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MATERNAL-FETAL MEDICINE

Does prenatal identification of fetal macrosomia change management and outcome?

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Abstract

Purpose To assess whether there is an association between predicted fetal macrosomia and adverse outcomes in macrosomic newborns (> 4000 g), based on a sonographic evaluation up to 2 weeks prior to delivery.

Methods A retrospective cohort study of 3098 mothers of macrosomic babies who were delivered at our institution (2000–2015). We compared the management and outcomes of women with predicted fetal macrosomia with that of women with unknown fetal macrosomia. The primary outcomes were cesarean section (CS) rate and postpartum hemorrhage. Secondary outcomes were composite maternal and neonatal outcomes and birth injuries.

Results In 601 (19.4%) women fetal macrosomia was predicted, and in 2497 (80.6%) women, fetal macrosomia was unknown. CS rate was more than 3.5 times higher in the group of predicted macrosomia (47.2% vs. 12.7%, $P < 0.001$) than those with unpredicted macrosomia; not only due to non-progressive labor, but for non-reassuring heart rate as well. However, predicted fetal macrosomia reduced the risk of postpartum hemorrhage (aOR 0.5, 95% CI 0.2–1.0), maternal (aOR 0.3, 95% CI 0.2–0.5) and neonatal composite adverse outcomes (aOR 0.7 95% CI 0.6–0.9). It was also associated with increased risk for induction of labor, episiotomy, 3rd- or 4th-degree tears and a longer maternal hospitalization. Birth injuries and shoulder dystocia were not different between the groups.

Conclusions Antepartum CS was found to be associated with predicted fetal macrosomia. Moreover, a planned CS due to macrosomia was associated with reduced risk for postpartum hemorrhage, maternal and neonatal outcome, even for babies with a mean birth weight < 4500 g.

Keywords Birth injuries · Cesarean section · Fetal macrosomia · Maternal outcome · Postpartum hemorrhage · Shoulder dystocia

Introduction

Fetal macrosomia is predicted beyond an absolute birth weight, 4000 g or 4500 g, regardless of gestational age [1, 12]. The American College of Obstetrics and Gynecologists (ACOG) recognizes the continuum risk of increased fetal growth, and divides fetal macrosomia into three categories according to birth weight (4000–4499 g, 4500–4999 g, and ≥ 5000 g) acknowledging that each category holds additional risk for complications [1]. Other reports suggest that a statistical approach to define fetal macrosomia, using customized growth curves based on ethnicity or individual characteristics, may improve precision in evaluating fetal growth [6, 7].

The number of infants with a birth weight exceeding 4000 g has increased over the years, reaching as high as 9% of all live-born neonates worldwide [12]. The macrosomic fetus predisposes women and babies to a variety of adverse maternal and perinatal outcomes, including operative vaginal delivery; cesarean section (CS); genital tract lacerations; postpartum hemorrhage; shoulder dystocia, leading to birth trauma (brachial plexus injury and fractures); and neonatal hypoglycemia [8, 13, 14, 20, 26]. As birth weight

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increases, the risk of adverse outcomes increases as well [1, 4]. Although the risk when birth weight is between 4000 g and 4500 g is greater than that of the general obstetric population, it increases sharply when the birth weight is more than 4500 g [1]. In order to reduces those complications, one must aim to have as accurate as possible an estimated fetal growth; a clinical estimation or by an ultrasound exam. By underestimating birth weight, we may expose women and their babies to complications. In contrast, overestimation has been shown to increase the likelihood of a CS [5]. Recently, The World Health Organization (WHO), the National Institute of Child Health and Diseases (NICHD), and the International Fetal and Newborn Growth Consortium for the twenty-first century have published international estimated fetal weight standards that might be adoptable worldwide and estimate fetal weight more accurately [10, 15, 16, 24].

For non-diabetic women, there are no proven interventions to treat predicted macrosomia, and planned CS is controversial. Prophylactic CS may be considered for suspected fetal macrosomia with an estimated fetal weight of at least 5000 g in women without diabetes and at least 4500 g in women with diabetes [1, 2]. However, the clinical effectiveness of offering prophylactic CS to women with any specific estimated fetal weight must still be established in randomized clinical trials [1].

Currently, there is limited information regarding the effect of predicted fetal macrosomia on clinical decision-making. Some studies do not specifically relate to macrosomic neonates [17, 19] and others are based either on a clinical estimation of fetal macrosomia, or sonographic estimates exceeding the 90th percentile for gestational age and measured between 4 and 12 weeks prior to delivery date [25, 27].

The objective of our study was to compare predicted fetal macrosomia with unpredicted fetal macrosomia in macrosomic neonates, based on a sonographic evaluation of fetuses larger than 4000 g measured up to 2 weeks prior to delivery, and evaluate whether expecting a macrosomic baby reduces or increases adverse outcomes.

Materials and methods

Study population

This was a retrospective cohort study of all live-born singleton pregnancies with macrosomic newborns who were delivered at our institution between 1 January 2000, and 1 June 2015. We also included women who were scheduled for a planned CS due to macrosomia. We compared the management and outcomes of women with predicted fetal macrosomia with that of women with unpredicted fetal macrosomia.

We excluded multiple pregnancies, fetal presentations other than cephalic, any contraindications for induction of labor or vaginal delivery, or women who were prescheduled to undergo a CS (except a planned CS due to macrosomia), such as previous multiple CS, breech presentation, or any other indication for a planned CS. Cases where mode of delivery was missing were also excluded.

This study was approved by the local Institutional Review Board (the Helsinki Committee). Due to the retrospective nature of the study, consent for participation was not required.

Data were retrieved from the computerized perinatal database of the center and was cross-tabulated using an individualized identification number per patient. Data from the neonatal unit and the neonatal intensive care unit (NICU) was integrated with the birthing unit database by using a unique admission number assigned to each parturient and her offspring. Collected data included demographic and obstetrical parameters, labor and delivery data, as well as short-term neonatal and maternal outcome (up to discharge).

Outcomes

The primary outcomes were CS rate and postpartum hemorrhage (PPH) ($\geq$ 500 cc blood loss in a vaginal delivery or $\geq$ 1000 cc blood loss in a CS).

Secondary outcomes were composite maternal and neonatal outcomes and birth injuries. Composite maternal outcome included at least one of the following: postpartum hemorrhage (PPH) ($\geq$ 500 cc blood loss in a vaginal delivery or $\geq$ 1000 cc blood loss in a CS), manual extraction of placenta, episiotomy or third- or fourth-degree tear.

Composite neonatal outcomes included at least one of the following: shoulder dystocia (any additional obstetric maneuvers following failure of gentle downward traction on the fetal head to effect delivery of the shoulders, or head-to-shoulder delivery time exceeding 60 s [21]), cord blood pH < 7, convulsions, hypoglycemia, anemia, polycythemia, clavicle fracture, Erb's palsy, cephalohematoma, subgaleal hemorrhage, transient tachypnea of the newborn (TTN), or respiratory distress syndrome (RDS).

Birth injuries included at least one of the following: cephalohematoma, subgaleal hemorrhage, clavicle fracture, or Erb's palsy.

Gestational age was based on last menstrual period (LMP) or ultrasound (US) examination earlier in pregnancy. Fetal macrosomia was predicted when an estimated fetal growth of 4000 g or above was demonstrated, based on an ultrasound examination up to 2 weeks prior to delivery, using Hadlock's formula.

Non-progressive labor (NPL) included prolonged first- or second-stage of labor. Prolonged first stage of labor was defined as women at or beyond 6 cm dilatation with

ruptured membranes who fail to progress despite 4 h of adequate uterine activity, or at least 6 h of oxytocin administration with inadequate uterine activity and no cervical change [4]. Prolonged second stage was established after 3 h of full dilatation among nulliparous with regional analgesia (RA), 2 h among nulliparous with no RA or multiparous with RA, and 1 h among multiparous without RA. Fetal heart rate tracing was categorized using the National Institute of Child Health and Human Development (NICHD) criteria [3].

Oligohydramnios was defined as amniotic fluid index (AFI) ≤ 5 cm.

As per our departmental protocol, amnioinfusion was executed only in cases when severe variable decelerations were identified. The procedure was performed by a transcervical route.

Statistical analysis

Statistical analysis was performed with the SPSS v25.0 package (IBM, Armonk, NY). Continuous variables were compared using Student's *t* test or Mann–Whitney *U* test. The Chi-square and Fisher's exact tests were used for the comparison of categorical variables, as appropriate. Logistic regression analysis was used to adjust outcomes for potential confounders. Differences were considered significant when *P* value was less than 0.05.

Results

Characteristics of study population

Overall, 3098 patients with macrosomic newborns were included in the study, of which in 601 (19.4%) women fetal macrosomia was predicted, and in 2497 (80.6%) women, fetal macrosomia was unknown (Fig. 1). Of note, we did two separate analyses, one including elective CS solely due to macrosomia, and a second analysis excluding those elective CS and including only women attempting a trial of labor.

The characteristics of the groups are presented in Table 1a. Women in the predicted macrosomia group were more likely to be nulliparous, with pregestational and gestational diabetes mellitus (GDM) than women with unpredicted fetal macrosomia. They also delivered a few days earlier. There were no other differences in characteristics between the groups (Table 1a). When we included only women who attempted a trial of labor, the predicted macrosomia group was still more likely to be nulliparous, with GDM. However, there was no difference in the rate of pregestational diabetes mellitus. Furthermore, they had a higher rate of hypertensive disorders of pregnancy (Table 1b).

Pregnancy outcome

Induction of labor and mode of delivery are presented in Table 2. We have referred only to women who attempted

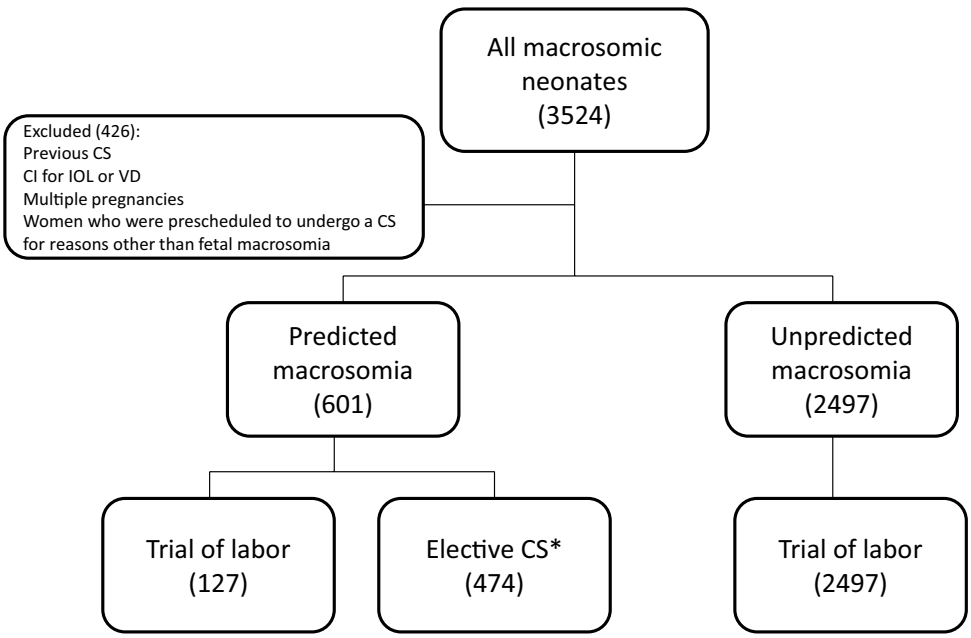

Fig. 1 Description of the study and control groups

CS: cesarean section; CI: contraindication; IOL: induction of labor; VD: vaginal delivery
*CS due to suspected fetal macrosomia

Table 1 Baseline characteristics in the predicted macrosomia and the unpredicted macrosomia groups

	Predicted macrosomia	Unpredicted macrosomia	*P* value
(a) Including scheduled cesarean sections for macrosomia (3098)			
N	601	2497	
Mean maternal age, years	30.4 ± 4.9	30.1 ± 4.9	0.3
Age > 35 years	106 (17.6)	389 (15.6)	0.2
Parity	1 ± 1.4	1.1 ± 1.5	< 0.001
Nulliparity	159 (26.5)	366 (14.7)	< 0.001
Gestational age at delivery	39.9 ± 1.1	40.1 ± 1	< 0.001
Chronic HTN	1 (0.2)	4 (0.2)	0.9
Hypertensive disorders of pregnancy[a]	17 (2.8)	51 (2)	0.2
GDM	48 (8)	10 (0.4)	< 0.001
Pregestational diabetes	45 (7.5)	11 (0.4)	< 0.001
Male neonate	202 (33.6)	846 (33.9)	0.9
Cord pH	7.28 ± 0.06	7.28 ± 0.08	0.9
Cord pH < 7 @ 5 min	0 (0)	2 (0.8)	0.1
(b) Excluding scheduled cesarean sections for macrosomia (2619)			
N	127	2497	
Mean maternal age, years	30.1 ± 4.6	30.1 ± 5.0	0.9
Age > 35 years	17 (13.4)	388 (15.6)	0.5
Parity	1.1 ± 1.3	1.2 ± 1.5	0.9
Nulliparity	32 (25.2)	363 (14.6)	0.001
Gestational age at delivery	40.0 ± 1.0	40.2 ± 1.0	0.07
Chronic HTN	1 (0.8)	4 (0.2)	0.1
Hypertensive disorder of pregnancy[a]	6 (4.7)	50 (2)	0.04
GDM	9 (7.1)	9 (0.4)	< 0.0001
Pregestational diabetes	2 (1.6)	11 (0.4)	0.08
Male neonate	38 (29.9)	846 (33.9)	0.4
pH	7.27 ± 0.07	7.28 ± 0.08	0.2
pH < 7 @ 5 min	0 (0)	2 (0.8)	0.6

Numbers are mean (SD), *n* (%), or *n*/*N* (%)

HTN hypertension, *GDM* gestational diabetes

[a]Hypertensive disorders of pregnancy include one of the following: gestational hypertension, preeclampsia

a trial of labor. CS rate was more than 3.5 times higher in the group of predicted macrosomia (47.2% vs. 12.7%, $P < 0.001$) than those with unpredicted macrosomia; not only due to NPL (29.1% vs. 5.6%, $P < 0.001$), but also due to non-reassuring heart rate (NRHR) (14.2% vs. 3.9%, $P < 0.001$). Induction of labor, including all four techniques (oxytocin infusion, prostaglandin E2, Foley catheter, or artificial rupture of membranes), was also more prevalent in the predicted macrosomia group than the unpredicted macrosomia group (65.4% vs. 31.9%, $P < 0.001$) (Table 2).

Maternal outcomes are presented in Table 3a, b. PPH was less likely to occur in the predicted fetal macrosomia group than in the unpredicted fetal macrosomia group when we included all macrosomic newborns (1.7% vs. 3.2%, $P < 0.04$) (Table 3a). When we included solely women who attempted a trial of labor, we found no difference between the groups (Table 3b). The predicted macrosomia group was also more prone to episiotomy (50% vs. 31.3%, $P < 0.001$), 3rd- or 4th-degree tear (2.7% vs. 0.6%, $P < 0.05$) and a longer duration of maternal hospitalization. There was no difference in composite maternal outcome between the groups (Table 3b).

Neonatal outcomes

Neonatal outcomes are presented in Table 4a, b. When we included all macrosomic babies, we found that the mean birth weight was higher in the predicted macrosomia group (4339 ± 296 g vs. 4209 ± 214 g, $P < 0.001$) than in the unpredicted macrosomia group, along with an increased rate of hypoglycemia (2.2% vs. 0.5%, $P < 0.001$) and hospital stay. Conversely, polycythemia and partial sepsis workup were found to be less likely in the neonates with predicted fetal macrosomia than the neonates with unpredicted fetal macrosomia. Neonatal composite outcome was significantly

Table 2 Pregnancy outcome and mode of delivery in the predicted macrosomia and the unpredicted macrosomia groups, excluding scheduled cesarean sections for macrosomia (2619)

	Predicted macrosomia	Unpredicted macrosomia	*P* value
N	127	2497	
Induction of labor	83 (65.4)	795 (31.9)	< 0.001
Pitocin	67 (52.8)	712 (28.6)	< 0.001
PG E2	20 (15.7)	106 (4.3)	< 0.001
Foley catheter	9 (7.1)	21 (0.8)	< 0.001
ARM	16 (12.6)	61 (2.4)	< 0.001
Vaginal delivery	61 (48)	2095 (84.1)	< 0.001
Vacuum-assisted delivery	13 (10.2)	152 (6.1)	0.06
NRHR	6 (4.7)	80 (3.2)	0.4
PSS	7 (5.5)	72 (2.9)	0.09
Cesarean section	60 (47.2)	315 (12.7)	< 0.001
NPL	37 (29.1)	139 (5.6)	< 0.001
NRHR	18 (14.2)	97 (3.9)	< 0.001
CS duration, min	30.4 ± 15.3	28.1 ± 14.0	0.4

Numbers are mean (SD), *n* (%), or *n*/*N* (%)

PG prostaglandin, *ARM* artificial rupture of membranes, *NRHR* non-reassuring heart rate, *PSS* prolonged second stage, *NPL* non-progressive labor, *CS* cesarean section

Table 3 Maternal outcome in the predicted macrosomia and the unpredicted macrosomia groups

	Predicted macrosomia	Unpredicted macrosomia	*P* value
(a) Including scheduled cesarean sections for macrosomia (3098)			
N	601	2497	
PPH	10 (1.7)	81 (3.2)	0.04
Shoulder dystocia[a]	12 (15.8)	218 (9.7)	0.08
Manual extraction of placenta[a]	8 (10.5)	193 (8.6)	0.6
Episiotomy[a]	37 (48.7)	703 (31.3)	0.001
Oligohydramnios	3 (0.5)	43 (1.7)	0.03
Amnioinfusion	8 (1.3)	50 (2)	0.3
Meconium-stained amniotic fluid	23 (3.8)	65 (2.6)	0.1
3rd or 4th degree tear[a]	2 (0.3)	14 (0.6)	0.5
Positive GBS colonization	1 (0.2)	29 (1.2)	0.03
SBE prophylactic treatment	2 (0.3)	9 (0.4)	0.9
Maternal hospital stay, days	4.7 ± 1.3	3.3 ± 1.3	< 0.001
Composite maternal complications[b]	18 (3)	250 (10)	< 0.001
(b) Excluding scheduled cesarean sections for macrosomia (2619)			
N	127	2497	
PPH	6 (4.7)	81 (3.3)	0.4
Shoulder dystocia[a]	12 (16.2)	218 (9.7)	0.07
Manual extraction of placenta[a]	8 (10.8)	192 (8.5)	0.5
Episiotomy[a]	37 (50)	703 (31.3)	0.001
Oligohydramnios	1 (0.8)	43 (1.7)	0.4
Amnioinfusion	7 (5.5)	50 (2)	0.008
Meconium-stained amniotic fluid	11 (8.7)	65 (2.6)	< 0.001
3rd or 4th degree tear[a]	2 (2.7)	14 (0.6)	0.03
Positive GBS colonization	1 (0.8)	29 (1.2)	0.7
SBE prophylactic treatment	0 (0)	9 (0.4)	0.5
Maternal hospital stay, days	4.3 ± 1.4	3.2 ± 1.3	< 0.001
Composite maternal complications[b]	24 (18.9)	463 (17.5)	0.7

Data are *n* (%), unless otherwise indicated

PPH postpartum hemorrhage, *GBS* group B streptococcus, *SBE* subacute bacterial endocarditis

[a]Percentage out of vaginal delivery or vacuum-assisted delivery

[b]Composite maternal outcomes include at least one of the following: PPH, manual extraction of placenta, episiotomy, or third- or fourth-degree tear

lower in the study group than in the controls (32% vs. 38%, $P < 0.05$). Birth injuries and shoulder dystocia were not found to be different between the groups (Table 4a). When we excluded the elective CS due to macrosomia, mean birth weight and neonatal stay were still significantly higher in the study group, however, less distinct than before. Hypoglycemia was still more prevalent in the predicted macrosomia group. There was no difference in birth injuries or in neonatal composite outcomes between predicted and unpredicted macrosomia (Table 4b).

Association of predicted fetal macrosomia with adverse outcome: multivariable analysis

To evaluate the independent contribution of predicted fetal macrosomia to adverse outcomes, a multivariable logistic regression model was calculated. Included predictors were maternal age, parity, GDM, pregestational diabetes, hypertensive disorders of pregnancy and birth weights.

Predicted fetal macrosomia increases the risk of CS (Table 5a); however, it reduces the risk of PPH (aOR 0.5, 95% CI 0.2–1.0), maternal (aOR 0.3, 95% CI 0.2–0.5) and neonatal composite outcomes (aOR 0.7 95% CI 0.6–0.9) (Table 5a).

After analyzing a subgroup of neonatal birth weight of 4000–4500 g, we identified reduced odds of maternal (aOR 0.2, 95% 0.2–0.4), and neonatal composite outcomes (aOR 0.6, 95% CI 0.5–0.8). Conversely, we did not find an association within this subgroup between predicted fetal macrosomia and birth injuries.

When analyzing the cases where trial of labor was attempted, predicted fetal macrosomia was associated with a higher CS rate (aOR 6.4, 95% CI 4.3–9.5). However, there was no association between predicted fetal macrosomia and PPH, maternal or neonatal outcomes (Table 5b).

Table 4 Neonatal composite adverse outcome in the predicted macrosomia and the unpredicted macrosomia groups

	Predicted macrosomia	Unpredicted macrosomia	*P* value
(a) Including scheduled cesarean sections for macrosomia (2115)			
N	447	1668	
Birth weight, g	4339 ± 296	4209 ± 214	< 0.001
Neonatal hospital stay, days	4.1 ± 2.4	3 ± 2.4	< 0.001
NEC	0 (0)	1 (0.1)	0.6
Anemia	0 (0)	2 (0.1)	0.5
Thrombocytopenia	1 (0.2)	7 (0.4)	0.6
Polycythemia	6 (1.3)	62 (3.7)	0.01
Hypoglycemia	10 (2.2)	8 (0.5)	< 0.001
Cephalohematoma	1 (0.2)	15 (0.9)	0.14
Subgaleal hemorrhage	0 (0)	1 (0.1)	0.6
Birth fracture of clavicle	4 (0.9)	23 (1.4)	0.4
Erb's palsy	1 (0.2)	5 (0.3)	0.8
RDS	0 (0)	0 (0)	> 0.99
Perinatal asphyxia[a]	0 (0)	0 (0)	> 0.99
Assisted ventilation	0 (0)	0 (0)	> 0.99
Neonatal resuscitation	0 (0)	0 (0)	> 0.99
TTN	7 (1.6)	20 (1.2)	0.5
Convulsions	1 (0.2)	2 (0.1)	0.6
Meconium aspiration	0 (0)	4 (0.2)	0.3
Pneumonia	0 (0)	0 (0)	> 0.99
Partial sepsis workup[b]	2 (0.4)	32 (1.9)	0.03
Birth injuries[c]	5 (1.1)	43 (2.6)	0.06
Composite neonatal complication[d]	143 (32)	635 (38)	0.02
(b) Excluding scheduled cesarean sections for macrosomia (1767)			
N	103	1663	
Birth weight, g	4272 ± 207	4209 ± 214	< 0.0001
Neonatal hospital stay, days	3.6 ± 1.4	3.0 ± 2.4	< 0.0001
NEC	0 (0)	1 (0.1)	0.8
Anemia	0 (0)	2 (0.1)	0.7
Thrombocytopenia	1 (1)	7 (0.4)	0.4
Polycythemia	5 (4.9)	62 (3.7)	0.6
Hypoglycemia	4 (3.9)	8 (0.5)	< 0.0001
Cephalohematoma	1 (1)	15 (0.9)	0.9
Subgaleal hemorrhage	0 (0)	1 (0)	0.8
Birth fracture of clavicle	3 (2.9)	23 (1.4)	0.2
Erb's palsy	1 (1)	5 (0.3)	0.3
RDS	0 (0)	0 (0)	> 0.99
Perinatal asphyxia[a]	0 (0)	0 (0)	> 0.99
Assisted ventilation	0 (0)	0 (0)	> 0.99
Neonatal resuscitation	0 (0)	0 (0)	> 0.99
TTN	1 (1)	20 (1.2)	0.8
Convulsions	1 (1)	2 (0.1)	0.04
Meconium aspiration	0 (0)	4 (0.2)	0.6
Pneumonia	0 (0)	0 (0)	> 0.99
Partial sepsis workup[b]	0 (0)	32 (1.9)	0.2
Birth injuries[c]	4 (3.9)	43 (2.6)	0.4
Composite neonatal complication[d]	49 (47.6)	719 (43.2)	0.4

Data are *n* (%), unless otherwise indicated

OR odds ratio, *NEC* necrotizing enterocolitis, *RDS* respiratory distress syndrome, *TTN* transient tachypnea

Table 4 (continued)

of newborn

[a]Perinatal asphyxia: pH < 7 and base deficit ≥ 12 mmol/L at birth in a newborn exhibiting early signs of moderate or severe encephalopathy [11]

[b]Partial sepsis workup, including blood culture and blood count, were done based on the APP guidelines [18]. Further sepsis evaluation, including lumbar puncture (LP) and urine culture, was done based on the clinical impression of the medical team

[c]Birth injuries include at least one of the following: cephalohematoma, subgaleal hematoma, clavicle fracture, or Erb's palsy

[d]Composite neonatal outcomes include at least one of the following: shoulder dystocia, cord blood pH < 7, convulsions, hypoglycemia, anemia, polycythemia, clavicle fracture, Erb's palsy, cephalohematoma, subgaleal hemorrhage, TTN, or RDS

Table 5 Association of predicted macrosomia (vs. macrosomia not predicted) with adverse maternal and neonatal outcome: multivariable analysis

	Predicted macrosomia Adjusted OR (95% CI)
(a) Including scheduled cesarean sections for macrosomia (3098)	
PPH	0.5 (0.2–1.0)
Cesarean section	52.4 (38.9–70.7)
Shoulder dystocia[a]	1.5 (0.8–3.0)
Composite maternal complications[b]	0.3 (0.2–0.5)
Birth injuries[c]	0.5 (0.2–1.2)
Composite neonatal outcome[d]	0.7 (0.6–0.9)
(b) Excluding scheduled cesarean sections for macrosomia (2619)	
PPH	1.3 (0.5–3.2)
Cesarean section	6.4 (4.3–9.5)
Shoulder dystocia[a]	1.6 (0.8–3.2)
Composite maternal complications[b]	1.1 (0.7–1.7)
Birth injuries[c]	1.5 (0.5–4.4)
Composite neonatal outcome[d]	1.1 (0.7–1.7)

OR odds ratio, *CI* confidence interval, *PPH* postpartum hemorrhage, *GDM* gestational diabetes mellitus, *TTN* transient tachypnea of newborn, *RDS* respiratory distress syndrome

[a]Percentage out of vaginal delivery or vacuum-assisted delivery

Values reflect the results of a multivariable logistic regression analysis and are adjusted for age, parity, pregestational diabetes, GDM, and birth weight

[b]Composite maternal outcomes include at least one of the following: PPH, manual extraction of placenta, or 3rd or 4th degree tear

[c]Birth injuries include at least one of the following: cephalohematoma, subgaleal hematoma, clavicle fracture, or Erb's palsy

[d]Composite neonatal outcomes include at least one of the following: shoulder dystocia, cord blood pH < 7, convulsions, hypoglycemia, anemia, polycythemia, clavicle fracture, Erb's palsy, cephalohematoma, subgaleal hemorrhage, TTN, or RDS

The association of predicted fetal macrosomia with maternal and neonatal adverse outcomes was then recalculated within a subgroup of nulliparous women. We could demonstrate similar results (Table 6a, b).

Table 6 Association of predicted macrosomia (vs. macrosomia not predicted) with adverse maternal and neonatal outcome in a subgroup of nulliparous women: multivariable analysis

	Predicted macrosomia Adjusted OR (95% CI)
(a) Including scheduled cesarean sections for macrosomia (525)	
PPH	0.3 (0.08–1.5)
Cesarean section	30.5 (14.7–63)
Shoulder dystocia[a]	1.4 (0.2–12)
Composite maternal complications[b]	0.2 (0.06–0.6)
Birth injuries[c]	0.2 (0.01–2.0)
Composite neonatal outcome[d]	0.6 (0.4–1.0)
(b) Excluding scheduled cesarean sections for macrosomia (395)	
PPH	0.7 (0.1–5.3)
Cesarean section	5.6 (2.4–13.2)
Shoulder dystocia[a]	1.6 (0.2–13.9)
Composite maternal complications[b]	0.7 (0.2–2.3)
Birth injuries[c]	0.6 (0.05–8.4)
Composite neonatal outcome[d]	0.8 (0.4–1.8)

OR odds ratio, *CI* confidence interval, *PPH* postpartum hemorrhage, *GDM* gestational diabetes mellitus, *TTN* transient tachypnea of newborn, *RDS* respiratory distress syndrome

[a]Percentage out of vaginal delivery or vacuum-assisted delivery

Values reflect the results of a multivariable logistic regression analysis and are adjusted for age, parity, pregestational diabetes mellitus, GDM, and birth weight

[b]Composite maternal outcomes include at least one of the following: PPH, manual extraction of placenta, or 3rd or 4th degree tear

[c]Birth injuries include at least one of the following: cephalohematoma, subgaleal hematoma, clavicle fracture, or Erb's palsy

[d]Composite neonatal outcomes include at least one of the following: shoulder dystocia, cord blood pH < 7, convulsions, hypoglycemia, anemia, polycythemia, clavicle fracture, Erb's palsy, cephalohematoma, subgaleal hemorrhage, TTN, or RDS

Discussion

Principal findings of the study

In this study we compared management and outcomes

of macrosomic neonates, of which one group included neonates which were predicted to be macrosomic versus neonates who were not known to be macrosomic in the other group. Our main findings were as follows: (1) the prediction of a macrosomic fetus itself is associated with increased odds of CS, either planned or intrapartum; (2) birth injuries and shoulder dystocia were not found to be different between the groups; (3) PPH may be reduced by a planned CS when fetal macrosomia is predicted; (4) maternal and neonatal composite outcome can also be minimized by an elective CS when macrosomia is suspected; (5) predicted fetal macrosomia is associated with higher rates of induction of labor; and (6) predicted fetal macrosomia is associated with higher rates of episiotomy, and 3rd- or 4th-degree tears.

Looking at our cohort, it seems that expecting a big baby affects our management, and this have been shown in other studies as well [17, 25, 27]: not only by scheduling a planned CS more prominently, but also by performing more episiotomies and CS during labor. Those findings were present even after adjusting for confounders, including birth weight. We can only speculate that the anticipation of a macrosomic infant may have influenced the intrapartum management, although we cannot exclude the possibility that other variables have also had some influence.

The general consensus among experts is that a planned CS is indicated in some clinical settings to reduce the possibility of shoulder dystocia and related neonatal morbidity [12]. As opposed to Vendittelli et al. [25] who demonstrated the neonatal traumatic injury to be 1.8 times higher in the predicted macrosomia group, we did not demonstrate a difference in shoulder dystocia or birth injuries in our study between the groups, even when we included all macrosomic neonates, though these results should be interpreted with caution due to a small number of cases. Furthermore, the care provider might not have the ability to prevent shoulder dystocia even when anticipating a big baby. It has been well known that more than half of pregnancies complicated by shoulder dystocia have no identifiable risk factors [12].

Fetal macrosomia is known to be associated with maternal adverse outcomes, including PPH [12]. By scheduling a planned CS for women with predicted fetal macrosomia, we may have inadvertently reduced the rate of PPH. We could not demonstrate that finding when we separately analyzed the group of women who attempted a trial of labor. This may be explained by the fact that in our hospital, the common practice is that a nurse-midwife delivers the baby unless she encounters any difficulties, at which point a physician is called to assist. Nurse-midwives and physicians may manage their cases differently. It could also be explained by the fact that surgeons, as it has been shown in previous studies [22, 23], underestimate the blood loss in the CS, or that they take additional steps in order to reduce the risk of PPH, steps which may not be taken in a vaginal delivery by nurse-midwives.

We also presented lower odds of maternal and neonatal outcomes by scheduling those planned CS, a difference that was not present in the attempted trial of labor group when it was separately analyzed. These findings have been supported in the literature [1, 9, 21], though we have presented those outcomes even for babies weighting between 4000 and 4500 g.

Induction of labor and macrosomia is still a matter of debate. As long as we do not have unified guidelines, care providers will use their clinical judgment, experience, and maybe even common practice within their institution to decide whether they should induce their patients with predicted fetal macrosomia to avoid additional fetal growth or manage them expectantly.

One of the main roles of a physician is to facilitate the women we care for in the decision-making process. Care providers are strongly encouraged to share information and have an open communication with their patients. Pregnant women with predicted fetal macrosomia should be provided individualized counseling about the risks and benefits of vaginal and cesarean delivery based on the degree of macrosomia.

Strength and limitations

Given the retrospective design of this study, some information was not available. In addition, this study is powered insufficiently to assess rare outcomes. Furthermore, the frequency of adverse outcomes that has been reported was based on actual neonatal weights, not the estimated fetal weights on which decisions would need to be made. Nonetheless, our study included a relatively large cohort of women with macrosomic neonates demonstrating a different aspect of fetal macrosomia and its effect on care provider management. This management may affect maternal and neonatal outcomes.

Conclusion

In summary, we have found an association between anticipation of a macrosomic newborn and the rate of an antepartum CS, and not only due to NPL. We also demonstrated that by scheduling a planned CS for predicted fetal macrosomia, we can reduce the rate of PPH, maternal and neonatal outcome, even for babies with a mean birth weight of 4000–4500 g.

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Compliance with ethical standards

Conflict of interest All authors declare no conflict of interests. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. IRB was approved on 28th April 2015. Number of approval was 0044-15-BNZ.

Informed consent Due to the retrospective nature of the study, there was no need for informed consent.

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