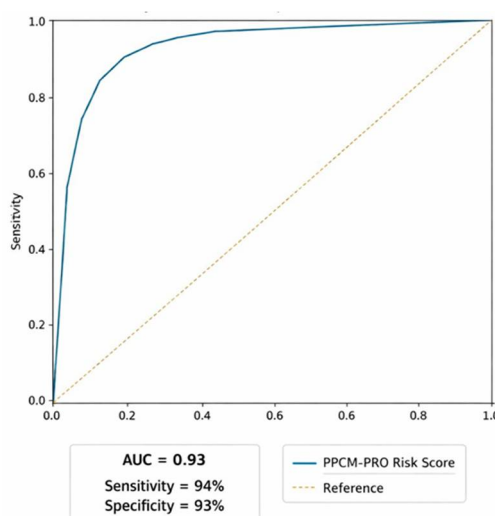


Figure 1. ROC curve of the PPCM-PRO risk score for predicting adverse outcome in peripartum cardiomyopathy.

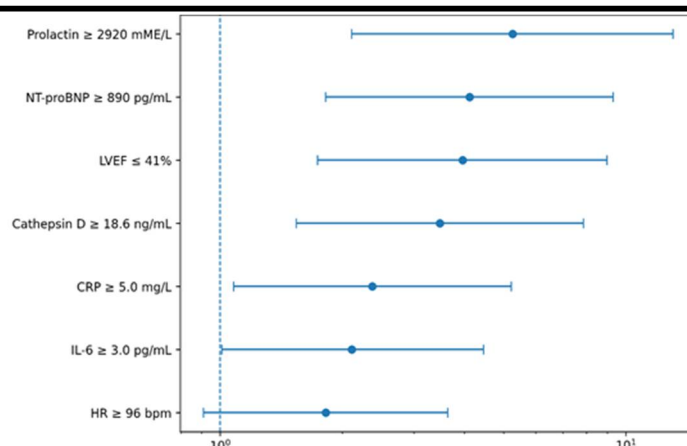


Figure 2. Forest plot showing odds ratios of independent predictors of adverse outcome in PPCM.

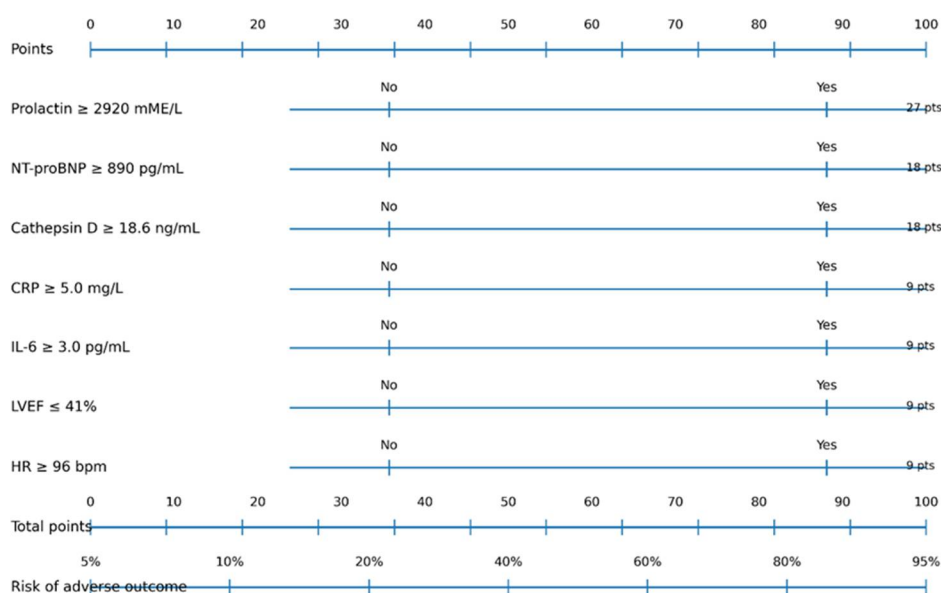


Figure 3. Nomogram of the PPCM-PRO risk model for estimating the probability of adverse outcome.

Discussion

The present study confirms that the clinical course of PPCM is highly heterogeneous and is determined by the interplay of hemodynamic, hormonal, inflammatory, and structural-functional myocardial abnormalities. These findings are consistent with contemporary evidence indicating substantial variability in clinical presentation, disease progression, and recovery patterns in PPCM [17-19]. The principal objective of this work was to identify clinical, laboratory, and echocardiographic parameters capable of predicting adverse disease course and incomplete recovery of myocardial contractile function within the first 6 months postpartum.

Among the laboratory variables, NT-proBNP emerged as one of the most important predictors. Increased concentrations were strongly associated with more severe left ventricular systolic dysfunction and adverse outcome. These findings are in line with previous reports indicating that higher NT-proBNP levels in PPCM are associated with a reduced probability of left ventricular recovery and a more severe clinical course [8-10]. This observation is also consistent with the broader concept that natriuretic peptides reflect myocardial stress and hemodynamic overload in heart failure [1,3].

Another important observation was the association between adverse outcome and elevated prolactin and cathepsin D levels. These findings are biologically plausible in the context of the prolactin-dependent hypothesis of myocardial injury, according to which oxidative stress promotes the cleavage of full-length prolactin by cathepsin D, resulting in a cardiotoxic 16-kDa prolactin fragment [7]. This mechanism has been implicated in endothelial dysfunction, impaired microcirculation, and progressive myocardial dysfunction [3,6,7]. Current experimental and clinical evidence further supports the role of prolactin-mediated pathways in PPCM pathogenesis [20].

Inflammatory activation also appeared to play a relevant role. Patients with more severe disease demonstrated higher CRP and IL-6 levels, supporting the concept that systemic inflammation contributes to myocardial injury and adverse remodeling in PPCM [3,6]. These observations are in agreement with recent studies emphasizing the contribution of inflammatory and immune-mediated mechanisms to disease progression and unfavorable outcome [18,19].

Echocardiographic parameters were likewise highly informative. Lower LVEF, increased chamber dimensions, and impaired diastolic function characterized patients with the least favorable profiles. These observations are consistent with earlier studies showing that baseline LVEF is among the strongest predictors of long-term outcome in PPCM [9,10,17]. Furthermore, previous investigations have shown that impaired baseline systolic function is associated with delayed myocardial recovery and persistent ventricular dysfunction [21,22].

Importantly, PPCM is increasingly recognized as a multifactorial disease with contributions from vascular, genetic, and hormonal pathways. Recent studies suggest a shared genetic predisposition between PPCM and dilated cardiomyopathy, further supporting the heterogeneity of the disease [23]. Moreover, angiogenic imbalance has been proposed as a key mechanism contributing to myocardial dysfunction in PPCM [24]. The association between PPCM and hypertensive disorders of pregnancy, particularly pre-eclampsia, has also been widely reported and may further aggravate myocardial stress and endothelial injury [25].

On the basis of these findings, we developed the integrated PPCM-PRO model, which combines biomarkers of myocardial stress, prolactin-dependent injury, inflammatory response, and left ventricular systolic function. The excellent AUC suggests strong discriminatory performance and supports the use of multicomponent prognostic tools

rather than reliance on individual indicators alone. This approach is in line with current concepts of risk stratification in PPCM [26].

From a clinical perspective, the proposed model may help identify high-risk patients at an early stage and thereby facilitate closer echocardiographic surveillance, serial biomarker assessment, and timely referral to specialized cardiology centers. In this way, the model may serve not only as a research tool but also as a practical instrument for personalized management, in accordance with current clinical recommendations [2,20].

Several limitations should be acknowledged. First, the sample size was relatively small, which may limit generalizability. Second, the single-center design may have contributed to overestimation of model performance. Third, although the selected biomarkers showed strong prognostic value, their interpretation should be standardized in future multicenter studies. Accordingly, larger prospective investigations are needed for external validation of the proposed model [17,26].

Conclusion

The present study demonstrated that adverse outcomes in PPCM are driven by a complex interplay of pathogenic mechanisms, including myocardial stress, activation of prolactin-cathepsin D-dependent myocardial injury, systemic inflammatory response, and marked structural-functional remodeling of the left ventricle.

The most significant prognostic factors for adverse outcome were elevated NT-proBNP, prolactin, and cathepsin D levels, together with reduced LVEF. Additional prognostic information was provided by

inflammatory markers such as CRP and IL-6, as well as by clinical indicators reflecting the severity of hemodynamic disturbances.

Based on these findings, the integrated PPCM-PRO model was developed by combining laboratory and echocardiographic parameters. The model demonstrated high diagnostic accuracy and may represent a promising tool for early risk stratification in patients with PPCM.

Practical Implications

In patients with suspected PPCM, comprehensive assessment should include not only clinical and echocardiographic parameters but also laboratory biomarkers, particularly NT-proBNP, prolactin, and cathepsin D. Elevated values of these markers may indicate a higher likelihood of adverse outcome and justify closer dynamic follow-up. Reduced LVEF, increased chamber dimensions, and signs of diastolic dysfunction should also be incorporated into risk stratification. Use of the PPCM-PRO model may facilitate early identification of high-risk patients and support optimization of management strategies, including more frequent echocardiographic monitoring, serial biomarker assessment, and timely referral to specialized cardiology centers.

Study Limitations

The proposed prognostic model was developed on the basis of a relatively small patient sample. In addition, marked separation between clinical groups may have contributed to overestimation of diagnostic performance. Therefore, the results require external validation in larger independent cohorts.

References

1. Bauersachs J, König T, van der Meer P, et al. Pathophysiology, diagnosis and management of peripartum cardiomyopathy. *Eur J Heart Fail.* 2019;21(7):827-843. doi:10.1002/ejhf.1493
2. Arbelo E, Protonotarios A, Gimeno JR, et al. 2023 ESC Guidelines for the management of cardiomyopathies. *Eur Heart J.* 2023;44(37):3503-3626. doi:10.1093/eurheartj/ehad194
3. Sigauke F, Mayosi BM, Sliwa K. Peripartum cardiomyopathy: a comprehensive review. *Heart Fail Rev.* 2024.
4. Sliwa K, Hilfiker-Kleiner D. Peripartum cardiomyopathy. *Lancet.* 2025.
5. Arany Z, Elkayam U. Peripartum cardiomyopathy. *Circulation.* 2016;133(14):1397-1409. doi:10.1161/CIRCULATIONAHA.115.020491
6. Kryczka KE, et al. Biomarkers in peripartum cardiomyopathy. *Biomolecules.* 2024.
7. Hilfiker-Kleiner D, Kaminski K, Podewski E, et al. A cathepsin D-cleaved 16-kDa form of prolactin mediates postpartum cardiomyopathy. *Cell.* 2007;128(3):589-600. doi:10.1016/j.cell.2006.12.036
8. Forster O, Hilfiker-Kleiner D, Ansari AA, et al. Relevance of NT-proBNP in peripartum cardiomyopathy. *Clin Res Cardiol.* 2008;97:808-814.
9. Golland S, Modi K, Bitar F, et al. Predictors of recovery in peripartum cardiomyopathy. *Int J Cardiol.* 2009.
10. McNamara DM, Elkayam U, Alharethi R, et al. Clinical outcomes for peripartum cardiomyopathy in North America: results of the IPAC study. *J Am Coll Cardiol.* 2015;66(8):905-914. doi:10.1016/j.jacc.2015.06.1309
11. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults. *Eur Heart J Cardiovasc Imaging.* 2015;16(3):233-270. doi:10.1093/ehjci/jev014
12. Tuck MK, Chan DW, Chia D, et al. Standard operating procedures for serum and plasma collection. *J Proteome Res.* 2009;8(1):113-117. doi:10.1021/pr800545q
13. Rai AJ, Gelfand CA, Haywood BC, et al. Processing of serum and plasma samples for proteomic studies. *Clin Proteomics.* 2005;2(1-2):17-23.
14. Turk V, Stoka V, Turk D. Human cathepsin D: structure, function and role in disease. *Biol Chem.* 2012;393(9):1091-1103. doi:10.1515/hsz-2012-0168
15. Melmed S, Casanueva FF, Hoffman AR, et al. Diagnosis and treatment of hyperprolactinemia. *J Clin Endocrinol Metab.* 2011;96(2):273-288. doi:10.1210/jc.2010-1802
16. Maisel A, Mueller C, Adams K, et al. Measurement of BNP and NT-proBNP in heart failure. *Nat Rev Cardiol.* 2008;5(10):594-606. doi:10.1038/ncpcardio1298
17. Davis MB, Arany Z, McNamara DM, Golland S, Elkayam U. Peripartum cardiomyopathy: JACC state-of-the-art review. *J Am Coll Cardiol.* 2020;75(2):207-221. doi:10.1016/j.jacc.2019.11.014
18. Sliwa K, Mebazaa A, Hilfiker-Kleiner D, et al. Clinical characteristics and outcome of patients with peripartum cardiomyopathy. *Eur Heart J.* 2020;41(39):3783-3791. doi:10.1093/eurheartj/ehaa455
19. Honigberg MC, Givertz MM. Peripartum cardiomyopathy. *BMJ.* 2019;364:k5287. doi:10.1136/bmj.k5287
20. Hilfiker-Kleiner D, Haghikia A, Nonhoff J, Bauersachs J. Peripartum cardiomyopathy: current management and future perspectives. *Eur Heart J.* 2015;36:1090-1097. doi:10.1093/eurheartj/ehu454
21. Biteker M, Ilhan E, Biteker G, et al. Predictors of recovery in peripartum cardiomyopathy. *Clin Cardiol.* 2012;35(9):589-593. doi:10.1002/clc.22023
22. Golland S, Modi K, Bitar F, et al. Clinical profile and predictors of complications in peripartum cardiomyopathy. *J Card Fail.* 2009;15:645-650. doi:10.1016/j.cardfail.2009.02.008
23. Ware JS, Li J, Mazaika E, et al. Shared genetic predisposition in peripartum and dilated cardiomyopathy. *N Engl J Med.* 2016;374:233-241. doi:10.1056/NEJMoa1505517

24. Patten IS, Rana S, Shahul S, et al. Cardiac angiogenic imbalance leads to peripartum cardiomyopathy. *Nature*. 2012;485:333-338. doi:10.1038/nature11040
25. Bello N, Rendon ISH, Arany Z. The relationship between pre-eclampsia and peripartum cardiomyopathy. *J Am Coll Cardiol*. 2013;62:1715-1723. doi:10.1016/j.jacc.2013.08.717
26. Elkayam U. Risk stratification in peripartum cardiomyopathy. *Heart Fail Clin*. 2019;15(1):45-55. doi:10.1016/j.hfc.2018.08.007