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Original Article

Women with gestational diabetes mellitus in the ACHOIS trial: Risk factors for shoulder dystocia

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Abstract

Background: Gestational diabetes mellitus (GDM) is associated with increased risk of fetal macrosomia and shoulder dystocia. However, not all women with GDM and fetal macrosomia have shoulder dystocia.

Aims: To identify the risk factors for shoulder dystocia in women with gestational diabetes using data from women recruited into the routine care group of the ACHOIS trial.

Methods: A secondary analysis was performed on data collected from women enrolled in the ACHOIS trial. Bivariate analyses were performed using the Fisher exact test. Variables found to be significantly associated with shoulder dystocia and previously identified risk factors were used as explanatory variables in multivariate analyses.

Results: A positive relationship was found between the severity of maternal fasting hyperglycaemia and the risk of shoulder dystocia, with a 1 mmol increase in fasting oral glucose-tolerance test leading to a relative risk (RR) of 2.09 (95% CI 1.03–4.25). Shoulder dystocia occurred more often in births requiring operative vaginal delivery (RR 9.58, 95% CI 3.70–24.81, $P < 0.001$). Macrosomic and large-for-gestational-age infants were more likely to have births complicated by shoulder dystocia (RR 6.27, 95% CI 2.33–16.88, $P < 0.001$ and RR 4.57, 95% CI 1.74–12.01, $P < 0.005$, respectively). Fetal macrosomia was the only variable to maintain its significance in all multivariate analyses.

Conclusions: Fetal macrosomia is the strongest independent risk factor for shoulder dystocia. Effective preventative strategies are needed.

Key words: ACHOIS trial, gestational diabetes mellitus, macrosomia, risk factors, shoulder dystocia.

Background

Gestational diabetes mellitus (GDM) is defined as carbohydrate intolerance of varying degrees of severity with onset or first recognition during pregnancy.[1] GDM complicates 5–9% of all pregnancies and is associated with increased perinatal morbidity. The most frequent and significant morbidity is fetal macrosomia, which increases the risk of shoulder dystocia and birth trauma.[2]

Macrosomia is most often defined in the obstetric literature as a birthweight above 4000 g. The best predictor of macrosomia is maternal obesity, however, there is evidence that impaired glucose tolerance is a significant and independent risk factor.[3–7] Moreover, intensive treatment of GDM reduces the risk of macrosomia and shoulder dystocia.[8–10]

Shoulder dystocia is an undesirable consequence of fetal macrosomia and may be accompanied by additional birth

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Caroline Crowther was a chief investigator in the ACHOIS trial.
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trauma including brachial plexus injuries and bone fractures. Shoulder dystocia complicates 0.6–1.4% of all births and in infants with a birthweight of 4000–4500 g the incidence rises to 5–9%.[11,12] Accordingly, higher birthweight is the common denominator connecting maternal and fetal risk factors for shoulder dystocia which include obesity, diabetes, high parity and a prior birth complicated by shoulder dystocia. Labour risk factors for shoulder dystocia include induction of labour and instrumental vaginal delivery.[12–14]

Attempts to prevent shoulder dystocia using the knowledge of risk factors to identify women at risk and implementing strategies, such as Caesarean section and induction of labour, have not been proven effective in randomised controlled trials.[15–19] A review of literature by the American College of Obstetricians and Gynecologists (ACOG) found that there was no level 1 evidence to support a level A recommendation for performing a Caesarean section in all cases of suspected macrosomia. However, based primarily on consensus and expert opinion, the ACOG recommends that prophylactic Caesarean section be considered for suspected fetal macrosomia with estimated fetal weights greater than 5000 g in women without diabetes and greater than 4500 g in women with diabetes.[19] Induction of labour has been identified as a risk factor for shoulder dystocia; however, it has been suggested as a means to prevent further fetal weight gain and improve outcome in large-for-gestational-age infants. Two *Cochrane* systematic reviews assess the role of induction of labour in preventing pregnancy complications, which included shoulder dystocia, in cases of suspected macrosomia and in pregnant women with diabetes.[20,21] Both reviews conclude that there was insufficient evidence regarding the effect of inducing labour on preventing shoulder dystocia. Manoeuvres used in the treatment of shoulder dystocia have been used prophylactically to aid the passage of the fetus and try and prevent shoulder dystocia and other birth trauma. However, two randomised trials of such manoeuvres included in a *Cochrane* systematic review have failed to identify clinical evidence to support this practice.[22–24] The failure of the interventions described above to provide adequate prophylaxis against shoulder dystocia reflects difficulties faced in predicting its occurrence.

Because of the risk of macrosomia associated with abnormal glucose metabolism, women with GDM have traditionally been classified as 'at risk' of shoulder dystocia and other birth trauma. However, not all women with GDM and macrosomia develop this complication.

The aim of this study was to identify maternal, antenatal, intrapartum, fetal risk factors for shoulder dystocia in women with gestational diabetes using data from women recruited into the control group of the ACHOIS trial.

Methods

A secondary analysis was performed on data collected from women enrolled into the routine care group of the ACHOIS study.[10] As previously described[10] women with a singleton or twin pregnancy between 16 and 30 weeks gestation and with risk factors for gestational diabetes or a positive 50-g oral glucose-challenge test and an abnormal 75-g oral glucose-tolerance test (OGTT) at 24–34 weeks were eligible for inclusion in the trial. Of the 1000 women enrolled, 510 were randomised to the routine-care group. These women gave birth to 524 infants, amongst whom there were 16 cases of shoulder dystocia.[10] The presence and severity of shoulder dystocia were assessed by means of a standardised checklist completed by the caregiver present at birth.[10]

The protocol for the ACHOIS trial was approved by the ethics committee at each of the 18 collaborating centres (14 in Australia and four in the UK). All women who participated provided written informed consent.

For this study, the analyses were conducted in two stages. Firstly, for the 510 women in the routine care group we compared baseline characteristics (age, body mass index (BMI), ethnicity, parity, previous pregnancies complicated by shoulder dystocia, two-hour OGTT results, fasting OGTT results) for women with births complicated by shoulder dystocia and those that did not have shoulder dystocia using the Fisher exact test. Log binomial regression was used to analyse continuous predictors such as two-hour OGTT results and fasting OGTT results. Intrapartum factors (operative vaginal delivery, Caesarean) and fetal factors were similarly compared.

Secondly, all variables found to be significantly associated with shoulder dystocia were used as explanatory variables in multivariable analyses (log binomial regression). The purpose of this analysis was to examine the impact of each explanatory variable on the incidence of shoulder dystocia while controlling for the impact of the remaining variables. For all analyses, a $P < 0.05$ was used to indicate statistical significance.

Results

The results suggest that there is a positive relationship between the severity of maternal hyperglycaemia and the risk of shoulder dystocia, with a 1-mmol increase in fasting OGTT leading to a relative risk (RR) of 2.09 (95%CI 1.03–4.25, $P = 0.04$; Table 1). In women with a BMI ≥ 25 kg/m^2, there was a trend towards an increased risk of shoulder dystocia (RR 2.31, 95% CI 0.64–8.44, $P = 0.15$). A similar trend was seen among Caucasian women (RR 4.31, 95% CI 0.58–32.31, $P = 0.09$), however, neither of these results were statistically significant (Table 1).

Shoulder dystocia occurred more often in births requiring operative vaginal intervention (RR 9.58, 95% CI 3.70–24.81, $P < 0.001$). The increased rate of shoulder dystocia was seen in operative vaginal births using forceps and ventouse extraction (RR 6.35, 95% CI 2.34–17.26, $P = 0.0025$ and RR 6.14, 95% CI 2.12–17.77, $P = 0.0067$, respectively). No cases of shoulder dystocia were found among women undergoing Caesarean delivery (Table 2).

Macrosomic and LGA infants were more likely to have births complicated by shoulder dystocia (RR 6.27, 95% CI 2.33–16.88, $P < 0.001$ and RR 4.57, 95% CI 1.74–12.01, $P < 0.005$, respectively) (Table 3).

Table 1 Associations between maternal characteristics and the risk of shoulder dystocia

	Shoulder dystocia $n = 16$ (no.%)	No shoulder dystocia $n = 508$	Relative risk 95% CI†	*P*-value
Maternal age				
≥ 35 years	3 (2.6)	116	0.79 (0.23,2.71)	0.49
< 35 years	13 (3.3)	392		
Parity				
Multigravida	7 (2.7)	258	0.76 (0.29,2.01)	0.38
Primigravida	9 (3.6)	250		
Ethnicity				
Caucasian	15 (3.8)	392	4.31 (0.58,32.31)	0.09
Other‡	1 (0.9)	116		
Body mass index§				
= 25 kg/m^2	9 (3.7)	241	2.31 (0.64,8.44)	0.15
< 25 kg/m^2	3 (1.6)	190		
Shoulder dystocia in previous pregnancy				
Yes	0 (0)	2		0.94
No	16 (3.2)	506		
OGTT results				
Fasting (mmol/L)				
1 unit increase			2.09 (1.03,4.25)	0.04
2 h (mmol/L)				
1 unit increase			1.49 (0.89,2.48)	0.13

†CI denotes confidence interval.
‡Includes Asian, aboriginal and remaining races excluding Caucasians.
§Data only available for 443 participants.

When operative vaginal delivery, fetal macrosomia and fasting OGTT were all included in the regression model, there was a reduction in the RR of shoulder dystocia associated with fasting OGTT linear (RR 1.40 95% CI 0.69–2.87, $P = 0.35$).

A regression model including fetal macrosomia and fasting OGTT demonstrated that macrosomia played a mediating role in the increased risk of shoulder dystocia seen with worsening hyperglycaemia. The adjusted RR associated with fetal macrosomia maintained its significance (RR 5.47 95% CI 1.96–15.23, $P = 0.0011$) while a reduction was seen in the RR associated with fasting OGTT (RR 1.50 95% CI 0.74–3.05, $P = 0.25$).

Table 2 Associations between intrapartum factors and the incidence of shoulder dystocia

	Shoulder dystocia $n = 16$ (no.%)	No shoulder dystocia $n = 508$	Relative risk 95% CI	*P*-value
Induction of labour				
Yes	3 (2.0)	149	0.56 (0.16,1.95)	0.27
No	13 (3.6)	359		
Operative vaginal birth				
Yes	9 (17.0)	53	9.58 (3.70,24.81)	< 0.001
No	7 (1.5)	455		
Ventouse				
Yes	4 (17.4)	23	6.14 (2.12,17.77)	< 0.01
No	12 (2.5)	485		
Forceps				
Yes	5 (16.7)	30	6.35 (2.34,17.26)	< 0.005
No	11 (2.3)	478		
Caesarean				
No	16 (4.8)	331		< 0.005
Yes	0 (0)	177		

Discussion

Fetal macrosomia was the strongest predictor for shoulder dystocia. Statistical analysis showed that this variable played a mediating role in the association between the degree of maternal fasting hyperglycaemia and the risk of shoulder dystocia. This is consistent with previous findings that show that increasing fasting plasma glucose levels on the OGTT correlate with increasing infant birthweight.[26]

Our findings showed an increased risk of shoulder dystocia associated with operative vaginal birth with both the use of forceps and of vacuum extraction. Operative vaginal birth has previously been identified as a risk factor for shoulder dystocia.[12–14] Some studies have reported that this risk is increased with the use of vacuum compared with forceps delivery.[13,14] Indications for operative vaginal birth, including a prolonged second stage of labour, are associated with shoulder dystocia and therefore it is difficult to determine

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Table 3 Association between fetal size and the risk of shoulder dystocia

	Shoulder dystocia $n = 16$ (no.%)	No shoulder dystocia $n = 508$	Relative risk 95% CI	*P*-value
Macrosomia				
≥ 4000 g	10 (9.0)	100	6.27 (2.33,16.88)	< 0.001
< 4000 g	6 (1.5)	408		
Large for gestational age‡				
Yes	9 (8.5)	106	4.57 (1.74,12.01)	< 0.005
No	7 (1.7)	402		
Small for gestational age§				
No	16 (3.4)	470		0.29
Yes	0 (0)	38		

‡Birthweight exceeding the 90th percentile on standard charts.[25]
§Birthweight below the tenth percentile on standard charts.[25]

whether the relationship between the two variables is causal. The debate continues over a standard objective definition for shoulder dystocia and hence the perceived difficulty of births requiring instrumental assistance may result in over-diagnosis.[27,28]

No reports of shoulder dystocia were found among women who underwent Caesarean delivery. Rarely, there may be cases of shoulder dystocia among women undergoing emergency Caesarean section after cephalic replacement with the Zavenelli manoeuvre.

Previous studies have shown that increasing maternal weight is associated with an increased risk of shoulder dystocia.[29,30] Caucasian race has been identified as a risk factor for shoulder dystocia. Current literature attributes this risk to increased rates of obesity and macrosomia among these women.[29,30]

We found no association between parity or a prior birth complicated by shoulder dystocia and the risk of shoulder dystocia. Retrospective studies of births complicated by shoulder dystocia have reported recurrence rates of between 1% and 16.7%.[31–33] Only two women in the study sample had a previous birth complicated by shoulder dystocia. Large retrospective studies show that parity is associated with an increased risk of giving birth to macrosomic infants and hence an increased risk of shoulder dystocia.[34,35]

Conclusion

The purpose of this study was to identify new risk factors for shoulder dystocia and confirm previously reported risk factors in women with gestational diabetes using data from women enrolled in the routine care group of the ACHOIS trial. The sample of 510 women included 16 cases of shoulder dystocia. Fetal macrosomia was found to play a mediating role between the degree of maternal hyperglycaemia and the risk of shoulder dystocia. Among all the risk factors identified in the empirical analysis, only fetal macrosomia remained statistically significant in multivariate analyses. This finding supports the hypothesis that fetal macrosomia is the strongest predictor for shoulder dystocia in patients with GDM and emphasises the need for effective preventative strategies.

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References

1 Metzger BE, Coustan DR. Summary and recommendations of the Fourth International Workshop Conference on Gestational Diabetes Mellitus: The Organizing Committee. *Diabetes Care* 1998; Suppl **2**: B161–B167.
2 Hoffman L, Nolan C, Wilson JD, Oats J, Simmons O. Gestational diabetes mellitus – management guidelines: The Australasian Diabetes in Pregnancy Society. *Med J Aust* 1998; **169**: 93–97.
3 Naylor CD, Sermer M, Chen E, Sykora K. Caesarean delivery in relation to birthweight and gestational glucose tolerance: Pathophysiology or practice style? Toronto Trihospital Gestational Diabetes Investigators. *JAMA* 1996; **275**: 1165–1170.
4 Wollschlaeger K, Nieder J, Koppe I, Hartlein K. A study of fetal macrosomia. *Arch Gynecol Obstet* 1999; **263**: 51–55.
5 Casey BM, Lucas MJ, Mcintire DD, Leveno KJ. Pregnancy outcomes in women with gestational diabetes compared with the general obstetric population. *Obstet Gynecol* 1997; **90**: 869–873.
6 Ostlund I, Hanson U, Bjorklund A *et al.* Maternal and fetal outcomes if gestational impaired glucose tolerance is not treated. *Diabetes Care* 2003; **26**: 2107–2111.
7 Jensen DM, Sorensen B, Feilberg-Jorgensen N, Westergaard JG, Beck-Nielsen H. Maternal and perinatal outcomes in 143 Danish women with gestational diabetes mellitus and 143 controls with a similar risk profile. *Diabet Med* 2000; **17**: 281–286.
8 Buchanan TA, Kjos SL, Montoro MN *et al.* Use of fetal ultrasound to select metabolic therapy for pregnancies complicated by mild gestational diabetes. *Diabetes Care* 1994; **17**: 275–283.

9 Langer O, Rodriguez DA, Xenakis EM, McFarland MB, Berkus MD, Arrendondo F. Intensified versus conventional management of gestational diabetes. *Am J Obstet Gynecol* 1994; **170**: 1036–1046.

10 Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS. Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) Trial Group. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. *N Engl J Med* 2005; **352**: 2477–2486.

11 American College of Obstetricians and Gynecologists. *ACOG Practice Bulletin*, Vol. 40. Washington DC: American College of Obstetricians and Gynecologists, 2002.

12 Nesbitt TS, Gilbert WM, Herrchen B. Shoulder dystocia and associated risk factors with macrosomic infants born in California. *Am J Obstet Gynecol* 1998; **179**: 476–480.

13 Bofill JA, Rust OA, Devidas M, Roberts WE, Morrison JC, Martin JN Jr. Shoulder dystocia and operative vaginal delivery. *J Matern Fetal Med* 1997; **6**: 220–224.

14 Caughey AB, Sandberg PL, Zlatnik MG, Thiet MP, Parer JT, Laros RK Jr. Forceps compared with vacuum: Rates of neonatal and maternal morbidity. *Obstet Gynecol* 2005; **106**: 908–912.

15 Gonen O, Roses DJ, Dolfin Z, Tepper R, Markov S, Fejgin MD. Induction of labor versus expectant management in macrosomia: A randomized study. *Obstet Gynecol* 1997; **89**: 913–917.

16 Kjos SL, Henry OA, Montoro M, Buchanan TA, Mesman JH. Insulin-requiring diabetes in pregnancy: A randomized trial of active induction of labor and expectant management. *Am J Obstet Gynecol* 1993; **169**: 611–615.

17 Tey A, Eriksen NL, Blanco JD. A prospective randomized trial of induction versus expectant management in nondiabetic pregnancies with fetal macrosomia. *Am J Obstet Gynecol* 1995; **172**: 293.

18 Rouse DJ, Owen J. Prophylactic cesarean delivery for fetal macrosomia diagnosed by means of ultrasonography: A Faustian bargain? *Am J Obstet Gynecol* 1999; **181**: 332–338.

19 American College of Obstetricians and Gynecologists. *Fetal macrosomia*. In: Practice Bulletin No. 22. Washington, DC: American College of Obstetricians and Gynecologists, November 2000.

20 Irion O, Boulvain M. Induction of labour for suspected fetal macrosomia. *The Cochrane Database of Systematic Reviews* 1998, Issue 2. Art. No.: CD000938. DOI: 10.1002/14651858.CD000938.

21 Boulvain M, Stan C, Irion O. Elective delivery in diabetic pregnant women. *The Cochrane Database of Systematic Reviews* 2001, Issue 2. Art. No.: CD001997. DOI: 10.1002/14651858.CD001997.

22 Beall MH, Spong CY, Ross MG. A randomized controlled trial of prophylactic maneuvers to reduce head-to-body delivery time in patients at risk for shoulder dystocia. *Obstet Gynecol* 2003; **102**: 31–35.

23 Poggi SH, Allen RH, Patel CR, Ghidine A, Pezzullo JC, Spong CY. Randomized trial of McRoberts versus lithotomy positioning to decrease the force that is applied to the fetus during delivery. *Am J Obstet Gynecol* 2004; **191**: 874–878.

24 Athukorala C, Middleton P, Crowther CA. Intrapartum interventions for preventing shoulder dystocia. *Cochrane Database of Systematic Reviews* 2006; Issue 4. Art. No.: CD005543. DOI: 10.1002/14651858.CD005543.pub2.

25 Roberts CL, Lancaster PA. Australian national birth weight percentiles by gestational age. *Med J Aust* 1999; **170**: 114–118.

26 Schrader HM, Jovanovic-Peterson L, Bevier WC, Peterson CM. Fasting plasma glucose and glycosylated plasma protein at 24–28 weeks of gestation predict macrosomia in the general obstetric population. *Am J Perinatol* 1995; **12**: 247–251.

27 Beall MH, Spong C, McKay J, Ross MG. Objective definition of shoulder dystocia: A prospective evaluation. *Am J Obstet Gynecol* 1998; **179**: 934–937.

28 Spong CY, Beall M, Rodrigues D, Ross MG. An objective definition of shoulder dystocia: Prolonged head-to-body delivery intervals and/or the use of ancillary obstetric maneuvers. *Obstet Gynecol* 1995; **86**: 433–436.

29 Cedergren MI. Maternal morbid obesity and the risk of adverse pregnancy outcome. *Obstet Gynecol* 2004; **103**: 219–224.

30 Usha Kiran TS, Hemmadi S, Bethel J, Evans J. Outcome of pregnancy in a woman with an increased body mass index. *BJOG* 2005; **112**: 768–772.

31 Baskett TT, Allen AC. Perinatal implications of shoulder dystocia. *Obstet Gynecol* 1995; **86**: 15–17.

32 Ginsberg NA, Moisidis C. How to predict recurrent shoulder dystocia. *Am J Obstet Gynecol* 2001; **184**: 1427–1429.

33 Smith RB, Lane C, Pearson JF. Shoulder dystocia: What happens at the next delivery? *Br J Obstet Gynecaecol* 1994; **101**: 713–715.

34 Orskou J, Henriksen TB, Kesmodel U, Secher NJ. Maternal characteristics and lifestyle factors and the risk of delivering high birth weight infants. *Obstet Gynecol* 2003; **102**: 115–120.

35 Yasmeen S, Danielsen B, Moshesh M, Gilbert WM. Is grandmultiparity an independent risk factor for adverse perinatal outcomes? *J Matern Fetal Neonatal Med* 2005; **17**: 277–280.

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