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Prediction of adverse maternal outcomes in preeclampsia at term

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ABSTRACT

Preeclampsia complicates 5–8% of all pregnancies and is associated with high rates of maternal and perinatal morbidity. The majority of cases occur at term gestations where the baby can be safely delivered. The preeclampsia disease process however can progress in the mother resulting in significant morbidity. In this study we were interested in examining the number of patients developing preeclampsia with severe features at term. We also investigated whether factors at admission might be predictive of disease progression.

We performed a retrospective cohort study at a tertiary obstetric hospital in Melbourne, Australia from 2015 to 2017. There were 124 participants presenting with preeclampsia at term and included in our study. After admission, 44.4% progressed to preeclampsia with severe features. Disease features at admission associated with disease progression were chronic hypertension, elevated systolic blood pressure, reduced haemoglobin and elevated creatinine. Using predictive modelling, we determined that a combination of these features showed good discrimination (area under ROC = 0.88 (95% confidence interval 0.82–0.94)) with good performance (negative predictive value 80% and positive predictive value 87%) for predicting progression to preeclampsia with severe features.

Almost half of the women presenting with preeclampsia at term will progress to preeclampsia with severe features. Admission characteristics can be used to predict those at risk of disease progression.

1. Introduction

Preeclampsia complicates 5–8% of all pregnancies and accounts for 100 maternal and 400 perinatal deaths globally each day [1,2]. It can rapidly progress to preeclampsia with severe features, resulting in multi-system organ damage and failure [1,3]. In the long-term, preeclampsia with severe features is an independent risk factor for cardiovascular disease, including hypertension, coronary artery disease, renal disease, type 2 diabetes mellitus and cerebrovascular accidents [4].

The majority of preeclampsia cases occur at term (≥37 weeks' gestation). Whilst delivery is safe at term gestations for the baby, preeclampsia can progress to disease with severe features, which can lead to significant morbidity for the mother in both the short and long term [5]. Determining which women might progress to preeclampsia with severe features at term might allow us to prioritise their delivery and allocate increased monitoring to this group. Furthermore, if therapeutics for preeclampsia become available in the future this cohort of women could be treated and the risk of disease progression reduced.

We were interested in assessing the proportion of women progressing to preeclampsia with severe features at term. Additionally, we investigated the point at which disease progression to preeclampsia with severe features occurred: antenatally, intrapartum or postpartum. Furthermore, we determined whether admission characteristics could be used to predict which patients were more likely to progress to preeclampsia with severe features.

2. Methods

We conducted a retrospective cohort study at the Mercy Hospital for Women, a tertiary obstetric hospital in Melbourne, Australia from July 2015 to April 2017. We included women admitted with preeclampsia at

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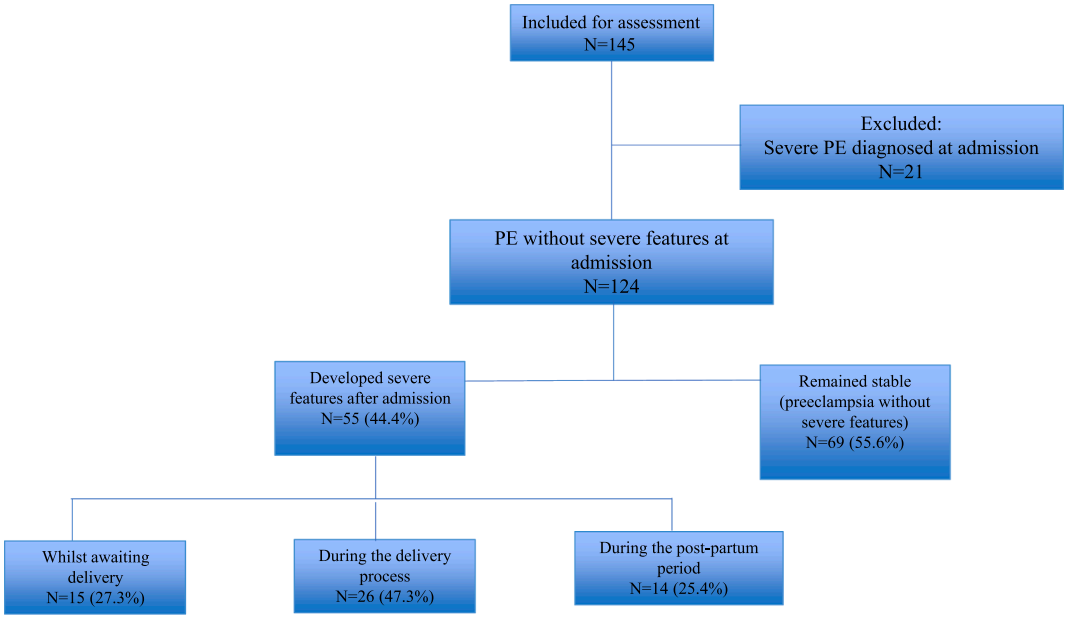

Fig. 1. Flow diagram of patient recruitment and timing of progression of preeclampsia to preeclampsia with severe features.

term ($\geq 37^{+0}$ weeks' gestation), diagnosed using the American College of Obstetrics and Gynaecology (ACOG) guideline. Women were excluded if they had severe preeclampsia at admission, preeclampsia diagnosed before 37^{+0} weeks' gestation or if there was uncertainty regarding the diagnosis or insufficient patient information. The included participants with preeclampsia at term were divided into two groups: women with stable disease and women that progressed to severe preeclampsia. We compared the baseline characteristics and admission characteristics of those that progressed to severe disease after admission and those that remained stable (Fig. 1).

2.1. Clinical practice for managing patients with preeclampsia at term

Pregnant women are reviewed weekly from 36 weeks' gestation, with blood pressure and urine dipstick performed at clinic visits. If the patient is hypertensive or has proteinuria determined by 1 + or more on urine dipstick they are admitted for preeclampsia monitoring. During monitoring, history review and examination is performed as well as blood pressure monitoring for 4 hours, formal proteinuria assessment, full blood count and biochemistry. If the patient is ≥37 weeks' gestation and diagnosed with preeclampsia then birth is arranged as soon as possible[5].

2.2. Data collection

Patients with preeclampsia were identified using Birthing Outcomes Systems (BOS), an electronic database utilised by hospital staff to record patient characteristics, labour and delivery outcomes as part of routine care. The terms "severe preeclampsia", "term preeclampsia" and "eclampsia" were searched. We obtained the files of all participants identified and accessed pathology results using the electronic pathology database, Clinical Patient Folder. Baseline characteristics including maternal age, parity, body mass index (BMI), chronic medical history, obstetric history and family history of preeclampsia were recorded.

2.3. Outcomes

Our primary outcome was the proportion of participants progressing to severe preeclampsia at term using ACOG criteria [6] which are systolic blood pressure of 160 mm Hg or diastolic blood pressure of 110 mm Hg or higher on two occasions, thrombocytopenia (platelet count less than 100,000/microliter), impaired liver function (abnormally elevated liver enzymes to twice the normal concentration), progressive renal insufficiency (elevated serum creatinine concentration), pulmonary oedema or new onset cerebral/visual disturbance. We evaluated maternal factors and preeclampsia disease characteristics upon admission to determine their predictive value for disease progression. These include maternal characteristics (maternal age, smoking status, body mass index, medical history, obstetric history, family history of preeclampsia), clinical characteristics (signs and symptoms of preeclampsia, systolic and diastolic blood pressures, gravidity, parity, gestational age at diagnosis) and laboratory findings (proteinuria, platelets, haemoglobin, alanine transaminase (ALT), aspartate transaminase (AST), creatinine, uric acid). These predictors were selected based on previous studies [7–11].

Secondary outcomes were preeclampsia disease characteristics at delivery including clinical and biochemical characteristics and gestational age at delivery, mode of delivery, antihypertensive medicine use and magnesium sulphate use; postpartum outcomes including maternal complications of labile blood pressure, intensive care admission, postpartum haemorrhage and neonatal outcomes including birth weight and neonatal intensive care admission. This information was obtained from patient files and electronic pathology databases.

2.4. Statistical analysis

Data was expressed as a median and interquartile range (IQR) or a percentage of total. We used a Student's t-test for continuous variables that approximated a normal distribution and Mann-Whitney U tests for skewed data and a Fisher's exact test for categorical variables. Statistical significance was defined as a p value ≤ 0.05. Statistical analysis was performed using GraphPad Prism 7 (GraphPad Software, La Jolla, CA).

We also performed predictive modelling. We transformed all continuous variables to values greater than data minimums prior to modelling. Univariable associations between outcome and covariates were assessed using the likelihood ratio chi-squared statistic from a logistic regression model. We used backward stepwise logistic modelling, with the probability of variable removal set at 0.05, commencing with all variables with univariable p-values < 0.2. Five thousand bootstrap replications were performed with the final predictors chosen if they occurred in greater than 50% of replications [12]. Regression coefficients

Table 1
Baseline patient characteristics at admission.

	No severe features at term (N = 69)	Severe disease progression at term (N = 55)	*p*-value
Maternal age (years)	31 [26.5–35.0]	32 [28–34]	0.30
Multipara	25 (36.0%)	17 (30.9%)	0.53
Singletons	66 (95.6%)	53 (96.4%)	0.84
Gestational age at diagnosis (weeks)	38.4 [37.4–40.0]	38.4 [37.2–39.6]	0.60
Chronic medical disease			
• Essential hypertension	1 (1.4%)	11 (20.0%)	< 0.001
• Diabetes	0	2 (3.6%)	0.07
• Autoimmune disease	8 (11.6%)	4 (7.3%)	0.41
Body mass index median, kg/m^2	27 [23–30]	26 [24–30]	0.67
Current smoker	2 (2.9%)	4 (7.3%)	0.26
Obstetric History:			
• Past history of preeclampsia	8 (11.6%)	13 (23.6%)	0.08
• Gestational diabetes	13 (18.8%)	11 (20.0%)	0.87
Family history of preeclampsia	2 (2.9%)	6 (10.9%)	0.07

Data presented as median [25th − 75th percentile] and number (%).

of the final model underwent parameter wise shrinkage to improve future prediction and was assessed for calibration, using logistic regression of outcome against linear predictor; discrimination, using area under ROC curve and sensitivity at 90 % specificity; and negative (NPV) and positive (PPV) predictive values assessed at a prevalence of 0.44. Graphical presentation of the final model included predictive probability against outcome status and observed proportion against quantiles of predicted probability. Statistical analysis was performed using both Stata (StataCorp. 2017. Stata Statistical Software: Release 15. College Station, TX: StataCorp LLC) and R (R Core Team (2013). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, AustriaURL http://www.R-project.org/). Software programs used were mfpboot (Stata) and shrink package [13].

2.5. Ethics

Ethics approval was obtained prior to commencing this study through The Mercy Health Human Research Ethics Committee (approval project number 2017–045). As this was a retrospective cohort study, individual patient consent was not required.

3. Results

A total of 145 women were diagnosed with term preeclampsia between 1 July 2015 and 30 April 2017 at the Mercy Hospital for Women, Heidelberg, Australia. Of these, 21 women were diagnosed with preeclampsia with severe features at admission and were excluded from analysis. Our final cohort thus consisted of 124 participants at term with preeclampsia but without features of severe disease at initial presentation (Fig. 1).

3.1. Incidence of severe preeclampsia

Of 124 women, 44.4% (n = 55) progressed to preeclampsia with severe features. This occurred whilst awaiting birth in 27.2% of women, during delivery in 47.3% or immediately postpartum in 25.5% of women (Fig. 1). Most women were diagnosed with preeclampsia with severe features based on severe hypertension alone (65.5%, n = 28). Another 26 (47.2%) women had severe hypertension plus additional feature of severe disease (doubling of LFT's (n = 9), thrombocytopenia (n = 4), raised creatinine (n = 10) and new onset cerebral disturbance (n = 3). One patient had doubling of LFTs alone.

3.2. Clinical factors associated with progression to preeclampsia with severe features

Baseline characteristics including maternal age, parity, body mass index (BMI), smoking status, obstetric history, past history of preeclampsia or family history of preeclampsia were similar between the groups. Interestingly, women progressing to severe preeclampsia were more likely to have a medical history of essential hypertension compared to women that remained stable (20.0% compared with 1.4%, $p < 0.001$) (Table 1).

3.3. Preeclampsia at diagnosis

At the time preeclampsia was diagnosed, preeclampsia-specific symptoms (chest pain, dyspnoea, visual changes, headache, vaginal bleeding and decreased fetal movements) and signs (oxygen saturation, right-upper quadrant tenderness, hyperreflexia, peripheral oedema) were similar between groups.

Women that developed preeclampsia with severe features had a higher systolic blood pressure (150 [142–155] compared to 145 [140–150] mmHg $p = 0.001$) and diastolic blood pressure (95 [90–100], 90 [85–95] mmHg ($p = 0.002$) at admission. They had a higher serum creatinine (67 [56–92] compared to 53 [41–44]] μmol/L ($p < 0.001$) and lower haemoglobin levels (116 [107–130] compared to 124 [118–131] g/L $p = 0.01$) compared to participants that remained stable. Whilst proteinuria was elevated in those that progressed to severe disease (urine protein/creatinine ratio 0.05, interquartile range (IQR) of [0.03–0.11]) this was not statistically significant. There was no difference in platelet count, uric acid and abnormal liver function tests between groups (Table 2).

Antihypertensive medication use (90.9% compared to 50.7% $p < 0.001$) and difficulty controlling blood pressure despite medication use (27.3% compared to 1.4% $p < 0.001$) were both significantly higher in patients that progressed to severe disease after admission (Table 2).

3.4. Preeclampsia at delivery

The average length from diagnosis to delivery for women with preeclampsia at term was 1.65 days (standard deviation of 1.9 days), which was similar between those progressing to severe disease and those remaining stable ($p = 0.10$). Gestational age at delivery and rates of spontaneous labour were also similar between groups. Furthermore, vaginal birth and caesarean section were also similar.

Those with severe preeclampsia had significantly higher systolic (165, IQR 160–180 mm Hg) and diastolic (100, IQR 95–110 mm Hg) blood pressures during delivery compared to systolic (145, IQR 140–150 mmHg) and diastolic (90, IQR 8–195 mmHg) blood pressures in women remaining stable. There were also significantly more participants receiving intravenous antihypertensive medication (0 compared to 23.6% ($p < 0.0001$) and magnesium sulphate (4.3% compared to 45.5% $p < 0.0001$) in the severe preeclampsia group. Interestingly, more women with severe features had non-reassuring fetal CTG results (43.6% compared to 13.0%) (Table 3).

3.5. Postnatal and neonatal outcomes

There was no difference in postnatal infection or postpartum haemorrhage, however admission to high dependency unit (38.2% compared to 4.3% $p < 0.0001$) and antihypertensive medication use (52.7% compared to 18.8% $p = 0.0001$) was significantly higher in women with severe disease. In the severe preeclampsia group, 1 (1.8%) patient developed HELLP syndrome and 2 (3.6%) developed eclampsia. For the neonate, there were no differences in birth weight or intensive care unit admission (Table 4).

Table 2
Disease characteristics of preeclampsia at study entry.

	No severe features at term (N = 69)	Severe disease progression at term (N = 55)	*p*-value
Symptoms			
Chest pain	0	1 (1.8%)	0.21
Dyspnoea	0	1 (1.8%)	0.21
Visual changes	5 (7.2%)	4 (7.3%)	0.99
Headache	15 (21.7%)	13 (23.6%)	0.80
Vaginal bleeding	1 (1.4%)	3 (5.5%)	0.21
Decreased fetal movements	2 (2.9%)	1 (1.8%)	0.69
Signs			
Right upper quadrant tender	2 (2.9%)	2 (3.6%)	0.82
Hyperreflexia	1 (1.4%)	1 (1.8%)	0.87
Peripheral oedema	11 (15.9%)	10 (18.2%)	0.74
Systolic blood pressure (mmHg)	145 [140–150]	150 [142–155]	0.001
Diastolic blood pressure(mmHg)	90 [85–95]	95 [90–100]	0.002
Urine protein creatinine ratio (PCR)	0.04 [0.03–0.07]	0.05 [0.03–0.11]	0.15
Multisystem disease involvement			
• Haemoglobin (g/L)	124 [118–131]	116 [107–130]	0.01
• Platelet count ($x10^9$/L)	202 [173–238]	196[(157–245]	0.25
• Uric acid (mmol/L)	0.34 [0.29–0.38]	0.36 [0.32–0.42]	0.18
• Elevated transaminases (U/L)	9 (13.0%)	13 (23.6%)	0.13
• Serum creatinine (μmol/L)	53 [41–44]	67 [56–92]	< 0.001
Antihypertensive medicationUse (Labetalol, Nifedipine, IV) antenatally			
	35 (50.7%)	50 (90.9%)	< 0.001
Increased need for antihypertensive medications antenatally	1 (1.4%)	15 (27.3%)	< 0.001
Fetal CTG - non-reassuring	0	1 (1.8%)	0.21

Data presented as median [25th − 75th percentile] and number (%).

Table 3
Delivery characteristics.

	No severe features at term (N = 69)	Severe disease progression at term (N = 55)	*p*-value
Gestation at delivery (weeks), median (IQR)	38.6 [38–40.2]	38.5 [37.6–40.0]	0.80
Spontaneous labour	12 (17.4%)	11 (20.0%)	0.82
Mode of Delivery			
• Vaginal	42 (60.9%)	25 (45.5%)	0.10
• Caesarean section	27 (39.1%)	30 (54.5%)	0.10
Systolic BP during delivery (mmHg)	145 [140–150]	165 [160–180]	< 0.0001
Diastolic BP during delivery (mmHg)	90 [85–95]	100 [95–110]	< 0.0001
Antihypertensive medication infusion, n (%)	0	13 (23.6%)	< 0.0001
Magnesium sulphate infusion, n (%)	3 (4.3%)	25 (45.5%)	< 0.0001
Fetal CTG - non-reassuring	9 (13.0%)	24 (43.6%)	0.0002

Data presented as median [25th–75th percentile] and number (%).

3.6. Progression of preeclampsia to preeclampsia with severe features using admission characteristics

We investigated whether characteristics at diagnosis were predictive of progression to severe disease. Eleven variables with univariate p-values < 0.2 were identified: chronic hypertension, diabetes mellitus, past history of preeclampsia, family history of preeclampsia, systolic blood pressure, diastolic blood pressure, urine protein creatinine ratio, haemoglobin, plasma urate, plasma creatinine, abnormal liver function and antihypertensive medication use (Tables 1 and 2). Using a backward stepwise model combined with parameterwise shrinkage we found four variables (chronic hypertension, systolic blood pressure, haemoglobin and serum creatinine) associated with development of severe preeclampsia (Table 5). This model displayed good

Table 4
Maternal and neonatal outcomes.

	No severe features at term (N = 69)	Severe disease progression at term (N = 55)	*p*-value
Maternal Outcomes			
High Dependency Unit (HDU) admission	3 (4.3%)	21 (38.2%)	< 0.0001
Infection	0	3 (5.5%)	0.08
Eclampsia	0	2 (3.6%)	0.19
Haemolysis, Elevated Liver enzymes and Low Platelets (HELLP)	0	1 (1.8%)	0.44
Post-Partum Haemorrhage (> 500 mis)	12 (17.4%)	12 (21.8%)	0.65
Discharged home on antihypertensives	13 (18.8%)	29 (52.7%)	0.0001
Neonatal Outcomes			
Birth weight (grams)	3322 [2918–3519]	3240 [2936–3610]	0.28
Neonatal Intensive Care Unit (NICU)	2 (2.9%)	3 (5.5%)	0.65
Special Care Nursery (SCN)	8 (11.6%)	6 (10.9%)	0.99
Fetal death in-utero (FDIU)	0	1 (1.8%)	0.44

Data presented as median [25th − 75th percentile] and number (%).

Table 5
Multivariate analysis of predictors used to determine progression to pre-eclampsia with severe features.

	Odds Ratio*	95% CI	*p*-value
Essential hypertension	1.38	1.11–1.71	0.02
Systolic blood pressure (> 125 mmHg)	1.10	1.01–1.17	0.002
Hemoglobin (> 70 g/L)	0.95	0.89–0.97	0.002
Serum creatinine (> 30 μmol/L)	1.09	1.04–1.14	< 0.001

Essential hypertension = history of essential hypertension; Systolic blood pressure = systolic blood pressure > 125 mmHg, Haemoglobin = haemoglobin > 70 (g/L); Serum creatinine = serum creatinine > 30 (μmol/L).

* Based upon the linear predictor = −2.5788 + 0.3196 × essential hypertension + 0.0949 × (systolic blood pressure −125) + − 0.0480 × (haemoglobin −70) + 0.0843 × (serum creatinine −30).

discrimination with a ROC curve area 0.88 (95%, CI 0.82 to 0.94) and 70% (95%, CI 57 to 82) sensitivity at 90% specificity (Fig. 2a) and a negative predictive value (NPV) of 79.7% and positive predictive value (PPV) of 86.7%. The predicted probability of developing severe pre-eclampsia displayed linearity between observed and predicted proportions based upon these disease characteristics (Fig. 2b).

4. Discussion

4.1. Main findings

Our study demonstrated that almost half of participants at 44.4% developed preeclampsia with severe features following a diagnosis of preeclampsia at term. This occurred whilst awaiting induction in 15 (27.2%) women, during delivery in 26 (47.3%) women or immediately postpartum in 14 (25.5%) women. Furthermore, we discovered that admission characteristics were able to differentiate women at risk of disease progression from those remaining stable. Utilising essential hypertension, an elevated systolic blood pressure, reduced haemoglobin and elevated serum creatinine we were able to construct a prediction model for disease progression with good performance (NPV 79.7% and PPV 86.7%). Potentially prioritising the delivery and increasing monitoring of these patients could be instituted to reduce progression to severe disease. Furthermore if a medical therapeutic did become available in the future this could be administered to reduce progression of disease and the associated short and long-term burden of severe disease.

4.2. Interpretation

We found a past history of hypertension was predictive of progression to severe disease. Our findings are similar to those by Zwertbroek et al. [8] who found that chronic hypertension was one of the strongest predictors of progression to severe disease (odds ratio 2.4) in patients with late preterm preeclampsia between 34 and 37 weeks' gestation. This contrasts early preterm models where hypertension was not predictive of adverse maternal outcomes [11]. This may reflect a difference in the pathogenesis of disease at early compared to late gestations. Whilst early onset preeclampsia is largely thought to be related to placental implantation defects, perhaps later onset disease may reflect the maternal vasculature where pre-existing hypertension has a greater influence on this [14]. Considering chronic hypertension appears to be an important risk factor for progression to severe disease near or at term, it should be considered for analysis in future models and when managing patients presenting with preeclampsia at term. It would be important to specify target blood pressure parameters for women with chronic hypertension prospectively in future studies. Whilst we follow tight blood pressure control for women with chronic hypertension following the CHIPS trial [15], as our study was retrospective we could not control for this. It would be interesting to see whether the variable of chronic hypertension remains predictive when patients are prospectively recruited with chronic hypertension with a target of tight blood pressure control aiming for a diastolic blood pressure of 85 mmHg.

Regarding maternal signs at admission, we found that systolic blood pressure was significantly higher at admission among women who progressed to preeclampsia with severe features at term. This contrasts the findings of the multicentre, prospective fullPIERS study [11] that examined admission characteristics of the 261 participants that had adverse outcomes and compared these with the cohort of 1762 women admitted with preterm preeclampsia. They found blood pressure at admission was not predictive of any adverse outcome. This might be explained by the treatment paradox, where antihypertensive therapy may have diluted the predictive value of blood pressure. This issue was addressed by a recent prospective, observational study in the United Kingdom that developed and validated a prediction model for adverse outcomes in preterm preeclampsia [9]. By including treatment of

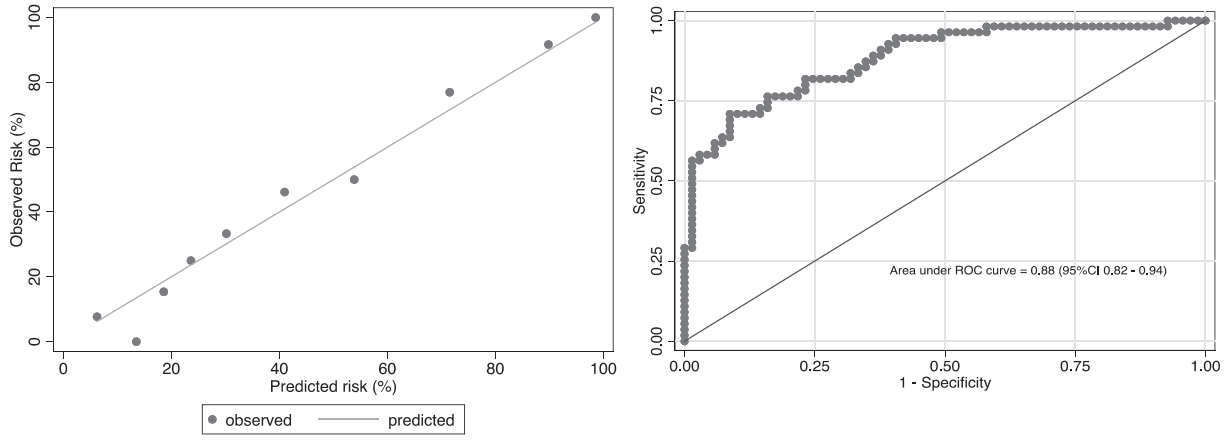

Fig. 2. Using admission characteristics of essential hypertension, systolic blood pressure, haemoglobin and creatinine we were able to show that they were predictive of disease progression as demonstrated by (a). This was based on the linear predictor equation −2.5788 + 0.3196 × essential hypertension + 0.0949 × (systolic blood pressure −125) + −0.0480 × (haemoglobin −70) + 0.0843 × (serum creatinine −30) which was used to create (a). Incorporating these characteristics, we were able to produce a receiver operating curve (b) with high sensitivity and specificity.

antihypertensives, magnesium sulphate and early delivery as predictors of adverse outcomes, this study reduced treatment bias. Similarly, we found a significantly larger percentage of women were prescribed antihypertensive medication antenatally in the group that progressed to severe disease. However, as this information is not available at admission therefore we did not include it in our predictive model.

We found that biochemical parameters routinely ordered to diagnose preeclampsia were able to predict the likelihood of progression to severe disease. Hemoglobin levels and serum creatinine were also predictive of progression whilst uric acid and transaminases were not. Similar to our study, elevated serum creatinine was a marker for adverse outcome in preterm and near-term preeclampsia studies [8,9,11]. Haemoglobin levels were reduced in women who progressed to severe disease at term, although this was not significant in pre-term studies [9,11], it could perhaps reflect the clinical distinction between pre-term and term disease. The sflt-1/placental growth factor ratio has shown promise in identifying which patients at preterm gestations will not develop preeclampsia in the subsequent week. Perhaps it might have value in determining which patients might progress to preeclampsia with severe features. Whether the sflt-1/placental growth factor ratio can predict preeclampsia disease progression would be interesting to examine in future studies[16].

Although women progressing to severe preeclampsia had increased proteinuria in our study, this was not statistically significant. This is consistent with the fullPIERS study where degree of dipstick proteinuria did not correlate with adverse outcome [11]. Both studies support recent conclusions that the degree of proteinuria is not reflective of disease severity [17]. Although the presence or absence of proteinuria is important for diagnosing preeclampsia, severe proteinuria ≥ 5 grams per day has been removed from classification systems as it is a poor marker of adverse outcome in cases of severe disease [18]. Furthermore, our study excluded 24-hour urine collection from our prediction model as the delay of this test would impede timely delivery at term.

This research is significant for patient care in several ways. Firstly, past history of hypertension, admission blood pressure and biochemical markers can be used to stratify maternal risk. These parameters are routinely collected at admission, making them pragmatic tools for the prediction and management of severe preeclampsia at term. Women at increased risk of progression to severe preeclampsia could have their delivery prioritised. Furthermore, small therapeutics are being developed and could be used to reduce immediate progression of disease and subsequent long-term morbidity for women with term disease. Secondly, it furthers our clinical understanding of preeclampsia evolution. This is particularly important for severe disease progression during the delivery process, as it highlights the need for heightened monitoring during delivery at term. Thirdly, it further acknowledges that tests such as urine protein creatinine ratio do not need to be repeated beyond diagnosis of term preeclampsia as they are not useful in predicting the progression to severe disease.

The use of maternal and disease characteristics at admission to predict the development of severe preeclampsia at term is promising. Data were obtained from a large tertiary centre in Melbourne, and we believe this group is representative of women admitted with preeclampsia without severe features at term. Due to its retrospective nature, this study is a pragmatic representation of hospital admission and management of women with preeclampsia at term.

Close to 50% of patients presenting with preeclampsia at term progressed to preeclampsia with severe features. Potentially prioritising the delivery of patients presenting with the predictive admission characteristics, increasing surveillance in this group and in the future, directing therapeutics towards them may reduce disease progression and the long term side effects of preeclampsia with severe features. This has potential to reduce maternal morbidity and perhaps prevent the life-long burden of cardiovascular disease resulting from severe disease.

Author contribution

Tarini Paul obtained the data, analysed the data and wrote the manuscript. Fiona Brownfoot, Roxanne Hastie and Stephen Tong designed the study, assisted with statistical analysis of the data and edited the manuscript. Richard Hiscock and Emerson Keenan assisted with statistical analysis and edited the manuscript.

Ethics approval

Ethics approval for the project was obtained on the 12th December 2017 from the Mercy Hospital for Women Human Research Ethics Committee (approval project number 2017–045). As this was a retrospective cohort study, individual patient consent was not required (in accordance with the ethics board).

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

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