ABSTRACT ID

PNC191

Chronic Postpartum Changes of Mesenteric Arteries in Nω-Nitro-L-Arginine Methyl Ester Hydrochloride (L-NAME)- induced Hypertension in Pregnant Rats Model

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Introduction

- Women with a history of hypertensive disorders of pregnancy (HDP) have two- to four-fold increased risk to develop cardiovascular diseases later in life [1].
- A balanced production of vasoconstrictor endothelin-1 (ET-1) and vasodilator nitric oxide (NO) is important for blood pressure regulation [2,3].
- There are very few information on the structural changes on the vessels following HDP during chronic postpartum period.

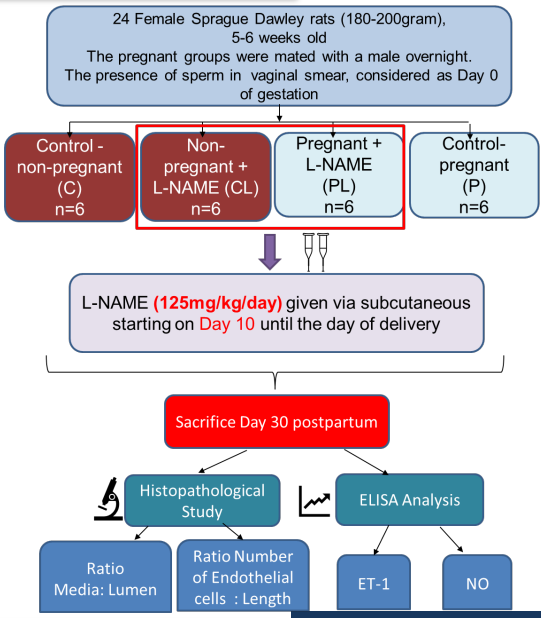
Hypothesis

- Transient turbulence blood flow during gestational hypertension causes permanent and ongoing vascular changes that eventually leads to cardiovascular diseases.

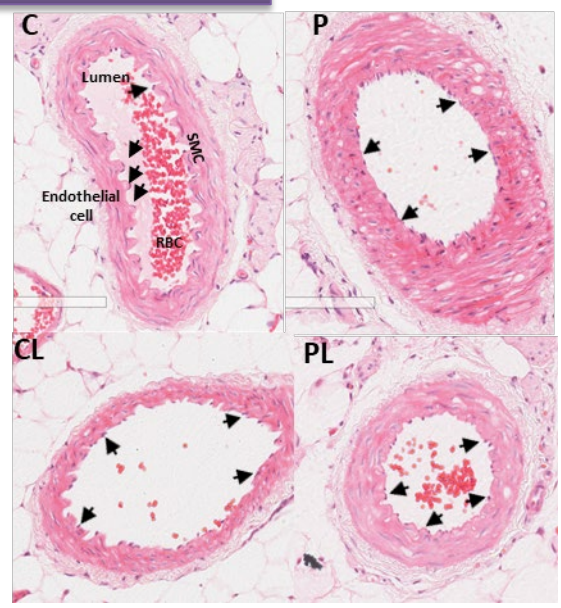
Objective

- To investigate the histopathological effects on mesenteric arteries of L-NAME-induced hypertension in pregnant rat model during chronic postpartum period.
- To quantify the concentration of ET-1 and NO as the key biomarkers in BP regulation in the L-NAME-induced hypertension in pregnant rat model during chronic postpartum period.

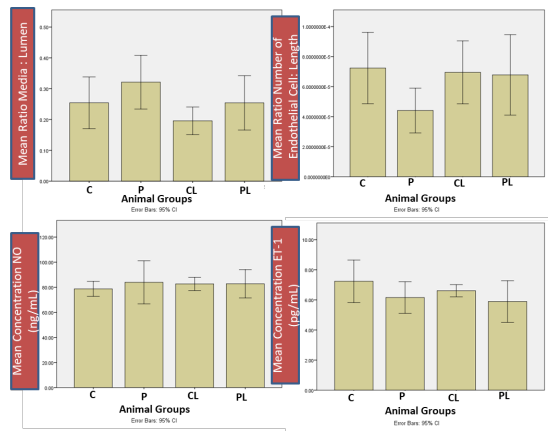
Methodology



Result



The figures are the representative photomicrograph of the mesenteric arteries. Arrow indicates endothelial cells seen as simple squamous cells with flattened nucleus lining lumen of artery. No obvious abnormalities were observed in endothelium and tunica media in all groups. (H&E 20x). SMC; Smooth muscle cell, RBC; Red blood cell.



The bar charts show means of all the parameters studied in this experiment. The statistical tests were done to compare means between groups. The analysis show no significant differences between groups ($p > 0.05$) for all parameters.

Discussion & Conclusion

- In this study, the rats were sacrificed at Day 30 postpartum which is equivalent to 2.5 years of human's year [4]. However, there is no evidence that demonstrates abnormal changes to the endothelium and tunica media of the resistance artery in long-term duration following HDP.
- Thus, further investigation of its potential chronic effect warrants a deeper analysis at the endothelial receptors and alteration at the ultrastructure level.

Reference

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Acknowledgement

IIUM Research Acculturation Grant Scheme (IRAGS)