Dual dangers: Navigating pre- eclampsia complicated by peripartum cardiomyopathy

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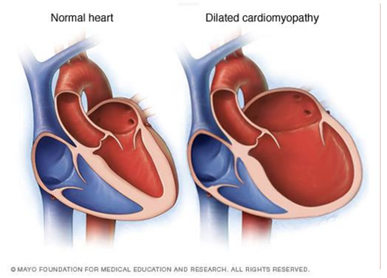
Background

In Australia, Pre – eclampsia is a major contributor to maternal and perinatal morbidity and mortality, affecting approximately ~2-8% of pregnancies. This hypertensive disorder occurs after 20th week of gestation and is characterized by elevated high blood pressure and involvement of multi – organ system dysfunction. Pre – eclampsia is progressive in nature and can lead to serious complications, one of which is peripartum cardiomyopathy. Peripartum cardiomyopathy (PPCM) is a serious but rare type of heart failure that occurs in the latter stages of pregnancy or within 5 months of the postpartum period. The precise mechanism of PPCM is poorly understood, however it is proposed that three potential common factors between pre- eclampsia and PPCM is thought to instigate their development. The combination of endothelial dysfunction, genetic predisposition and; inflammatory and immune responses in pregnancy. Peripartum cardiomyopathy (PPCM) is diagnosed based on National Heart, Lung, and Blood Institute guidelines, which include heart failure onset in late pregnancy or within five months postpartum, absence of preexisting heart disease, and left ventricular dysfunction with an ejection fraction below 45%. Despite increasing research, PPCM has a high mortality rate (30%–60%), and survivors face a significant risk (50%–80%) of future cardiac failure. Symptoms like fatigue, oedema, and dyspnoea often overlap with normal pregnancy and other conditions, making diagnosis challenging. Additionally, comorbidities such as pulmonary embolism, toxic cardiomyopathy, and Takotsubo syndrome further complicate detection. As a result, PPCM is often unrecognised or diagnosed late, leading to poor outcomes for both mother and baby.



Case study

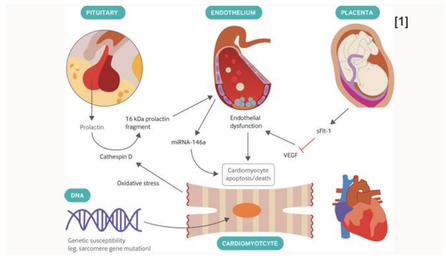
Our case involves a 28-year-old woman presenting with worsening respiratory symptoms, desaturation, and refractory hypertension in setting of diet controlled gestational diabetes and pre-eclampsia. She has nil significant medical history and a BMI of 39. She underwent and emergency caesarean section (EmCS) at 38+2 when she developed cardiogenic shock requiring intensive care support. Imaging revealed a severely reduced left ventricular ejection fraction (LVEF 35%), consistent with PPCM. Postoperatively, she remained in cardiogenic shock, requiring inotropic support and mechanical ventilation, prompting transfer to a tertiary centre. Her course was further complicated by acute kidney injury, hyperkalaemia, and RSV pneumonia .

Treatment

Initially, required ionotropic support and intubation post CS delivery in ICU (post tertiary transfer). Her electrolytes were also corrected and she eas managed with IV antibiotics for her pneumonia. Once stabilised and downgraded to ward, she was managed with sacubitril/valsartan 24/26mg, frusemide, spironolactone and bisoprolol. She was discharged with cardiology follow up

Discussion

Recent studies indicate that pregnancy hormones may trigger vascular damage, leading to cardiomyopathy in women with a genetic predisposition, such as a family history of dilated cardiomyopathy. This occurs due to increased secretion of microRNA-146a by the endothelium, which is stimulated by sFlt-1, a placental protein released in high amounts toward the end of pregnancy. Additionally, elevated serum prolactin levels during pregnancy and postpartum contribute to myocyte damage, further increasing the risk of cardiomyopathy.



Conclusion

In conclusion, pre-eclampsia remains a significant contributor to maternal and perinatal complications, with its association with PPCM highlighting the need for early recognition and intervention. The overlapping pathophysiological mechanisms, including endothelial dysfunction, genetic predisposition, and inflammatory responses, suggest a complex interplay contributing to disease progression. Our case demonstrates the severe consequences of PPCM, emphasizing the importance of prompt diagnosis and multidisciplinary management. Advances in understanding the hormonal and vascular factors involved in PPCM development may lead to improved preventative and therapeutic strategies in the future. Early identification and comprehensive postnatal follow-up remain crucial to optimising maternal and neonatal outcomes.

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