

A Delayed Diagnosis of Peripartum Cardiomyopathy - A Case Report

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OBJECTIVE

We report a case of Peripartum Cardiomyopathy, initially diagnosed as preeclampsia and sepsis. This led to delayed management of true diagnosis.

PRESENTATION

A 30 year old woman (G3P0) presented to triage feeling generally unwell at 37+4. She was contracting and 3cm on examination. Observations were normal, proteinuria present and ALT, creatinine and urea were elevated. A provisional diagnosis of early pre-eclampsia was made.

This was a consultant led pregnancy due to significant past medical history of Stage IV alveolar rhabdomyosarcoma (treated with chemotherapy in childhood) and spina bifida occulta. She was a smoker and received serial growth scans during pregnancy which were all normal.

Due to past medical history she had planned for an elective section. As a result of labouring prior to her booked date, an emergency caesarean was performed with EBL 1100mls. Immediately post delivery she became unwell with oxygen saturations of 94%, pulse of 116 and unrecordable blood pressure. On examination, she appeared pale, uterus was well contracted, and no obvious signs of bleeding were noted. The working diagnosis at this stage was sepsis and dehydration. Sepsis 6 was initiated, she received 4L normal saline IV and she was transfused with 2 units of RBCs.

POSTNATAL CARE

Following initial management, maternal observations stabilised, however urine output was reduced. Blood tests remained deranged and maternal health deteriorated. A multidisciplinary team meeting was held and the decision to transfer to intensive care unit was made.

CT abdomen and pelvis showed extensive intra-abdominal ascites and bilateral pleural effusions likely secondary to underlying cardiogenic failure or fluid overload. An echocardiogram performed in ITU showed evidence of heart failure:

Severe left ventricular systolic dysfunction without valvular abnormalities

Ejection fraction of 25 per cent

A diagnosis of peripartum cardiomyopathy was made. She was commenced on Bisoprolol and Ramipril and is now under follow-up by Cardiology and the heart failure team. She is awaiting cardiac MRI as an outpatient.

PERIPARTUM CARDIOMYOPATHY

Cardiovascular disease is one of the leading causes of maternal death (1) and of these, one third were associated with cardiomyopathy. The estimated incidence of peripartum cardiomyopathy in the Western world is 1000-4000 pregnancies, with a mortality rate of 10% (2). Risk factors include Afro- Caribbean Ethnicity, Multiparity, Multiple Pregnancy, Obesity, Chronic Hypertension, Pre-eclampsia and Maternal Age (2).

The European Society of Cardiology defines peripartum cardiomyopathy as idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or up to 6 months following delivery, where no other cause of heart failure is found. It is a diagnosis of exclusion. The left ventricle may not be dilated but the ejection fraction is nearly always reduced below 45% (3).

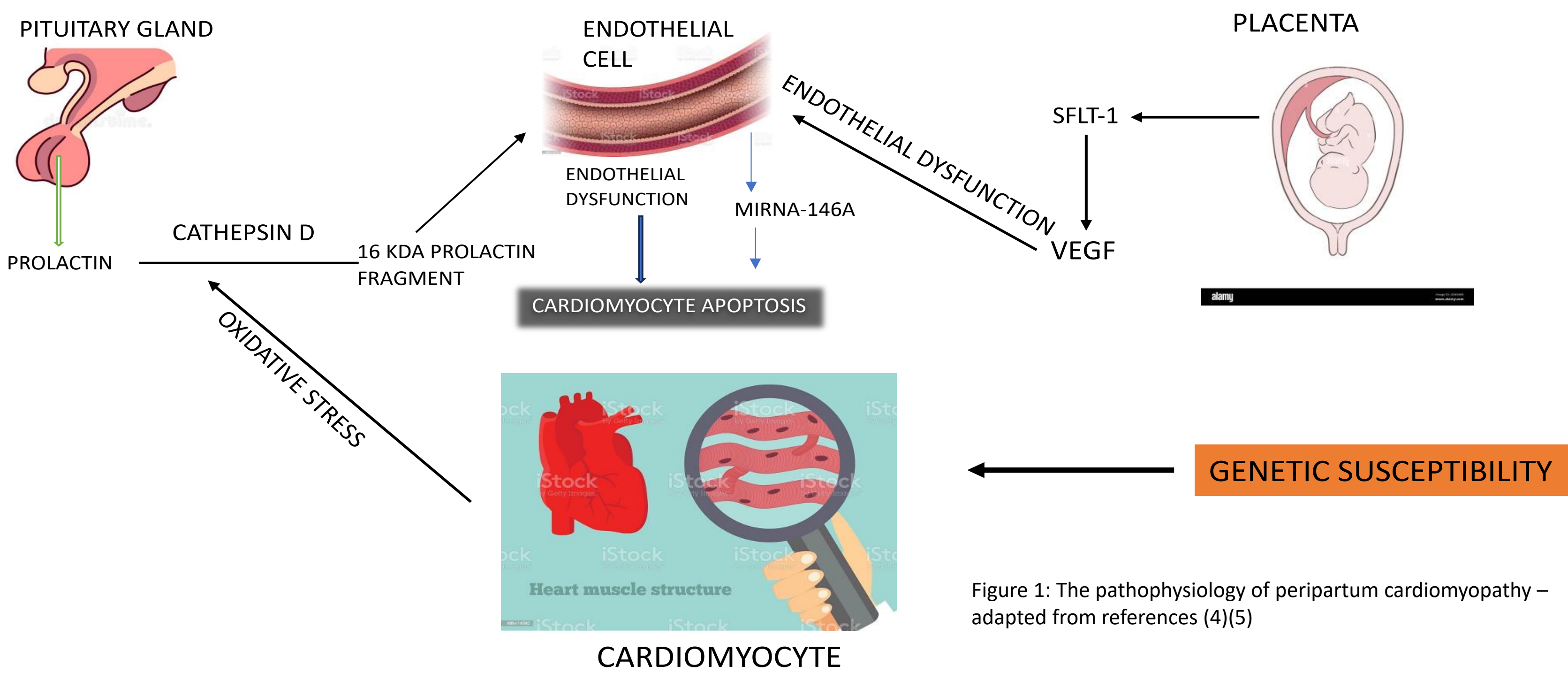


Figure 1: The pathophysiology of peripartum cardiomyopathy – adapted from references (4)(5)

PATHOGENESIS OF CARDIOMYOPATHY

The true pathogenesis of peripartum cardiomyopathy is unclear (4). It is thought to be related to both genetic factors as well as vascular injury from sFlt-1, VEGF and PlGF (see figure 1 (4) (5). Symptoms include shortness of breath, chest pain, cough, palpitations, dizziness and leg swelling (3). Signs may include sinus tachycardia and raised JVP (ibid). Differentials to think about include other cardiac causes, amniotic fluid embolism, asthma, hypertension and pulmonary embolus (3) (5) . Pre-eclampsia can be a differential or a co-existing pathology, also related to sFlt-1, VEGF and PlGF levels.

DIAGNOSING PERIPARTUM CARDIOMYOPATHY

The majority of presentations are in the 4 months following pregnancy. As it is a diagnosis of exclusion (3), it is important to also consider the other differentials mentioned previously. ECG, CXR and BNP testing are useful, but echocardiography is the most helpful imaging modality, with cardiac MRI being used later in diagnosis to identify further features of tissue injury (6). Management depends on if pregnant or postpartum (7). Pregnancy management relies on volume control with loop diuretics, beta-blockers for arrhythmia, hydralazine for afterload and managing risk of VTE with LMWH (8) (9). Post partum, ACE inhibitors and ARBs can be used with good effect. If hemodynamically unstable, management should follow an A-E approach and consideration of intensive care involvement (7).

If diagnosed antenatally, delivery timing depends on maternal wellbeing versus gestational age. For anyone deteriorating, delivery should be made regardless if premature or not. Vaginal delivery should be the aim on a delivery suite, with continuous fetal monitoring and cardiac monitoring. Consideration of instrumental to shorten the second stage can also be made (2).

Risk of relapse in subsequent pregnancy is more likely if ongoing left ventricular dysfunction, with a 50% chance of worsening LVF. If previously full recovery, the risk of relapse is less, at 20% (10). Pre-conception counselling and LARC to avoid unplanned pregnancy is recommended (4).

CONCLUSION

Due to how rare the incidence of peripartum cardiomyopathy is, it isn't surprising the diagnosis was delayed. In contrast to the regular symptoms of PPCM, this patient gave an unusual presentation with reduced urine output, raised LFTs and urea. She was initially managed as sepsis, dehydration and potential pre-eclampsia. An atypical presentation of pre-eclampsia could have led to PPCM.

It was only when referred to ITU by the MDT that an echocardiogram was requested, and diagnosis of cardiomyopathy made. We appreciate the importance of accurate diagnosis due to the high risk of maternal and foetal morbidity and mortality and encourage our obstetric colleagues to take note of the increasing burden of cardiovascular disease in pregnancy.

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