Singapore Journal of Obstetrics and Gynaecology
March 2004; 35(1): 48-52

NORMAL VAGINAL DELIVERY OF A 5600 GRAM MACROSOMIC FEMALE FETUS WITH SHOULDER DYSTOCIA : A CASE REPORT AND REVIEW OF LITERATURE

Ahmed Zakaria Rabie

INTRODUCTION

The improvement in perinatal morbidity and mortality in modern obstetrics mostly involves the advance in obstetric and neonatal care given to premature and small for gestation age (SGA) infants. Conversely there has been less concern about the large for gestational age (LGA) fetus which is subjected to the danger of birth asphyxia and trauma[1]. Obstetric interest is heightened by the predisposition of the mother of the large infants dystocia. The diagnosis of dystocia is made with increasing frequency as the fetal weight increases. Mothers with macrosomic infants are more prone to cephalopelvic disproportion, failure to progress in labor, prolonged labor, operative vaginal delivery and emergency caesarean section (C.S.) compared with normal weight babies[2]. Genital tract lacerations are more common and associated with the need for a episiotomy and additional manoevres are required to deliver the shoulder. Uterine rupture may occur in association with suprabupic or fundal pressure. Postpartum haemorrhage is more likely due to a combination of uterine atony due to prolonged labor, a large infant and increased blood loss from lacerations and extensive episiotomy[3].

Traumatic midpelvic vertex delivery occurs during dystocia prior to full dilatation of the cervix which is often followed by the serious shoulder dystocia. Brachial plexus injury of the nerve root C5 & C6 occurs in 5-15% of neonates. The range of permanent palsy is from 4-32% but usually less than 10%[4]. One half of all cases occurs in infants weighing less than 4500g (9 lb, 15 oz)[5]. Fractures of the clavicles occurs in 15% of cases of shoulder dystocia[6]. Fractures of the humerus occurs rarely and that of cervical spine are extremely rare. A combination of adverse events including traumatic manipulation and obstructed cerebral venous return, along with hypoxia for 4-6 minutes may result in fetal brain damage[3]. The majority of macrosomic fetuses are subjected to anterpartum intensive surveillance testing. The recommendations from the American College of Obstetrics & Gynecology are : No single method of anterpartum screening appears to be superior to another. Anternatal surveillance of post term pregnancies should begin at 42 weeks. Surveillance between 40-42 weeks has not been shown to improve outcome. No clear difference in outcome has been found between induction of labor and expected management[7].

Department of Obstetrics and Gynaecology
Faculty of Medicine,
Suez Canal University -
Ismailia,
Egypt

Correspondence:
A/Prof. Ahmed Zakaria Rabie,
Department of Obstetrics & Gynaecology,
Faculty of Medicine,
Suez Canal University,
Ismailia,
Egypt

CASE REPORT

A 38 year old multiparous woman (para2) presented at AL-Khafji Hospital for the first time during the end of the first stage of labor with strong frequent uterine contractions. The patient had no information on antenatal care. On diagnosing macrosomia by careful cinical examination and history taking, she had uneventful past history of previous similar pregnancies with spontaneous vaginal delivery of two macrosomic babies. She had no history of diabetes. The data of antenatal care of the previous or the ongoing pregnant was not written or registered. Based on their previous experience patient and her husband were refusing the C.S. method of delivery.

The management depended on the fact that, with expectation of shoulder dystocia the clinical acumen has been reported to be as accurate as ultrasound[8]. The head may deliver first but does not undergo spontanous external rotation and recoils tightly against the perineum. Gentle traction on the head downwards and backwards may fail to deliver the anterior shoulder. Once the head has been delivered the uterus contracts down and this causes a reduction or cessation of blood flow to the intervillous space. In addition the fetal chest is compressed so that adequate respiratory effort is impossible even though the infants mouth and nose are delivered. After delivery of the head the supply of oxygen to the fetus is reduced and the umbilical artery pH falls at a rate of 0.04 units/min. Thus provided the fetus is not hypoxic up to the delivery of the head, there is probably 4-6 minutes during which delivery of the infant can be achieved without hypoxic brain damage.

In delivery of the shoulders, the bisacromial diameter is large and/or the pelvic brim is more flat than gynaecoid. The anterior shoulder may become impacted behind the pubic symphysis. Usually the posterior shoulder will descend below the sacral promontory. The anterior shoulder is more likely to become impacted above the pelvic brim if it attempts to enter the pelvis in the antero-posterior diameter which is the narrowest of the pelvic inlet. On very rare occasions usually associated with mid pelvic delivery, both shoulders may be arrested above the pelvic brim. Sometimes referred to as bilateral shoulder dystocia it appears logical to try to move the fetal shoulders to the oblique and transverse diameter of the pelvic inlet as these are longer than the antero-posterior diameter[3]. In McRobert's maneuver the maternal hips are hyperflexed by bringing the knee up beside the chest. This straightens the maternal lumbar lordosis and lumbosacral angle, reducing the obstructive effect of the sacral promontory. In addition, the angle of inclination of the pelvis is reduced from approximately 25° to 10° so that plane of the pelvic inlet is perpendicular to the maternal expulsion forces. As a result, the symphysis rotates superiorly which lifts the fetus and flexes the fetal spine towards the anterior shoulder pushing the posterior fetal shoulder below the pelvic brim. This position effectively reduces fetal trauma by reducing shoulder extraction forces responsible for stretching of theplexus[17]. In the plan for delivery of the posterior shoulder, following episiotomy, the hand is inserted into the vagina and sacral hollow with palpation of the fetal humerus and following it to the elbow. The posterior shoulder is pushed off from midline to the oblique diameter which is a favorable diameter that also assists in the adduction of the fetal shoulders. This makes the fetal back always directed anterior placing the fingers in the posterior axilla and sweeping the arm across the fetal chest with delivery of the posterior shoulder. Delivery of the baby proceeds without difficulty[2,3].

So, once the problem was recognized with the patient's refusal of C.S. the situation was explained to her. Additional personnel was summoned to provide anesthesia and neonatal resuscitation. The strong frequent uterine contraction was pressed rapidly for full cervical dilatation and spontaneous delivery of the large fetal head. By providing inhalation analgesia and episiotomy the delivery was conducted using the abovementioned manoevres. Following delivery, exploration and closure of the episiotmy was done. On following the baby in the intensive care unit, her Apgar sore was 10/10 after 1 and 5 minutes and her birth weight was 5600g.

DISCUSSION

There is disagreement as to precisely where the threshold should be to assign a neonate as macrosomic. The birth weight found the 90th percentile at term varied considerably. For 40-42 weeks it ranged from 3720-3 995g in Denver[18] and from 3970-4 363g in Portland[9]. After 41 weeks it was greater than 4,000g in San Antonio[10]. The most commonly proposed criteria for macrosomia is a birth weight greater than 4,000g (8lb, 13oz) or 4,500g (9lb, 15oz) or birth weight over ± 2 SD the mean birth weight by age[11]. Birth weight which exceeds 4,000g has an incidence between 3.1-7.7% and > 2,500g is 10%[2]. The medical literature confirms that prediction of fetal macrosomia is difficult and ultrasound estimation of fetal weight adds little additional useful information. What clinicians really expect to predict is not macrosomia per se but the serious complications, such as shoulder dystoia or brachial plexus injury associated with these babies. Such complications, however are not determined by birth weight alone but by a complex and poorly understood relationship between fetal and maternal anatomy and other factors[5].

The incidence of shoulder dystocia is proportional to birth weight with the fetal macrosomia being the predominant strong risk factor. However, most cases of shoulder dystocia occurs in infants weighing less than 4,500g and the majority of cases occur without any risk factor[3]. The overall incidence shoulder dystocia is between 0.15-0.3% that increases to 3.3% in nondiabetic women and between 4,100-4,325g; for infant of diabetic mothers (IDM) it is 7% for weight between 4,250-4,500g and 25% for those > 4,500g[10]. In vast majority of cases there will not be recurrence of shoulder dystocia. The range of increased risk of recurrence is 1.1-13.8%[3]. The most commonly used definition for shoulder dystocia is failure of the shoulders to deliver spontaneously and/or with gentle downward traction on the fetal head. The perinatal death due to complication of shoulder dystocia is rare but damage to the infant from associated asphyxia and trauma is not uncommon. Attempts to incorporate shoulder width as a predictor for shoulder dystocia have not been successful[13]. Infants with shoulder dystocia had significantly longer abdominal diameter minus biparietal diameter (AD-BPD) measurement with no difference in both weight versus a normal delivery group. No infant with antenatal AD-BPD difference < 2,6cm suffered shoulder dystocia within 2 weeks of delivery in IDM with estimated fetal weight (EFW) of 3,800-4,200g[14]. The management of shoulder dystocia is required almost as often as neonatal resuscitation and much more ferquently than maternal cardiopulmonary resuscitation (CPR) in the labor ward. In any presentation of cases with shoulder dystocia, the hospital chart shoulder clearly document the time, the type and sequences of maneuvers used to manage the dystocia. There are many techniques proposed to deal with shoulder dystocia. No single manoeuvre or combination of techniques have been proven to be superior in any trial[15]. Because macrosomia has the commonest association with shoulder dystocia and neonatal injury, it has been proposed that elective C.S. of estimated fetal weight of between 4,000 and 4,500g shoulder be pursued. However, reconsideration ocurred because of the high cost of the total number of C.S. to prevent one permanent brachial plexus injury[16]. Overall, the majority of cases of shoulder dystocia have normal progression in labor, and spontaneous or low pelvic assisted delivery. Thus, as with the antepartum markers, evaluation of currently known intrapartum risk factors lacks clinical predictive value. No consistent pattern of labor and/or delivery could also reliably predict the onset of shoulder dystocia[3].

The need for antepartum surveillance testing for the large fetus is determined by the aetiological factors for excessive growth of the developing fetus. For

macrosomia which result from either genetic, constitutional, environmental or hormonal factors, each cause has its own paradigm for antepartum management. A number of risk factors for fetal macrosomia have been recognized. The strongest risk factor is Gestational diabetes mellitus (GDM) which results in a two-fold increase in the incidence of macrosomia[2]. In addition to GDM, heavy babies can result from diseases that cause hydrops, such as erythroblastosis fetalis, placental chorioangiomas, or heart disease. Certain syndromes also are associated with overgrowth, including transposition of the great vessels, the Marshall's syndrome Wearver's syndrome Sotos Syndome (cerebral gigantism) and Beckwith-wiedemann (exomphalos-macroglossia-gigantism syndrome)[19].

A specific glucose threshold that constitutes ideal glucose control has not been determined as yet. The appearance of phosphatidyl glycerol (PG) in the amniotic fluid is believed to be the most sensitive indicator of fetal lung maturity (FLM).

Kjos et al reported that surfactant deficient respiratory distress syndrome (RDS) does not occur in any term neonates[20]. Hyaline membrance disease occured in infants before 36 weeks gestation in diabetic pregnancies[21]. Thus, in accurately dated term GDM pregnancies a lecithin / sphingomyelin ratio of ≥ 2:1 may be sufficient to indicate lung maturity particularly with well controlled glucose levels. In Bekwith Wiedemann syndrome both omphalocoele and macroglossia can be detected by ultrasound. Elevated α-fetoprotein and amniotic fluid insulin may assist in diagnosing omphalocele. Amniotic fluid insulin levels would be normal or low in Sotos syndrome. Nesidioblastosis (which is a proliferation of islet cells) can be diagnosed by ultrasound and in elevated amniotic fluid insulin levels. In addition, asymmetric overgrowth pattern as determined by ultrasound could diagnose Weaver's syndrome[1].

Most of the antenatal risk factors are inter-related and when two or more of these risk factors are present, the risk of macrosomia is only 32%. It is noteworthy that 34% of macrosomic infants are born to mothers without any risk factors and 38% of pregnant women have at least one risk factor[2]. Approximately 20% of birthweight is determined by fetal genotype[22]. Male gender contributes approximately 150-200g of increased weight compared with the female infant at term[23]. Symmetric overgrowth represents the genetic effect of overgrowth, whereas the overgrowth asymmetric is more characteristic of the pregnant diabetic women and is associated with altered body composition[1]. Parous women are 2-3 times more likely than control women to have macrosomic infants. Multiparity and age 35 years have 1.5-2 times the risk of macrosomia[12]. Enhanced maternal bodyweight before pregnancy have a greater risk of giving birth to excessively large infants when sex and gestational age are controlled[24]. Maternal weight gain during pregnancy has a poor correlation with birth weight but does make an independent contribution[1]. It has been reported that maternal birthweight >8 pounds could increase the risk of macrosomia by 2.8 times[25]. Ponderal index (weight/ height) in the upper 10 percentile and height exceeding 169cm tall increase the risk s ignificantly[2]. The major strategies used to predict macrosomia are the clinical risk factors, clinical estimation of birthweight by Leopold's manoeuvres by ultrasonography. Each method has its substantial limitation in term of accuracy[5]. The volume of amniotic fluid, the size and configuration of the uterus and maternal body habitus complicate estimation of the fetus palpation through the abdominal wall. The mean error documented is about 300g (11-1,6 oz)[26]. Using Leopold's manoeuvre the actual weight is underestimated by approximately 1 lb in 50% of fetuses weighing > 4,000 gm and in up to 80% of fetuses weighing > 4,500 g[27]. Ultrasonographic estimation of fetal weight has typical mean error ranges from 300-550gm (11.6 to 19.4 oz)[26]. Ultrasound estimation at best are 10% of the actual fetal weight two-thirds of the time. There is a potential source of error when dealing with the very large infant. In several instances[28], actual birth weight was underestimated by more than 800g. When abdominal circumference (Ac) increases ≥1.2cm/week after 32 weeks gestation an LGA infant resulted in 79% of cases. Growth velocity less than this ocurred in appropriate for gestational age infant 89% of the time[29]. Postterm pregnancy is associated with a high incidence of macrosomia and shoulder dystocia. Boyd et al[2] found that the incidence of macrosomia was 12% at 40 weeks and 21% at 42 weeks gestation. With advancing gestation, the fetal chest and shoulders continue to grow steadily whereas the biparietal diameter growth slows considerably increasing the likelihood of an unfavorable shoulder / head circumference ratio[3]. Accurate determination of gestational age, identifying the estimated day of confinement (EDC) could distinguish the true postterm from wrong dates. Clinical signs such as quickening, first auscultation of fetal heart, early uterine size and uterine fundal height at 20 weeks as well as Naegel's rule all help in diagnostic accuracy. Decreased amniotic fluid volume even in the presence of a reactive non stress test (NST), thick meconium stained amniotic fluid, abnormal intrapartum monitoring indicates the need for further evaluation of delivery[1].

Optimizing outcomes still have much controversies about spontaneous versus induced labor and selecting candidates for elective C. S. delivery as opposed to vaginal birth. Given that the fetus continues to gain 230 gm (8.1oz) per week after the 37th week elective induction of labor before or near term has been suggested to prevent macrosomia and its complications[30]. Since fetal weight cannot reliably predict the presence or absence of macrosomia, then operative interventions are performed unnecessarily. Labor induction itself result in higher rates of C.S. delievry than spontaneous labor regardless of condition of the cervix controlled gestational age and without favorably altering perinatal outcomes[5]. Clearly there is a distinction between a genetically predetermined large fetus which is macrosomic, heavier than 4,000 g or 4,500 g and the macrosomic infant of a diabetic mother. The incidence of shoulder dystocia is higher in IDM[31] especially in women with an increased body fat in the upper torso. In such cases, inductions for IDM is both medically and cost efficient, preventing the majority of injury without a substantial increase in the C.S. rate[32]. Induction of the nondiabetic macrosomic infants, however increases the C.S. delivery rate by about

two-fold[33]. Haram et al[11] suggest that labor should not be induced or C.S. performed in non-diabetic pregnancies unless the estimated fetal weight is above 5,000 g or if the gestational age is above 42 weeks. In diabetic pregnancies selective induction or C.S. is usually performed when weight is above 4,000 g. The patients obstetric history, her progress during labor, the adequacy of her pelvis and other evidence suggestive of fetopelvic disproportion should be used in determining an intervention, such as C.S.[5].

REFERENCES:

1. ***Berkus M. D., Conway D. and Langer O. (1999) :*** *The large fetus. Clin. Obstet Gynecol. 42 (4) : 766-784.*

2. ***Boyd M. E., Usher R. H., and Mclean F. H., (1983) :*** *Prediction, risk, proposed management.. Obstet Gynecol; 61 : 715-721.*

3. ***Baskett T. F. (2001) :*** *Prediction and management of shoulder dystocia. In Bonnar J. (eds). Recent advances in obstetrics and gynecology (21) chapters (4) : 45-54.*

4. ***Chauvergus J. O., Berg I. ; Erichs K. and Jerr I (1998) :*** *Cause and effect of obstetric (neonatal) brachial plexus palsy. Acta Pediatr Scand. 77 : 357-364.*

5. ***Zamorski M. A, and Biggs W. S. (2001) :*** *Management of suspected fetal macrosomia. Am. Fam. Physician. 63 : 302-6.*

6. ***Oppenheim W., Davis A., Growdon W., Dory F. and Davlin L., (1990) :*** *Clavicle fractures in the newborn Clin-Orthop. 250 : 176-180.*

7. ***ACOG, (1993) :*** *ACOG technical bulletin 187. Washington, DC :*

8. ***Sherman D. J., Arieli S., Tovobin Siegal G., Caspi E., Bukovsky I. (1998) :*** *A comparison of clinical and ultrasonic estimation of fetal weight. Obstet. Gynecol. 91 : 212-217.*

9. ***Babson S. G., Berhrman R. E. and Lessel R.,*** *Liveborn with weight for gestational age of white middle class infants. Pediatrics. 45 : 937-944.*

10. ***Langer O., Berkus M. D., Huff R. W., and Samueloff A (1991) :*** *Shoulder dystocia should the fetus weight > 4,000 gram be deliver by cesarean section. Am J. Obstet. Gynecol. 165 : 831-837.*

11. ***Haram K., Bergesjo P. and Pirhonen J., (2001) :*** *Suspected large fetus in the last period of pregnancy-a difficult problem. J. perinatol Mar (2) : 93-6.*

12. ***Mondanlou H. D., Dorchester W. L., Thorosian A., and Freeman R. K., (1980) :*** *Macrosomia maternal, fetal and neonatal implications Obstet. Gynecol., 55 : 420-424.*

13. ***Verspyck E., Goffinet E., Hellot M. F., miliiez J., Marpau L. (1999) :*** *Newborn shoulder width : A prospective study of 2222 consecutive measurements. Br. J. Obstct. Gynecol, 106 : 589-593.*

14. ***Cohen B., Penning S., Major C., Ansley D., Porto M., and Garite T., (1996) :*** *Sonographic prediction of shoulder dystocia in infants of diabetic mothers thus. Obstet. Gynecol. 88 : 10-13.*

15. ***Mcfarland M. B., Langer O, Piper J. M, and berkus M. D., (1996) :*** *Perinatal outcome and the type and number of maneuvers in shoulder dystocia Int. J. Gynecol. Obstet. 55 : 219-224.*

16. ***Rouse D. J., Owen J., Goldenberg R. I., and Cliver S.p., (1996) :*** *The effectiveness and costs of elective cesarean section for fetal macrosomia diagnosed by ultrasound. JAMA, 276 : 1480-1486.*

17 ***Conick B. Stringer C. A and held B. (1983) :*** *An alternative maneuver for management of shoulder dystocia. Am J. Obstet. Gynecol. 145 : 828-884.*

18. ***Lubchenco L. O., Hansman C, Dressler M., and Boyed E., (1963) :*** *Intrauterine growth as estimated from liverborn birth weight data at 24 to 42 weeks of gestation. Pediatrics, 32 : 793-800.*

19. ***Stevenson D. K., Hopper A. O. and Cohen R. S. (1982) :*** *Macrosomia : Cause and consequences J. Pediatrics, 100 : 515-520.*

20. ***Kjos S. L., Walther F. J., Montoro M., Paul R. H., Diaz F. and Stabler M., (1990) :*** *Prevalence and etiology of respiratory distress in infants of diabetic mothers. Predictive value of fetal lung maturation tests. Am J. Obstet Gynecol. 163 : 898-903.*

21. ***Piper J. M., Samueloff A. and Langer O., (1995) :*** *Outcome of amniotic fluid analysis and neonatal respiratory status in diabetic and nondiabetic pregnancy J. Reprod Med. 40 : 780-784.*

22. ***Polani P. E. (1974) :*** *Chromosomal and other genetic influence on birth weight variation : Elliot K, Knight J. (eds) : Size at birth Ciba Foundation Symposia vol 27). Amsterdam El-sevier-Excerpta Medica. : 127.*

23. ***Miller H. C. and Merritt T. A. (1979) :*** *Fetal growth in*

humans. Chicago 1L : year book medical.

24. ***Anderson G. D., Blinder I. N., MackClemont and Sinclair J. C. (1984) :*** *Determination of size at birth in a candian population Am. J. Obstet. Gynecol, 150 : 236-244*

25. ***Klebanoff F. M. A., Mills J. L and Brendes H. W. (1985) :*** *Mothers birth weight as a predictor of macrosomia. Am. J. Obstet. Gynecol. 153 : 253-257.*

26. ***Chauhan S. P., Lutton P. M., Bailey K. J., Guerrieri, J. P., and Morrison J., (1992) :*** *Intrapartum clinical sonographic and parous patients estimates of newborn birth weight Obstet. Gynecol., 79 : 956-8.*

27. ***Watson W. J., Soisson A. P. and Harlass F. E. (1988) :*** *Estimated weight of the term fetus. Accuracy of ultrasound versus clinical examination J. Reprod. Med., 33 : 369-371.*

28. ***Benacerraf B. R., Gelman R. and Figoletto F. D. (1988) :*** *Sonographically estimated fetal weights : Accuracy and limitation. Am J. Obstet Gynecol. 63 : 201-204.*

29. ***Landom M. B., Mintz M. C., and Gabbe S. B., (1989) :*** *Sonographic evaluation of fetal abdominal growth : Perdictor of the large for gestational age infant in pregnancies complicated by diabetes mellitus. Am. J. Obst. Gynecol. 60 : 115-121.*

30. ***Ott W. J. (1988) :*** *The diagnosis of altered fetal growth Obstet. Gynecol. Clin North. Am. 15 : 237-63.*

31. ***Defronzo R. A., Ferrannini E, and Koivistio V., (1983) :*** *New concepts in the pathogenesis and treatment of non insulin dependent diabetes mellitus Am J. Med. 17 : 52-81.*

32. ***Mcfarland M. B., Trylovich C. G., and Langer O., (1998) :*** *Anthropometric difference in macrosomic infant of diabetic and non-diabetic mothers. J. Matern, Fetal Med. 7 : 292-295.*

33. ***Friesen C. D., Miller A. M and Rayburn W. F. (1995) :*** *Influence of spontaneous or induced labor on delivering the macrosomic fetus. Am J Perinatal. 12 : 63-66.*