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Case Report

A case of foetal macrosomia

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ABSTRACT

Fetal macrosomia is an upcoming challenge in the field of obstetrics due to its rising incidence. The incidence varies according to ethnicity, genetic differences and anthropometric discrepancies between populations. Obesity, previous history of macrosomia, multiparity, diabetes and post-dated pregnancy are few risk factors associated with macrosomia. Management of macrosomia is a big challenge as no precise guidelines have been set. Macrosomia is associated with multiple maternal and foetal complications like operative delivery, post partum haemorrhage, perineal trauma, shoulder dystocia, brachial plexus injury, skeletal injury, birth asphyxia etc. We report a case of foetal macrosomia, weighing 5.5kg which was delivered by LSCS to a woman having BMI - 26.6kg/m² with 39 weeks of pregnancy with history of previous LSCS. There was no maternal or foetal complication. There was no history of diabetes in present pregnancy and inter conception period. Because of rarity of this condition we report this case of foetal macrosomia with a short review of literature.

Keywords: Foetal macrosomia, Gestational diabetes mellitus, Perinatal Injury, Perineal trauma, Post partum haemorrhage, Shoulder dystocia

INTRODUCTION

Fetal macrosomia defined as birth weight greater than 90% for gestational age is an upcoming challenge in the field of obstetrics due to its rising incidence.¹ The incidence varies according to ethnicity, genetic differences and anthropometric discrepancies between populations. Obesity, previous history of macrosomia, multiparity, diabetes and post-dated pregnancy are few risk factors associated with macrosomia. Management of macrosomia is a big challenge as no precise guidelines have been set. Macrosomia is associated with multiple maternal and foetal complications. These include prolonged obstructed labour due to fetopelvic or cephalopelvic disproportion. There is increased risk of caesarean section, prolonged labour, maternal haemorrhage and perineal trauma.

CASE REPORT

A 30 year old patient second gravida with one living issue at 39 weeks of pregnancy was admitted on 30 May 2019. She was referred from a civil hospital due to big baby and previous LSCS status, which was performed three years back for postdatism with big baby and failure of induction. She had delivered a 4.6 kg female baby and her postpartum period was uneventful.

There was no history of diabetes in previous pregnancy or during inter conception period.

There was no history of fever, rashes, spotting per vaginum, drug intake, and radiation exposure during this pregnancy. There was no history of polydypsia,

polyphagia or polyuria There was no record suggestive of gestational diabetes available. She had no addictions. There was no family history of diabetes mellitus, hypertension or thyroid dysfunction.



Figure 1: The macrosomic baby of 5.5 kg of weight.



Figure 2: The multigravida mother (BMI of 26.6 kg/m²) with the macrosomic baby.

At the time of admission her vitals were within normal limits. There was no pallor, edema, thyroid swelling or any significant lymphadenopathy. Her BMI was 26.6kg/m². No abnormality was detected on respiratory, cardiovascular or CNS examination. Per abdomen

examination - fundal height was term size with foetus in longitudinal lie and cephalic presentation.

Foetal heart rate was 138/min with birth weight clinically 4.5kg with no uterine contractions and scar tenderness. Her hematological, biochemical and serological parameters were normal and random blood sugar was 88 mg%. GCT was 84mg/dl.

She was taken for scheduled LSCS in view of previous LSCS and big baby. A term male baby, large for gestation age with birth weight 5.5kg and APGAR score 8/9 was delivered (Figure 1, 2). Length of the baby was - 59 cm, head circumference - 39cm and chest circumference - 43cm

Baby was kept in NICU for three days for observation. His regular blood sugar charting was done but none of the reading was below 60mg/dl or above 120mg/dl. His investigations - Complete blood with ESR, urine routine, LFT and KFT were within normal range. No abnormality was detected on ultrasound of cranium, liver, gall bladder, spleen and kidneys. Baby was discharged on 4th day. Post partum period of mother was uneven.

DISCUSSION

There is no precise definition of macrosomia. Macrosomia is described as a newborn with an excessive birth weight. According to ACOG foetal macrosomia has been defined in several different ways, including birth weight of 4000-4500g (8 lb, 13 oz to 9 lb, 15 oz) or greater than 90th for gestational age after correcting for neonatal sex and ethnicity (90th percentile).¹ A diagnosis of fetal macrosomia can be made only by measuring birth weight after delivery; therefore, the condition is confirmed only after delivery of the neonate. Fetal macrosomia is encountered in up to 10% of deliveries.¹ The criterion for the definition for macrosomia is related to the maximum birth weight of foetus that the human pelvis can effectively transport from the uterus to the exterior and it depends on pelvic size which varies according to geopolitical regions and level of nutrition.² The international birth weight cut off seems to be high for a country like India where there is poor nutritional support in majority in antenatal period, besides epidemiological studies have shown that Chinese and South Asian countries infants are small for gestational age. The incidence of macrosomia varies according to ethnicity, genetic differences and anthropometric discrepancies between populations. In a study by Koyanagiet al, the 90th percentile of birth weight was 3250g in India and the prevalence of a birth weight of 4kg or greater was 0.5%.³ Thus our case is relatively uncommon.

Numerous endocrinological changes occur in pregnancy to ensure adequate glucose supply to fetus. In pregnancy multiple hormones are involved in producing insulin resistance but it is counteracted by postprandial

hyperinsulinemia in mother. Those who are unable to mount a hyperinsulinemic response, relative hyperglycaemia may develop (gestational diabetes). Glucose crosses the placenta by facilitated diffusion and results in foetal hyperglycaemia which causes hypertrophy and hyperplasia of islet of langerhans of foetal pancreas. This produces foetal hyperinsulinemia with resultant transfer of glucose into foetal cells and accumulation of fat leading to macrosomia. Insulin like growth factors I and II are also involved in foetal growth and adiposity.

A number of risk factors associated with macrosomia have been identified. According to ACOG committee they are as follows in the decreasing order of importance; a history of macrosomia, maternal prepregnancy weight, weight gain during pregnancy, multiparity, male foetus, gestational age >40 weeks, ethnicity, maternal age younger than 17 years and a positive glucose tolerance test (excluding pre-existing diabetes mellitus).⁴ Maternal diabetes is one of the strongest risk factors associated with giving birth to an infant that is considered large for gestational age. Pregestational and gestational diabetes result in fetal macrosomia in as many as 50% of pregnancies complicated by gestational diabetes and in 40% of those complicated by type 1 diabetes mellitus. Our patient hadn't had GDM, however she was multiparous and she had macrosomic baby in previous pregnancy.

Studies of macrosomic infants of diabetic mothers revealed a greater amount of total body fat, thicker upper-extremity skin fold measurements, and smaller ratios of head to abdominal circumference than macrosomic infants of non diabetic mothers.⁵ Also maternal over nutrition and foods with high glycemic index such as sugary beverages, high energy dense carbohydrate diet and fatty diets have been suggested as capable of causing foetal macrosomia.⁶ Our patient had BMI of 26.6 kg/m² with normal blood sugar. Multi-parity and grand multiparity increase the risk of macrosomia. Race and ethnicity are associated with macrosomia. Macrosomia occurs with higher frequency in newborns of Hispanic origin. Because Hispanic women have a higher incidence of diabetes during pregnancy, part of the preponderance of macrosomia in this ethnic group is due to the higher incidence of diabetes in pregnancy. However, even when corrected for diabetes, Hispanic mothers tend to have larger new borns. Fetal sex influences macrosomia potential. Male infants weigh more than female infants at any gestational age. Recent studies have confirmed this association.⁷ The sex of our new born baby was male. Excessive amniotic fluid defined as greater than or equal to 60th percentile for gestational age has recently been associated with macrosomia.⁸

Weighing the newborn after delivery is the only way to accurately diagnose macrosomia, because the prenatal diagnostic methods (assessment of maternal risk factors, clinical examination and ultrasonography measurement

of the foetus) remain imprecise as can be seen in our case. Ultrasonography measurement is considered to be no more accurate than clinical examination.⁹ In our case the expected birth weight by clinical and ultrasound was approximately 4.5kg but after birth it measured 5.5kg.

Macrosomia is associated with multiple maternal and foetal complications. Morbidity and mortality associated with macrosomia can be divided into maternal, fetal, and neonatal categories. A study investigating the effects of birth weight on fetal mortality shows that higher fetal mortality rates are associated with a birth weight of greater than 4250g in nondiabetic mothers and a birth weight of 4000g in diabetic mothers.¹⁰ These include prolonged obstructed labour due to foetopelvic or cephalopelvic disproportion. There is increased risk of caesarean section, prolonged labour, maternal haemorrhage and perineal trauma. The rate of caesarean section significantly increased among the patients who delivered after labour induction as compared to those whose labour was not induced.¹¹ Caesarean delivery is justified in all cases of fetal weight estimation greater than 4500 gm.¹² Maternal complications include: uterine atony (11%), cervix/vaginal laceration (4.9%), uterine rupture (0.4%) and perineal tear (1.7%).¹² Maternal trauma such as obstetric fistulae, are socially devastating. Post partum haemorrhage is a frequent cause of maternal mortality. Neonatal complications such as neonatal asphyxia, skeletal and nerve injuries such as 0.96% for Erb's palsy, Klumpke's palsy, 9.6% for shoulder dystocia, and 1.4% for bone fracture etc may lead to childhood and adult disability as well as death.¹³ In our case no such complications were present due to timely decision taken for LSCS

Management of macrosomia is a big challenge as no precise guidelines have been set. ACOG doesn't support the policy of early induction in suspected macrosomia because induction does not improve maternal or foetal outcome. Results from large cohort study has revealed that it is safe to allow trial of labour for foetus >4kg. While the risk of birth trauma with vaginal delivery is higher with increased birth weight, caesarean delivery reduces, but does not eliminate this risk. Prophylactic caesarean delivery may be considered for suspected foetal macrosomia with estimated foetal weights >5kg in pregnant women without diabetes and >4.5kg in pregnant women with diabetes. Our patient was previous LSCS and big baby (birth weight approximately 4.5kg), so the decision of LSCS was taken. Most effective way to manage macrosomia is by prevention i.e. by improving modifiable risk factors like obesity and gestational diabetes. Weight loss and also reduction in body mass index between the first and second pregnancies can reduce the risk of large for gestational age births.

CONCLUSION

Clinical assessment and ultrasound can diagnose macrosomia but the precise determination of foetal

weight can be done only after delivery. Macrosomia is associated with multiple maternal and foetal complications, so management has to be individualised for every case for favourable outcome. The rate of perinatal and maternal morbidity can be reduced by the antenatal diagnosis, as can be seen in our case.

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