OPEN ACCESS

GAVIN PUBLISHERS

Journal of Diabetes and Treatment

Morrison JC and Magann EF. J Diabetes Treat: JDBT-152.

Short Commentary

DOI: 10.29011/ 2574-7568. 000052

Gestational Diabetes, Macrosomia and Shoulder Dystocia: A Short Commentary

John C. Morrison[1*], Everett F. Magann[2]

[1]Department of Obstetrics and Gynecology, University of Mississippi Medical Center, Jackson, Mississippi, USA

[2]Department of Obstetrics and Gynecology, University of Arkansas for the Medical Sciences, Little Rock, Arkansas, USA

[*]**Corresponding author:** John C. Morrison, Department of Obstetrics and Gynecology, University of Mississippi Medical Center, 2500 North State Street, Jackson, Mississippi 39216, USA. Tel: +16016682024; Email: jmorrison@umc.edu

Citation: Morrison JC, Magann EF (2018) Gestational Diabetes, Macrosomia and Shoulder Dystocia: A Short Commentary. J Diabetes Treat: JDBT-152. DOI: 10.29011/ 2574-7568. 000052

Received Date: 04 April, 2018; **Accepted Date:** 23 April, 2018; **Published Date:** 01 May, 2018

Abstract

Gestational Diabetes Mellitus (GDM) frequently complicates pregnancy, is very costly for the healthcare system/patient and causes catastrophic outcomes is some cases. In obstetric practices across the world, there are screening tests for GDM and if abnormal, diagnostic tests are employed. These pathways are very important as women with GDM having meticulous patient care results in euglycemia and outcomes for the mother, fetus, newborn can approach those without GDM. Particularly in cases where the glucose excursions are not well controlled (and even in some cases where it is) fetal macrosomia can occur and this can lead to an excess of abdominal births, shoulder dystocia with attendant brachial plexus injury as well as other newborn issues such as respiratory distress syndrome hyperbilirubinemia hypoglycemia and even brain damage. However, since most of the cases of shoulder dystocia involve birth weights that are not macrosomic, providers need to be prepared to appropriately deal with such cases as this will reduce the number of permanent brachial plexus injuries unless it has occurred in utero.

Commentary

Gestational Diabetes Mellitus (GDM) is a common, costly, and occasionally catastrophic complication of pregnancy for the mother, fetus, newborn and child. There were over 4 million births in the United States in 2014 and nearly 215,000 (approximately 17%)were complicated by GDM [1]. This ranks GMD, higher than hypertension, infection and a bit lower than preterm delivery among the most common complications affecting pregnancy. GDM is also costly as it involves screening every pregnant woman for this disorder and further diagnostic testing of those who fail the initial assessment. When GDM is diagnosed, intensive dietary and exercise instructions are needed as well as point of care glucose testing in the home (fasting and after each meal). Also, women with GDM require more frequent prenatal visits and additional ultrasound scans for growth assessment as well as numerous fetal health assessment tests (NST or BPP) later in pregnancy [2]. Treatment with oral hypoglycemic agents and/or insulin therapy may be necessary to achieve maternal euglycemia during gestation. This intensive diagnostic and treatment pathway is valuable as those with GDM demonstrate near normal pregnancy outcomes for mother and baby if maternal glucose is tightly controlled (fasting glucose <100 mg/dl (5.6mmol/L), 2 hours postprandial <120mg/dl (6.7 mmol/L) during pregnancy [3] (Table 1).

Diagnosis*
Universal one-hour screening glucose 24 - 28 weeks (>140 mg/dl [7.8 mmol/L])
Diagnostic three-hour glucose tolerance test (≥2 abnormal values)
Education/Diet/Exercise/Home Glucose Testing:
Achieve Euglycemia
<100 mg/dl (5.6 mmol/L) - fasting glucose
<120 mg/dl (6.7 mmol/L) - 2 Hour Postprandial Glucose
More Frequent Prenatal Visits
Additional Ultrasound Scans
Fetal Health Assessment Tests
Non-Stress Test (NST)
Biophysical Profile (BPP)
Consideration of:

Oral Hypoglycemic Agents
Insulin
*United States paradigm above; Europe, Australia and other countries test with hemoglobin A1c at first prenatal visit and use a 75g oral GTT as a diagnostic test (one abnormal value)

Table 1: Management of GDM.

If these criteria for glucose control are not met, lethal birth anomalies, stillbirth, macrosomia, shoulder dystocia, birth trauma, hypoglycemia, respiratory distress syndrome and NICU admissions are all more common and lead to more expense [2]. Later effects of GDM (particularly among macrosomic infants) include the development of metabolic syndrome during childhood which is diagnosed when three of five criteria are met (obesity, hypertension, carbohydrate intolerance, low high-density lipoproteins levels and hypertriglyceridemia) [4]. Finally, the parturient with GDM may develop ketoacidosis, infection, preeclampsia and if macrosomia is present, there is an increased rate of labor induction and a concomitant rise in cesarean section rates with all their attendant risk [5].

Fetal macrosomia, defined as ≥4500 grams in women with diabetes (≥5000 grams in those with no glucose intolerance) regardless of gestational age, is an important factor for associated maternal and fetal complications [6]. Excess fetal growth as described above can occur in the absence of GDM when it is due to genetic factors. Also, macrosomia is increased in obese women with and without GDM secondary to poor eating habits and lack of exercise. In those with GDM, fetal macrosomia usually occurs due to patient noncompliance with diet, exercise, glucose management etc. but it can occur despite meticulous patient/physician management. Risk factors for macrosomia include GDM, prior large baby (≥4500 GDM), maternal obesity, excessive weight gain during early pregnancy, gestational age ≥ 41weeks, high maternal birthweight, adolescent pregnancy and African American/Hispanic/Native American race [6]. While untreated GDM may have a risk of macrosomia as high as 20%, none of these risk factors or combination of risk factors has a high enough positive predictive value to be used an indicator of macrosomia for clinical management in the current gestation [7]. Unfortunately, ultrasound mensuration is also inaccurate, particularly in macrosomic fetuses, so that the estimated fetal weight cannot be used to predict the actual birthweight and subsequent neonatal injury [8]. Nevertheless macrosomia is of interest to the clinician as women with GDM have an increased risk of shoulder dystocia and brachial plexus injury [2].

Shoulder dystocia is an unpredictable and unpreventable event at delivery [9]. While the rate is higher in macrosomic fetuses, 50% of the shoulder dystocia occur in those with normal birthweight [10]. Shoulder dystocia occurs when the anterior shoulder is obstructed behind the symphysis pubis (or posterior shoulder by the maternal sacral promontory). Shoulder dystocia occurs in 1 to 2% of deliveries and is most commonly diagnosed with failure of the fetal shoulders to be delivered with gentle downward traction, thus requiring additional obstetric maneuvers to effect delivery [10]. Chauhan et al [11] found over a 23-year-old epoch that there were 85 Brachial Plexus Injuries (BPI) amongst 89,978 deliveries (1 per thousand) and there was permanent nerve injury in 1 per 10,000; a rate comparable to other studies in the literature [10]. Similar to macrosomia, there are maternal/fetal complications following shoulder dystocia. There is an increased maternal risk of postpartum hemorrhage, perineal lacerations, rectal sphincter injury and various maternal neuropathies while in the newborn BPI clavicular/humeral fractures, low Apgar scores, encephalopathy and death can occur [10].

Risk factors statistically associated with shoulder dystocia include diabetes, a family history of diabetes, macrosomia, advanced maternal age, term pregnancy, Hispanic ethnicity as well as an android pelvis [9] (Table 2). While increased birthweight and diabetes are statistically associated with shoulder dystocia, most cases occur in newborns with normal birthweights [11]. While these associations occur statistically more frequently in deliveries with shoulder dystocia, the positive predictive value is only 8 to 10%; therefore, risk factors can never be used to predict when shoulder dystocia will occur [12]. Prolonged first or second stages of labor were thought to be related to shoulder dystocia in the distant past but recent data has confirmed that protraction/arrest disorders have no effect on shoulder dystocia [10]. Likewise, studies on ultrasound predicted weight amongst patients with shoulder dystocia found that about 50% of the infants were not macrosomic even though 80% of the women were diabetic; thus fetal macrosomia can be accurately be predicted antenatally only 55% of the time [8].

Maternal Age at Delivery - (35 years or greater)	APGAR Score at Five Minutes ≤Three
Maternal Age at Delivery- (19 years or less)	Cord Ph <7.0
African American/Hispanic/ Native American Race	NICU Admission
Macrosomia >4500grams + GDM, (>5000grams - No GDM)	Neonatal Transfer to a Medical Center
Greater Than or Equal to 41 Weeks at Delivery	Neonatal Fracture (clavicle/ humerus)
Android Pelvis	Neonatal Encephalopathy
	Neonatal Death

Table 2: Risk Factors for Shoulder Dystocia/Erb's Palsy.

The important clinical question is how to prevent BPI and other serious sequala which may follow shoulder dystocia. Is there any benefit to labor induction before the baby becomes macrosomia

or elective C-section for fetuses suspected to be macrosomic? Several authors have studied the issue of labor induction to prevent macrosomia and thus reduce the rate of shoulder dystocia for women with GDM and estimated fetal weight ≥4500 grams. These studies did not reduce the rate of shoulder dystocia but there was an increase in abdominal delivery [13,14]. The benefit of planned cesarean to prevent shoulder dystocia in cases of suspected macrosomia has also been investigated. Rouse et al. [15] tested the sensitivity/specificity of ultrasound for detecting macrosomia and found 3695 cesareans would be required to prevent one permanent brachial plexus injury at the additional cost of 8.7 million for each BPI injury averted. Therefore, the recommendation of the American of Obstetricians and Gynecologists remains that elective cesarean section can be considered for women with GDM and an estimated fetal weight ≥4500 grams (women without diabetes and fetuses with an estimated to fetal weight of ≥ 5000 grams) [10]. Since shoulder dystocia and subsequent brachial palsy cannot be predicted and because at least 50% of Erb's palsy occur in cases where there is no shoulder dystocia, the obstetrician should be ready to manage this complication in every vaginal delivery. A detailed management plan for shoulder dystocia is beyond the scope of this Short Commentary, however there are substantial references within the ACOG monograph on Erb's Palsy on this subject [9].

Although we know that fetal macrosomia is statistically related to GDM and subsequent shoulder dystocia, determining which fetus is actually macrosomic prior to delivery is problematic due to ultrasound or physical estimation of fetal weight being so inaccurate [2,8,10]. Also, the majority of Erb's palsy cases occur most often amongst infants weighing <4000 grams [16]. While we cannot predict which infants will be beyond 4500 grams in women with GDM, we also cannot use risk factors for shoulder dystocia or macrosomia to assist us clinically due to low positive predictive value. In addition, the majority of Erb's palsy cases do not follow shoulder dystocia but rather occur after normal spontaneous deliveries [16]. It is obvious as the nerve injuries without shoulder dystocia, as well as BPI noted following cesarean section, are due to in utero forces which either result from pressure over a period of days or weeks on the affected nerves or rapid first and/or second stages of labor which stretch or avulse the nerves during the labor process [9,17].

In summary, although we cannot prevent shoulder dystocia and BPI in women with GDM, we can work to reduce the rate of macrosomia and many of the its other sequelae. Our best way to attack this problem would be to diagnose GDM and treat it effectively. If an abnormal glucose test is noted between 24 and 28 weeks, the thee hour glucose assessment should be performed to diagnose GDM [10]. Women who cannot or will not avail themselves of diagnostic glucose testing can be managed as if they had GDM, particularly if they had it in a previous gestation. Once a diagnosis of GDM is made, then education about diet, exercise and home glucose testing are helpful. Glucose assessment four times per day (by finger stick in the home) to achieve euglycemic is our most important task. If diet and exercise are not sufficient to control glucose excursions then oral hypoglycemic agents and/or insulin may be appropriate. Additional prenatal visits and ultrasound assessments as well studies of fetal well-being will ensure the mother as well as the fetus is infant as healthy as possible. The reduction of macrosomia should decrease newborn issues such as hypoglycemia, hyperblilemnia as well as respiratory issues and temperature instability problems thus leading to a reduction in NICU days. Also reducing macrosomia may be helpful in decreasing cesarean delivery rates and the complications associated with that procedure.

Conclusion

Although shoulder dystocia cannot be prevented in women with GDM, we should diagnose and manage gestational diabetes assiduously to as early diagnoses of GDM and appropriate treatment will decrease the number of complications in the mother and the baby.

Disclosure: No Funding Source, No Conflicts of Interest, Reprints will not be available

References

1. Hamilton BE, Martin JA, Osterman MJ, Curtin SC, Matthews TJ (2015) Births: final data for 2014. Natl Vital Stat Rep 210: e1-e11.
2. Metzger BE, Lowe LP, Dyer AR, Trimble ER, Chaovarindr U, et al. (2008) Hyperglycemia and adverse pregnancy outcomes. HAPO Study Cooperative Research Group. N Engl. J Med 358: 1991-2002.
3. American College of Obstetricians and Gynecologists. Gestational Diabetes Mellitus. #180. ACOG Practice Bulletin. Obstet Gynecol 130: e17-e31
4. Boney CM, Verna A, Tucker R, Vohr BR (2005) Metabolic syndrome in childhood: Association with birth weight, maternal obesity and gestational diabetes mellitus. Pediatrics 115: e290-e296.
5. Bofill JA, Morrison JC (2015) Gestational diabetes: A conundrum for mother, baby and physician. J Diabetes Metab 6: 1000567
6. American College of Obstetricians and Gynecologists. Fetal Macrosomia. #173. ACOG Practice Bulletin: 1-15
7. Boulet SL, Alexander GR, Salihu HM, Pass M (2003) Macrocosmic births in the United States: determinants, outcomes, and proposed grades of risk. AM J Obstet Gynecol 188: 1372-1378.
8. Byrant DR, Leonardi MR, Landwehr JB, Bottoms SF (1998) Limited usefulness of fetal weight in predicting neonatal brachial plexus injury. Am J Obstet Gynecol 179: 686-689.
9. American College of Obstetricians and Gynecologists. Neonatal brachial plexus palsy. ACOG Monograph; Washington DC
10. American College of Obstetricians and Gynecologists. Shoulder Dystocia. Practice Bulletin #178; ACOG. 2017

11. Chauhan SP, Rose CH, Gherman RB, Magann EF, Holland MW, et al. (2005) Brachial plexus injury: a 23-year experience from a tertiary center. AMJ Obstet Gynecol 192: 1795-802.

12. Gilbert WM, Nesbitt TS, Danielson B (1999) Associated factors in 1611 cases of brachial plexus injury. Obstet Gynecol 93: 536-540.

13. Vendittelli F. Riviere O. Neveu B. Lemery D (2014) Does induction of labor for constitutionally large-for-gestational-age fetuses identified in utero reduce maternal morbidity? BMC Pregnancy Childbirth 14: 156-159.

14. Boulvain M, Senat MV, Perrotin F, Winer N, Beucher G, Subtil D, et al. (2015) Induction of labour versus expectant management for large-for-date fetuses: a randomized controlled trial. Groupe de Recherche en Obstetrique et Gyncologie (GROG). Lancet: 2600-2605.

15. Rouse DJ, Owen J (1999) Prophylactic cesarean delivery for fetal macrosomia diagnosed by means of ultrasonography - A faustian bargain? Am J Obstet Gynecol: 332-338.

16. Chauhan SP, Briery C, Gherman RB, Magann EF, Klauser CK, et al. (2007) Shoulder dystocia without versus with brachial plexus injury: a case control study. J Maternal Fetal and Neonatal Medicine. 20: 313-317.

17. Sandmire H, Morrison J, Racinet C, Hankins G, Pecorari D, et al. (2008) Newborn brachial plexus injuries: the twisting and extension of the fetal head as contributing causes. J Obstet Gynecology 28: 170-172.