Non-cardiogenic pulmonary oedema in the setting of severe early-onset pre-eclampsia

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INTRODUCTION

Often an under-appreciated complication, pulmonary oedema secondary to pre-eclampsia can significantly impact outcomes and alter management priorities[1]. Our case report illustrates this, describing a patient with severe dyspnoea, orthopnoea and massive acute pregnancy weight gain of 47kg in the setting of severe early onset pre-eclampsia and non-cardiogenic pulmonary oedema.

CASE

A 40-year old G1P0 was admitted to hospital at 24+4 weeks gestation with rapidly increasing weight gain associated with mid thoracic pitting oedema, worsening shortness of breath and severely limited exercise tolerance. Prior to this the pregnancy had been uncomplicated, however she did have risk factors for hypertensive disorders of pregnancy including her age, parity and a pre-pregnancy BMI of 42 (104kg). The patient was also of indigenous background with a significant family history of diabetes and unspecified cardiac conditions. At time of presentation to hospital she was found to be severely hypertensive with nephrotic range proteinuria (urine protein/creatinine ratio of 868) and a serum uric acid level of 0.60 mmol/L. Obstetric ultrasound revealed asymmetric fetal biometry with abnormal umbilical artery doppler and redistribution of blood flow. The diagnosis of early onset pre-eclampsia was made and comprehensive investigations including transthoracic echocardiogram and respiratory function tests excluded other pathology. During her admission, the patient required increasing doses of three antihypertensive agents to control her blood pressure and despite close monitoring of fluid balance, continued to retain fluid to a weight of 151kg (+47kgs). At 26+0 weeks gestation, deteriorating renal function and worsening respiratory symptoms including significant shortness of breath at rest, warranted a decision for delivery on maternal grounds. The baby was delivered via emergency classical caesarean section in reasonable condition.

DISCUSSION

This case demonstrates the difficulty of timing delivery in the setting of severe pre-eclampsia at early preterm gestations. Rapidly worsening respiratory symptoms in the setting of progressive oedema prompted a difficult decision of delivery for maternal indications, despite the early gestation and suboptimal fetal growth.

The PEPE study[1] explored risk factors for development of pulmonary oedema in pre-eclampsia. It demonstrated increased rates of pulmonary oedema with increasing severity of pre-eclampsia. Markers such as pronounced thrombocytopenia, higher serum uric acid concentration and receipt of magnesium sulphate infusion were found to be associated with a higher risk of pulmonary oedema. Additionally, nulliparity nearly triples the risk for development of pre-eclampsia[2].

The patient in this case report was nulliparous and had a high serum uric acid level. Magnesium sulphate was administered pre-delivery, but only for fetal neuroprotection and not for treatment of pre-eclampsia. The platelet count remained normal.

Non-cardiogenic pulmonary oedema is a known complication of pre-eclampsia, but is rarely one of the deciding factors for timing of delivery. This case highlights some of the complexities of managing pre-eclampsia.

REFERENCES

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