Why don’t all women with preeclampsia with severe features develop pulmonary edema?

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Title: Why don’t all women with preeclampsia with severe features develop pulmonary edema?

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We thank Jha and Jha for their comments on our work. The determinants of myocardial and pulmonary oedema are likely multifactorial. The goal of this study, however, was to address the conflicting results in the echocardiography literature suggesting a degree of reduced left ventricular ejection fraction (LVEF) detected utilising various methods of LVEF quantification. We selected cardiac MRI to assess LVEF for its superior volumetric assessment and high degree of interobserver correlation. Currently, MRI based strain imaging is investigational without a clinical indication for use. For this reason, the finding of a normal LVEF, on cardiac MRI, in all groups, confirms that the cause of pulmonary oedema is not overtly reduced LVEF.

We expected to find a stark difference between women with pre-eclampsia complicated by pulmonary oedema and those without pulmonary oedema. Instead, the MRI findings in all women with pre-eclampsia with severe features were similar and they differed significantly from women with preeclampsia without severe features and normotensive controls. Jha and Jha note that the left atrial volumes indexed to body surface area (LAVI) were above the echocardiographically derived cut off of 34ml/m$^2$ which could suggest diastolic dysfunction in the women with pulmonary oedema.\(^1\) The left atrial volumes reported were, however, cardiac MRI derived volumes and the LAVI in all groups fell below 39ml/m$^2$, the normal mean LAVI for women on MRI.\(^2\)

The suggestion that pulmonary oedema occurs based on severity of hypertension is not supported by our findings. Women with pulmonary oedema had the highest mean systolic pressures and women with the severe features of HELLP syndrome and eclampsia had the highest diastolic pressures, but there was no statistically significant difference between these
two groups. The difference in blood pressures was driven by the relatively lower blood pressures in the group with preeclampsia without severe features and normotensive controls.

As a condition characterised primarily by endothelial dysfunction, a degree of increased capillary permeability is expected. While women with pre-eclampsia with severe features are thought to have more severe endothelial dysfunction, it is not clear that endothelial dysfunction and capillary permeability are more abnormal in one manifestation of preeclampsia with severe features than another. We feel that this does not satisfactorily differentiate the groups nor conclusively describe the mechanism underlying pulmonary oedema. Indeed, the question may not be why some women with pre-eclampsia develop pulmonary oedema, but rather why all women with pre-eclampsia with severe features do not.
