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Case Report

MACROSOMIA - AN OBSTETRICAL CHALLENGE

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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 5.4 kg which was delivered by LSCS to a woman having BMI – 35.2 kg/m² with 41 weeks pregnancy with history of previous LSCS and undetected gestational diabetes. There was no maternal or foetal complication. There was no history of diabetes in previous pregnancy and interconception period. Because of rarity of this condition we report this case of foetal macrosomia with a short review of literature.

Keywords: *Macrosomia*

INTRODUCTION

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. 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.

CASES

A 20 year old patient third gravida with one live child and one abortion with 41 weeks pregnancy was admitted from outpatient department of Hindu Rao Hospital. She was referred from a private centre due to oversize baby and previous LSCS which was performed three years back for postdatism and failure of induction. She had delivered a 3.5 kg female baby and her postpartum period was uneventful.

There was no history of diabetes in previous pregnancy or interconception period. She had a spontaneous abortion of three months gestation followed by dilatation and curettage.

There was no history of fever, rashes, spotting per vaginum, drug intake, and radiation exposure during this pregnancy. No history of polydypsia, polyphagia or polyuria. No record suggestive of gestational diabetes was available.

She was non smoker and non alcoholic and not addicted to any drug. There was no family history of diabetes mellitus, hypertension, thyroid dysfunction, congenital abnormalities, or tuberculosis. At the

time of admission her vitals were within normal limits. There was no pallor, oedema, thyroid swelling or any significant lymphadenopathy.

Her BMI was 35.2 kg/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 5 kg. Mild uterine contractions were present with no scar tenderness. On vaginal examination she was in early labour with poor Bishop's score. Her investigations – Complete blood with ESR, urine routine, random Blood sugar (103 mg%), LFT and KFT were within normal range. HIV, HBsAg and VDRL were non reactive.

She was taken for LSCS in view of postdatism, previous LSCS, large size baby with cephalopelvic disproportion. A term male baby, large for gestation age with birth weight 5.405 kg and apgar score 8/9/9 was delivered (Figure 1). Length of the baby was – 59 cm, head circumference - 39 cm and chest circumference – 43 cm.

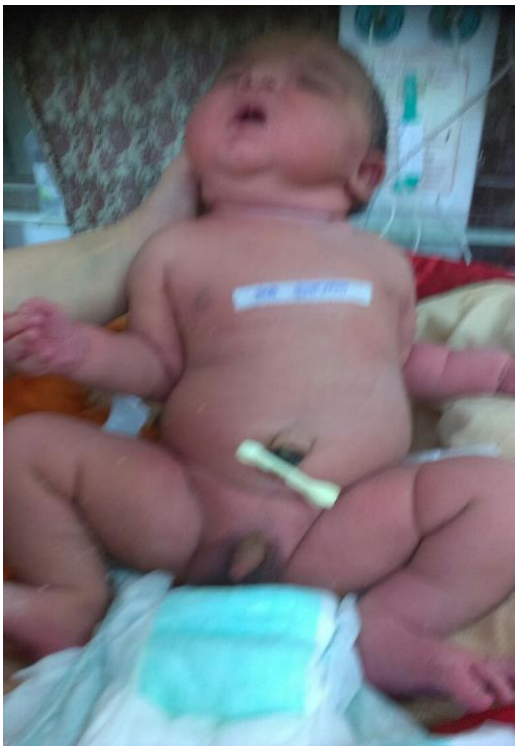

Figure 1: Large for gestation age (B. Wt – 5.4 kg and length – 39 cm)

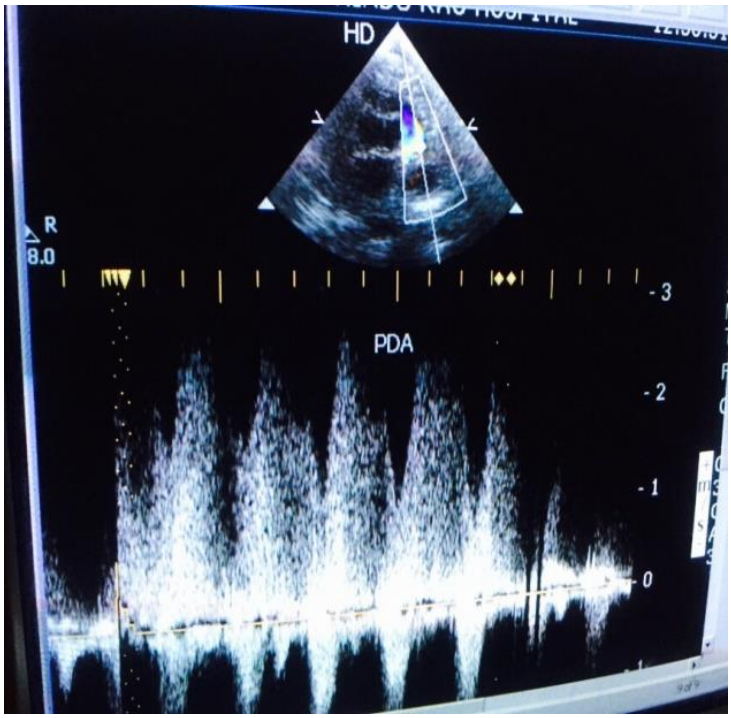

Figure 2: ECHO showing continuous flow (Systolic and Diastolic flow) in pulmonary artery across PDA

Baby was kept in NICU for three days for observation. His regular blood sugar charting was done but none of the reading was below 60 mg% or above 124 mg%. 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. ECHO showed small echo drop out area in mid inter atrial septum region (patent foramen ovale) with small left to right shunt and patent ductus arteriosus (Figure 2). Inter ventricular septum was intact. No significant valvular abnormality was recorded. There was trace TR and PR. Figure 3 shows the colour Doppler of pulmonary artery. Left ventricle showed good contractility with normal dimensions. Baby was discharged on 4th day.

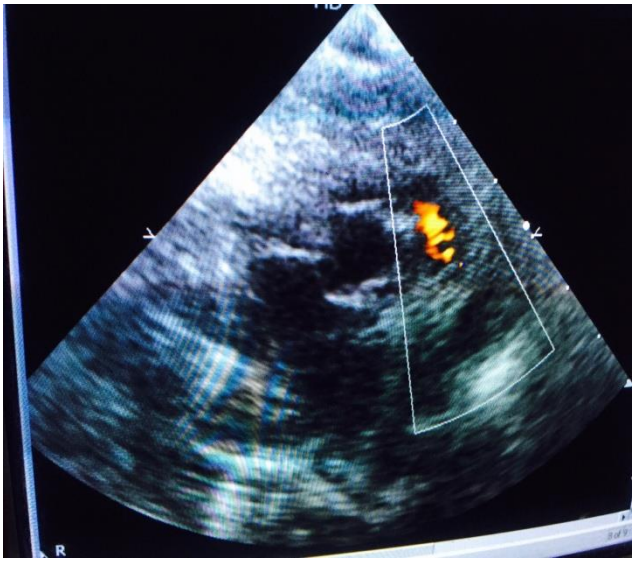

Figure 3: Colour Doppler of Pulmonary artery

In the immediate postpartum period mother was also kept on regular blood sugar charting. Her fasting blood sugar ranged from 90 mg% to 120 mg% while post prandial ranged from 110 mg% to 150 mg%. There was no need of Insulin or oral hypoglycemic drugs. Blood sugar was controlled on diet only. Rest of the postpartum period was insignificant.

DISCUSSION

Foetal macrosomia is an upcoming obstetrical challenge. There is no precise definition of macrosomia. Commonly infants exceeding 90th percentile or greater than 2 standard deviations above the mean weight for any specific gestation age are considered large for gestational age. Such babies comprise of the heaviest 10% of the newborns[1]. According to ACOG the term foetal macrosomia implies foetal growth beyond specific weight, usually 4 kg or 4.5 kg, regardless of foetal gestation age. Results from large cohort studies support the use of 4.5 kg as the weight at which a foetus should be considered macrosomic[2]. 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[3]. 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 Koyanagi *et al.*, (2013), the 90th percentile of birh weight was 3250 g in India and the prevalence of a birth weight of 4 kg or greater was 0.5%.

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 fetus, gestational age > 40 weeks,

ethnicity, maternal birth weight, maternal height, maternal age younger than 17 years and a positive 50 g glucose screen with a negative result on the three hour glucose tolerance test (excluding pre-existing diabetes mellitus. 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. Our patient had excessive weight gain and undetected gestational diabetes during this pregnancy. She was carrying a male foetus along with postdatism (>41 weeks). Anthropometric measurements of the new born are also suggestive of GDM in mother as chest circumference was 4 cm more than the head circumference (Figure 1).

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. Ultrasonography measurement is considered to be no more accurate than clinical examination (Leopold's manoeuvre). In our case the expected birth weight by clinical and ultrasound was approximately 4.5 kg but after birth it measured 5.4 kg.

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 > 5 kg in pregnant women without diabetes and > 4.5 kg in pregnant women with diabetes. Our patient was a case of post-dated pregnancy with previous LSCS and good size baby (birth weight approximately 4.5 kg), 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.

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. Maternal trauma such as obstetric fistulae, are socially devastating post partum haemorrhage is a frequent cause of maternal mortality. Neonatal complications such as shoulder dystocia, neonatal asphyxia, skeletal and nerve injuries such as Erb's palsy, Klumpke’s palsy etc may lead to childhood and adult disability as well as death. In our case no such complications were present due to timely decision taken for LSCS. The intra partum and post partum period were uneventful.

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. Suspected cases of macrosomia can undergo trial of labour but aim should be to minimize maternal and foetal complications. Both the diagnosis and management of macrosomia pose a big challenge in obstetrics world.

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