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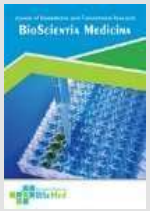

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The Role of NT-proBNP in the Diagnosis of Peripartum Cardiomyopathy with Multiple Left Ventricular Thrombus: A Case Report

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A B S T R A C T

Background: Peripartum cardiomyopathy (PPCM) is a rare but serious condition characterized by left ventricular dysfunction in late pregnancy or early postpartum. Early diagnosis is essential to prevent complications such as thromboembolism. N-terminal pro-B-type natriuretic peptide (NT-proBNP) is a promising biomarker for the diagnosis of PPCM. **Case presentation:** An 18-year-old female presented with progressive shortness of breath and generalized edema. Physical examination and echocardiography revealed acute heart failure with multiple left ventricular thrombus. Very high levels of NT-proBNP (1,796 pg/mL) supported the diagnosis of PPCM. The patient was successfully treated with standard heart failure therapy and anticoagulants. **Conclusion:** This case report highlights the importance of NT-proBNP in the early diagnosis of PPCM and detecting of thromboembolic complications. Significant increases in NT-proBNP levels, even without obvious symptoms, should be promptly evaluated for PPCM, especially in high-risk populations.

1. Introduction

Peripartum cardiomyopathy (PPCM) is a rare but potentially fatal cardiomyopathy condition that occurs late in pregnancy or within the first five months after delivery. This condition is characterized by left ventricular systolic dysfunction, which is characterized by a decrease in ejection fraction (EF) to below 45%. This reduction in the heart's ability to pump blood effectively can cause various symptoms such as fatigue, shortness of breath, and edema, which are often mistaken for normal pregnancy symptoms. PPCM is a diagnosis of exclusion, which means that its diagnosis requires the exclusion of all other causes of heart failure. This is a challenge in itself because PPCM symptoms are often non-specific and overlap with general symptoms in pregnancy and the postpartum period. Therefore, the diagnosis of PPCM requires a comprehensive approach involving a careful history, thorough physical examination, and appropriate supporting examinations. Left ventricular dysfunction in PPCM can cause a variety of serious complications, including congestive heart failure, arrhythmias, and thromboembolism. Congestive heart failure occurs when the heart is unable to pump enough blood to meet the body's needs, which can cause fluid to build up in the lungs and other parts of the body. Arrhythmias, or heart rhythm disturbances, can occur due to structural and functional changes in the heart caused by PPCM. Thromboembolism, namely the formation of blood clots in the heart that can break

loose and block blood vessels elsewhere, is also a significant risk in PPCM patients.[1,2]

Given the potential for these serious complications, early diagnosis of PPCM is critical to initiate timely therapy and prevent morbidity and mortality. PPCM therapy generally focuses on treating heart failure and preventing complications. Medications such as diuretics, ACE inhibitors, beta-blockers, and anticoagulants are often used to manage symptoms and prevent thromboembolic complications. In some cases, additional therapy such as bromocriptine, which is a dopamine agonist and prolactin inhibitor, may also be considered. Biomarkers such as N-terminal pro-B-type natriuretic peptide (NT-proBNP) have emerged as invaluable tools in the early diagnosis of PPCM. NT-proBNP is a prohormone produced and released by the ventricles of the heart in response to increased wall stress. Elevated NT-proBNP levels in the blood correlate with the degree of left ventricular dysfunction and the severity of heart failure. In PPCM, NT-proBNP levels are often very high, reflecting increased ventricular wall stress due to systolic dysfunction. The use of NT-proBNP as a biomarker in the diagnosis of PPCM has been supported by various studies. These studies show that NT-proBNP has high sensitivity and specificity in detecting PPCM, even in the early stages when symptoms may still be unclear. In addition, NT-proBNP levels can also be used to assess patient prognosis and monitor response to therapy.[3-5] This case report presents an 18-year-old female who presented with symptoms of acute heart failure and was found to have multiple left ventricular thrombus on echocardiography. The very high levels of NT-proBNP in these patients played an important role in leading to the diagnosis of PPCM and guiding subsequent therapy.

2. Case Presentation

An 18-year-old woman, with status G1P1A0H1 (1 pregnancy, 1 birth, 0 abortions, and 1 live child), came to the hospital with the main complaint of progressive shortness of breath which had gotten worse in the last three days. The patient reported that she had experienced this shortness of breath four months after giving birth (postpartum), and was getting worse by physical activity and lying on her back. In fact, this shortness of breath often woke her up at night, so she had to sleep with an elevated pillow to reduce the discomfort. These symptoms indicate the heart's inability to pump blood effectively, especially during physical activity or in certain positions that increase the heart's workload. Apart from shortness of breath, patients also complain of edema or swelling throughout their body. This edema has gotten worse over the past month, although initially it only occurred in both legs four months postpartum. This swelling then spread to her stomach, which got bigger and bigger, and eventually to her entire body. This generalized edema indicates an accumulation of fluid in the body's tissues, which can be caused by impaired heart function or other medical conditions.

The patient had a history of normal spontaneous labor four months previously, assisted by a midwife at 37-38 weeks' gestation. The delivery went smoothly, and the baby was born healthy with strong cries. During her pregnancy, the patient never experienced hypertension or high blood pressure, and there was no previous history of heart disease. This is important to note because hypertension in pregnancy is a risk factor for PPCM. The fact that the patient had no history of hypertension during pregnancy reduces the possibility of other causes of heart failure. The patient had breastfed her baby for the first month but was forced to stop due to complaints of disturbing shortness of breath. The decision to stop breastfeeding is most likely a response to worsening symptoms of heart failure. Breastfeeding increases the body's metabolic demands and may worsen symptoms in patients with compromised heart function. About one month before entering this hospital, the patient had been treated at a regional hospital due to complaints of fatigue and shortness of breath. During this treatment, she received one unit of red blood cell transfusion, but no further examination was carried out regarding the possibility of heart disease. This indicates that symptoms of heart failure have

appeared previously, but the cause has not been identified. Red blood cell transfusions may be given to treat anemia that may accompany heart failure or as part of preparation for a test or other medical procedure.

On physical examination, the patient appeared moderately ill but was still fully conscious (composable). Her blood pressure was measured at 108/70 mmHg, pulse 124x/minute with a regular rhythm and adequate filling, respiratory rate 28x/minute, and body temperature 36.5°C. Her oxygen saturation reached 98% with nasal oxygen assistance of 3 liters/minute. Her weight is 60 kg with a height of 157 cm, resulting in a body mass index (BMI) of 24.3 kg/m^2, which indicates being overweight. These findings provide a general picture of the patient's clinical condition upon admission to the hospital. Normal blood pressure with a rapid pulse (tachycardia) can indicate cardiac compensation for decreased heart function. A rapid breathing rate (tachypnea) indicates impaired lung function, possibly due to heart failure. Normal oxygen saturation with oxygen support indicates hypoxemia (decreased oxygen levels in the blood) which improves with oxygen therapy. Being overweight is a risk factor for various cardiovascular diseases, including PPCM.

Further physical examination revealed edema on both eyelids (palpebra), increased jugular venous pressure (JVP) which is a sign of increased pressure in the right atrium of the heart, enlargement of the heart (cardiomegaly), accumulation of fluid in the pleural cavity in both lungs (bilateral pleural effusion), accumulation of fluid in the abdominal cavity (ascites), and edema in all four limbs (extremities). These findings are consistent with a diagnosis of congestive heart failure, in which the heart is unable to pump blood effectively, causing fluid to build up in various parts of the body. Laboratory examination results showed very high levels of NT-proBNP, namely 1,796 pg/mL. NT-proBNP is a biological marker released by the heart in response to increased pressure or cardiac workload. Elevated NT-proBNP levels often indicate impaired heart function. Apart from that, the results of other laboratory tests showed hemoglobin levels of 12.1 g/dL, leukocytes 5,790/mm^3, platelets 191,000/mm^3, albumin 3.0 g/dL, and globulin 3.9 g/dL. Liver function tests, serum electrolytes, and hemostasis were within normal limits. Pleural fluid analysis shows the presence of transudate, which is fluid that accumulates due to an imbalance in hydrostatic and oncotic pressure, not due to inflammation. These laboratory findings provide further evidence of heart failure and indicate that the pleural effusion is most likely caused by increased hydrostatic pressure due to heart failure. An electrocardiogram (ECG) shows sinus tachycardia, a condition in which the heart beats faster than normal. In addition, the ECG also showed low voltage, which was likely caused by bilateral pleural effusions that disrupted the heart's electrical conduction. A chest x-ray confirmed cardiomegaly and bilateral pleural effusion. These ECG and chest X-ray findings support the diagnosis of heart failure and show an enlarged heart and fluid buildup in the pleural cavity. Echocardiography examination, which is an ultrasound examination of the heart, revealed a decrease in left ventricular systolic function with an ejection fraction (EF) of only 18%. Ejection fraction is a measure of how well the heart pumps blood out of the left ventricle throughout the body. Normal EF values usually range between 50-70%, so a value of 18% indicates a significant disturbance in the heart's ability to pump blood. Apart from that, echocardiography also shows eccentric left ventricular hypertrophy, a condition in which the left ventricular wall thickens and enlarges in response to an increase in the heart's workload.

Other findings on echocardiography include moderate mitral regurgitation and moderate tricuspid regurgitation, indicating leakage in the mitral and tricuspid valves of the heart. This regurgitation can occur due to left ventricular dilatation and papillary muscle dysfunction, which are often found in PPCM. There is also the possibility of pulmonary hypertension, namely increased blood pressure in the pulmonary arteries, which can occur as a result of left

heart failure. Minimal pericardial effusion, which is the accumulation of small amounts of fluid in the sac of the heart (pericardium), is also found in echocardiography. Most worryingly, echocardiography also revealed multiple thrombus or blood clots in the left ventricle. Intracardiac thrombus formation is a serious complication of PPCM and increases the risk of thromboembolism. This thrombus can break loose and block blood vessels in the brain, lungs, or other organs, causing stroke, pulmonary embolism, or organ infarction. Based on the overall clinical, laboratory, and echocardiographic findings, a diagnosis of peripartum cardiomyopathy (PPCM) with multiple left ventricular thrombus was made. PPCM is a type of cardiomyopathy that occurs in late pregnancy or early postpartum, while multiple left ventricular thrombus is a serious complication that can occur in PPCM. Once the diagnosis is made, the patient immediately receives standard heart failure therapy, which includes administering oxygen, diuretics (furosemide) to reduce fluid buildup, ACE inhibitors (ramipril) to reduce blood pressure and heart workload, and beta blockers (bisoprolol) to control heart rate. In addition, patients are also given anticoagulants (heparin) to prevent the growth of existing blood clots and prevent the formation of new blood clots. This therapy aims to improve heart function, reduce symptoms of heart failure, and prevent thromboembolism complications.

3. Discussion

The presented case clearly illustrates the crucial role of N-terminal pro-B-type natriuretic peptide (NT-proBNP) in diagnosing peripartum cardiomyopathy (PPCM). In this patient, very high NT-proBNP levels (1,796 pg/mL) were a strong indicator of PPCM, although the symptoms presented by the patient were nonspecific. The finding of multiple left ventricular thrombus (blood clots in the left ventricle) on echocardiography further strengthens this diagnosis and indicates the need for immediate anticoagulant therapy to prevent thromboembolic complications. NT-proBNP is a valuable biomarker in the early diagnosis of PPCM. These biomarkers are peptide fragments produced and released by the heart ventricles in response to increased wall stress. In heart failure conditions, including PPCM, there is an increase in ventricular filling pressure and systolic dysfunction, which triggers the release of greater amounts of NT-proBNP. Therefore, increased blood levels of NT-proBNP correlate with the degree of left ventricular dysfunction and the severity of heart failure.[6,7]

Peripartum cardiomyopathy (PPCM) is a condition characterized by left ventricular (LV) systolic dysfunction. This dysfunction means the left ventricle, the heart's main pumping chamber, cannot contract with enough force to eject blood efficiently throughout the body. This condition causes a decrease in cardiac output, namely, the amount of blood pumped by the heart per minute, and an increase in ventricular filling pressure, namely the pressure in the left ventricle at the end of the diastole (heart filling phase). The pathophysiology of PPCM is not completely understood, but several mechanisms have been proposed to explain the occurrence of left ventricular systolic dysfunction in this condition. One of the most prominent mechanisms is oxidative stress. Oxidative stress occurs when there is an imbalance between the production of free radicals, which are unstable and reactive molecules, and the body's ability to neutralize them. Free radicals can damage heart cells and cause myocardial dysfunction. In PPCM, oxidative stress is thought to be triggered by several factors, including increased activity of the proteolytic enzyme cathepsin D. This enzyme can break down prolactin, a hormone that plays a role in lactation, into 16-kDa prolactin fragments which are cardiotoxic. This 16-kDa prolactin fragment can disrupt the function of cardiomyocytes (heart muscle cells), cause apoptosis (programmed cell death), and inhibit angiogenesis (formation of new blood vessels). In addition to oxidative stress, inflammation also plays a role in the pathogenesis of PPCM. Increased levels of proinflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), have been observed in PPCM patients. These proinflammatory cytokines can cause myocardial

dysfunction through various mechanisms, including activation of apoptotic pathways, impaired energy metabolism, and myocardial fibrosis. Another factor that may contribute to left ventricular systolic dysfunction in PPCM is microvascular dysfunction. Disorders of the small blood vessels in the heart can cause myocardial ischemia (lack of blood flow to the heart muscle) and left ventricular dysfunction. The decrease in cardiac output and increase in ventricular filling pressure that occurs in PPCM triggers the release of brain natriuretic peptide (BNP) and NT-proBNP by ventricular cardiomyocytes as a compensatory response. BNP and NT-proBNP are peptide hormones that have vasodilatory (widening of blood vessels), natriuretic (increasing sodium excretion through the kidneys), and diuretic (increasing urine production) effects. The vasodilatory effects of BNP and NT-proBNP help reduce the workload of the heart by reducing peripheral vascular resistance (resistance to blood flow). The natriuretic and diuretic effects help reduce blood volume and ventricular filling pressure, thereby reducing the workload of the heart and improving heart function. NT-proBNP is a very useful biomarker in the diagnosis of PPCM. Increased levels of NT-proBNP in the blood reflect increased ventricular filling pressure and cardiac dysfunction. In PPCM, NT-proBNP levels are often very high, even in the early stages of the disease when symptoms may still be unclear. Studies have shown that high NT-proBNP levels in patients with symptoms of heart failure in the peripartum period have a high positive predictive value for PPCM. This means that the majority of patients with elevated NT-proBNP levels in this period did have PPCM. NT-proBNP can be used as a screening tool to identify women at risk of PPCM. Elevated NT-proBNP levels in late pregnancy or early postpartum, especially in women with risk factors for PPCM, should immediately be further evaluated with echocardiography to confirm the diagnosis. In addition, NT-proBNP can also be used to assess the prognosis of PPCM patients. Higher NT-proBNP levels at diagnosis correlate with an increased risk of complications and mortality. Serial monitoring of NT-proBNP levels may also help assess response to therapy and identify patients who require additional therapy or further intervention. Left ventricular systolic dysfunction is a characteristic feature of PPCM causing decreased cardiac output and increased ventricular filling pressure. This condition triggers the release of NT-proBNP as a compensatory response. NT-proBNP is a valuable biomarker in the early diagnosis of PPCM and assessment of prognosis. Increased NT-proBNP levels in patients with symptoms of heart failure in the peripartum period should be immediately evaluated further. Early diagnosis and appropriate therapy can improve clinical outcomes and prevent life-threatening complications in patients with PPCM.[8-10]

Oxidative stress and inflammation are two interrelated pathological processes and are thought to play an important role in the pathogenesis of peripartum cardiomyopathy (PPCM). Both of these processes can trigger cardiomyocyte damage, cause left ventricular dysfunction, and ultimately increase NT-proBNP levels in the blood. Oxidative stress occurs when there is an imbalance between the production of free radicals, which are reactive molecules containing oxygen, and the body's ability to neutralize them through the antioxidant defense system. Free radicals can damage various cellular components, including DNA, proteins, and lipids, which can disrupt cell function and cause cell death. Pregnancy and the postpartum period are periods with significantly increased metabolic demands. This increase in metabolism can increase the production of free radicals as a byproduct of the oxidation process. Mitochondria are cellular organelles that play a role in energy production. Mitochondrial dysfunction can lead to increased production of free radicals and impaired cellular function. In PPCM, there is evidence of mitochondrial dysfunction in cardiomyocytes, which may contribute to increased oxidative stress. Deficiencies in antioxidants, such as vitamin E and selenium, can reduce the body's ability to neutralize free radicals, thereby increasing oxidative stress. Several studies show that selenium deficiency can

increase the risk of PPCM. Activation of neurohormonal systems, such as the renin-angiotensin-aldosterone system (RAAS), can increase free radical production through various mechanisms, including increasing the activity of the NADPH oxidase enzyme. Oxidative stress can cause direct damage to cardiomyocytes through several mechanisms. Free radicals can attack lipids in cell membranes, causing lipid peroxidation and cell membrane damage. Damage to cell membranes can disrupt cellular function and cause cell death. Free radicals can modify proteins through oxidation of amino acid residues, causing changes in protein structure and function. Protein modifications can interfere with various cellular processes, including heart muscle contraction. Free radicals can cause DNA damage, which can trigger mutations and genome instability. DNA damage can disrupt DNA replication and transcription, which can lead to cell death or malignant transformation of cells. Cardiomyocyte damage due to oxidative stress can lead to left ventricular dysfunction, which is a characteristic feature of PPCM. This left ventricular dysfunction causes a decrease in cardiac output and an increase in ventricular filling pressure, which triggers the release of NT-proBNP.[11-13]

Inflammation is the body's natural response to injury or infection, aimed at eliminating harmful stimuli and initiating the healing process. However, excessive or chronic inflammation can damage body tissues and organs. In PPCM, inflammation is thought to play an important role in the pathogenesis of this disease. Viral infections, such as parvovirus B19 and coxsackie virus B, have been associated with an increased risk of developing PPCM. Viral infections can trigger an excessive immune response, causing inflammation and cardiomyocyte damage. In some cases of PPCM, it is thought that there is an autoimmune reaction, where the immune system attacks the heart tissue itself. This autoimmune reaction can cause inflammation and damage to cardiomyocytes. Increased cardiac workload during pregnancy and the postpartum period can cause hemodynamic stress, which can trigger inflammation. Hemodynamic stress can cause activation of endothelial cells, which are the inner lining of blood vessels, and the release of proinflammatory cytokines. Oxidative stress can trigger inflammation through activation of nuclear transcription factor kappa B (NF-κB), which is a key regulator of inflammatory responses. Activation of NF-κB causes increased production of proinflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). Inflammation can cause cardiomyocyte damage through several mechanisms. Proinflammatory cytokines, such as TNF-α and IL-6, can cause direct damage to cardiomyocytes through activation of the apoptotic pathway, that is, programmed cell death. Inflammation can trigger the infiltration of immune cells, such as macrophages and T lymphocytes, into heart tissue. These immune cells can release various inflammatory mediators, such as free radicals, proteases, and proinflammatory cytokines, which can damage cardiomyocytes. Chronic inflammation can cause myocardial fibrosis, which is the formation of scar tissue in the heart muscle. Myocardial fibrosis can disrupt heart function and cause heart failure. Inflammation-induced cardiomyocyte damage can lead to left ventricular dysfunction, which is a characteristic feature of PPCM. This left ventricular dysfunction causes a decrease in cardiac output and an increase in ventricular filling pressure, which triggers the release of NT-proBNP. Oxidative stress and inflammation are two pathological processes that are interrelated and can reinforce each other. Oxidative stress can trigger inflammation through activation of NF-κB, while inflammation can increase oxidative stress through the release of free radicals by immune cells. This interaction between oxidative stress and inflammation may cause more severe cardiomyocyte damage and worsen left ventricular dysfunction in PPCM. Oxidative stress and inflammation are two pathological processes that are interrelated and are thought to play an important role in the pathogenesis of PPCM. Both of these processes can trigger cardiomyocyte damage, cause left ventricular

dysfunction, and ultimately increase NT-proBNP levels in the blood. Elevated NT-proBNP levels may be a useful biomarker in the early diagnosis of PPCM and assessment of patient prognosis. A better understanding of the role of oxidative stress and inflammation in the pathogenesis of PPCM may open new opportunities for the development of more effective therapies in treating this disease.[14-16]

NT-proBNP has emerged as an invaluable biomarker in the diagnosis of PPCM due to its ability to accurately differentiate between patients with PPCM and those without. This is proven by various studies showing that NT-proBNP has high sensitivity and specificity in detecting PPCM, even in the early stages when symptoms may not be completely clear. Sensitivity refers to the ability of a test to identify individuals who actually have the disease. In the context of PPCM, the high sensitivity of NT-proBNP means that this biomarker can detect almost all cases of PPCM. In other words, very few cases of PPCM are missed by the NT-proBNP test. Specificity, on the other hand, refers to the ability of a test to identify individuals who truly do not have the disease. The high specificity of NT-proBNP means that this biomarker rarely gives false positive results in patients without PPCM. This means that if the NT-proBNP test shows a positive result, it is very likely that the patient indeed suffers from PPCM. A meta-analysis involving more than 1,000 PPCM patients found that NT-proBNP had a sensitivity of 94% and a specificity of 86% in diagnosing PPCM. These numbers show that NT-proBNP is a very reliable tool in detecting PPCM and differentiating it from other conditions that can cause similar symptoms. NT-proBNP threshold values for the diagnosis of PPCM may vary depending on the population and measurement method used. However, in general, values above 300 pg/mL are considered abnormal and may indicate heart failure, including PPCM. In the case presented, the patient's NT-proBNP level was very high (1,796 pg/mL), far exceeding the normal threshold value. This provides strong evidence of the presence of PPCM in these patients. It is important to note that these threshold values are only a guide, and interpretation of NT-proBNP levels should always be done in the patient's overall clinical context. Other factors such as age, kidney function, obesity, and other heart diseases can also influence NT-proBNP levels. Apart from being used in diagnosis, NT-proBNP can also be used to assess the prognosis of PPCM patients. Higher NT-proBNP levels at diagnosis correlate with an increased risk of complications such as thromboembolism, arrhythmia, and death. This suggests that NT-proBNP is not only useful for diagnosing PPCM, but also for predicting the disease course and clinical outcome of patients. Serial monitoring of NT-proBNP levels may also help assess response to therapy and identify patients who require additional therapy or further intervention. A decrease in NT-proBNP levels after therapy indicates improved cardiac function and a better prognosis. Conversely, if NT-proBNP levels remain high or increase despite therapy, this may indicate the need for adjustment of therapy or further evaluation to look for other causes of heart failure. NT-proBNP is a valuable biomarker in the early diagnosis of PPCM and assessment of prognosis. The high sensitivity and specificity of NT-proBNP allows early detection of PPCM, even at an early stage when symptoms may not yet be obvious. Serial monitoring of NT-proBNP levels can also help guide therapy and assess response to treatment. Therefore, the use of NT-proBNP in clinical practice may improve PPCM management and ultimately improve patient clinical outcomes.[17,18]

Although NT-proBNP has been shown to be a very useful biomarker in the diagnosis of peripartum cardiomyopathy (PPCM), its interpretation should not be done in isolation. There are a number of factors that can influence NT-proBNP blood levels, so accurate interpretation must consider the patient's overall clinical context. Age is an important factor that needs to be considered in interpreting NT-proBNP levels. As we age, physiological changes occur in the heart that can affect the production and release of NT-proBNP. Several studies have shown that NT-proBNP levels tend to increase with age, even in individuals without heart disease. This may be due to the decline in kidney

function that occurs with age, which can reduce the clearance of NT-proBNP from the body. In addition, structural and functional changes in the heart that occur with age, such as increased ventricular stiffness and decreased diastolic function, may also contribute to increased NT-proBNP levels. Therefore, the NT-proBNP threshold value for the diagnosis of PPCM may need to be adjusted in older patients. A higher threshold value may be necessary to avoid incorrect diagnosis of PPCM in older patients with elevated NT-proBNP levels caused by age rather than PPCM. Therefore, it is important to consider the patient's age when interpreting NT-proBNP levels and use threshold values appropriate to the patient's age. Kidney function is also an important factor that can influence NT-proBNP levels. NT-proBNP is mainly excreted via the kidneys, so impaired renal function can lead to decreased clearance of NT-proBNP and increased levels in the blood. In patients with chronic renal failure, NT-proBNP levels can increase significantly, even in the absence of cardiac disease. Therefore, the interpretation of NT-proBNP levels in patients with impaired renal function should be done with caution. It is important to evaluate the patient's renal function before interpreting NT-proBNP levels. Laboratory tests such as serum creatinine and glomerular filtration rate (GFR) can be used to assess kidney function. If the patient has impaired renal function, the NT-proBNP threshold value for the diagnosis of PPCM may need to be adjusted. In addition, it is also necessary to consider other possible causes of increased NT-proBNP levels in patients with impaired renal function, such as hypertension, left ventricular hypertrophy, and myocardial fibrosis. Obesity is a significant risk factor for various cardiovascular diseases, including PPCM. Several studies have shown that obese patients tend to have higher NT-proBNP levels compared with normal-weight individuals, even in the absence of heart disease. This may be caused by increased cardiac workload in obese patients, which may trigger the release of NT-proBNP. Additionally, obesity is also associated with an increased risk of insulin resistance, dyslipidemia, and hypertension, all of which may contribute to cardiac dysfunction and increased NT-proBNP levels. Therefore, obesity needs to be considered in the interpretation of NT-proBNP levels. If the patient is obese, the NT-proBNP threshold value for the diagnosis of PPCM may need to be adjusted. In addition, it is also important to evaluate other cardiovascular risk factors in obese patients, such as hypertension, diabetes mellitus, and dyslipidemia, which may influence the interpretation of NT-proBNP levels. NT-proBNP is not a specific biomarker for PPCM. Elevated NT-proBNP levels can also be found in various other heart diseases, such as chronic heart failure, acute myocardial infarction, hypertensive heart disease, and heart valve disease. Therefore, it is important to exclude other possible causes of heart failure before establishing a diagnosis of PPCM based on elevated NT-proBNP levels. This can be done by carrying out a careful history, thorough physical examination, and appropriate supporting examinations. Echocardiography is essential in differentiating PPCM from other causes of heart failure. In PPCM, echocardiography usually shows left ventricular systolic dysfunction without significant cardiac structural abnormalities. On the other hand, in other heart diseases, echocardiography can show structural heart abnormalities such as left ventricular hypertrophy, left ventricular dilatation, or heart valve abnormalities. NT-proBNP is a very useful biomarker in the diagnosis of PPCM, but its interpretation should be done with caution. NT-proBNP levels can be influenced by various factors other than PPCM, such as age, kidney function, obesity, and other heart diseases. Therefore, interpretation of NT-proBNP levels should always be done in the clinical context of the patient as a whole.[19,20]

4. Conclusion

This case report emphasizes the importance of NT-proBNP in the early diagnosis of PPCM and detecting thromboembolic complications. Significant increases in NT-proBNP levels, even without obvious symptoms, should be promptly evaluated for PPCM, especially in high-risk populations. Early diagnosis and therapy of

PPCM can improve clinical outcomes and prevent life-threatening complications.

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