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Constitutional Macrosomia – A Case Report

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Abstract: *Macrosomia, birth weights greater than 4000g, is said to occurs in about 10% of pregnancies and associated with complications affecting both mother and newborn. It is more common in the infant of the diabetic mother, but does occur in non-diabetic pregnancies. Mortality in this condition is associated with co-existing complications. We describe the case of a term female neonate who was delivered at a weight of 7230g with severe perinatal asphyxia complicated by a necrotising enterocolitis.*
Keywords: *Macrosomia, IDM, Constitutional.*

I. Introduction

Macrosomia has been defined as a specific birth weight above 4000g or 4500g regardless of gestational age.[1] in another definition, it is described as defined as a birth weight greater than the 90th percentile of the intrauterine growth curves of Lubchenco.[2] It affects 1-10% of all pregnancies.[3] The pathophysiology of macrosomia is related to the associated maternal or fetal condition that accounts for its development. Factors that have been implicated include genetics, diabetes (pre-gestational and gestational and the class of diabetes A, B, and C), race, ethnic influences and maternal weight gain.[4]

The incidence is highest in newborns of Hispanic origin, as compared with other races. It's incidence appears to be gradually increasing in Nigeria, though there have been few reported cases.[5] In UITH in 2013, a total of 49 cases of macrosomic babies were seen translating to an incidence of 33.2/1000 admissions.

In spite of associated risk factors, no combination of these factors can accurately predict the occurrence of macrosomia to allow for clinical use. Macrosomia is associated with birth injuries (both trauma and hypoxic injuries), increased incidence of neonatal morbidity and mortality, caesarean deliveries and maternal injuries.

II. Case Report

Baby O.K, a 15hr old female neonate delivered at an estimated gestational age of 40 weeks (Ballard assessment),[6] was admitted following referral from a private hospital outside Ilorin, on account of unusual baby size and poor cry at birth. She (baby) was delivered via emergency caeserian section on account of ruptured uterus following prolonged labour resulting from cephalopelvic disproportion to a 24 year old now $P_3{}^{+0}$ 2 alive woman. No record of the Apgar scores or maternal BMI were provided.

Pregnancy was booked at a primary health care facility and was essentially uneventful. She (mother) was not known to be diabetic before or during pregnancy. Random blood sugar done was said to be normal even though she had had two previous deliveries of large babies whose weight were said to have been greater than 4000g.

Baby presented unconscious with a Blantyre coma score of 2/5, and depressed cortical responses. She had an O_2 saturation of 90% in room air but in respiratory distress, afebrile at 37.2^0C with anthropometric measurements revealing a (7230g) and length (64cm) far above 90th percentile respectively, and her occipito-frontal circumference on the 90th percentile. Systemic examinations revealed tachpnoea with good air entry bilaterally, apparently normal CVS findings and CNS findings in keeping with severe perinatal asphyxia and Stage II hypoxic-ischaemic encephalopathy. Initial assessment was that of a macrosomic infant with SPA HIE II was made and she was managed accordingly. Haematocrit and random blood sugar were normal.

Eight hours into admission, progressive abdominal distension was noted and an impression of gut ischaemia secondary to asphyxia was entertained. A nasogastric tube was passed for bowel decompression and an abdominal x-ray was ordered. The x-ray revealed a gas filled peritoneum; at this point the assessment was changed to a Necrotizing Enterocolitis complicating Severe Perinatal Asphyxia with HIE II and the Paediatric Surgical Unit was invited. Intra-operative findings revealed ceacal and ileal perforations, thickened hypertrophied descending colon, and narrow recto-sigmoid junction with collapsed distal rectum. She had Ileostomy, ceacectomy and a rectal biopsy sample was taken for histology.
Unfortunately, on the 8th day of life (5th day post-operative) she succumbed to her illness.

III. Discussion

In modern obstetrics, fetal macrosomia is a major contributor to obstetric morbidity. It is an important cause of perinatal morbidity and mortality. In a 3-year retrospective study carried out from January 2005 December 2007, Ezeogwu et al found that the incidence of fetal macrosomia in UNTH Enugu was 8.1% with a mean birth weight of 3600 ± 1200g.[5] In UITH the incidence over a year period (2013) is 8.2% with the highest single weight of 7230g as highlighted in the index case. Abdul et al reported, in a cross sectional comparative study that was carried out over a five year period (Jan 2001-Dec 2005), a prevalence of 4.2% in Zaria.

There is strong association (in order of decreasing importance) between previous history of macrosomia, maternal pre-pregnancy weight, weight gain during pregnancy, multiparity, male fetus, gestational age more than 40 weeks, ethnicity, maternal birth weight, maternal height, maternal age younger than 17 years, and maternal pre-gestational diabetes or gestational diabetes.[1,7,8] In the case presented, the macrosomia was likely constitutional as the 2 eldr sibling were also macrosomic at birth.

Fetal growth can be considered the outcome of an interaction between the genetic cause of growth and constraints provided by limitations on substrate availability (selected amino acids, free fatty acids, and mainly glucose). Hyperglycemia in the fetus results in the stimulation of insulin, insulin like growth factors, growth hormone, and other growth factors, which, in turn, stimulate fetal growth and deposition of fat and glycogen. Classically as insulin does not affect brain growth, in the infant of the diabetic mother the head size is spared. Macrosomia in the infant of a diabetic mother result from a poor glucose control, and these infants need to be further investigated as they are at increased risk of intrauterine and extrauterine demise.

Although macrosomia may be associated with adverse maternal and perinatal outcome, there is paucity of recent data regarding fetal macrosomia in our setting. Prolonged labor, shoulder dystocia and injury to infant following instrumental delivery for mid-cavity arrest were the major fetal risks.[9] It was however difficult to ascertain the diabetic status of our patient's mother but there was significant history of deliveries of macrosomic babies in her two previous deliveries.

Complications arising from macrosomia can affect the mother, fetus and neonate. Labour is usually significantly prolonged, and there is associated prolonged hospital stay, primary postpartum haemorrhage and genital tract laceration in women with fetal macrosomia; this was the case with the mother of this patient. Fetal macrosomia usually indicates poor maternal glucose control: hyperglycaemia is a known teratogen, with infants of diabetic mothers being 5 to 8 times more likely to develop congenital anomalies, especially neural tube defects and cardiovascular system anomalies.[10] Still birth rates in macrosomic infants is twice as high as in control subjects.[7] The macrosomic newborn is at risk of both hypoxic (severe birth asphyxia with encephalopathy) and traumatic birth injuries inclusive of which are brachial plexus injuries, shoulder dystocia, clavicular fractures. The incidence of shoulder dystocia was found to range between 19.9% and 50% of pregnancies complicated by diabetes.[1] Other neonatal complications include hypoglycaemia, polycythaemia, hyperbilirubinaemia and respiratory distress syndrome.[2] Necrotising enterocolitis, a known complication that can occur in severe birth asphyxia, has never been described in the face of fetal macrosomia.

IV. Conclusion

The precise determination of fetal weight is only done at delivery. Clinical and ultrasound determination of fetal weight are highly imprecise especially at the third trimester. The route of delivery should therefore be individualized. Identifying the risk factors to fetal macrosomia during antenatal period will be useful to plan appropriate delivery management to optimise good perinatal and maternal outcome.

It should be noted that the majority of large infants are constitutionally large and do not require special intervention, which will result in adverse perinatal outcome. Efforts should therefore be directed to the accelerated (pathologic) overgrown fetus and to methods of primary prevention of this abnormality by appropriate management approaches for the mother and fetus.[11]

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