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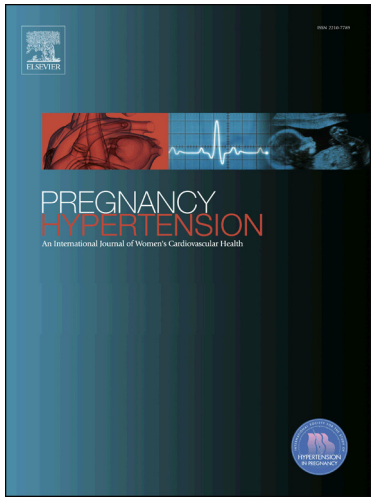

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Does induction of labour in nulliparous hypertensive women result in vaginal birth? – A descriptive study utilising birth registry data.

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Abstract

Background: Induction of labour (IOL) is a common procedure yet we have little information on the efficacy of the process for women with a hypertensive disorder of pregnancy (HDP).

Objective: To describe the birth type and associated factors in nulliparous HDP women undergoing an induction of labour.

Study design: Statutorily collected datasets on every birth and hospital admission which occurred in the state of NSW Australia between the years 2000-2011 were analysed. Hypertensive women were compared to normotensive women.

Results: Of the nulliparous women, 9.9% had a HDP. IOL for HDP women were 56.2% in a cohort of 447 558 women. The AOR for a woman with a HDP undergoing an IOL resulting in a vaginal delivery when compared to a normotensive woman is 0.86 (95%CI 0.83-0.88). Prior to 33 weeks, the lowest perinatal mortality rates (PMR) are seen in women who undergo elective caesarean section (C/S). For women with preeclampsia (PE), lower PMR are seen in women who undergo IOL.

Conclusion: For women with PE and SPE, IOL resulted in lower rates of vaginal delivery than spontaneous labour when compared to normotensive women who also underwent IOL. Women with PE at ≥33 weeks who underwent IOL had the lowest PMR.

Keywords: pregnancy hypertension, preeclampsia, induction of labour, gestational hypertension, chronic hypertension

Introduction:

Induction of labour (IOL) through the use of prostaglandins, syntocinon and amniotomy are common procedures in industrialised countries. Rates of induction and associated morbidity are both increasing [1] and it is known that elective induction for non-medical reasons increases the risk of adverse events in both mothers and babies [2]. From Level 1 evidence we know that the process may be feasible in outpatient settings for low risk women [3, 4], that women prefer the process to commence in the morning, although there is no increased efficacy when compared to evening commencement [4, 5]. The process may prevent infant macrosomia in the babies of insulin dependent diabetic women [6], although there is no evidence to support the process as preferable when compared to repeat elective caesarean section in women with previous caesarean section [7], but the induction process in all women may be of benefit in preventing perinatal death in women at or beyond term [8]. There is not enough evidence to support the routine use of acupuncture [9], amniotomy alone [10], castor oil [11], corticosteroids [12], extraamniotic prostaglandins [13], homeopathy [14], or sexual intercourse [15] although breast stimulation may be beneficial in low risk women [16] and membrane sweeping [17,18] has been shown to increase spontaneous labour rates in low risk women at term. Hyaluronidase injections may increase vaginal birth rates [19], intravaginal prostaglandin administration is optimal to intracervical [20], syntocinon is optimal in conjunction with prostaglandin administration in comparison to syntocinon alone [21], intravenous prostaglandin is not more efficacious than intravenous oxytocinon and has more side effects [22], mechanical methods may be preferable to prostaglandins in reducing caesarean section rates [23] and sub-lingual or buccal misoprostol need further trials to assess safety [24].

Even though we have a significant amount of evidence concerning the IOL process overall there is very little evidence of efficacy of the process in hypertensive women. The HYPITAT randomised controlled trial evaluated the efficacy of IOL in women with gestational hypertension and mild preeclampsia [25]. In this study women in the IOL arm had a reduced risk of the composite maternal outcome (serious morbidity or mortality) (relative risk 0·71, 95% CI 0·59—0·86, p<0·0001) when compared to women treated with expectant management with no overall difference in operative delivery or caesarean section rates. The study reported no increase in adverse neonatal outcomes but was not powered sufficiently for this outcome. HYPITAT II examined the effect of IOL in women 34-37 weeks gestation and found no difference in maternal outcomes but significantly more neonatal distress in the IOL arm [26]. The 2.5 year follow up on women in the HYPITAT study found no difference between the women's cardiovascular status between women who underwent IOL (and were therefore exposed to short (seven days on average) time periods of disease) and women who delivered following expectant management [27]. Following the publication of these results, induction of labour in hypertensive women increased in the Netherlands from 58.3% to 67.1% [28]. In regard to women with severe hypertensive disease who require delivery to optimise either or maternal or fetal safety, there is an absence of trial data examining the effect of IOL in comparison to elective caesarean section at either term or pre-term women. Expert opinion drives clinician decision making in the majority of cases [29].

The effectiveness of the varying methods and combination of methods of induction of labour used in HDP women also requires examination as this has not previously been examined.

The aim of this study was to describe the birth type and associated factors in nulliparous HDP women undergoing an induction of labour dependent upon diagnosis and method of IOL undertaken. Validated population registry datasets, such as this, are able to provide a large cohort for analysis and enable diagnostic groupings of HDP to be examined.

Materials and Methods:

Pregnancy and birth data for the time period July 1st 2000 till December 31st 2011 of all births were provided by New South Wales (NSW), Ministry of Health as recorded in the NSW Perinatal Data Collection (PDC). This population based surveillance system contains maternal and infant data on all births of greater than 400 grams birth weight and/or 20 completed weeks gestation. The NSW PDC contains statistics on all births in New South Wales - which amounts to one third of all births which occur in Australia annually. Data is provided on a variety of variables including maternal age, maternal hypertension, maternal diabetes, parity, fetal presentation, onset of labour, gestation at birth, delivery type, Apgar scores and admission to neonatal intensive care and resuscitation details for the neonate. This dataset (NSW PDC) was linked to the Admitted Patient Data Collection (APDC) for the same time period through the New South Wales Centre for Health Record Linkage (CheReL). Probabilistic data linkage techniques were utilised for data linkage and de-identified datasets were provided for analysis. Probabilistic record linkage software assigns a 'linkage weight' to pairs of records. For example, records that match perfectly or nearly perfectly on first name, surname, date of birth and address have a high linkage weight, and records that match only on date of birth have a low linkage weight. If the linkage weight is high it is likely that

the records truly match, and if the linkage weight is low it is likely that the records are not truly a match. This technique has been shown to have a false positive rate of 0.3% of records [30].

Ethical approval was obtained from the NSW Population and Health Services Research Ethics Committee, Protocol No.2010/12/291.

Subjects:

There are four types of hypertension recognised within the diagnostic criteria prescribed by the Society of Obstetric Medicine of Australia and New Zealand (SOMANZ) [31]. Women were coded as having preeclampsia if their PDC record was coded for the variable 'Pre-eclampsia', or 'Pregnancy Induced Hypertension – proteinuric' (variable available 2006-2011) or if their APDC record for the birth record was coded as including the International Statistical Classification of Diseases (ICD-10-AM) [32] codes O14.0, O14.1, O14.2, O14.9 (proteinuric hypertension). Cases of gestational hypertension were derived from the PDC code 'Pregnancy Induced Hypertension – non-proteinuric' or if their APDC record for the birth event was coded as including ICD-10-AM code O13.0 (gestational hypertension). Cases of chronic hypertension were derived from either the PDC, where a positive response was recorded for chronic hypertension or from the APDC records of women who had a birth admission which included the ICD-10-AM codes O10.0, O10.1, O10.2, O10.3, O10.4, O10.9 (chronic hypertension). Cases of preeclampsia superimposed on chronic hypertension were derived where a PDC record had a positive response for both preeclampsia and chronic hypertension or from APDC records of women who had a birth admission which included

the ICD-10-AM code O11 (superimposed preeclampsia on chronic hypertension). In cases where the type of hypertension differed between that recorded on the PDC and the APDC, the diagnosis considered more severe was used, for example a women coded as having gestational hypertension in one system and preeclampsia in the other was given a final diagnosis of preeclampsia. Women who received none of these hypertensive codes were coded as normotensive. The birth admission including the ICD-10-AM codes Z37.0 (single live birth), Z37.1 (single stillbirth) or Z38.0 (singleton born in hospital) was deemed the birth admission in the APDC dataset. Death may have been detected on any one of the following four datasets. The PDC 'Discharge status' variable or admissions in the APDC where the case mode separation was coded as 'Died' or the NSW RBDM or ABS Death Data where a death had been recorded.

Nulliparous women with a singleton pregnancy were only included in this study to eliminate the potential effect of previous delivery type and plurality.

Outcomes:

Stillbirth and neonatal deaths were calculated from multiple sources but were limited to those that occurred within 28 days of birth and they were only counted once. The maternal admission data for any admission that occurred during the pregnancy, as well as the birth admission for all cases of stillbirth or neonatal death were examined to determine any maternal medical or pregnancy related condition. This methodology of utilising multiple data sources to identify cases has been shown by Lain et al [2012] to be the most reliable way to increase ascertainment of cases [33].

Gestation is recorded at birth in the PDC and is also recorded in the database according to the woman's menstrual history, usually combined with a routine scan at 12-13 weeks. Onset of labour (spontaneous, induced or no labour) was as recorded in the PDC. The PDC also provided the delivery type data as well as neonatal outcomes, such as admission to neonatal intensive care (NICU) or special care nursery (SCN), resuscitation, APGAR scores, birth weight, as well as reason for caesarean section. Fetal distress was as recorded in the birth record in the APDC utilising the ICD-10-AM codes O68 – labour and delivery complicated by fetal stress. Vaginal delivery refers to both normal vaginal delivery and instrumental vaginal delivery in the context of this study.

Data analysis:

Demographic data is reported between the comparison groups according to HDP status utilising Chi square for dichotomous variables and mean or median comparison for continuous data. When examining delivery type, odds ratios were calculated using logistic regression with and without adjustment for maternal age and gestation at delivery. Taking into account the size of the cohort and the number of analyses undertaken, results were considered significant at the level $p<0.01$. Analysis was undertaken with IBM SPSS v.20®

Results:

Within the time period (2000-2011) there were 669 880 deliveries. This number was refined to 447 558 nulliparous women with a singleton pregnancy. The rate of HDP within this cohort was 9.9% (44 498 women). The demographic details and birth outcomes for all nulliparous women, stratified for the diagnosis of HDP, are contained in Table 1. An analysis of cases defined by induction, including outcomes, is displayed in Table 2. An analysis of the odds ratio of an induction of labour resulting in a vaginal birth both unadjusted and adjusted is contained in Table 3. Table 4and 5 contain a detailed analysis of maternal and neonatal outcomes for women and neonates undergoing induction of labour.

	Normotensive n=403 060	All hypertensive n=44 498	Preeclampsia n=16 869	Gestational Hypertension n=24 531	Chronic Hypertension n=1897	Preeclampsia Superimposed on Chronic hypertension n=1201
Age	28.7 (5.70)	28.8 (5.79)	28.7 (5.86)	28.6 (5.60)	31.6 (5.57)	28.7 (5.70)
Gestation at delivery	39.2 (2.18)	38.4 (2.60)	37.6 (3.07)	39.0 (1.97)	38.4 (2.57)	37.3 (3.37)
Smoking Yes No Missing	 46 351 (11.5%) 355 730 (88.3%) 978	 4219 (9.5%) 40 143 (90.2%) 136	 1552 (9.2%) 15 251 (90.4%) 66	 2398 (9.8%) 22 072 (90.0%) 61	 183 (9.7%) 1711 (90.2%) 3	 86 (7.2%) 1109 (92.3%) 6
Labour onset Spontaneous Induced No Labour Missing	 254 176 (63.1%) 108 255 (26.9%) 40 553 (10.1%) 76	 12 107 (27.2%) 24 976 (56.2%) 7409 (16.7%) 6	 3296 (19.5%) 9339 (55.4%) 4231 (25.1%) 3	 8071 (32.9%) 13 903 (56.7%) 2554 (10.4%) 3	 583 (30.7%) 937 (49.4%) 377 (19.9%) 0	 157 (13.1%) 797 (66.4%) 247 (20.6%) 0
Mode of birth	 206 367	 17 292	 5396	 10 833		

Normal vaginal birth	(51.2%)	(38.9%)	(32.0%)	(44.2%)	699 (36.8%)	364 (30.3%)
Forceps	29 290 (7.3%)	3197 (7.2%)	1118 (6.6%)	1884 (7.7%)	134 (7.1%)	61 (5.1%)
Vacuum	51 188 (12.7%)	5355 (12.0%)	1814 (10.8%)	3215 (13.1%)	196 (10.3%)	130 (10.8%)
Vaginal breech	1674 (0.4%)	138 (0.3%)	67 (0.4%)	62 (0.3%)	6 (0.3%)	30 (0.3%)
Total caesarean section						
Elective	114 372 (28.4%)	18 500 (41.6%)	8471 (50.2%)	8524 (34.8%)	862 (45.4%)	643 (53.6%)
Emergency	40 553 (10.1%)	7409 (16.7%)	4231 (25.1%)	2554 (10.4%)	377 (19.9%)	247 (20.6%)
Missing	73 819 (18.3%)	11 091 (24.9%)	4240 (25.1%)	5970 (24.3%)	485 (25.6%)	396 (33.0%)
	169	16	3	13	0	0
Maternal Mortality Rate (early death)	15 (3.7/10 000)	9 (20.2/100 000)	4 (23.7/100 000)	4 (16.3/100 000)	1 (52.7/100000)	0

Perinatal Mortality Rate Stillbirth NND Total	2494 (6.2/1000) 1303 (3.3/1000) 3797 (9.4/1000)	342 (7.7/1000) 188 (4.2/1000) 530 (11.9/1000)	171 (10.1/1000) 103 (6.2/1000) 274 (16.2/1000)	133 (5.4/1000) 69 (2.8/1000) 202 (8.2/1000)	23 (12.1/1000) 9 (4.7/1000) 32 (16.8/1000)	15 (12.5/1000) 7 (5.8/1000) 22 (18.3/1000)
Birthweight	3339.3 (572.23)	3196.3 (717.69)	3006 (814.60)	3340 (597.69)	3199 (691.53)	2937 (848.29)
5 minute Apgar<7	9277 (2.3%)	1521 (3.4%)	711 (4.2%)	677 (2.8%)	80 (4.2%)	53 (4.4%)
Resuscitation (any type)	22 479 (50.5%)	168 455 (41.8%)	9100 (54.0%)	11 813 (48.2%)	934 (49.2%)	650 (54.0%)
NICU/SCN admission	76 881 (19.1%)	14 691 (33.0%)	7180 (42.6%)	6266 (25.5%)	659 (34.7%)	585 (48.7%)

Table 1 Demographic and birth women details for all normotensive and HDP women

	Normotensive	**All HDP**	**PE**	**GH**	**CH**	**SPE**
Prostaglandin only	12557/18 540	3449/5612	1260/2226	1986/3011	117/187	86/188
Vaginal birth	67.7%	61.5%	56.6%	66.0%	62.6%	45.7%
Prostaglandin + oxytocin	4000/6558	1050/1808	402/680	583/1003	30/60	35/65
Vaginal birth	61.0%	58.1%	59.1%	58.1%	50.0%	53.9%
Prostaglandin + oxytocin +	14959/23 725	4279/6732	1582/2474	2404/3740	176/290	117/228
ARM	63.1%	63.6%	64.0%	64.3%	60.7%	51.3%
Vaginal birth						
Prostaglandin + ARM	5096/6717	1297/1730	475/657	754/982	42/51	26/40
Vaginal birth	75.9%	75.0%	72.3%	76.8%	82.4%	65.0%
Oxytocin only	14972/21 459	1273/2000	378/617	816/1247	54/96	25/40
Vaginal birth	69.8%	63.7%	61.3%	65.4%	56.3%	62.5%
Oxytocin + ARM	19812/26 694	4485/6089	1649/2308	2552/3368	155/209	129/204
Vaginal birth	74.2%	73.7%	71.4%	75.7%	74.2%	58.3%
ARM only	2604/3373	489/656	151/230	305/380	22/31	11/15
Vaginal birth	77.2%	74.5%	65.7%	80.3%	71.0%	73.3%
Other methods	824/1189	207/349	77/147	108/172	10/13	12/17

Vagainal birth	69.3%	59.3%	52.4%	62.8%	76.9%	70.1%
Total Vaginal birth	74 824/108 225 69.1%	16 529/24 976 66.2%	5974/9339 64.0%	9508/13 903 68.4%	606/937 64.7%	441/797 55.3%

Table 2 Induction of labour method and % resulting in vaginal birth expressed per diagnostic group as a % of women undergoing that form of induction. Highlighted cells reflect the highest % vaginal birth for that diagnosis

	Vaginal birth Odds ratio	Adjusted odds ratio	p
Normotensive induced women	1.00		
HDP induced women	**0.87 (0.85-0.90)**	**0.86 (0.83-0.88)**	<0.001
Preeclampsia induced women	**0.79 (0.76-0.83)**	**0.75 (0.72-0.79)**	<0.001
Gestational Hypertension induced women	0.97 (0.93-1.00)	0.96 (0.93-1.00)	0.06
Chronic Hypertension induced women	**0.82 (0.71-0.94)**	0.88 (0.77-1.01)	0.06
Superimposed Preeclampsia induced women	**0.55 (0.48-0.64)**	**0.54 (0.47-0.62)**	<0.001

Table 3 Unadjusted and adjusted odds ratio of an induction of labour resulting in a vaginal birth for both normotensive and hypertensive women adjusted for maternal age, gestation at delivery, maternal smoking, maternal diabetes and neonatal gender.

	Spontaneous labour vaginal birth n=2419 (14.3%)	Spontaneous labour caesarean section n=877 (5.2%)	Induced labour vaginal birth n=5974 (35.4%)	Induced labour caesarean section n=3362 (19.9%)	Elective caesarean section n=4231 (25.1%)
SCN/NICU admission	613 (25.3%)	325 (37.1%)	1807 (30.2%)	1362 (40.5%)	3073 (70.6%)
Second APGAR <7	97 (4.0%)	30 (3.4%)	286 (4.8%)	78 (2.3%)	220 (5.2%)
Resuscitation	1074 (44.4%)	515 (58.7%)	2640 (44.2%)	1912 (56.9%)	2959 (69.9%)
Eclampsia Antenatal Labour Puerperium Total (rate)	 4 4 8 (3.3/1000)	 2 1 2 5 (5.7/1000)	 1 6 5 12 (2.0/1000)	 1 3 5 9 (2.7/1000)	 5 No labour 14 19 (4.5/1000)
Stillborn	34	1	115	8	13

NND Total PMR	18 21.5/1000	2 3.4/1000	12 21.3/1000	8 4.8/1000	63 18.0/1000
Maternal Mortality	1 (41.3/100 000)	1 (114/100 000)	1 (16.7/100 000)	0	1 (23.6/100 000)

Table 4 Neonatal and maternal outcomes for women with preeclampsia only

Reason for caesarean section	Normotensive	Preeclampsia	Gestational hypertension	Chronic hypertension	Superimposed Preeclampsia	p
Failure to progress	57.4%	48.1%	54.2%	58.0%	52.0%	<0.001
Fetal distress	28.2%	30.9%	28.7%	28.1%	32.6%	<0.001
Other	13.4%	20.2%	16.1%	13.6%	14.9%	<0.001
Not stated/missing	1.0%	0.8%	1.0%	0.3%	0.5%	<0.001

Table 5 Reason for caesarean section in induced women stratified by hypertensive diagnosis utilising chi-square

The eclampsia rate statistically differed between those HDP women who laboured spontaneously (1.4/1000), were induced (1.2/1000) or underwent elective pre-labour caesarean section (2.8/1000) (p=0.008). Eclamptic events which occurred during labour were not different between women who laboured spontaneously (0.58/1000 HDP deliveries) and those whose labour was induced (0.52/1000 HDP deliveries) (p=0.97). Fetal distress occurred in 25.5% of preeclampsia cases, 23.4% of gestational hypertension cases, 25.1% of chronic hypertension cases and 29.2% of superimposed preeclampsia cases (p<0.001).

For all HDP women, as well as women with preeclampsia and chronic hypertension, the IOL method which resulted in the highest vaginal delivery rate was prostaglandin and ARM (75.0%, 72.3% and 82.4% respectively). For normotensive women and women with gestational hypertension and superimposed preeclampsia, the method which resulted in the highest vaginal delivery rate was ARM only (77.2%, 80.3% and 73.3% respectively). See Table 3.

Rates of fetal distress in fetuses of induced women were higher in all HDP diagnostic groups than for women who laboured spontaneously (p<0.001) but did not differ significantly between diagnostic groups for HDP induced women. See Figure 1. Reason for caesarean section for induced women is illustrated in Table 6, with failure to progress being highest (58.0%) in women with chronic hypertension and lowest in women with preeclampsia (48.1%).

Figure 1 Rates of fetal distress as recorded in the mother's birth record utilising ICD-10-AM codes compared between HDP diagnostic groups

Following adjustment for gestation, maternal age, presence of maternal diabetes, smoking and neonatal gender, the odds ratio for a woman with preeclampsia or superimposed preeclampsia of delivering vaginally following an induction of labour was lower than normotensive women (0.75 95%CI 0.72-0.79 and 0.54 95%CI 0.47-0.62 respectively).

Discussion:

The differences in gestation at birth, induction rates, birthweight, need for special care or intensive care for neonates clearly differentiates the hypertensive cohort as one at greater risk for adverse outcomes when compared to the normotensive. Women with preeclampsia deliver earlier, are induced more frequently and give birth to lower weight infants than normotensive women and women with gestational hypertension [34]. In this study hypertensive women were induced at greater than twice the rate as normotensive women (26.9 %v 56.2%) and this highlights the interventionist management of the HDPs in the Australian setting, where only 27.2% of hypertensive women labour spontaneously. In comparison to expectant management, intervention for women with HDP has been shown to lower both mortality and morbidity for mother and baby [35, 36, 37].

Although women with preeclampsia have a reduced OR of delivering vaginally following IOL than normotensive women, this finding was not replicated in the women with gestational hypertension or chronic hypertension indicating that women with preeclampsia may have a reduced receptiveness to induction pharmacological agents or that clinicians have a lowered threshold for making the decision to deliver via

caesarean section in these women. The absence of blood pressure, pharmacological treatment and biochemical data in this dataset limits the conclusions which can be drawn around this issue.

The higher rates of vaginal birth following IOL occurred in both hypertensive and normotensive women who delivered pre-term. There is very little in the literature assessing the safety and efficacy of the IOL process in hypertensive women pre-term. Those studies which have addressed this pre-term issue have not included hypertensive women [38-41]. Although the numbers in the HDP pre-term groups were not large (n=5456) when compared to the term deliveries (127 525) these numbers were still greater than any which have been reported previously.

The method of induction appeared to influence the vaginal delivery rate. Differences in vaginal delivery rates as high as 32.4% were seen between induction methods. The absence of syntocinon usage in all HDP groups and normotensive women was associated with the highest rates of vaginal birth, whether it be through the use of prostaglandins and ARM or ARM alone. Many other factors have been indicated as influencing IOL success, with parous, tall women with a low BMI having higher vaginal delivery rates [42] although other factors such as cervical length on transvaginal ultrasound as well as the Bishops Score (most importantly the dilatation component) have higher positive predictive values for vaginal delivery [43, 44]. Insulin like growth factor binding protein 1 and fetal fibronectin also appear to play a part in predicting IOL success [44] although we were not able to control for these in this study.

Maternal mortality is higher in the HDP group than the normotensive (20.2 v 3.9/100 000 births). In a systematic review of maternal mortality worldwide, hypertensive disease accounted for a 16.1% of maternal deaths in the developed world [45] with variation in rates between 6.7% and 24.3%. The 20.2/100 000 maternal mortality rate associated with hypertensive disease in this study equates to 27.3% of all maternal mortality (in this total cohort), higher than reported World Health Organisation developed country rates [45]. In this study 30% of deaths

occurred in women at ≤32 weeks gestation, 22% occurred between 33-36 weeks and the remaining 48% occurred at term. In this study, none of the cases of maternal death were directly associated with women with eclampsia.

Following removal of all known fetal deaths in utero, the women with preeclampsia who laboured spontaneously and delivered vaginally had the highest rates of perinatal mortality (21.5/1000) when compared to women who laboured spontaneously or were induced and delivered via caesarean section or vaginally or underwent elective caesarean section. The overall perinatal mortality rate in this cohort was 10/1000 births or 1% of nulliparous, singleton births. This equates to World Health Organisation estimates [46].

Elective caesarean section when compared to induced or spontaneous labour was not protective against the incidence of eclampsia. The overall eclampsia rate per 1000 preeclamptic births was 3.1. In the spontaneous cohort it was 3.9/1000, in the induced cohort 2.3/1000 and the elective caesarean section cohort 4.5/1000. Even if only postpartum seizures were examined, the incidence of eclampsia in the elective caesarean section cohort was 3.3/1000 compared to 1.1/1000 within the induced women. When comparing women with preeclampsia who gave birth vaginally to women who delivered via caesarean section [regardless of onset on labour], the incidence of eclampsia was 2.4/1000 births compared to 3.9/1000 births respectively. When examining postpartum eclampsia this difference was even greater with 0.6/1000 in the vaginal birth cohort and 2.5/1000 in the caesarean section group. The increased incidence in women postpartum following caesarean section could be due to these women being more severely unwell, larger volumes of intravenous fluids being administered, rebound hypertension following spinal/epidural removal or the use of non-steroidal anti-inflammatory drugs which have previously been associated with postpartum eclampsia [47].

Conclusions:

The adjusted odds ratio for a woman with preeclampsia or superimposed preeclampsia undergoing an induction of labour which results in a vaginal birth in lower than for normotensive women.Superimposed preeclampsia carries the highest maternal and perinatal mortality rates. Clinicians are able to use these results as a guide accompanying the wide variety of clinical and laboratory findings regarding maternal and fetal well-being.

Limitations and future directions:

Large datasets are powerful tools to study incidence and associated factors. These results do not imply causation. The datasets used in this study lack data on maternal BMI, blood pressure readings, disease symptoms haematological findings, treatment variations, incidence of fetal distress, cardiotocograph and other measurements of fetal well-being - all factors which may be potential confounders in assigning causal associations. These data items are not recorded in the PDC nor the APDC and, hence, could not be included in variables in any statistical modelling. Event timeline information is also not able to be established. Multifactorial clinician assessment of individual cases can never be modelled into this type of equations. These more refined details are able to be detected in smaller cohorts and trials yet such tools lack the power often required to answer specific questions which often leads to the use of composite outcomes which are neither precise nor specific. Large datasets which provided greater detail on baseline maternal characteristics would meet the needs of researchers and clinicians alike.

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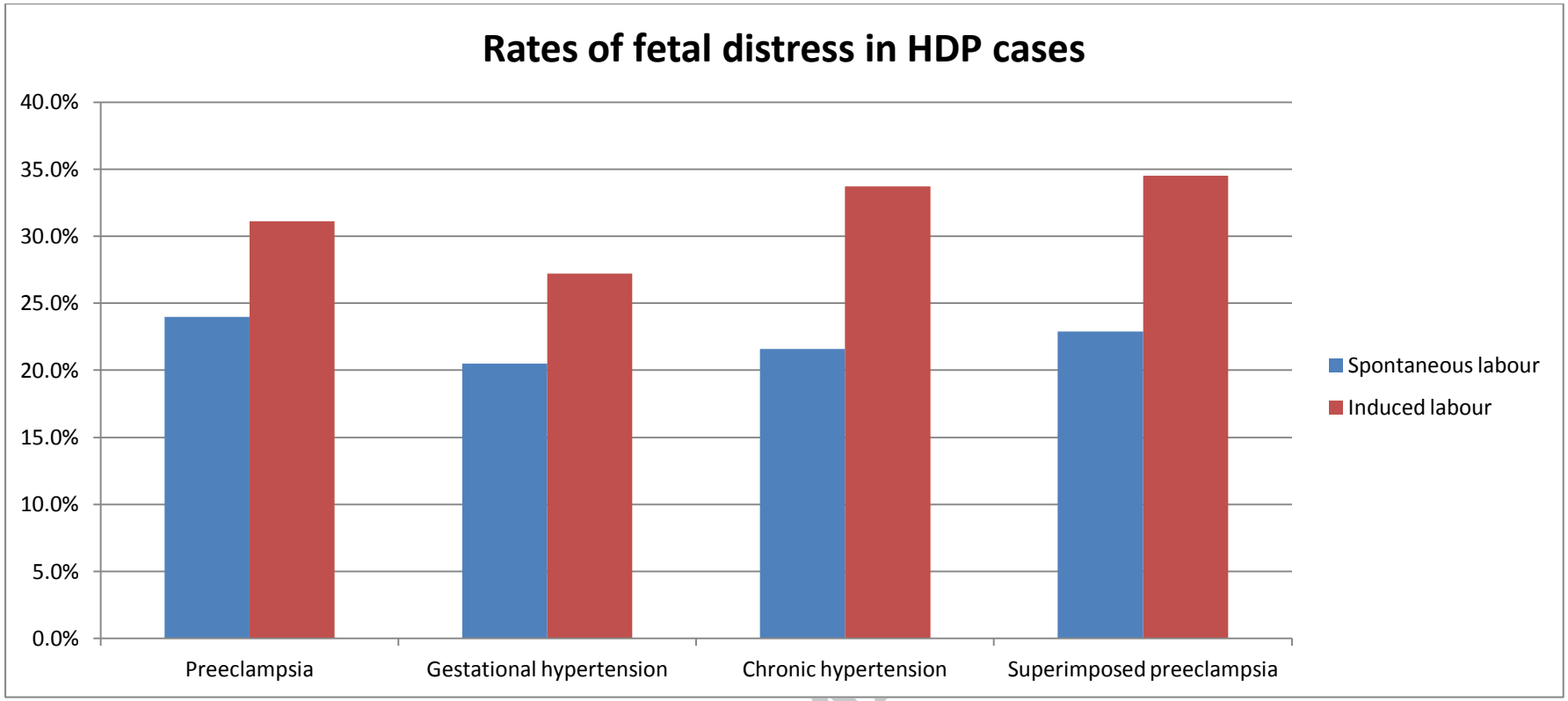

Highlights

- 56.2% of women with a HDP underwent an IOL during the 11 year period of the study
- The AOR of a women with HDP undergoing an IOL resulting in a vaginal delivery was 0.86 (95% CI 0.83-0.88) when compared to normotensive women undergoing an IOL