

Prevalence of pre-eclampsia and adverse pregnancy outcomes in women with pre-existing cardiomyopathy: a multi-centre retrospective cohort study.

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Abstract

Objective: To determine the prevalence of pre-eclampsia and fetal growth restriction (FGR; <3rd centile) in women with pre-existing cardiac dysfunction. Design: Retrospective cohort study. Setting: Maternity units in UK and Australia. Population: Pregnant women with impaired left ventricular ejection fraction <55%. Methods: Routine clinical data, including medical history and pregnancy outcome were collected retrospectively. Main Outcome Measures: Pre-specified outcomes included pre-eclampsia and FGR prevalence in women with pre-existing cardiac impairment, compared with the general population; and the relationship between pregnancy outcome and pre-pregnancy cardiac phenotype. Results: In this cohort of 282 pregnancies,

pre-eclampsia prevalence was not significantly increased (4.6% [95% C.I. 2.2-7.0%] versus population prevalence of 4.6% [95% C.I. 2.7-8.2], $p=0.99$); 12/13 of these women had additional obstetric/medical risk factors. However, prevalences of preterm pre-eclampsia (<37 weeks) and FGR were increased (1.8% versus 0.7%, $p=0.03$; 15.2% versus 5.5%, $p<0.001$, respectively). Neither systolic nor diastolic function correlated with pregnancy outcome; however, left ventricular mass index (LVMI) weakly correlated with pre-eclampsia (5g/m² increase: OR 1.18 [95% C.I. 1.01-1.38], $p=0.04$). Antenatal β blockers ($n=116$) were associated with lower birthweight Z score (adjusted difference -0.33 [95% C.I. -0.63- -0.02], $p=0.04$). Conclusions: This study demonstrated a modest increase in preterm pre-eclampsia and significant increase in FGR in women with cardiac dysfunction. These results do not support a causal relationship between cardiac dysfunction and pre-eclampsia, especially accounting for the background risk status of the population. The mechanism underpinning the relationship between cardiac dysfunction and FGR merits further research but could be influenced by concomitant β blocker use.

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