AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 4, Issue 04, 2026 ISSN (E): 2993-2149

Pathomorphological Features of the Myocardium in Peripartum Cardiomyopathy Depending on Maternal Age and Parity

Zafar. K. Narbaev.,
Andijan state medical institute**,** candidate of Medical sciences., associate Professor.,
E-mail: zafarnarbaev96@gmail.com
Shahnozakhon Z. Narbayeva.,
Andijan state medical institute**,**
E-mail: shakhnoza.narbaeva23@gmail.com

Abstract: Peripartum cardiomyopathy is a serious cardiac disorder associated with pregnancy and the postpartum period. This study investigated the pathomorphological features of the myocardium in women with peripartum cardiomyopathy, with particular attention to the influence of maternal age and parity. Histopathological examination showed that myocardial damage became more pronounced with increasing age and parity. The main findings included degenerative changes in cardiomyocytes, interstitial fibrosis, vascular alterations, and inflammatory cell infiltration. These results suggest that maternal age and parity are important factors influencing myocardial remodeling and disease severity in peripartum cardiomyopathy. The findings may contribute to improving the morphological assessment and prognosis of this condition.

Keywords: peripartum cardiomyopathy, myocardium, pathomorphology, maternal age, parity, fibrosis, histopathology.

Introduction

Peripartum cardiomyopathy (PPCM) is a serious form of heart failure associated with pregnancy. It usually develops during the last month of pregnancy or within the first months after delivery. The disease is characterized by left ventricular systolic dysfunction without another identifiable cause. Although uncommon, PPCM remains an important cause of maternal morbidity and mortality worldwide, and its incidence varies among different populations [1].

Early diagnosis is challenging because symptoms such as dyspnea, fatigue, and peripheral edema are similar to the normal physiological changes of pregnancy. As a result, many women are diagnosed only after significant cardiac dysfunction has developed, increasing the risk of pulmonary edema, arrhythmias, thromboembolism, cardiogenic shock, and death [2].

The pathogenesis of PPCM is complex and remains incompletely understood. Current evidence indicates that hormonal changes, oxidative stress, vascular dysfunction, inflammation, immune imbalance, and genetic susceptibility contribute to myocardial injury. Pregnancy may act as a trigger in women with an underlying predisposition, leading to structural and functional changes in the myocardium [3].

Pregnancy places considerable hemodynamic stress on the cardiovascular system through

increased blood volume, heart rate, and cardiac output. In susceptible women, these adaptations may exceed myocardial reserve, resulting in cardiomyocyte damage, ventricular dilation, and myocardial remodeling [4].

Maternal age and parity are considered important factors affecting the development and severity of PPCM. Advanced maternal age is associated with reduced cardiovascular reserve and a higher prevalence of hypertension, diabetes, and preeclampsia, while repeated pregnancies expose the myocardium to recurrent hemodynamic and hormonal stress. However, the relationship between these factors and myocardial structural changes has not been fully clarified [5].

Hypertensive disorders of pregnancy, particularly preeclampsia, are closely associated with PPCM. Both conditions share mechanisms such as endothelial dysfunction and vascular imbalance, suggesting a common pathway of myocardial injury during the peripartum period [6].

Most published studies have focused on clinical manifestations, echocardiographic findings, and treatment outcomes. Although echocardiography is essential for evaluating cardiac function, it cannot fully demonstrate microscopic myocardial alterations. Histopathological examination provides valuable information on cardiomyocyte degeneration, inflammatory infiltration, vascular abnormalities, and fibrosis, thereby improving the understanding of disease progression [7].

Despite recent advances, information on the influence of maternal age and parity on myocardial morphology remains limited. Further pathological studies are needed to identify structural changes associated with these risk factors and to improve disease assessment and prognosis [8].

Therefore, investigating the age- and parity-related pathomorphological features of the myocardium in PPCM is highly relevant. A better understanding of myocardial remodeling may improve morphological diagnosis, support prognostic evaluation, and contribute to more individualized management of women with peripartum cardiomyopathy [9].

Aim of the study

The aim of this study was to investigate the age- and parity-related pathomorphological features of the myocardium in women with peripartum cardiomyopathy and to evaluate the relationship between these morphological changes and the severity of myocardial injury [10].

Materials and Methods

This study was carried out at the Andijan Regional Pathology Center. The study included 64 autopsy cases and medical records of women who died with a diagnosis of peripartum cardiomyopathy between 2020 and 2025. Only 64 cases with a confirmed diagnosis of peripartum cardiomyopathy based on clinical and pathological findings were included in the study. Example only: The maternal age ranged from 19 to 42 years, with a mean age of 31.8 ± 5.4 years. For comparative analysis, the cases were classified into three age groups: <25 years, 25–34 years, and ≥35 years. According to parity, the patients were divided into primiparous and multiparous groups. Myocardial tissue samples were collected from the left ventricle, right ventricle, and interventricular septum during routine autopsy. The specimens were fixed in 10% neutral buffered formalin, embedded in paraffin, and cut into 4–5 μm sections. The sections were stained with hematoxylin and eosin (H&E) and Masson's trichrome. The histopathological examination focused on cardiomyocyte degeneration, hypertrophy, interstitial edema, inflammatory cell infiltration, microvascular changes, and the degree of myocardial fibrosis. Statistical analysis was performed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation (Mean ± SD). A p value of <0.05 was considered statistically significant.

Results and Discussion

During the study, myocardial tissue obtained from women who died with a diagnosis of peripartum cardiomyopathy was examined pathomorphologically. In all cases, both the macroscopic and microscopic structure of the myocardium was evaluated. Particular attention was paid to changes in cardiomyocytes, the interstitial tissue, and the myocardial microvasculature. The morphological findings were also compared according to maternal age and parity [11].

Macroscopic examination showed that most hearts were enlarged. Dilatation of the left ventricle and enlargement of the cardiac chambers were the most common findings. The myocardium had a soft consistency, and the cut surface appeared uneven and congested with blood. In some cases, mild thinning of the left ventricular wall was observed, whereas in others the myocardial wall thickness was relatively preserved despite marked chamber dilatation [12]. No characteristic pathological changes were found in the endocardium or heart valves. Overall, the macroscopic findings indicated that dilatation was the main structural change in the hearts of women with peripartum cardiomyopathy [13]. These findings were further evaluated together with the histopathological changes observed on microscopic examination (Figure 1).

Figure 1. Gross appearance of the heart in a woman with peripartum cardiomyopathy. The heart is enlarged with dilatation of the left ventricle and enlargement of the cardiac chambers. The myocardium appears soft, and the cut surface is uneven and congested with blood, indicating dilated structural changes characteristic of peripartum cardiomyopathy.

Microscopic examination of the myocardium showed pathological changes in both cardiomyocytes and the interstitial tissue. As shown in Figure 2, the myocardial fibers were irregularly arranged in most cases. Degenerative changes were observed in the cardiomyocytes. The cytoplasm of many cells was strongly eosinophilic. Some cardiomyocytes showed cytoplasmic vacuolization and pyknotic nuclei. Interstitial edema was found in most cases. Small blood vessels were dilated and congested with blood. In several samples, erythrocytes were seen outside the blood vessels. Mild inflammatory cell infiltration was also present in the interstitial tissue. The infiltrate mainly consisted of lymphocytes and macrophages [14]. These findings indicate that the main histopathological features of peripartum cardiomyopathy are cardiomyocyte degeneration, interstitial edema, and microvascular changes (Figure 1).

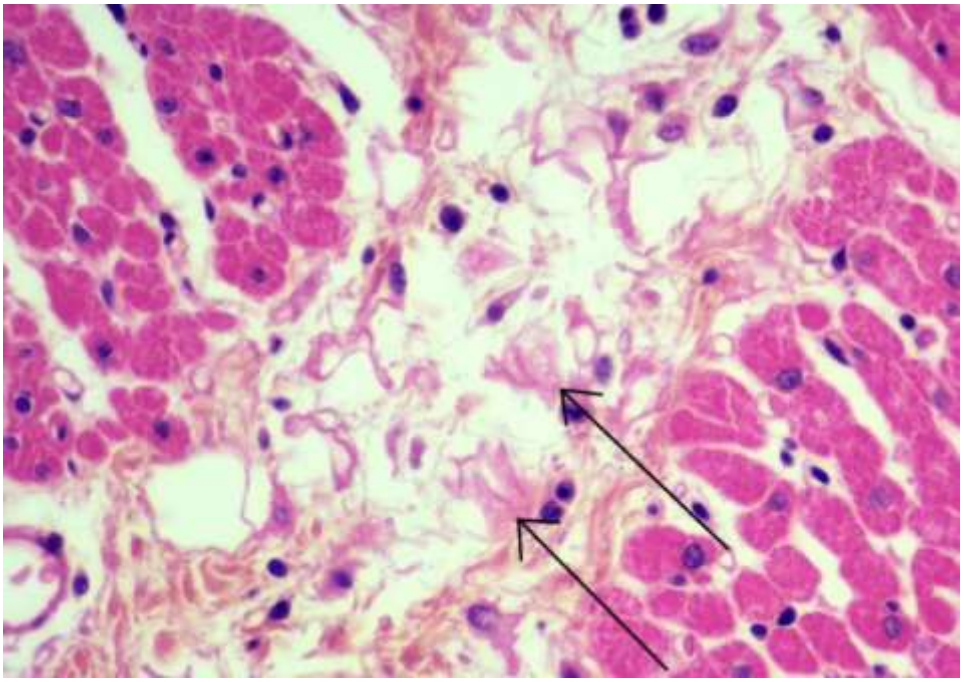

Figure 2. Histopathological appearance of the myocardium in peripartum cardiomyopathy (Hematoxylin and Eosin stain, ×200). The myocardial fibers are irregularly arranged. Cardiomyocytes show degenerative changes, including increased eosinophilia, cytoplasmic vacuolization, and pyknotic nuclei. Interstitial edema, dilated congested capillaries, and mild inflammatory cell infiltration are also observed.

Microscopic examination showed hypertrophy of cardiomyocytes in many cases. The cells were enlarged, and their nuclei were bigger than normal. These changes were more obvious in the left ventricular myocardium. Interstitial fibrosis was also observed. Collagen fibers were increased between the myocardial fibers. In some cases, fibrosis was focal, while in others it was more diffuse. As shown in Figure 3**,** fibrotic areas separated the myocardial fibers and changed the normal structure of the myocardium. The degree of fibrosis was higher in women of older age and in multiparous women [15]. These findings suggest that myocardial remodeling becomes more severe with increasing age and parity in patients with peripartum cardiomyopathy (Figure 1).

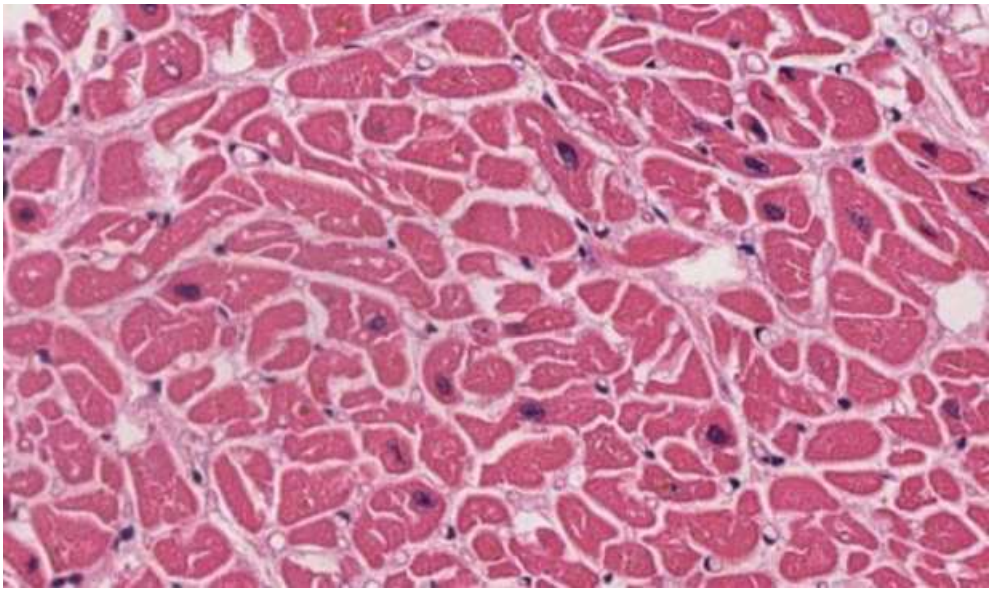

Figure 3. Microscopic appearance of the myocardium showing enlarged cardiomyocytes with enlarged nuclei and interstitial fibrosis. Fibrotic areas separate the myocardial fibers and alter the normal myocardial structure (Hematoxylin and Eosin stain, ×200).

The histopathological findings were compared according to maternal age and parity. Degenerative changes of cardiomyocytes, interstitial edema, myocardial hypertrophy, and fibrosis were identified in all study groups. However, the severity of these pathological changes differed between the groups. Women aged 35 years and older showed more pronounced myocardial remodeling than younger women. Cardiomyocyte hypertrophy was more evident in this group, with enlarged myocardial fibers and hyperchromatic nuclei. Interstitial fibrosis was also more extensive, resulting in disruption of the normal myocardial architecture. In contrast, younger women mainly demonstrated degenerative changes and mild interstitial edema, while fibrosis was less pronounced. Similar differences were observed according to parity. Multiparous women showed more marked cardiomyocyte hypertrophy and interstitial fibrosis than primiparous women. Dilatation and congestion of small intramyocardial blood vessels were also observed more frequently in multiparous cases. Mild inflammatory cell infiltration was detected in both groups without obvious differences in distribution. Overall, the findings indicate that increasing maternal age and higher parity are associated with more advanced myocardial remodeling in peripartum cardiomyopathy. These changes were characterized mainly by cardiomyocyte hypertrophy, increased interstitial collagen deposition, and progressive myocardial fibrosis (see. Table 1 and 2).

Table 1. Distribution of the study population according to maternal age and parity (n = 64)

Variable	Number of cases (n)	Percentage (%)
Maternal age		
<25 years	12	18.8
25–34 years	34	53.1
≥35 years	18	28.1
Total	64	100.0
Parity		
Primiparous	27	42.2
Multiparous	37	57.8
Total	64	100.0

Table 2. Comparison of the main histopathological findings according to maternal age and parity

Histopathological finding	<25 years	25–34 years	≥35 years	Primiparous	Multiparous
Cardiomyocyte degeneration	+	++	+++	++	+++
Cardiomyocyte hypertrophy	+	++	+++	++	+++
Interstitial edema	++	++	+++	++	++
Inflammatory cell infiltration	+	+	++	+	++
Microvascular congestion	++	++	+++	++	+++
Interstitial fibrosis	+	++	+++	+	+++

Abbreviations: *+, mild changes; ++, moderate changes; +++, severe changes.*

Table 1 presents the distribution of the study population according to maternal age and parity. Among the 64 women included in the study, the largest proportion belonged to the 25-34 years age group, followed by women aged ≥35 years and those younger than 25 years. According to parity, multiparous women accounted for a greater proportion of cases than primiparous women. These findings indicate that peripartum cardiomyopathy was observed more frequently among women aged 25-34 years and in multiparous patients. This distribution provided the basis for the subsequent comparative histopathological analysis according to maternal age and parity.

Table 2 shows that the severity of histopathological changes increased with both maternal age and parity. Women aged ≥35 years demonstrated the most pronounced cardiomyocyte degeneration, hypertrophy, microvascular congestion, and interstitial fibrosis. In comparison, women younger than 25 years showed predominantly mild histopathological changes. According to parity, multiparous women exhibited more severe myocardial remodeling than primiparous women. Cardiomyocyte hypertrophy and interstitial fibrosis were the most prominent findings in

the multiparous group. Degenerative changes and microvascular congestion were also more evident in these patients, whereas inflammatory cell infiltration and interstitial edema showed only minor differences between the parity groups.

These observations indicate that advanced maternal age and multiparity are associated with more severe structural changes of the myocardium in patients with peripartum cardiomyopathy.

Conclusion

The present study demonstrated that peripartum cardiomyopathy is associated with significant structural alterations of the myocardium, including cardiomyocyte degeneration, hypertrophy, interstitial edema, microvascular congestion, inflammatory cell infiltration, and progressive fibrosis. The severity of these histopathological changes increased with maternal age and parity. Women aged 35 years and older, as well as multiparous women, showed more pronounced myocardial remodeling characterized by marked cardiomyocyte hypertrophy, extensive interstitial fibrosis, and greater microvascular alterations than younger and primiparous women. These findings indicate that maternal age and parity are important factors influencing the morphological progression of peripartum cardiomyopathy. Histopathological evaluation of myocardial tissue provides valuable information for understanding disease severity and myocardial remodeling. The results of this study contribute to a better understanding of the pathological basis of peripartum cardiomyopathy and may support improved morphological diagnosis, risk assessment, and future .

References

1. J. Bauersachs, T. König, P. van der Meer, D. Hilfiker-Kleiner, A. Mbakwem, R. Hamdan, A. M. Jackson, et al., "Pathophysiology, Diagnosis and Management of Peripartum Cardiomyopathy: A Position Statement from the Heart Failure Association of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy," *European Journal of Heart Failure*, vol. 21, no. 7, pp. 827–843, Jul. 2019, doi: 10.1002/ejhf.1493.
2. M. B. Davis, Z. Arany, D. M. McNamara, S. Goland, and U. Elkayam, "Peripartum Cardiomyopathy: JACC State-of-the-Art Review," *Journal of the American College of Cardiology*, vol. 75, no. 2, pp. 207–221, Jan. 2020, doi: 10.1016/j.jacc.2019.11.014.
3. K. Sliwa, M. C. Petrie, P. van der Meer, A. Mebazaa, D. Hilfiker-Kleiner, A. M. Jackson, et al., "Clinical Presentation, Management and 6-Month Outcomes in Women with Peripartum Cardiomyopathy: An ESC EORP Registry," *European Heart Journal*, vol. 41, no. 39, pp. 3787–3797, Oct. 2020, doi: 10.1093/eurheartj/ehaa455.
4. M. F. Hoes, Z. Arany, J. Bauersachs, D. Hilfiker-Kleiner, et al., "Pathophysiology and Risk Factors of Peripartum Cardiomyopathy," *Nature Reviews Cardiology*, vol. 19, no. 9, pp. 555–571, Sep. 2022, doi: 10.1038/s41569-021-00664-8.
5. K. Sliwa, J. Bauersachs, Z. Arany, T. F. Spracklen, and D. Hilfiker-Kleiner, "Peripartum Cardiomyopathy: From Genetics to Management," *European Heart Journal*, vol. 42, no. 32, pp. 3094–3102, Aug. 2021, doi: 10.1093/eurheartj/ehab344.
6. S. Carlson, C. M. Trankle, K. Kessler, et al., "Peripartum Cardiomyopathy: Risks, Diagnosis, and Management," *International Journal of Women's Health*, vol. 15, pp. 89–99, 2023, doi: 10.2147/IJWH.S389788.
7. M. B. Davis and K. Sliwa, "To Infinity and Beyond: Evolving Understanding of Peripartum Cardiomyopathy," *JACC: Heart Failure*, vol. 11, no. 9, pp. 1243–1245, Sep. 2023, doi: 10.1016/j.jchf.2023.06.035.
8. D. M. McNamara, U. Elkayam, R. Alharethi, D. D. Damp, D. H. Hsich, T. E. Ewald, J. D. Modi, et al., "Clinical Outcomes for Peripartum Cardiomyopathy in North America: Results of the IPAC Study," *Journal of the American College of Cardiology*, vol. 66, no. 8, pp. 905–914, Aug. 2015, doi: 10.1016/j.jacc.2015.06.1309.
9. K. Sliwa, M. C. Petrie, D. Hilfiker-Kleiner, J. Mebazaa, J. Bauersachs, et al., "Long-Term Prognosis, Subsequent Pregnancy, Contraception and Overall Management of Peripartum

Cardiomyopathy: Practical Guidance Paper from the Heart Failure Association of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy," *European Journal of Heart Failure*, vol. 20, no. 6, pp. 951–962, Jun. 2018, doi: 10.1002/ejhf.1178.
10. D. Hilfiker-Kleiner, A. Haghikia, J. Nonhoff, et al., "Bromocriptine for the Treatment of Peripartum Cardiomyopathy: A Multicentre Randomized Study," *European Heart Journal*, vol. 38, no. 35, pp. 2671–2679, Sep. 2017, doi: 10.1093/eurheartj/ehx355.
11. U. Elkayam, "Risk Factors of Peripartum Cardiomyopathy," *JACC: Heart Failure*, vol. 11, no. 8, pp. 1025–1027, Aug. 2023, doi: 10.1016/j.jchf.2023.05.011.
12. J. G. Demakis and S. H. Rahimtoola, "Peripartum Cardiomyopathy," *Circulation*, vol. 44, no. 5, pp. 964–968, Nov. 1971, doi: 10.1161/01.CIR.44.5.964.
13. J. Bauersachs, K. Sliwa, D. Hilfiker-Kleiner, et al., "Peripartum Cardiomyopathy: A Comprehensive and Contemporary Review," *European Heart Journal*, vol. 45, no. 8, pp. 693–706, Feb. 2024, doi: 10.1093/eurheartj/ehad728.
14. Z. Arany and U. Elkayam, "Peripartum Cardiomyopathy," *Circulation*, vol. 133, no. 14, pp. 1397–1409, Apr. 2016, doi: 10.1161/CIRCULATIONAHA.115.020491.
15. M. B. Gentry, J. K. Dias, A. Luis, R. Patel, J. Thornton, and G. L. Reed, "African-American Women Have a Higher Risk for Developing Peripartum Cardiomyopathy," *Journal of the American College of Cardiology*, vol. 55, no. 7, pp. 654–659, Feb. 2010, doi: 10.1016/j.jacc.2009.09.043.