

Short communication

High sensitivity cardiac troponin I levels in preeclampsia[☆]Adam Morton^{a,*}, Anthony Morton^b^a Mater Health and University of QLD, Raymond Tce, South Brisbane, QLD 4101, Australia^b Medical Statistician, 40 Garioch St, Tarragindi, QLD 4121, Australia

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ABSTRACT

Pre-eclampsia may be associated with cardiac complications including pulmonary oedema. Nine studies examining whether pre-eclampsia is associated with elevated levels of cardiac troponin (cTnI) revealed inconsistent results. In this study high sensitivity cardiac troponin I (hscTnI) levels were measured in 40 asymptomatic women with pre-eclampsia. HscTnI was elevated in ten (25%) women. A linear correlation between peak mean arterial pressure and log hscTnI was demonstrated.

1. Introduction

Acute cardiovascular complications occur in approximately 6% of women with pre-eclampsia [1]. Acute pulmonary oedema has been reported in 2.9–5.6% of pre-eclamptic women [2]. Other cardiac complications of pre-eclampsia include Takotsubo and peripartum cardiomyopathy, pleural and pericardial effusions, and dissection of aortic, coronary and vertebral arteries. Pre-eclampsia is associated with an increased risk of cardiovascular complications in later life.

Myocardial infarction during pregnancy is rare, the incidence estimated to be 0.6–1.0 cases per 10 000 pregnancies [3]. cTnI and cTnT levels are unchanged compared with non-pregnant values during normotensive pregnancy (NTP). A modest rise in cTnI may be seen 24 h postpartum however peak values remain below cut-off values for myocardial ischaemia irrespective of the mode of delivery [4]. Previous studies have revealed inconsistent results regarding whether cTnI is elevated in pre-eclampsia (Table 1). Five studies demonstrated elevated cTnI levels in pre-eclampsia compared with NTP. The incidence of cTnI higher than the non-pregnant reference interval in women with pre-eclampsia in these studies ranged between 2.6 and 70%. Four other studies found no difference in cTnI between pre-eclampsia and NTP, leading authors to conclude other pathologies causing myocardial damage should be investigated in women with pre-eclampsia and elevated cTnI.

High sensitivity assays detect troponin in more than 50% of a normal reference population and identify people above or below the 99th centile with optimal precision. A pilot study of women at high and low-risk for myocardial strain in pregnancy found the incidence of elevated high sensitivity cTnT to be 4.3% [5]. To date no studies have

reported hscTnI levels in NTP or pre-eclampsia.

2. Methods

HscTnI was measured pre and post-delivery in 40 women with pre-eclampsia who had no clinical evidence of cardiac complications other than hypertension. Definitions of pre-eclampsia were as per the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy [6]. Women with abnormal renal function, cardiac disease, chronic hypertension, obstructive sleep apnoea, known illicit drug use or suspected pulmonary embolism were excluded. HscTnI measurements were made on an Architect Stat chemiluminescent microparticle immunoassay system where the 99th percentile for healthy non-pregnant women is 15.6 pg/mL [7]. Repeated samples were collected where hscTnI was elevated to exclude heterophile antibody interference or chronic disorders causing abnormal values.

3. Statistical analysis.

The data were analysed using R v 3.4.1. (R Core Team (2017). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria). Variables tested in multilinear regression included age, parity, gestation, BMI, proteinuria and blood pressure. A linear regression model employing log (troponin) was fitted to these fairly widely dispersed data: $\log(\text{troponin}) = -3.964 + 0.047 * \text{MAP}$. The standardised residuals were normally distributed. When the log was omitted the standardised residuals indicated a poor fit. A nonlinear (additive) model did not improve the analysis.

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Table 1
Previous studies comparing cTnI levels in preeclampsia and normotensive pregnancies.

Beigi et al. [19]	cTnI higher in severe PET vs NTP cTnI elevated in 0% NTP, 2% women with mild PET and 14.6% women with severe PET
Yang et al. [20]	cTnI higher in women with PET vs NTP cTnI elevated in 70% women with PET
Fleming et al. [21]	Mean cTnI higher in GH and PET vs NTP Median cTnI higher with PET vs GH
Atalay et al. [10]	Proteinuric hypertension associated with highest levels Mean cTnI higher in PET vs healthy pregnancy Significant correlation between systolic BP, diastolic BP and cTnI No correlation between cTnI and proteinuria cTnI lower post Mg SO ₄ than pre
Pasupathi et al. [22]	Mean cTnI higher in PET vs NTP and non-pregnant controls Proteinuric hypertension associated with highest levels cTnI
Bozkurt et al. [23]	cTnI levels higher in eclamptic women vs PET and NTP No difference in cTnI between PET and healthy pregnancy
Aydin et al. [24]	No difference in cTnI between severe PET and NTP
Sprawka et al. [25]	No significant difference in cTnI between mild PET, severe PET and NTP
Joyal et al. [26]	No significant difference in cTnI between PET and NTP

4. Results

The mean age of the study population was 29 years (range 17–41), mean gestation at delivery was 35 weeks (23–41 weeks), and 24 women were primigravida. Four women had pre-eclampsia in a previous pregnancy. Mean body mass index (BMI) was 29.5 kg/m² (range 17–67), with 8 of the women (20%) had a BMI greater than 35 kg/m². Five of the women had ceased smoking once they were aware of the pregnancy, the remainder were non-smokers. Predominant ethnicities were White (68%), Pacific Islander (10%) and African (10%). All of the women had proteinuria. The median urine protein: creatinine ratio was 228 mg/mmol creatinine (normal < 30 – range 35–2174). The average peak mean arterial pressure (MAP) prior to delivery was 124 mm Hg (range 105–155). Pre-eclampsia with severe features was present in 16 women including 2 women with haemolysis, elevated liver enzymes and low platelets and 1 woman with eclampsia. Twenty-four women received infusion of magnesium sulphate for eclampsia prophylaxis. Vaginal delivery occurred in 15 women. There was 1 stillbirth. There was no significant difference in antepartum or postpartum hscTnI levels. Mean and median hscTnI were 20 ng/L and 5 ng/L respectively. HscTnI was elevated in 10 women (25%), values ranging from 19 to 145 ng/L. In those women with elevated values, repeated testing

revealed a decline to normal values within 72 h postpartum. HscTnI was elevated in 1 of the 4 women with recurrent pre-eclampsia. Seven women (44%) with pre-eclampsia with severe features had elevated hscTnI. Four of the 24 primigravida women had elevated hscTnI compared with 6 of the 16 multigravida women. This difference was not significant ($p = .26$). Gestation was divided into 3 groups – 28 weeks gestation or earlier (4 women), 29–36 weeks gestation (18 women), and after 36 weeks gestation (18 women). Difference in proportions for gestation was not significant (0/4,5/18,5/18) ($p = .7$) nor was a trend test for proportions significant ($p = .4$). Age, BMI and proteinuria were also not predictive of elevation of hscTnI.

The average MAP of women with elevated hscTnI was 135 mmHg compared with 120.5 mm Hg in women with normal hscTnI ($p = .006$). Statistical analysis revealed a significant linear relationship between log hscTnI predicted by MAP ($p = .013$) (Fig. 1). Predicted hscTnI was $\exp(-3.964 + 0.047 * \text{MAP})$. The distribution of hscTnI values is illustrated in Fig. 2.

5. Discussion

The utility of cardiac markers may be affected in pregnancy. While CK-MB is unchanged in pre-eclampsia and NTP prior to delivery, CK-MB levels rise significantly on the first postpartum day, with values above the non-pregnant reference interval in 36% of healthy postpartum women in one study [8]. CK-MB is therefore not useful in diagnosing myocardial ischaemia peripartum. Brain natriuretic peptide (BNP) levels are higher during pregnancy than in the non-pregnant state, though stable throughout the trimesters, and generally below 20 pg/mL. BNP levels rise 2–3 fold in the first 48 h postpartum, with 6.1% of asymptomatic early postpartum women having levels greater than 100 pg/mL after uncomplicated pregnancy [9]. BNP levels return to baseline 6–12 weeks after birth. Chronic hypertension, gestational hypertension and pre-eclampsia are all associated with significantly elevated levels of BNP. In pre-eclampsia this elevation may persist for 3–6 months postpartum.

This study found that hscTnI was elevated in 25% of women with pre-eclampsia, and that there was a relationship between hscTnI levels and peak MAP. Similarly Atalay et al found a significant correlation between cTnI levels, diastolic and systolic BP, but no correlation with proteinuria [10]. Endomyocardial biopsy of women with pre-eclampsia has shown microvascular abnormalities as well as changes in the mitochondrial structure of the myometrial muscle cells [11]. Echocardiography studies reveal evidence of diastolic dysfunction in approximately 20% of women with pre-eclampsia. The prevalence of diastolic dysfunction, left ventricular remodeling and maternal BNP levels are significantly higher in early-onset pre-eclampsia (prior

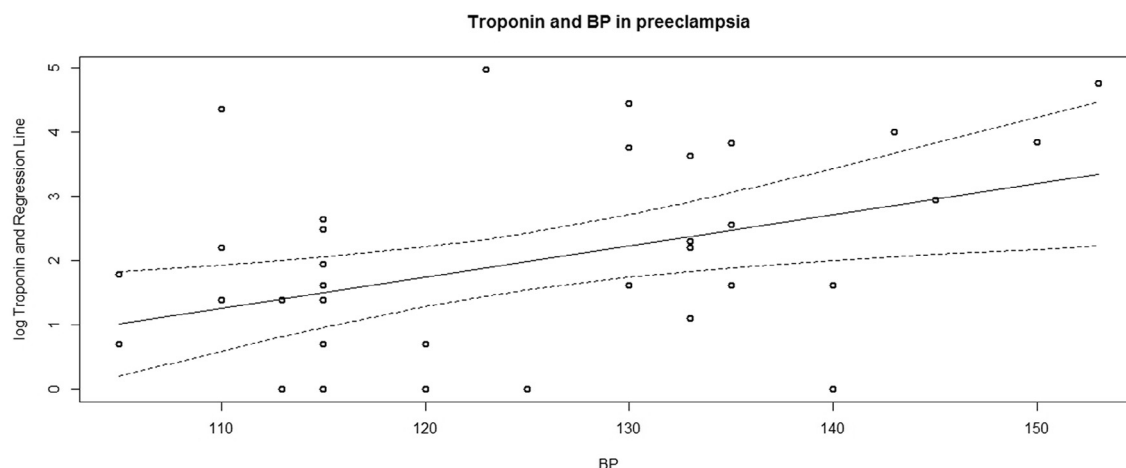


Fig. 1. Log hscTnI and peak MAP in Preeclampsia.

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