

# Risk factors and perinatal outcome of pregnancies complicated with cephalopelvic disproportion: a population-based study

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## Abstract

**Objectives** To characterize risk factors and perinatal outcome following cephalopelvic disproportion (CPD).

**Methods** A retrospective population-based study comparing all singleton deliveries of women with and without CPD, between 1988 and 2010, was conducted. A multiple logistic regression model was used to control for confounders.

**Results** Out of 242,520 patients, 0.3% ( $n = 673$ ) were diagnosed with CPD. Using a multivariable analysis, the following obstetric risk factors were significantly associated with CPD: fetal macrosomia (birth weight above 4 kg, OR = 3.3, 95% CI 2.7–4.1,  $P < 0.001$ ), infertility treatment (OR = 2.6, 95% CI 1.8–3.8,  $P < 0.001$ ), previous caesarean delivery (OR = 2.2, 95% CI 1.9–2.7,  $P < 0.001$ ), maternal obesity (OR = 2.1, 95% CI 1.3–3.4,  $P < 0.001$ ), and polyhydramnios (OR = 1.7, 95% CI 1.3–2.3,  $P < 0.001$ ). Deliveries complicated by CPD resulted in Caesarean delivery in 99%, and were more likely to have laceration of the cervix (1.2 vs. 0.3%,  $P < 0.001$ ), rupture of uterus (0.4 vs. 0.1%,  $P < 0.001$ ), intrapartum mortality (0.6 vs. 0.1% in control,

$P < 0.001$ ), and low 1-min Apgar scores ( $<7$ ; 27.2 vs. 6.5%,  $P < 0.001$ ).

**Conclusions** In our population, independent risk factors for CPD include fetal macrosomia, infertility treatment, previous caesarean delivery, maternal obesity and polyhydramnios. These pregnancies had higher rates of adverse perinatal outcomes and accordingly high index of suspicion should be pursued when commencing trial of labor of such pregnancies.

**Keywords** Cephalopelvic disproportion (CPD) · Pregnancy outcome · Caesarean delivery · Pelvic fractures

## Introduction

Dystocia is an abnormal progression of the birth process which can result from cephalopelvic disproportion (CPD). There are three main components responsible for dystocia (“the 3 P’s”): passageway (the pelvic canal), passenger (the size, lie, position and presentation of the fetus), and power (uterine contractions). CPD is defined as a mismatch between the maternal birth canal (the pelvis), and the fetal head [1]. Attempts to predict which maternal pelvis bears the tendency for CPD and dystocia have been made [2–4], suggesting nulliparity [2, 3, 5], fetal macrosomia, epidural analgesia [6], hydramnios, hypertensive disorders and gestational diabetes mellitus [2, 7–12] as risk factors for second stage of labor arrest. Measurement of the intertrochanteric distance and transverse diagonal of Michaelis sacral rhomboid area have been suggested as screening procedure in remote areas [13, 14]. Clinical pelvimetry is a fading skill, and X-ray pelvimetry was not proven effective in predicting CPD and is not recommended for ruling out dystocia due to CPD [1, 15–17]. Retrospective studies

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utilizing CT or MRI pelvimetry subsist, but no consensus exists for its application, and imaging studies are certainly not part of the routine care. Current recommendation of the American College of Obstetricians and Gynecology (ACOG) [18] is that the clinical course during delivery will determine the diagnosis of CPD.

Suggested maternal CPD etiologies for the relatively narrow pelvis include a moderate association with malnutrition or young maternal age [19, 20], as well as advanced maternal age [3] and short stature [20, 21]. A meta-analysis investigating the association between short stature and CPD revealed that one of five short stature women were referred to caesarian section (CD) for having small pelvises [22]. Nevertheless, short stature is not necessarily an indication for a small size pelvis [23]. Maternal obesity has also been suggested as a risk factor for CPD [7, 24, 25]. Rickets is known to be a cause for small or distorted pelvis [20, 23]. One study of an immigrant population of women from countries with high rates of malnutrition to the USA, reports that this population had higher rates of CPD following their immigration, probably due to malnutrition during puberty [26].

Multiple hereditary exostoses and other cases with exostoses might cause a mechanical obstruction of the birth canal [27–29]. Limited data exist regarding obstetric consequences following pelvic fractures [30–35]; most of the literature discusses the implications of pregnant woman in the acute multitrauma setting [36–40].

Cephalopelvic disproportion can be ruled out when the biparietal diameter has passed through the pelvic brim, and the leading edge of the vertex is in the mid-pelvis at the level of the ischial spines, but if descent is too slow and engagement does not occur, CPD is suspected. Palpation of fetal head suturae molding status, caput succedaneum or the degree of asynclitism can raise suspicion of dystocia related to CPD [1]. Nevertheless, the diagnosis of dystocia should not be made before an adequate trial of labor has been achieved [18].

Cephalopelvic disproportion can lead to severe maternal morbidity (including rupture of the uterus, severe perineal and vaginal tears) and fetal morbidity and even mortality, specifically in remote underdeveloped areas [20, 41]. Accordingly, the ability to predict impending arrest, can prevent adverse perinatal outcome. The present study was aimed to determine risk factors and perinatal outcome of patients diagnosed with CPD during labor. A secondary aim was to find whether there is a relationship between CPD and a previous pelvic fracture.

## Materials and methods

A retrospective population-based analysis of all singleton pregnant women who delivered at the Soroka University

Medical Center from 1988 to 2010 was performed. A comparison was made between women with and without the diagnosis of CPD (diagnosed during labor, or documented in her medical care files).

Data were extracted from the computerized perinatal database of the hospital. The computerized perinatal database consists of obstetric and perinatal information recorded directly during and after delivery by an obstetrician, which is then examined by skilled medical secretaries before being entered into the computerized database. Coding is done following assessment of the medical prenatal care records as well as the routine hospital documents, measures that assure minimal bias.

Data extracted from the computerized perinatal database and the hospital's archives were related to the following categories: (1) maternal characteristics: age, fertility treatment, parity; (2) pregnancy outcomes: gestational age, birth weight; (3) maternal characteristics and outcomes: labor induction, labor dystocia, premature rupture of membranes, polyhydramnion, diabetes mellitus, hypertensive disorders and Caesarian delivery (CD); (4) perinatal outcomes: Apgar scores, and perinatal mortality.

The definition of labor dystocia (failure of labor to progress during the first and second stages) was based on deviations from Friedman's plots [2–4]. The length of the second stage of labor was limited to 2 h in nulliparous women (or 3 h if epidural analgesia was applied), and 1 h in multiparous women (or 2 h if epidural analgesia was applied). Patients were managed with oxytocin augmentation. The diagnosis of labor dystocia was made by the attending physician [2–4]. The diagnosis of CPD was done following a trial of labor, when labor dystocia was established, as recommended in the current literature.

The study was approved by the local Ethics Institutional Board. Statistical analysis was performed with the SPSS package. Statistical significance was calculated using the Chi-square test for differences in qualitative variables and the ANOVA test for differences in continuous variables. A multivariable analysis was constructed to control for confounders. The variables that were included in the model were chosen according to their statistical and clinical relevance to CPD. Odds ratios (OR) and their 95% confidence interval (CI) were computed. *P* value <0.05 was considered statistically significant.

## Results

Out of 242,520 patients which were included in our cohort, 0.3% (*n* = 673) were diagnosed with CPD.

Table 1 shows clinical characteristics of women with and without CPD. Patients with CPD tended to be nulliparous, younger, of Jewish ethnicity, to be involved with

**Table 1** Clinical characteristics of women with and without CPD

Characteristics		CPD (n = 673)	Cohort (n = 241,847)	OR (95% CI)	P value
Maternal age (years)	<18	3.0%	2.0%		0.049
	18–29	60.9%	57.3%		
	29–39	32.7%	36.8%		
	>40	3.4%	3.9%		
Ethnicity	Bedouin	40.0%	51.0%		<0.001
	Jewish	60.0%	49.0%	1.6 (1.3–1.8)	
Pregnancies number	1	41.9%	19.5%	3 (2.6–3.5)	<0.001
	2–4	40.7%	47.5%		<0.001
	>5	17.4%	33.0%		<0.001
Parity	1	50.7%	23.4%	3.4 (2.9–3.9)	<0.001
	2–4	36.4%	50.8%		<0.001
	>5	12.9%	25.8%		<0.001
Fertility treatment	IVF	1.5%	0.6%		<0.001
	OI	2.5%	1.1%		
	Total	4.0%	1.7%	2.5 (1.7–3.7)	
Gender	Female	40.0%	48.7%		<0.001
	Male	60.0%	51.3%	1.4 (1.2–1.7)	
Gestational age (weeks)	Mean	39.8 ± 1.5	39 ± 2.3		<0.001
	<36	2.7%	8.0%		<0.001
	37–41	88.0%	87.6%		
	42+	9.4%	4.5%		
Birth weight (Gr)	Mean	3,514.8 ± 505.3	3,181.5 ± 552.1		<0.001
	<2,500	2.5%	8.0%	0.3 (0.2–0.5)	<0.001
	2,500–3,999	82.5%	87.2%		<0.001
	≥4,000	15.0%	4.8%	3.5 (2.9–4.4)	

**Table 2** Obstetric risk factors for patients with and without CPD

Characteristics	CPD (n = 673)	Cohort (n = 241,847)	OR (95% CI)	P value
Previous caesarian delivery	23.8%	11.9%	2.3 (1.9–2.8)	<0.001
Recurrent abortions	3.7%	5.2%	0.7 (0.5–1.1)	0.84
Polyhydramnion	7.4%	3.6%	2.2 (1.6–2.9)	<0.001
Oligohydramnion	1.9%	2.3%	0.8 (0.5–1.4)	0.477
Lack of prenatal care	9.1%	9.3%	1 (0.7–1.3)	0.809
Gestational diabetes mellitus	7.7%	5.8%	1.4 (1–1.8)	0.028
Diabetes mellitus type 2	0.4%	0.6%	0.8 (0.3–2.4)	0.666
Diabetes Mellitus type 1	0.1%	0.1%	2.2 (0.3–15.5)	0.43
Hypertensive disorders	7.1%	5.6%	1.3 (1–1.7)	0.89
Maternal obesity	2.8%	1.0%	2.9 (1.8–4.6)	<0.001

fertility treatment and to have male newborns as compared to the control group. Preterm delivery was less common in CPD patients. Mean birth-weight for the CPD was 333 g higher than the comparison group. Likewise, macrosomia was found to be significantly higher in CPD group.

Table 2 presents obstetric risk factors for patients with and without CPD. Factors significantly associated with

CPD were status after CD, fertility treatments, polyhydramnios, obesity and gestational diabetes.

Table 3 presents complications and outcomes related to pregnancy and labor of patients with and without CPD. Failure to progress in both delivery stages 1 and 2, non reassuring fetal heart rate (FHR) patterns and meconium stained amniotic fluid, as well as first minute low Apgar

**Table 3** Pregnancy and labor complications and outcomes of patients with and without CPD

Characteristics	CPD ( <i>n</i> = 673)	Cohort ( <i>n</i> = 241,847)	OR (95% CI)	<i>P</i> value
Failure to progress in labor first stage	13.7%	1.8%	8.8 (7.1–11)	<0.001
Failure to progress in labor second stage	25.3%	1.5%	22.3 (18.7–26.6)	<0.001
Non-reassuring FHR patterns	15.0%	4.8%	3.5 (2.8–4.3)	<0.001
Meconium stained amniotic fluid	24.1%	15.4%	1.7 (1.5–2.1)	<0.001
Post partum hemorrhage	0.1%	0.6%	0.3 (0–1.8)	0.139
Laceration of cervix	1.2%	0.3%	4.6 (2.3–9.2)	<0.001
Rupture of uterus	0.4%	0.1%	7.8 (2.5–24.7)	<0.001
Labor induction	42.9%	26.3%	2.1 (1.8–2.5)	<0.001
Caesarian delivery	99.0%	13.1%	633.3 (300.7–1,333.6)	<0.001
Blood transfusion	5.2%	1.4%	3.9 (2.8–5.5)	<0.001
Low Apgar 1 min (<7)	27.2%	6.5%	5.4 (4.5–6.4)	<0.001
Low Apgar 5 min (<7)	3.1%	3.1%	1 (0.7–1.6)	0.935
Perinatal mortality (total)	0.9%	1.4%	0.7 (0.3–1.5)	0.29
Intrauterine fetal death	0.1%	0.7%	0.2 (0–1.4)	0.073
Intrapartum death	0.6%	0.1%	7.7 (2.8–20.8)	<0.001

**Table 4** Multiple logistic regression model, with backward elimination, of factors associated with CPD

Variables	OR	95% CI	<i>P</i> value
Macrosomia (birth weight above 4 kg)	3.3	(2.7–4.1)	<0.001
Infertility treatment	2.6	(1.8–3.8)	<0.001
Previous caesarean delivery	2.2	(1.9–2.7)	<0.001
Obesity	2.1	(1.3–3.4)	0.001
Polyhydramnios	1.7	(1.3–2.3)	<0.001

The initial model included, in addition, maternal age and gestational diabetes mellitus

(but not of fifth minute Apgar) were significantly associated with CPD. Laceration of cervix, rupture of uterus during labor, induction of labor and maternal blood transfusion were also found to occur more in the group of CPD.

Intrapartum intrauterine fetal mortality was found to be significantly higher with CPD, but overall fetal mortality was not significantly different.

Using multiple logistic regression models (Table 4), with CPD as the outcome variable, the following variables were found to be independent risk factors: macrosomia, infertility treatments, previous CD, obesity and polyhydramnios. Maternal age and gestational diabetes were not found to be independent risk factors after controlling for confounders.

Pelvic fractures were found among 28 women of the cohort population; only 3 (11%) women had proceeded to normal labor. In all the surgical reports, previous pelvic fracture was registered as a cause for CD, besides one patient (3.5%) who also had CD before her pelvic fracture.

## Discussion

Cephalopelvic disproportion is a clinical condition with possible devastating perinatal obstetrical outcomes. Patients with CPD in our population had higher rates of maternal morbidities including cervical lacerations and uterine rupture, and these pregnancies had higher rates of perinatal complications including low 1-min Apgar scores as well as intrapartum mortality. Therefore, our aim was to isolate the most significant risk factors. Our multivariate analysis produced the profile of obese pregnant women, these with a previous cesarean delivery, carrying a macrosomic fetus and parturients with polyhydramnios.

Interestingly, although in other studies diabetes mellitus was found as an independent risk factor for CPD [2, 7–12], it was not found as an independent risk factor in our multivariate analysis. It seems that it is not the diabetes mellitus per-se but rather the result of uncontrolled disease, which is illustrated by the presence of polyhydramnios and fetal macrosomia. The association of maternal obesity and fetal macrosomia is also known in the literature [11, 12, 25], and our findings surely confirm that both factors are important contributors to CPD.

Of 28 women following pelvic fracture, only 3 (11%) had proceeded to normal delivery. Nevertheless, the discussion of this population in light of the restricted data [30–34] and our small sample size is limited, and decision should be individualized according to the clinical judgment of the attending physician.

Our study offers several strengths. Our large sample size allowed us to study the association of a relatively rare diagnosis with several clinically important risk factors as well

as outcomes in our population. Additionally, the comprehensive database allowed us to access pregnancy information that was actually obtained in a prospective manner (by the attending physician). However, our study has several weaknesses; mostly due to its retrospective design, such as the potential for missing data. Nevertheless, data were reported by an obstetrician directly after delivery and skilled medical secretaries routinely reviewed the information prior to entering it into the database thereby minimizing recall bias. Coding was done after assessing the medical prenatal care records together with the routine hospital documents. Also, deliveries occurred over a 20-year period, in a tertiary medical center. One bias may be the changing diagnosis of CPD, influenced by different meanings and attitudes of different physicians. However, as the practice did not change over the years regarding CPD, this is unlikely to have a significant effect. Unfortunately, data regarding several risk factors such as malnutrition was not available in our database.

In conclusion, independent risk factors for CPD, identified in our population include fetal macrosomia, infertility treatment, previous caesarean delivery, maternal obesity and polyhydramnios. These pregnancies had higher rates of adverse perinatal outcomes including intrapartum mortality, and low Apgar scores at 1 min. High index of suspicion should be pursued when commencing trial of labor of such pregnancies. In the prenatal and perinatal clinical setting, the clinician should bear in mind these associated factors, with close monitoring during trial of labor.

**Conflict of interest** None.

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