Suspected fetal macrosomia and the risk of shoulder dystocia as an indication for cesarean section

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Abstract

Macrosomia is associated with increased risk for perinatal complications including prolonged labor, shoulder dystocia, perinatal mortality and maternal morbidity including cesarean delivery, severe postpartum hemorrhage, and vaginal lacerations. Among maternal risk factors for fetal macrosomia are high body-mass index, diabetes mellitus, postterm pregnancy, previous macrosomic infant. Shoulder dystocia occurs in 0.2-3% of all births and represents an obstetric emergency. Although, in general, clinical estimates of birth weight perform favorably, ultrasound immediately prior to labour is more accurate at predicting the high birth-weight fetus. Ultrasound measurement of abdominal circumference and fetal biometry are the only practical methods used to detect fetal weight over 4000 g, but they are characterized by low sensitivity, low positive predictive value and high negative predictive value. Serial sonographic measurements can increase the positive predictive value. By combining three-dimensional volumetric measurements with two-dimensional measurements, should increase the ability to predict macrosomia. Nowadays, clinicians needs to examine all the information available to take a decision on whether the risk of macrosomia and shoulder dystocia is high, and if so then an elective caesarean section is indicated. Predicting fetal macrosomia does not imply that elective caesarean section is the method of choice, but it should be made clear to the couple that elective caesarean section is the low-risk option. Important to note is that each woman should be informed of the particular risks associated with a macrosomic fetus and shoulder dystocia. The mother should be supported in the decision, because it is a situation in which maternal autonomy is paramount.

Keywords: *macrosomia, shoulder dystocia, cesarean section*

Introduction

Macrosomia refers to growth beyond a specific threshold[1]. In developed countries, the most common thresholds that have been proposed are weight above 4000 g or 4500 g[2,3]. A grading system has also been suggested: grade 1 for infants 4000 to 4499 g, grade 2 for 4500 to 4999 g, and grade 3 for over 5000 g[4]. The American College of Obstetricians and Gynecologists supports use of the 4500 g threshold for diagnosis of macrosomia because morbidity increases sharply beyond this weight, but acknowledges there is some increased risk of morbidity at weights >4000 g[5]. Such a birth weight is associated with an increased risk for maternal morbidity and a number of perinatal complications including prolonged labor, shoulder dystocia with brachial palsy, facial nerve palsy, fractures of the clavicular and humeral bones, perinatal mortality, asphyxia[6,7] and cesarean section. Delivery of large fetuses is still a source of anxiety among obstetricians, despite major progress in obstetrics in the last century. The worldwide prevalence of birth of infants ≥4000 g is about 9 percent and about 0.1 percent of newborns weigh ≥5000 g, with wide variations among countries[8]. The prevalence of birth weight ≥4000 g in developing countries is typically 1 to 5 percent, but ranges from 0.5 to 14.9 percent[9]. In population statistics, normal weight is defined as between the 10th and 90th percentile for gestational age (i.e. assuming a normal population distribution). Using a statistical approach, any fetus weighing >90th percentile for gestational age would be considered large for gestational age (LGA). The use of country-specific centiles may be the best approach, particularly in the developing world, since it accounts for differences between populations[10]. The 95th and 97.75th percentiles have also been used as thresholds. Weight percentile for gestational age is the best means for identifying the preterm or term macrosomic fetus.

About 70 percent of infants with birth weight over 4500 grams are male[11]. Racial and ethnic differences influence birth weight. The proportion of newborns with birth weight >4000 g has increased during the past two decades, in parallel with an increasing prevalence of maternal body mass index (BMI)≥25 kg/m^2 related to alimentary habits and with an increase in maternal age[12]. Although the fetal genome is the central controller of growth in an uncomplicated pregnancy[13], maternal clinical characteristics and fetal gender are associated with fetal growth[14,15]. Both transient and permanent fetal and maternal injuries are seen as a consequence of delivering a large fetus, and for the neonate it might result in impairment to health later in life[16].

Macrosomia occurs in approximately 42-62% of pregnancies complicated by Type-1 diabetes mellitus (DM1)[17-19], in 30–56% of pregnancies complicated by Type-2

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diabetes mellitus (DM2)[20] and in 10-20% of pregnancies complicated by gestational diabetes mellitus (GDM)[21]. In women with DM1 and DM2, indicators of poor placentation in early pregnancy are related to normal birth weight, and indicators of normal placentation to increased birth weight (i.e. fetal overgrowth in both instances)[22]. The growth pattern of fetuses of women with diabetes, especially when glycemic control has been poor, is different from that in fetuses of nondiabetic mothers[23]. There is an altered fetal growth in macrosomic and non-macrosomic fetuses of women with DM1, DM2 and GDM. Growth profiles differ among these groups, with the most prominent growth deviations in fetuses of women with DM1, probably due to poor glucose control. Maternal hyperglycemia due to diabetes leads to increased secretion of insulin by the fetus and muscle growth, deposition of excess fat and organomegaly in the fetus. Previous studies on fetal macrosomia have confirmed that preconception and first-trimester glucose control has the greatest effect on fetal size[24]. Macrosomic infants of diabetic mothers have increased mass, larger shoulders and greater amounts of body fat, decreased head-to-shoulder ratio, more muscle growth in the inter-scapular areas and abdomen after 32 weeks and increased skin folds in the upper extremities[25]. Increased fetal size starts from the second trimester onwards, and that the difference in size persists despite improvements in diabetic control[26]. Several studies have used this information in an attempt to predict the risk of shoulder dystocia in diabetic pregnancies, but no method has proven to be reliable until present[27].Accelerated mid trimester growth in abdominal circumference (AC) was often associated with the birth of a heavy or LGA baby, and poor maternal glycemic control (HbA1c >7.0%) in early pregnancy was a modest predictor of both. These morphologic differences are responsible for substantially higher rates of shoulder dystocia among macrosomic fetuses of diabetic pregnancies compared to macrosomic fetuses of non-diabetic pregnancies[28].

Higher maternal age and obesity are other risk factors for excessive fetal growth. In 2004, a large review of maternal and neonatal data in the USA[29] showed that the statistical association between GDM and LGA infants strengthened as pre-gravid BMI increased, thus suggesting that maternal overweight was an independent risk factor for LGA newborns among those patients. An increase in BMI 25% during pregnancy has a sensitivity of 86.2%, specificity of 93.6%, positive predictive value of 71.4% and negative predictive value of 97.45% for macrosomia[30]. There is a strong association between waists to hip ratio and the delivery of a macrosomic newborn independent of BMI. This association suggests that central adiposity may be linked with the mechanism leading to macrosomia in the newborn[31].Women with a history of one macrosomic infant are at significantly increased risk of another macrosomic infant in a subsequent pregnancy. For women with two or more macrosomic infants, the risk is even greater[32,33].

Shoulder dystocia occurs in 0.2 to 3 percent of all births and represents an obstetric emergency. It occurs when the shoulders fail to traverse the pelvis after delivery of the head. The problem lies at the pelvic inlet. In most cases, the posterior shoulder enters the pelvis but the anterior shoulder remains lodged above the symphysis pubis[34]. Few shoulder dystocia can be anticipated and prevented, as most occur in the absence of risk factors. Brachial plexus injury is one of the most serious fetal complications, and occurs in 2 to 16 percent of shoulder dystocia. Most cases could be resolved, but up to 30 percent result in permanent neurologic impairment[35].

Among fetal factors, LGA status is generally considered the most predictive of shoulder dystocia. The incidence of shoulder dystocia increases progressively as birth weight increases over 4000 g[36], and morbidity and mortality from shoulder dystocia increase significantly when birth weight is ≥4500 g[37]. Although birth weight is a risk factor for shoulder dystocia, it is not highly useful for predicting its occurrence because:

- The majority of extremely high birth weight infants do not have shoulder dystocia. It was reported in only 15.5 percent of 7859 infants with birth weight ≥5000 g delivered vaginally in one series[38].
- Approximately 50 percent of shoulder dystocia occur in infants with birth weight <4000 g[11].
- It is difficult to predict birth weight prior to delivery. The sensitivity of ultrasonographic examination to detect fetal weight >4500 g ranged from 22 to 69 percent only in one study[39]. Clinical estimates of birth weight based upon Leopold's maneuvers are also insensitive.

Among maternal risk factors, diabetes in pregnancy is considered the most significant risk factor[40]. The likelihood of shoulder dystocia increases several fold over the non-diabetic population, due, in part, to the higher prevalence of macrosomia in women with diabetes compared with non-diabetic women[11]. The chest-to-head and shoulder-to-head ratios are increased in DM, thereby increasing the risk of shoulder dystocia independent of fetal weight and at weights <4000 g[8]. Even a single abnormal glucose value in a 75 g two-hour glucose tolerance test is associated with adverse pregnancy outcome, including macrosomia and shoulder dystocia[2].

Shoulder dystocia recurs in 1-25 percent of subsequent pregnancies in retrospective studies[41]. This may be underestimated since many patients and clinicians choose an abdominal delivery in pregnancies subsequent to an episode of shoulder dystocia. The combination of a previous shoulder dystocia and LGA is particularly worrisome[42]. Of note, the absence of shoulder dystocia in a previous pregnancy does not preclude its occurrence in a subsequent pregnancy[43].

A relationship between shoulder dystocia and abnormal labor progress, including both precipitous and prolonged second stage, has been reported at both high and average birth weights, but data are inconsistent[44] because of the high frequency of labor abnormalities in the general obstetrical population and the relatively low frequency of shoulder dystocia[45].

Post-term pregnancy is a risk factor for shoulder dystocia, presumably because of higher birth weights with

advancing gestational age. In a cohort study of term (n = 379.445) and post-term (n = 65.796) births from Norway, the relative risk of shoulder dystocia in post-term births was increased by 30 percent (RR 1.3, 95% CI 1.2-1.4)[46].

Male gender is more common in pregnancies complicated by shoulder dystocia than in the overall birth population (55 to 68 percent versus 51 percent)[47]. Several fetal biometric parameters (i.e. difference between the fetal abdominal and biparietal diameters, chest circumference, humerospinous distance, cheek-to-cheek diameter, and shoulder width) have been used to predict shoulder dystocia, primarily in fetuses of diabetic gravidas[48]. The value of this approach has not been tested in large prospective studies. However, most often shoulder dystocia presents without any identifiable prior risk factors and over 90% of the cases of shoulder dystocia occur in babies weighing less than 4500 g[49]. For predicting shoulder dystocia, risk factors have a limited use. Currently, the one most used is estimated fetal weight, but the positive predictive value of macrosomia alone is only 3.3%[50,51].

LGA infants are more likely to develop respiratory distress than AGA infants[52], due to the increased risk of respiratory distress syndrome in infants of diabetic mothers who are more likely to be delivered prematurely. In a report based upon data from the Netherlands Perinatal Registry from 1997 to 2002, the incidence of hypoglycemia was about 19 percent in all LGA infants and 15 percent in LGA infants of nondiabetic mothers[53].

It was seen that polycythemia occurs more frequently in LGA infants of both diabetic and nondiabetic mothers compared with AGA infants[54].

Ultrasound assessment

Fetal macrosomia is a continuing challenge in obstetrics practice. Attempts at the prenatal diagnosis have proven difficult with many series reporting a positive predictive value of only 50%. Sonography is most predictive, albeit not highly accurate, even when performed near term in singleton, cephalic presenting, non-diabetic pregnancies. Performing a single estimation at 29 to 34 weeks of gestation has very poor predictive value for birth weight at term. At this time can significantly underestimate birth weight, probably because of accelerated growth in the later part of the third trimester[55].

AC is the most common and reliable single parameter used to assess risk of macrosomia[56]. It is measured on a defined plane incorporating the liver since growth abnormalities are often reflected by changes in liver size[57]. The most commonly used thresholds for prediction of macrosomia are ACs of 35 to 38 cm[58]. The sensitivity of the AC measurement depends upon the cut-off chosen, definition of macrosomia, and gestational timing of the examination. The AC measurement is equally accurate whether determined in two dimensions or by an elliptical estimate[59].

Ultrasound biometry, the only practical method used to detect fetal weight over 4000 g, is characterized by low sensitivity, low positive predictive value, high negative predictive value[60] and inherent inaccuracies, with large intra- and inter-observer variability[11]. Studies at term and intrapartum give a 6-11% mean absolute error when compared with the actual birth weight[61]. Since the fetus is an irregular, three-dimensional structure of varying density, the ability of formulas to predict fetal weight has been limited, without good sensitivity and specificity. In addition, sonographic measurement does not permit differentiation between pathologically large and large but healthy infants[62].

Most commonly, a combination of biparietal diameter (BPD), head circumference (HC), AC, and femur length (FL) is used. The most popular formulas are Hadlock's[63] and Warsof's(64) with Shepard's modification[65]. Comparisons of these formulas concluded that the formula using BPD, FL and AC (second Hadlock formula) resulted in the best estimate of fetal weight, while the formula using only BPD and AC (Shepard formula) had the least accurate estimate[66]. Formulas for estimating fetal weight perform better for normal sized fetuses than for macrosomic ones[8,67]. The majority of studies conducted to date used a fixed threshold (i.e. estimated fetal weight >4000 g or 4500 g) to compare the accuracy of different models for the detection of macrosomia, a threshold which, as described earlier, does not necessarily represent the optimal threshold for this purpose[68]. O'Reilly-Green and Divon found that the optimal threshold for the detection of macrosomia using the model of Hadlock and contributords was 3711 g rather than 4000 g[69]. As a result, comparison of models using such a fixed threshold may not reflect the true relative accuracy of the different models. A systematic review that compared the accuracy of sonographic (16 different formulas with various combinations of BPD, HC, AC, and FL) and AC in the prediction of macrosomia analyzed 63 studies included 19.117 women[70].

A rounder fetal head, quantified by a small occipito frontal diameter (OFD) and normal HC (with a larger BPD/OFD ratio), may contribute to shoulder dystocia by either transiting the pelvis more quickly than a more oval-shaped head, or failing to rotate such that the fetal shoulders incorrectly present at the pelvic inlet[71].

Estimating fetal weight by ultrasonographic measurement prior to induction of labor could potentially be even more problematic owing to the low position of the head and an increased risk of abdominal circumference distortion or posterior position of the femur at this late gestation[72,73]. Similarly, including maternal weight in weight-estimation formulas also improves the accuracy, to a mean absolute percentage error of 3.69 percent with 97.1 percent of fetuses within 10 percent of actual birth weight[74,75]. However, predictions of macrosomia by these techniques are limited by the substantial false-positive and false-negative rates inherent in these tests[76].

The majority of sonographic formulas do not take body composition into account. Because body composition can vary greatly, even in the fetus, significant variation in birth weight can occur among fetuses with similar biometric parameters. Body fat accounts for 14 percent of the birth weight in neonates, but 46 percent of birth weight variance[77]. Ultrasound has been used to assess subcutaneous

fat at the mid humerus[78], shoulder[79], abdominal wall[80], thigh[81], and peribucal area[82] to provide better evaluation of normal and disturbed growth[77]. Combinations of soft tissue measurements or other parameters (i.e. umbilical cord cross section or amniotic fluid volume) with estimation of fetal weight (EFW) may be more useful for predicting macrosomia than any method alone[82,83].

The sonographic measurements described above estimate weight using two-dimensional principles on a three-dimensional subject. Improvements in imaging technologies have helped alleviate this problem, leading to better weight estimation.Volumetric measurement by two-dimensional ultrasound can be calculated using the formula: EFW = (0.23718 X AC2 X FL) + (0.03312 X HC3). When compared to the traditional calculation of EFW using Shepard or Hadlock formulas, this method had fewer systematic and absolute errors (mean percent error was 6.2)[84].

Three-dimensional ultrasound has heightened interest in using volumetric estimates to predict fetal macrosomia. Validation studies for EFW showed similarities between three-dimensional and two-dimensional measures in systemic error measurements of fetal thigh, AC and estimated weight precision[85]. Most predictions were within 10 percent of true birth weight. Subsequent studies incorporated measurement of single parameters, such as the fetal upper arm[86] and thigh[87], which also correlated well to birth weight. Better qualitative analysis of fetal soft tissue may be possible with three-dimensional ultrasound, allowing for improved estimation of actual birth weight[88].The best ability to predict macrosomia comes from combining three-dimensional volumetric measurements (i.e. volume of upper arms, thigh and abdomen) with two-dimensional measurements (formula = -1478.557 + 7.242 X thigh vol +13.309 X upper arm vol + 852.998 X log10 AC vol + 0.526 X BPD3)[89]. With combined measurements, the mean absolute percentage of error was 6.5 percent versus 10 to 15 percent with two-dimensional alone.

Magnetic resonance imaging (MRI) should be, in theory, a superior technique for evaluation of macrosomia because it evaluates fat better than ultrasound[90]. Some results have been encouraging. In one study, the median difference between MRI-derived EFW and actual birth weight was 3 percent, as opposed to 6.5 percent for ultrasound-derived EFW[91]. In addition, a small, but well designed, study demonstrated a significant correlation between MRI prediction of fetal shoulder measurements of fetuses with suspected macrosomia and the actual shoulder width[92].

Sonographic EFW has been combined with results of the glucose challenge test to better predict macrosomia. When sonographic EFW was >4000 g, the presence of a glucose challenge test (GCT) ≥120 mg/dL had a positive predictive value of 71 percent for macrosomia compared to 60 percent with GCT ≤120 mg/dL[93]. The mean absolute percent error is greater in infants weighing above 4500 g (12.6 versus 8.4 percent if below 4500 g), regardless of diabetic status[94].

A study comparing three EFW formulas using multiple parameters versus prediction of birth weight by AC alone concluded that measurement of AC was quicker and similarly accurate. All formulas were associated with an error of ± 20 to 25 percent[95]. Another study reported that AC >70 percentile is predictive of poor glycemic control and increased risk of macrosomia[96]. Based on these findings, the American Diabetes Association recommended the use of AC >75th percentile as a measure of glycemic control and risk for macrosomia in diabetic gravidas[97].

Some investigators have combined ultrasonography with pregnancy-specific data (i.e. parity, ethnicity, BMI, maternal height, weight and weight gain) to create nomograms for detecting fetal macrosomia, but these methods have not performed well consistently[98-100]. Mazouni and contributors[99] published a nomogram for individual prediction of macrosomia (birth weight>4000 g) based on maternal characteristics and the presence or absence of an EFW≥4000 g at ultrasound examination performed within 1week of delivery. Their formula was found to be superior to the four Hadlock formulae for EFW. In contrast to the results of Nahum and Stanislaw, Ben-Haroush and contributors[101] did not find any evidence that the prediction of LGA at birth could be improved by adding clinical information to the ultrasonically EFW. The longer the period between ultrasound examination and delivery, the more time there is for maternal factors to influence fetal growth[102].

The 39-week scan being performed on a high risk population with a higher prevalence of large fetuses would therefore be more likely to achieve clinically useful, especially if a targeted formula for macrosomia and the latest ultrasound three-dimensional volumetric studies or even MRI were utilized[85,102].

Clinical assessment

Fetal weight can be estimated clinically by palpation of the fetus through the maternal abdomen (i.e. Leopold maneuvers) and/or by measurement of fundal height. The capacity for antepartum diagnosis of fetal macrosomia in the general obstetrical population by clinical means is limited, but is somehow better in patients at higher risk. Symphyseal-fundal height varies with gestational age and maternal characteristics and the combination of these two parameters gives improved EFW than when symphyseal-fundal height alone is considered[99]. Although, in general, clinical estimates of birth weight perform favorably, ultrasound immediately prior to labor is more accurate at predicting the low- or high-birth weight fetus[72]. All women thought to be at risk should be delivered in a fully-equipped maternity unit[103].

Planning delivery in pregnancies at risk

Patient safety is about minimizing error and preventing harm. Reasons for errors include human fallibility, medical complexity, system deficiencies, and defensive barriers. Medical errors do not spare obstetric population and it is likely that strategies to reduce these errors would benefit pregnant women and their children[104].

If there is delay in the late active phase of labour (7-10 cm) or failure of descent in the second stage, careful evaluation is advised before vaginal delivery is allowed. If there is evidence of fetal hypoxia or the possibility of mid-pelvic extraction, cesarean section may be indicated. Clinical judgment and appropriate caution should replace any hard-and-fast rules requiring caesarean delivery[105]. The clinical approach that has evolved is to attempt identification of those pregnancies most likely to result in shoulder dystocia with long-term complications and then avoid vaginal delivery of them. The American College of Obstetricians and Gynecologists Task Force on Neonatal Brachial Plexus Palsy, identified the following clinical situations as high risk for shoulder dystocia and brachial plexus injury[106]:

- Estimated fetal weight >5000 g in women without diabetes or >4500 g in women with diabetes
- Prior shoulder dystocia, especially with a severe neonatal injury
- Mid pelvic operative vaginal delivery of a fetus with estimated weight >4000 g

Cesarean delivery in these scenarios is a reasonable option as it should reduce the occurrence of shoulder dystocia and associated morbidity. In fact, brachial plexus injury has been reported even after caesarean delivery. Thus, cesarean delivery reduces but does not eliminate the risk of birth trauma associated with macrosomia[107]. A policy of routine prophylactic cesarean delivery for all cases of suspected high birth weight would likely result in many unnecessary cesarean births of both normal weight and high birth weight neonates[108]. Even if newborns weighing over 4000 g could have been accurately predicted, routine cesarean section would have prevented just 57% of the shoulder dystocia cases but not one permanent injury[109].

The medical literature does not support elective cesarean section for suspected fetal macrosomia in non-diabetic women[110]. A woman with a height >175 cm has a significantly lower chance of shoulder dystocia and trial of labor might be chosen. No pressure should be placed on the couple and, once they have decided, there is no need to revisit the issue unless the couple requests it. The couple should be supported in the decision they make. This is a situation in which maternal autonomy is paramount. For babies weighing 4.5 kg or more, the emergency cesarean section rate is 45%[111] and the instrumental delivery rate 19%[112].

Induction of labour does not improve outcomes in the setting of suspected fetal macrosomia and may increase cesarean deliveries. Induction of labour in women with diabetes in pregnancy have been showed to reduces fetal macrosomia[113].

Conclusions

In the light of the worldwide obesity epidemic, perinatal complications caused by fetal macrosomia will be an increasing phenomenon in the near future. Early identification of fetuses at risk for macrosomia will therefore be an issue of increasing importance in obstetrics. These women could then be offered extra ultrasound examinations enabling fetal growth follow-up, possibly employing more sophisticated techniques such as three dimensional ultrasound, and individual planning for time and mode of delivery. Such a clinical protocol might prevent perinatal complications due to fetal macrosomia, and thus benefit both the mother and the neonate. ■

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