Macrosomia due to maternal obesity: A case report

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ABSTRACT

Macrosomia, birth weight >4000 g is said to occur in <10% of the pregnancies and is associated with morbidity and mortality in both mother and baby. Although it is more common in diabetic mother, it is also associated with maternal obesity and high-body mass index. We describe the case of near-term male neonate who was delivered by cesarean section and birth weight was 5000 g who developed hypoglycemia and hyperbilirubinemia which was treated successfully.

Key words: Birth weight, Macrosomia, Maternal obesity, Neonate

macrosomic infant is generally defined as one with a birth weight of 4000 g and above irrespective of gestational age or a birth weight >90th centile for that population [1]. The first report of fetal macrosomia in the literature was reported by doctor monk Francois Rabelais in 16th century. Fetal development is regulated by multiple factors comprising genetic, fetal nutrition, environmental factors and two mechanisms, including hormone (insulin) dependent and nonhormone dependent (placental structure, placental circulation, and substrate such as oxygen, glucose, and amino acid delivery), and play an important role. A recent systematic review and metaanalysis confirms that maternal obesity, maternal pre-gestational or gestational diabetes, previous macrosomic birth, post-term pregnancy, male gender, and a high maternal body mass index (BMI >29) are the risk factors for macrosomia [2]. It occurs in 1-10% of all deliveries. Although the prevalence of macrosomia is more in developed countries, nowadays, it is increasing in developing countries, especially in urban population [3]. It not only increases the perinatal morbidity and mortality but also increases the risk of obesity, diabetes, cardiovascular diseases, and cancers in adolescence and in advance age group [4]. There is paucity of data on macrosomia from central India and only sporadic cases are reported in Indian literature. Here, we present a case of macrosomic infant of obese mother.

CASE REPORT

A 25-year-old second gravida without history of still birth or abortion delivered a near-term male neonate by cesarean section with respiratory difficulty and normal Apgar score. On further enquiry, mother was booked case and received adequate antenatal care at primary health-care level. Mother was obese (BMI 30.7 kg/m²) and she gained excess of weight during pregnancy, but she was not diabetic before or during pregnancy.

Her previous baby was average birth weight (2750 g) without any significant perinatal event. Her investigations including blood sugar, glycated hemoglobin, and thyroid profile was within normal limit except polyhydramnios on antenatal ultrasound.

On neonatal intensive care unit (NICU) arrival, baby was normothermic, irritable, tachypnoeic, and mild respiratory distress with SPO2 97%. His weight was 5000 g, length 57 cm, and head circumference 36 cm (length and head circumference were above 90th centile) (Fig. 1). On close monitoring, his respiratory distress was resolved in an hour and hypoglycemia was documented. Systemic examination was within normal limit. Hypoglycemia was managed as per standard guidelines and breast feeding was advised on 3rd day of life. Baby developed jaundice on 4th day of life and treated with double surface phototherapy. On investigation of his hemogram, septic screen, serum calcium, renal function test, electrolytes, chest radiograph, and ultrasound abdomen was within normal limit except two-dimensional



Figure 1: Macrosomic infant with birth weight 5000 g

echocardiography was suggestive of tiny closing patent ductus arteriosus and hypertrophic left ventricle. Baby was treated as per our NICU protocol and discharged. On discharge, baby was well accepting feeds and without any evidence of hypoglycemia.

DISCUSSION

A diagnosis of fetal macrosomia can be confirmed after measuring birth weight after delivery. A cross-sectional study done at Turkey on 10,898 newborns over a period of 4 years reported 4.7% non-diabetic macrosomic infant while Najafian et al. from Iran reported 9% fetal macrosomia on 20,000 live birth deliveries on 5 year cohort [5,6]. Highest prevalence (35-38%) of macrosomia is reported from Quebec Canada in Cree birth [7]. Sporadic cases are reported in Indian literature but there is paucity of data from central India. Macrosomia are subdivided into Class I (birth weight 4000-4499 g), Class II (4500-4999 g), and Class III (≥5000 g) by Gaudet et al. [2]. The reported incidence of Class III macrosomia varies from 0.1% to 1% by various authors [5,6].

Fetal growth can be considered the outcome of an interaction between genetic cause of growth and constraints provided by limitation on substrate availability. Hormonal and non-hormonal mechanisms play an important role of fetal development. Hyperglycemia in the fetus results stimulation of insulin, insulin like as growth factor, growth hormone, and other growth factor, which, in turn, stimulate fetal growth and deposition of fat and glycogen. Classically insulin does not affect brain growth. In studies on macrosomic infants of non-diabetic mothers, a significant relationship was found between birth weight and cord leptin and insulin like growth factor 1 but no relationship with cord insulin level [8].

There is strong association between maternal obesity, history of previous macrosomic infant, weight gain during pregnancy, maternal pre-gestational or gestational diabetes, multiparity, post-term pregnancy, high-maternal BMI, and male gender are reported by various authors [2,5,6]. Macrosomic infants themselves are at elevated risk of shoulder dystocia, brachial plexus injury, skeletal injury, clavicular fracture, perinatal asphyxia, muconeium aspiration syndrome, hypoglycemia, hypocalcemia, hyperbilirubinemia, respiratory distress, and low Apgar score. Still birth rate in macrosomic infants is twice as high as in controls. Mother is also at risk with macrosomic infant and

likely to complicate with post-partum hemorrhage, prolonged labor, cesarean delivery, prolonged hospital stay, infections, and anesthetic events.

CONCLUSION

Although no intervention has been proven to significantly reduce the risk of macrosomia, several potentially useful strategies may be helpful such as tight glucose control before and during pregnancy, prevention of maternal obesity, and thoroughly evaluation of risk factor by clinician can reduce the morbidity and mortality associated with macrosomic infant. It is hoped that with close cooperation of obstetrician, pediatrician and dieticians along with training of mothers, the number of such incidence would be minimized.

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