Title: Pathophysiology of pulmonary and myocardial edema in preeclampsia

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Joubert et al have made a commendable effort to elucidate the pathophysiology of pulmonary and myocardial oedema in various preeclampsia phenotypes. Nevertheless, we do have a few alternative explanations. The authors ruled out overt left ventricular systolic dysfunction (LVSD) as a cause of pulmonary oedema in preeclampsia. Therefore, the absence of LVSD despite myocardial oedema is indeed noteworthy. Nevertheless, a normal LV systolic function was demonstrated in several previous studies that reported myocardial oedema. Acute onset myocardial oedema without myocardial injury may not reflect systolic dysfunction. Ejection fraction is ubiquitously reported as an indicator of systolic function and it has its limitations. Myocardial strain analysis is now frequently used to detect subclinical systolic dysfunction. Ejection fraction tends to be normalize in the initial stages of evolving myocardial pathophysiology due to alterations in LV end-diastolic volume, wall thickness, circumferential strain, longitudinal strain, preload, afterload and chronotropic activity. Moreover, the increased afterload (elevated systemic vascular resistance) increases LV contractility and normalizes ejection fraction through Anrep effect. Therefore, subclinical LVSD in these patients cannot be ruled out without a detailed myocardial strain analysis.

The left atrial volume index (LAVI) was 38.9 ml/m² in women with preeclampsia and pulmonary oedema. In these patients, the LAVI exceeded the cut-off (>34 ml/m²) suggested to diagnose diastolic dysfunction. The presence of at least one echocardiographic parameter beyond cut-off (tricuspid regurgitant velocity, E/e’ (early mitral inflow filling velocity/mitral annular early diastolic velocity), septal or lateral e’ velocity) would have established the presence of diastolic dysfunction. Pulmonary oedema has been commonly noted in patients with heart failure and preserved ejection fraction (HFpEF). In these subsets of patients with HFpEF, elevated LV end-diastolic pressure, and subclinical LVSD could be implicated in the causation of pulmonary oedema.
In this study, systemic blood pressure appears to be positively correlated with pulmonary and myocardial oedema development. The preeclampsia with pulmonary oedema phenotype had higher systolic (189.4 mm Hg vs 163.9 mm Hg) and diastolic blood pressure (110.6 mm Hg vs 103.6 mm Hg) than those without oedema. Furthermore, preeclampsia with severe features phenotype had significantly higher diastolic blood pressure (122 mm Hg vs 110.6 mm Hg vs 92.1 mm Hg) than those with pulmonary oedema and without systemic features. Therefore, the presence of pulmonary oedema and a greater extent of myocardial oedema in those patients with higher mean blood pressure are not surprising. An elevated capillary hydrostatic pressure could partly explain the mechanism of myocardial and pulmonary oedema due to higher mean blood pressure. Additionally, we expect a lower plasma oncotic pressure due to protein loss in preeclampsia. Moreover, disruption in the endothelial barrier could lead to a higher efflux fluid in preeclampsia.

Therefore, based on the findings of this study, we can reasonably speculate on the pathophysiology of pulmonary and myocardial oedema in preeclampsia. Moreover, the addition of a few echocardiographic indices (tricuspid regurgitant velocity, E/e’, septal or lateral tissue doppler velocity, myocardial strain), and biochemical parameters (serum albumin, protein) could have reexplored the pathophysiology of pulmonary and myocardial oedema further.
References


