A retrospective audit of maternal and fetal outcomes associated with fetal macrosomia (≥ 4000 g) at King Edward VIII Hospital from 1st July 2012 to 1st July 2013

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Submitted in Partial Fulfillment of the Academic Requirement for the Degree of the Fellowship of the College of Obstetrics and Gynaecology of South Africa FCOG (Part II)
Declaration

1. Dr Kiresha Naicker, declare that this dissertation entitled “A retrospective audit of maternal and fetal outcomes associated with fetal macrosomia (≥ 4000 g) at King Edward VIII Hospital from 1st July 2012 to 1st July 2013 is my original work and has not been submitted in any form to another university. Where use was made of the work of others, it has been duly acknowledged in the text.

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Dedication

I would like to dedicate my dissertation to:

First and foremost to God Almighty for granting me the knowledge, wisdom and strength to complete this study.

My Parents

My father, Dr Ramakrishnan Soobramoney Naicker. Without his inspiration, guidance and support I may not be the person I am today. For being my best teacher.

My mother, Renuka Devi Naicker who has always been a constant source of love, motivation and strength during my moments of despair and discouragement.

My siblings

Revesh and my dearest sister Sanisha for their patience, encouragement and eternal love. You have been my greatest supporter.

My beloved brother Renesh who is my best friend, angel and inspiration. I wish I will be born being your sister in every life...

My grandparents

For all their love and blessings throughout my life.

My precious pets

For giving me such joy and wonderful memories.
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1. The KZN Department of Health

2. The Biomedical Research Ethics Committee (BREC), UKZN for ethical approval to conduct the study (BE: 055/15)

3. The Manager, King Edward VIII Hospital

4. Department of Postgraduate Research and Education
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List of Abbreviations

CS Caesarean Section
RPR Rapid Plasma Reagin
WHO World Health Organisation
BMI Body Mass Index
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Abstract

Introduction
Pregnancies with a macrosomic fetus are high-risk pregnancies and the incidence appears to be rising. The aim of our research was to identify the clinical profile of mothers who gave birth to macroscopic infants, and to study the maternal and neonatal complications associated with delivering infants with a birth weight of 4000 grams or greater.

Methods
Retrospective study involving a total of 238 deliveries of macrosomic babies from 1st July 2012 to 1st July 2013 at King Edward VIII Hospital, Durban. The study concerned the clinical profile of mothers who give birth to a macrosomic infant including the risk factors, mode of delivery and the incidence of maternal and perinatal complications. Data was analyzed using SPSS (version 23).

Results
Macrosomia occurred in 3.4% of all deliveries. Main risk factors for macrosomia were previous history of macrosomia, male sex, hypertensive disorders of pregnancy, body mass index ≥25, para 1 and 2, diabetes and higher gestational age at delivery. Majority of macroscopic infants were born to non-diabetic women in our audit.

Moreover, macrosomia increased delivery complications for both mothers and newborns. Neonatal complications included: shoulder dystocia was noted in 2.4%, respiratory distress (3.4%) neonatal jaundice (5.1%), and admission to nursery was noted in 99.6% of the cases and for a median duration of 1 day (range 1–11 days). Hypoglycemia complicated 18.6% of deliveries. Twenty (8.4%) infants were resuscitated. The stillbirth rate was 0.4%. Maternal complications included prolonged labour (5.9%), caesarean delivery (64.3%), post- partum haemorrhage documented in 25.2% of cases and perineal tears and cervical lacerations was noted in 34.1% of vaginal deliveries. There was a significant difference in the percentage of neonatal morbidity in the infants delivered vaginally compared to caesarean delivery (48.2% vs 7.8%; p<0.02). No maternal deaths occurred.

Conclusion
The prevalence of macrosomia was 3.4%. Main risk factors for macrosomia were previous history of macrosomia, male sex, hypertensive disorders of pregnancy, body mass index ≥25, para 1 and 2, diabetes and higher gestational age at delivery. Mother and neonate are at increased risk of complications.
Introduction

1.0 Background

Macrosomia is a term used to describe a large fetus or neonate weighing ≥ 4000g at term. The Pedersen’s hypothesis, which was suggested more than sixty years ago, links fetal macrosomia to the transplacental passage of excessive maternal glucose, which leads to fetal hyperglycaemia and excessive fetal insulin release. Since its introduction, the Pedersen hypothesis has been further extended by other investigators and accepted as the pathophysiologic basis for increased risk of macrosomia among infants of women with diabetes during pregnancy.

There is no universally accepted definition of fetal macrosomia. While some clinicians believe that infants with birth weight ≥ 4000 g or above the 90th percentile for the population and sex-specific growth curve can be said to be macrocosmic, others have used birth weight ≥ 4500 g. The American College of Obstetricians and Gynecologists (ACOG) defined macrosomia as birth-weight over 4,000 g irrespective of gestational age or greater than the 90th percentile for gestational age after correcting for neonatal sex and ethnicity. There has been further interest in the group of infants whose birth weight exceeds 5000 g.

1.1 Prevalence of macrosomia

The prevalence of fetal macrosomia varies between 0 -15% but the higher prevalence have been reported in higher income countries compared to low and middle income countries. The prevalence of macrosomia varies in sub-Saharan countries between 1.9 % in Ethiopia and 14.6 % in Nigeria. In Cameroon, its prevalence in 1995 was 6.4 %. In South Africa, the prevalence was 3.4% in 1995. A study from Denmark indicated an increase in the frequency of macrosomia from 16.7% in 1990 to 20.0% in 1999.
A number of risk factors associated with macrosomia have been identified, and include maternal body mass index, weight gain, advanced maternal age, multiparity, diabetes, and gestational age >41 weeks\textsuperscript{17, 18}. However, it is well known that prediction based on clinical risk factors alone and together with first trimester nuchal translucency and biochemical markers (free beta-human chorionic gonadotropin and pregnancy associated plasma protein A has a very low positive predictive value\textsuperscript{19, 20}). Non-modifiable factors include genetics, fetal sex, parity, maternal age and height. Modifiable factors include pre-gestational maternal anthropometric characteristics (BMI), gestational weight gain and maternal glucose metabolism.

Race and ethnicity are associated with macrosomia\textsuperscript{1, 21}. The incidence of macrosomia varies according to ethnicity, and is lower in the Chinese population. This difference in birth weight distribution is likely due to the genetic differences and anthropometric discrepancies between populations. From a recent study, the incidence of macrosomia in Chinese population was reported to be only 3.4\%\textsuperscript{21}.

Factors associated with fetal macrosomia include genetics, duration of gestation, presence of gestational diabetes, and diabetes mellitus types I and II. Genetic, racial, and ethnic factors influence birth weight and the risk of macrosomia\textsuperscript{22}. Maternal diabetes is one of the strongest risk factors associated with giving birth to an infant that is considered large for gestational age. Pre-gestational 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. Esseland Opai-Tetteh(1995) showed that the risk of macrosomia increases with maternal age\textsuperscript{15}.

Primary concern about the birth of a macrosomic fetus is adverse neonatal outcomes including stillbirth and neonatal mortality secondary to birth asphyxia, shoulder dystocia, birth injury, metabolic disorders, and meconium aspiration syndrome. The occurrence of these unfavourable outcomes and their risks factors have been widely studied\textsuperscript{23 - 25}.
Similarly, maternal complications such as increased risk of caesarean delivery, postpartum haemorrhage and perineal lacerations are increased in the setting of fetal macrosomia. Maternal and neonatal complications are shown in boxes 1 and 2.

1.2 Maternal complications

1.2.1 Prolonged labour

The duration of labour is more prolonged for women carrying macrosomic babies, and the risk is increased with increasing birth weight. Both the first and second stages of labour are longer than for normosomic pregnancies, and arrest of descent in the second stage of labour can occur secondary to macrosomia. In a study of macrosomic infants weighing more than 4,500 g, the risk of shoulder dystocia is higher when the second stage is longer than 2 hours, with a crude odds ratio (OR) of 1.17 (95% confidence interval [CI] 0.82–1.66). Prolonged labour associated with macrosomia is, in turn, a contributor to other maternal complications, including operative delivery and postpartum haemorrhage.
1.2. 2 Operative deliveries

The incidences of vaginal operative delivery and caesarean section are higher for macrosomic infants 24, 29, 31, 32. The overall rate of caesarean section in babies with a birth weight >4,000 g varies widely between different studies and ranges from 14% to 44% 33, 34. The risk of caesarean section escalates with increasing birth weight, and the proportion of vaginal instrumental delivery decreases with increasing birth weight 29, 31. The increased risk of caesarean section is a consistent finding in different countries and in different ethnic groups, and the odds are particularly high for primiparous mothers 35. In macrosomic births, the risk of shoulder dystocia is associated with the need for vaginal instrumental delivery 24.

1.2.3 Postpartum haemorrhage

Postpartum haemorrhage occurs more commonly following delivery of macrosomic babies 29, 36 and again, the risk increases with increasing birth weight 31.

1.2.4 Perineal trauma

The risk of perineal tears increases 1.5-fold to 2-fold in cases of macrosomia 37, 38. Some investigators suggest that the incidence of major perineal tear rises significantly with greater birth weight 39 but this has been refuted 31. The risk appears to be higher in Asian, Filipino, and Indian women than in Caucasian women 37. Such differences in the anatomy of the perineum, such as perineal body length and thickness among different ethnic groups, may be contributing factors 40. Major perineal trauma, including third and fourth degree tear, can cause significant long-term anal incontinence, which can have a negative impact on the woman’s quality of life.

1.3 Fetal and neonatal complications

Although the literature frequently and consistently demonstrates an increase in perinatal morbidity and mortality with increasing birth weight, the overall incidence of neonatal complications remains low 40.
1.3.1 Shoulder dystocia

The incidence of shoulder dystocia ranges between 0.58% and 0.70% in Caucasians\textsuperscript{41}. It also appears to vary with ethnicity, with an incidence of only 0.3% in the Chinese population\textsuperscript{42}. It has been reported consistently in the literature that the risk of shoulder dystocia escalates with increasing birth weight\textsuperscript{42–44}. However, the incidence of shoulder dystocia in different birth weight groups varies widely between studies. In a recent study in Norway, the incidence was approximately 1%, 2%, 4%, and 6% for birth weights of 4,000–4,199 g, 4,200–4,399 g, 4,400–4,599 g, and ≥4,600 g\textsuperscript{44}, respectively, whereas another study reported an incidence of over 20% when the birth weight was above 4,500 g. Nevertheless, despite such an association, half or even more of the births complicated by shoulder dystocia occur in babies with a birth weight less than 4,000 g\textsuperscript{42}.

1.3.2 Birth trauma

The incidence of birth trauma, namely brachial plexus and skeletal injuries, increases with rising birth weight\textsuperscript{25,29}.

1.3.3 Brachial plexus injury

Congenital brachial plexus injury is defined as flaccid paresis of an upper extremity due to traumatic stretching of the brachial plexus at birth, with passive greater than active range of motion. The incidence varies between countries and is approximately 1.5 cases per 1,000 live births\textsuperscript{42,45}.

Brachial plexus injury is characteristically related to shoulder dystocia; however, such complications can occur following normal spontaneous vaginal delivery and caesarean section\textsuperscript{46}. Both excessive exogenous traction and strong endogenous pushing forces contribute to brachial plexus injury BPI\textsuperscript{47}. The second most important risk factor for BPI is heavy birth weight\textsuperscript{43}, which is associated with a 14-fold increase in risk\textsuperscript{45}. In one study, the prevalence of BPI progressively increased with infant weight, occurring in only 3% of neonates in the 4,500–5,000 g group and 6.7% in the >5,000 g group\textsuperscript{48}.
Moreover, the risk is further increased when macrosomia and gestational diabetes coexist, with an adjusted OR of 42 (95% CI 4.05–433.64).

It has also been reported that BPI among infants weighing ≥4,000 g is more likely to be severe and persistent than in the normosomic group. Because the two main risk factors for congenital BPI, i.e., shoulder dystocia and macrosomia, are not easily predictable, it is difficult to foresee and prevent its occurrence.

1.3.4 Skeletal injuries

Skeletal injuries commonly occur in the presence of shoulder dystocia and are associated with large infants. Fracture of the clavicle is five times more common in macrosomic infants, and occurs more often in vaginal delivery than in caesarean section. Humeral fractures are less frequent, but also occur in big babies. Gregory et al in 1998 analysed neonatal complications following shoulder dystocia and reported that, unlike brachial plexus injury, the risk of having skeletal injuries in macrosomic infants is not higher than in those with normal birth weight. Clavicular fractures are usually managed conservatively and the outcome is most often benign, with complete recovery and no associated neurologic complications. Humeral fractures are managed mainly by closed reduction followed by splinting or traction techniques, and usually do not have long-term sequelae.

1.3.5 Chorioamnionitis

Significant maternal and neonatal complications can result from the birth of a macrosomic infant, and includes chorioamnionitis. The risk of chorioamnionitis slowly and steadily increases as birth weight increases, and the ORs are 1.94, 2.17, and 2.42 for birth weight groups of 4,000–4,499 g, 4,500–4,999 g, and ≥5,000 g, respectively.

1.3.6 Aspiration of meconium

Aspiration of meconium is a risk associated with macrosomia. Again, the risk increases with rising birth weight. The ORs are 1.28, 1.65, and 2.61 for babies with birth weights of 4,000–4,499 g, 4,500–4,999 g, and >5,000 g, respectively. However, other investigators reported that the association was not statistically significant.
1.3.7 Perinatal asphyxia

The risk of macrosomic neonates suffering from perinatal asphyxia increases 2–4-fold compared with that in normosomic infants\textsuperscript{23, 40}. The odds of perinatal asphyxia increase considerably with rising birth weight; in one study, the OR was 2.3 if birth weight was 4,500–4,999 g and increased further to 10.5 if birth weight was >5,000 g\textsuperscript{25}.

1.3.8 Poor Apgar scores

There are reports that Apgar scores are poor in infants with macrosomia. The greater the birth weight, the higher the risk of low Apgar scores\textsuperscript{25, 29}. Boulet et al in 2003 showed the OR for a 5-minute Apgar score ≤6 was 1.65 and 3.49 for infants with birth weight 4,500–4,999 g and >5,000 g, respectively, whereas that for a 5-minute Apgar score ≤3 was even higher, with corresponding ORs of 2.01 and 5.20\textsuperscript{29}. Furthermore, the risk of a low Apgar score is eight times higher in macrosomic babies when the delivery is complicated by shoulder dystocia\textsuperscript{24}. In contrast, Weissmann-Brenner et al in 2012 could not demonstrate any statistically significant difference in low Apgar scores between normal and big babies\textsuperscript{31}.

1.3.9 Neonatal hypoglycemia

The risk of neonatal hypoglycemia is higher in heavy babies\textsuperscript{43}, and the risk increases with increasing birth weight. Neonates with a birth weight >4,500 g had a seven-fold higher risk of having neonatal hypoglycemia, compared with those appropriate for gestation age\textsuperscript{51}. This risk further increases in the presence of gestational diabetes. Infants with a birth weight ≥4,000 g delivered by no diabetic mothers had a 2.4% risk of neonatal hypoglycemia, whereas those whose mothers had gestational diabetes had an incidence of 5.3%\textsuperscript{43}.

1.3.9.1 Intrauterine fetal death

Macrosomia has been consistently shown to be associated with a 2–3-fold increase in intrauterine fetal death\textsuperscript{52}.
Zhang et al in 2008 showed that birth weights of 4,000–4,499 g were not at increased risk of mortality compared with those born at 3,500–3,999 g; however, those born at 4,500–4,999 g had a significantly increased risk of stillbirth (OR 2.7, 95% CI 2.2–3.4) and the risk rose dramatically with a birth weight $\geq$5,000 g (OR 13.2, 95% CI 9.8–17.7)$^{25}$. Because maternal diabetes is closely related to macrosomia and fetal death, Mondestin et al in 2002 addressed this complex interaction and showed that the fetal death rate increased in macrosomic fetuses in both diabetic and non-diabetic pregnancies, but the cut off birth weight was different, being $\geq$4,250 g in non-diabetic women and $\geq$4,000 g in their diabetic counterparts$^{53}$.

1.3.9.2 Neonatal and infant mortality

Numerous epidemiologic studies have shown a distinct relationship between birth weight and neonatal and infant mortality, and have consistently demonstrated a reverse J pattern of weight-specific mortality in all populations, where the mortality rates increase at the extremes of birth weight$^{54}$. Compared with a normosomic group of infants with a birth weight of 3,000–3,999 g, babies with a birth weight $>5,000$ g had a 2–3-fold increase in risk of neonatal death, and a 1.6–2.0-fold increased risk of post neonatal and infant mortality, respectively. Such an association was not identified in babies with a birth weight of 4,000–4,999 g$^{29}$. However, a recent study by Zhang et al in 2008, which included close to 6 million births from the USA, showed that neonates with a birth weight $>4,500$ g also had a higher early neonatal death rate (OR 1.8), but there was no increase in late or post neonatal death$^{25}$. Early, late, and post neonatal deaths were all significantly increased in those weighing $\geq$5,000 g, with ORs of 6.4, 5.2, and 2.3, respectively. The leading cause of early neonatal death in macrosomic babies was asphyxia.

Sudden infant death syndrome is another concern for macrosomic babies, but the current data are conflicting. The majority of post neonatal deaths reported by Zhang et al in 2008 were due to sudden infant death syndrome. Infants with a birth weight $\geq$5,000 g have a more than 2-fold increase in risk$^{25}$. However, such a detrimental effect was not identified in other studies, and excessive intrauterine growth (birth weight $>90$th percentile) has even been shown to have a protective role in sudden infant death syndrome$^{55}$. 

8
1.4 Long-term complications

The Barker hypothesis explains the concept of fetal programming in utero, such that events during early development have a profound impact on the risk for development of future adult disease. Birth weight has been shown to be predictive of a number of adult diseases, such as hypertension, obesity, and insulin resistance. Alternative explanations for the association between fetal growth and later diseases, mainly genetic factors, have also been proposed.

Increased birth weight has been shown to have a positive association with overweight, insulin resistance, and metabolic syndrome in later life. The risk of developing metabolic syndrome in childhood is highest when there is coexistence of macrosomia and maternal gestational diabetes, and is comparatively less marked in the group with macrosomia alone.

Interestingly, breast cancer has been found to be associated with high birth weight in numerous studies. Those with particularly high birth weight (≥4,500 g) had the most pronounced elevation in risk (OR 3.10, 95% CI 1.18–7.97). It is postulated that this association is mediated in part by hormonal mechanisms that positively influence fetal growth and mammary gland development.

1.5 Prenatal diagnosis of fetal macrosomia

Prenatal estimation of fetal weight is notoriously known to be inaccurate, with errors exceeding 10% of the actual birth weight. In fact, sonographic estimates of birth weight are no better than clinical assessment. The sonographic detection of macrosomic infants >4,000 g is even more unreliable, with a low sensitivity, low positive predictive value. Different formulae for estimated fetal weight have been evaluated and the prediction of macrosomia is poor. The mean detection rates for fetuses with a birth weight of ≥4,000 g, ≥4,300 g, and ≥4,500 g were 29%, 24%, and 22%, respectively, and false positive rates were 12% (for ≥4,300 g) and 7% (for ≥4,500 g). Moreover, many researchers have developed additional assessment methods to improve the detection of macrosomia, including two-dimensional and three-dimensional assessment of fetal subcutaneous and soft tissue.
However, these methods are more time-consuming and technically demanding. Recently, a new formula has been shown to be superior to the traditional formulae for prediction of macrosomia, where 78% of estimates fell within ±5% of the actual weight at birth, 97% within ±10%, and 100% within ±15% and ±20%.

1.6 Management of fetal macrosomia

The management of suspected fetal macrosomia continues to be an obstetric challenge. This is due to the inaccuracy of prenatal clinical or sonographic diagnosis as discussed above, and also because of the difficulty in prediction of its complications during labour, in particular, the risk of shoulder dystocia.

The most effective way to manage macrosomia is probably by prevention. Two of the most important risk factors for macrosomia which can be modifiable are maternal obesity and gestational diabetes. The risk of macrosomia increases with the severity of maternal obesity. 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. Achieving optimal glycaemic control in diabetic women, especially postprandial glucose control, can also prevent macrosomia and reduce the incidence of shoulder dystocia and birth trauma.

The idea of inducing labour for suspected macrosomia before the baby grows too big, with an aim to reduce operative deliveries and birth trauma, has not been supported by clinical evidence. Induction of labour for suspected macrosomia in non-diabetic women has not been shown to improve either maternal or neonatal outcome. On the other hand, because women with diabetes have a higher risk of shoulder dystocia and birth trauma, the National Institute for Health and Care Excellence guideline currently suggests that pregnant women with diabetes should be offered elective birth by induction of labour after 38 weeks of gestation.
Whether elective caesarean section should be performed to prevent BPI is another controversial issue. It has been estimated that 443 caesarean sections are required to prevent one permanent BPI in diabetic women with an estimated fetal weight >4,500 g, and an exceedingly high number (3,695) of caesarean sections are needed to prevent one permanent BPI in the non-diabetic population\(^6^9\).

The Royal College of Obstetricians and Gynaecologists and the American College of Obstetricians and Gynaecologists recommend elective caesarean delivery in diabetic and non-diabetic women with estimated fetal weight >4,500 g and >5,000 g, respectively\(^4^1,\)^\(^7^0\). However, these guidelines may not be appropriate for the Asian population because the birth weight cut-off is too high\(^4^2\).

### 1.7 Rationale

Since there has not been any study conducted on macrosomic newborns in Kwa Zulu Natal, the present study will help to understand the prevalence, risk factors and maternal, fetal and neonatal complications of macrosomic newborns in the region. It will also draw attention of policy makers to improve the maternal and child health status in the region along with helping the fight for the present obstetrics challenges in Kwa Zulu Natal. It will therefore contribute to the academic discourse on reproductive health within the discipline of public health and most likely will come up with the ideas for future research on the subject. Considering increased risks of complications related to delivery of macrosomic fetuses the aim of this research was to determine the incidence, risk factors and perinatal outcome associated with giving birth to macrosomic babies weighing four or more kilograms.

### 1.8 Objectives

1. The profile of pregnant women with risk factors for fetal macrosomia
2. The maternal outcome associated with fetal macrosomia
3. The fetal outcome associated with fetal macrosomia

### 1.9 Hypothesis

Fetal macrosomia is associated with an increased risk of maternal and fetal complications
Methodology

2.0 Study Location

King Edward VIII Hospital, Durban, Kwazulu Natal.

2.1 Study period

1st July 2012 to 1st July 2013.

2.2 Study Design

A retrospective chart audit. All information was obtained from chart reviews.

2.3 Study Population

All women who delivered macrosomic infants at King Edward VIII Hospital from 1st July 2012 to 1st July 2013 were included.

2.4 Sample Size

Using a single proportion formula with degree of confidence of 1.65 and prevalence of 3.4% as according to the previous study 15, 50 mothers were required as study subjects. However a total of 238 mothers were enrolled in this study.

2.5 Inclusion Criteria

Singleton pregnancies

Gestational age of term pregnancies (37 to 42 weeks)

2.6 Exclusion Criteria

Multiple pregnancies

Pregnancies complicated by intrauterine growth restriction

Patients with incomplete data were excluded

2.7 Data collection

Demographic, obstetrical characteristics and maternal, fetal, neonatal, and pregnancy outcomes of macrosomic infants were recorded in a structured format
The following parameters were recorded in a structured format (Appendix 1).

- Demographic profiling
- Co morbidities
- Socio-economic status
- Past obstetric history
- Maternal complications
- Neonatal complications.
- Length of mothers stay in hospital

BMI was categorized in the following groups, according to the Guidelines of American Clinics for the identification, evaluation and treatment of obesity and overweight in adults: normal weight (BMI 18.5–24.9 kg/m²), overweight (BMI 25–29.9 kg/m²) and obese (BMI ≥30 kg/m²) 71. Age was grouped as follows: < 25 years, 25-35 years and > 35 years.

**2.8 Statistical Planning**

Data were captured on a customised MS Excel spread sheet and analysed using SPSS version 23. The Pearson’s correlation coefficient was utilised for the correlation between weight of the newborn and gestational age at delivery or neonatal complications. A p value <0.05 was considered significant.

**2.9 Ethical Consideration**

Ethics approval was obtained from the University of KwaZulu Natal Biomedical Research Ethics Committee (BE: 055/15) for a retrospective review of data, analysis and publication.
Results

3.0 Demographic data

There were 6932 deliveries during the one year study period. Of this 238 were macrosomic deliveries. The prevalence of macrosomic newborns was 3.4%. The mean age of mothers that had macrosomic babies was 26.6 ± 5.8 (range: 14 – 41) years. Eighty nine (37.4%) mothers were aged ≤ 20 years, 122 (51.3%) were aged between 21 – 30 years, 26 (10.9%) were aged between 31 – 40 years and one (0.4%) was aged between 41-50 years. The maximum frequency of macrosomic births occurred in women in the 21-30 year age group. There was no significant difference in the mean birth weight of macrosomic babies with shoulder dystocia and those without shoulder dystocia (4180.7 ± 278.7g vs. 4204. 0 ± 188.6g, \( P = 0.64 \)).

Body mass index was categorized as follows: Two (0.8%) were of normal BMI, 90 (36.2%) mothers were overweight and 159 (63%) were obese. The mean (SD) parity was 1.3 ± 1.2 (range: 0 – 7). Seventy two (30.3%) of the mothers were para 0, 132 (55.5%) were between para 1 and para 2, 30 (12.6%) were between para 3 and para 4 and 4 (1.6%) between para 5 and para 7. The mean (SD) gravidity was 2.4 ± 1.3 (range: 1 – 8). Two hundred and thirty four (98.3%) mothers attended antenatal care with a mean number of antenatal visits of 6.6 ± 2.5 (range: 1 – 16). The mean gestational age at booking for antenatal care was 21.3 ± 7.4 (range 4 – 40) weeks. The mean (SD) gestational age at delivery of 209 (87.8%) mothers was 39.7 ± 1.2 (range: 37 – 42) weeks. In 29 (12.2%) mothers gestational age at delivery was 42+ weeks. (Table 1)
### Table 1: Patient demographic profiles

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number (N=238)</th>
<th>Percentages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (26.6 ± 5.8 (range: 14 – 41) years.</strong></td>
<td></td>
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<tr>
<td>Age groups</td>
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<td><strong>Body mass index (35.6±6.4) range (24 - 61)</strong></td>
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<td><strong>Parity: 1.3 ± 1.2 (range: 0 – 7)</strong></td>
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<tr>
<td>1 – 2</td>
<td>132</td>
<td>55.5</td>
</tr>
<tr>
<td>3 – 4</td>
<td>30</td>
<td>12.6</td>
</tr>
<tr>
<td>5 – 7</td>
<td>4</td>
<td>1.6</td>
</tr>
<tr>
<td><strong>Gravid 2.4 ± 1.3 (range: 1 – 8)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 - 2</td>
<td>197</td>
<td>82.8</td>
</tr>
<tr>
<td>3 – 4</td>
<td>23</td>
<td>9.7</td>
</tr>
<tr>
<td>5 – 8</td>
<td>18</td>
<td>7.6</td>
</tr>
<tr>
<td><strong>Antenatal visits: 6.6 ± 2.5 (range: 1 – 16)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 - 4</td>
<td>46</td>
<td>19.3</td>
</tr>
<tr>
<td>5 - 8</td>
<td>154</td>
<td>64.7</td>
</tr>
<tr>
<td>9 - 16</td>
<td>34</td>
<td>14.3</td>
</tr>
<tr>
<td><strong>Gestational age (weeks)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At booking 21.3 ± 7.4 (range 4 - 40)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At term(n=209) 39.7 ± 1.2 (37 – 42)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At post term (n=29) 42+</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3.1 Laboratory variables

The mean (SD) haemoglobin was 10.9 ±1.2 (range: 7.5- 14.5). Two hundred and thirty five (98.7%) was rhesus positive. The RPR was negative in 98.3% of the patients. One hundred and sixty (67.2%) were HIV negative.

Table 2: Laboratory variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number n=238</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Haemoglobin:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mean : 10.9 ±1.3) (range: 7.5- 14.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Rhesus factor</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>235</td>
<td>98.7</td>
</tr>
<tr>
<td>Negative</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td><strong>RPR</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>4</td>
<td>1.7</td>
</tr>
<tr>
<td>Negative</td>
<td>234</td>
<td>98.3</td>
</tr>
<tr>
<td><strong>HIV status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td>160</td>
<td>67.2</td>
</tr>
<tr>
<td>Positive</td>
<td>78</td>
<td>32.8</td>
</tr>
</tbody>
</table>

3.2 Obstetric condition at presentation

Table 3 lists the 95 (39.9%) mothers who presented with obstetric conditions: 3 (1.3%) mothers were diabetic and 2 (0.8%) mothers developed gestational diabetes but of the medical conditions in pregnancy, hypertensive complications were the main problem.
Table 3: Obstetric condition at presentation

<table>
<thead>
<tr>
<th>Obstetric condition at presentation</th>
<th>Frequency n=95</th>
<th>% of the total macrosomic population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Gestational diabetes</td>
<td>2</td>
<td>0.8</td>
</tr>
<tr>
<td>Previous CS (x1 and 2 or more)</td>
<td>45</td>
<td>18.9</td>
</tr>
<tr>
<td>Hypertension complications of pregnancy (n=21)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-eclampsia</td>
<td>12</td>
<td>5.0</td>
</tr>
<tr>
<td>Gestational hypertension</td>
<td>9</td>
<td>3.8</td>
</tr>
<tr>
<td>Abruptio placenta</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Multifibroid uterus</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Breech</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>High body mass index</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Termination of pregnancy</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Advanced maternal age</td>
<td>4</td>
<td>1.7</td>
</tr>
<tr>
<td>Asthma</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Polyhydramnios</td>
<td>5</td>
<td>2.1</td>
</tr>
<tr>
<td>Teenage pregnancy</td>
<td>6</td>
<td>2.5</td>
</tr>
</tbody>
</table>

3.3 Socio-economic variables of the mothers

Majority of the mothers were single (90.3%); 14.3% were employed and 0.8% consumed alcohol. Among mothers, 0.4% had smoking habits. Details of the socio-economic variables are shown in Table 4.
Table 4: Socio-economic variables of the mothers

<table>
<thead>
<tr>
<th>Socioeconomic details</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Employment</td>
<td>34 (14.3%)</td>
<td>204 (85.7%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>1 (0.4%)</td>
<td>237 (99.6%)</td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td>2 (0.8%)</td>
<td>236 (99.2%)</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>215 (90.3%)</td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>23 (9.7%)</td>
<td></td>
</tr>
</tbody>
</table>

3.4 Previous macrosomic history

Thirty five (14.7%) mothers gave previous history of delivering macrosomic infants. All mothers delivered at term. Neonatal outcome showed that 227(95.4%) were live births, 10 (4.2%) stillbirths and one (0.4%) neonatal death.

3.5 Co-morbidities

Co-morbidities are listed in Table 5. Four (1.7%) had asthma, twenty one (8.8%) had hypertensive complications of pregnancy, two (0.8%) had thyroid disorder, three (1.3%) had diabetes and two (0.8%) had tuberculosis successfully treated.

Table 5: Co-morbidities

<table>
<thead>
<tr>
<th>Co-morbidities (n=33)</th>
<th>Number (n)</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>4</td>
<td>1.7</td>
</tr>
<tr>
<td>Anaemia</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Peripartum cardiomyopathy</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Hypertensive complications of pregnancy</td>
<td>21</td>
<td>8.8</td>
</tr>
<tr>
<td>Thyroid disorder</td>
<td>2</td>
<td>0.8</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3</td>
<td>1.3</td>
</tr>
</tbody>
</table>
3.6 Maternal outcomes
Labour was induced in 13.4% of the mothers. Fourteen (5.9%) mothers experienced prolonged second stage of labour and shoulder dystocia occurred in 2.4% of the deliveries.

3.6.1 Mode of delivery
One hundred and fifty three (64.3%) delivered by CS, 109 (71.2%) by emergency CS and 44 (28.8%) by elective CS. Eighty five (35.7%) delivered by normal vaginal delivery. Elective episiotomy was done in most cases of vaginal deliveries. The three main indications for CS were previous CS (29.4%), fetal distress (27.5%) and cephalo-pelvic disproportion (14.4%). Indications for CS are shown in Table 6.
Table 6: Indications for CS

<table>
<thead>
<tr>
<th>Indications for CS (n=153)</th>
<th>Number (n)</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Emergency CS (n=109)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fetal distress</td>
<td>42</td>
<td>27.5</td>
</tr>
<tr>
<td>Cephalo pelvic disproportion</td>
<td>22</td>
<td>14.4</td>
</tr>
<tr>
<td>Poor progress</td>
<td>8</td>
<td>5.2</td>
</tr>
<tr>
<td>Ante partum haemorrhage</td>
<td>4</td>
<td>2.6</td>
</tr>
<tr>
<td>Previous CS</td>
<td>11</td>
<td>7.2</td>
</tr>
<tr>
<td>MSL 2/3</td>
<td>6</td>
<td>3.9</td>
</tr>
<tr>
<td>Failed induction</td>
<td>3</td>
<td>1.9</td>
</tr>
<tr>
<td>Failed VBAC</td>
<td>3</td>
<td>1.9</td>
</tr>
<tr>
<td>Breech</td>
<td>5</td>
<td>3.3</td>
</tr>
<tr>
<td>Delayed 2\textsuperscript{nd} stage</td>
<td>3</td>
<td>1.9</td>
</tr>
<tr>
<td>Gestational hypertension</td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td>Preeclampsia</td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td><strong>Elective CS (n=44)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous CS</td>
<td>34</td>
<td>22.2</td>
</tr>
<tr>
<td>Failed induction of labour</td>
<td>4</td>
<td>2.6</td>
</tr>
<tr>
<td>Big baby</td>
<td>6</td>
<td>3.9</td>
</tr>
</tbody>
</table>

3.6.2 Maternal complications

Overall, 58 patients (24.4%) presented with maternal complications. First degree perineal tears occurred in 9 (10.6%) women, 2nd degree in 19 (22.4%), cervical lacerations in 1 (0.4%). Twenty nine (12.2%) experienced postpartum hemorrhage. (Table 7)
### Table 7. Complications experienced by mothers following delivery of macrosomic babies either by normal vaginal delivery and caesarean delivery

<table>
<thead>
<tr>
<th>Normal vaginal delivery (n=85)</th>
<th>Number (n)</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st degree tear</td>
<td>9</td>
<td>10.6</td>
</tr>
<tr>
<td>2nd degree tear</td>
<td>19</td>
<td>22.4</td>
</tr>
<tr>
<td>3rd degree tear</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cervical lacerations</td>
<td>1</td>
<td>1.2</td>
</tr>
</tbody>
</table>

Post-partum haemorrhage 12 14.1

(2nd degree tear (n=8); cervical laceration (n=2); unknown (n=1) and episiotomy (n=1))

### Caesarean delivery (n=153)

| Post-partum haemorrhage | 17 | 11.1 |

(elective CS (n=2) and emergency CS (n=15))

### 3.7 Neonatal outcome

There were 237 (99.6%) live birth infants and one (0.4%) stillbirth. There was a preponderance of male infants with macrosomia with the male to female ratio of 2.0 to 1. One hundred and fifty three (64.3%) of the infant were male and 85 (35.7%) were female.

Birth weight ranged between 4000 and 5500 g. The majority (92%) of newborns had a birth weight between 4000 and 4499 g (Table 8), the mean and median birthweight was 4201 ± 201.8 g and 4160 g respectively. Subgroup analysis showed that there was no difference in the mean birth weight of macrosomic babies delivered by CS compared to macrosomic babies delivered vaginally (4210.3 ± 212.5 g vs 4178.5 ± 172.0g; p=0.2).
<table>
<thead>
<tr>
<th>Variable</th>
<th>Number (n)</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight (gm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4000 – 4499</td>
<td>219</td>
<td>92.0</td>
</tr>
<tr>
<td>4500 – 4999</td>
<td>17</td>
<td>7.2</td>
</tr>
<tr>
<td>≥5000</td>
<td>2</td>
<td>0.8</td>
</tr>
<tr>
<td>Sex of newborn</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>153</td>
<td>64.3</td>
</tr>
<tr>
<td>Female</td>
<td>85</td>
<td>35.7</td>
</tr>
</tbody>
</table>

The 5 minute Apgar score was greater than 7 in 98.8% of cases. Eighty (33.8%) macrosomic infants experienced complications (Table 9). Two hundred and thirty seven (99.6%) newborns were admitted to nursery for observations and as a precautionary measure for a median duration of 1 day (range 1–11). Twenty (8.4%) infants were resuscitated. Commonly observed complications were respiratory distress (3.4%), hypoglycemia (18.6%) and neonatal jaundice (5.1%). The stillbirth rate in the macrosomic infants was 0.4%, but no maternal deaths occurred. One of the macrosomic infants died at two years.
Table 9: Neonatal complications observed in macrosomic infants

<table>
<thead>
<tr>
<th>Neonatal complications</th>
<th>Number (n)</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meconium stained liquor (2/3)</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Erbs palsy</td>
<td>2</td>
<td>0.8</td>
</tr>
<tr>
<td>Birth trauma</td>
<td>2</td>
<td>0.8</td>
</tr>
<tr>
<td>Transient tachypnea of the newborn</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Cardiac abnormality</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Sepsis</td>
<td>2</td>
<td>0.8</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>44</td>
<td>18.6</td>
</tr>
<tr>
<td>Respiratory distress syndrome</td>
<td>8</td>
<td>3.4</td>
</tr>
<tr>
<td>Neonatal jaundice</td>
<td>12</td>
<td>5.1</td>
</tr>
<tr>
<td>Rapid plasma reagin exposure</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Neonatal seizures</td>
<td>2</td>
<td>0.8</td>
</tr>
</tbody>
</table>

No correlation was found between the weight of the newborn and the different parameters. However correlation was found between weight of newborn and gestational age at delivery (R=0.172; p=0.008). Moreover, macrosomia increased neonate hypoglycemia and CS delivery.
4.0 Discussion

The main risk factors for macrosomia in our study were delivery of a previous macromomic baby, hypertensive disorders of pregnancy, male sex, BMI > 25 kg/m², parity ≥1, diabetes and increased gestational age at delivery. This is consistent with risk factors with regards to previous delivery of macromomic babies 72, 73, postmaturity 72, 74, 75, diabetes 76 - 78 and increased BMI 79, 80.

It has been reported that 38 – 40% of macromomic babies are born to mothers with at least one identifiable risk factor 81. Strehlow et al (2007) in this study reported that fewer than 40% of mothers had at least one risk factor for macromomia 82. In addition, in contrast to findings in other studies, maternal age and parity 17, 81, 83, were not significantly associated with macromomic deliveries in our study. This may be due to the small size of the study population or the influence of genetic, racial or ethnic factors 22.

Diabetes, pre-existing or gestational diabetes has been reported to be between 1-2% in the mothers of macromomic babies 84 with incidence increasing to 5–7% with births of 4500 g and greater 81, 85. Some studies have reported incidence of diabetes as high as 12.7 -19.5 % 1, 86. In our study, 2.1% of our patients who delivered macromomic babies had pre-existing diabetes and gestational diabetes.

The maternal complications were high in our audit compared to other studies. The overall maternal complications in this study was 24.4% (n=58) which is much higher than the reported overall rate of 3.1 – 7.3% 87, 88. Main maternal complications in this study were postpartum hemorrhage, perineal tears and cervical lacerations in 25.2%, 33% and 1.2% respectively versus 1.2%, 1.7% and 0.7% in an alternate study 88. However, another study reported postpartum haemorrhage and perineal tears in 17% and 37% patients respectively 87. Perineal tears and postpartum hemorrhage increases 2 and 3-5 fold respectively in macromomic deliveries 37, 89.

The overall complications in our macromomic infants was 33.6% (n=80) in this study which is in accordance with other studies 90, 91. Complication rates as low as 5.3- 16% 1, 43, 92, 93 and high as 44.3 – 88% have been reported 10, 75. The frequency of neonatal hypoglycaemia was the most common complications reported in majority of the studies. In our study neonatal hypoglycemia occurred in 18.6% cases compared to 34% in another study 94.
Stillbirth rate (0.4%) is much lower than reported in other studies, 6 – 12% \(^{95, 96}\). The low stillbirth rate in this study is similar to rate in a recent study, 1.3% \(^{86}\). The low stillbirth rate was probably due to knowledgeable anticipation and astute supervision with timely decision on the labour and delivery process and was vital to a desirable outcome.

The incidence of fetal macrosomia in our study was 3.4% which was similar to other studies \(^{15, 86}\), lower than the rates of 5.5- 10% reported elsewhere \(^{73, 74, 97, 98}\) but higher than 1.3 – 2.3 % \(^{75, 99}\). These differences in incidence may be due to differences in the definition of fetal macrosomia, differences in geographical and socioeconomic factors of the study population.

Shoulder dystocia, one of the main perinatal difficulties with the delivery of macrosomic babies, occurs infrequently with an incidence ranging from 0.2–9.5% of all vaginal deliveries \(^{86, 100}\). In an earlier study, El Fekih et al (2011) reported shoulder dystocia occurred in 1.9% of all vaginal deliveries\(^{101}\). In our study, shoulder dystocia was noted in 2.4% cases. Labour was induced in 13.4%, probably for maternal - fetal reasons such as hypertension, diabetes or oligohydramnios.

The birthweights of the new born was between 4000 and 4499 g in 92% of cases; 7.2% between 4500-4999g and 0.8% for 5000g and above. Bekdas et al (2013) reported 88% of the macrosomic infants had birth weights between 4000-4499g, 11% between 4500-4999g and 1% 5000g and above\(^{102}\). In the study of Demiroren et al (2008), these rates were 68%, 24% and 8% respectively\(^{103}\), and in the study of Akin et al (2010) these rates were 80%, 17% and 3% respectively\(^{104}\). The same results were reported by most authors \(^{10, 105}\). Fetal sex influences macrosomia potential. Male infants weigh more than female infants at any gestational age. Recent studies have confirmed this association \(^{106}\). In our study, sex of the infant influenced the birth weight, macrosomia was more dominant in male with 64%, and this is consistent with other authors \(^{10, 107}\). Previous history of macrosomic baby is the main maternal risk factor to macrosomia \(^{85, 108}\). It has 95% positive predictive value for macrosomia \(^{109}\). In our study, 14.16% of women had a past history of macrosomia. Other studies have shown rates as high as 25.9% or more \(^{1, 18, 86, 110, 111}\).
Multiparity ≥ 3 has been associated with macrosomia\textsuperscript{14,101,112,113}. This study did not find this association, majority of the macrosomic babies were born to para 1 and 2. Body mass index more or equal to 25 has been shown by several authors to be a risk factor for macrosomia \textsuperscript{18,110,114-117}. Our study corroborate the BMI ≥ 25 as a risk factor.

According to Kraïem et al (2004) CS is justified in all cases of fetal weight estimation greater than 4500 g\textsuperscript{118}. Many studies reported a higher rate of vaginal delivery compared to caesarean delivery when macrosomia is concerned \textsuperscript{86,101,108,119}.

Caesarean section delivery rate of 64.3\% was high in our study compared to other studies \textsuperscript{86,101,108}. In our study, the high CS rate was as a result of an increased number of women with previous CS and our study site policy was to deliver mothers with previous CS carrying a fetus weighing ≥ of 3400 g by CS.

\textbf{4.1 Limitations}

This being a retrospective study, the following were observed: The history of previous fetal macrosomia in the patients or their relations was not documented in most of the files of the patients.

\textbf{4.2 Conclusion}

The prevalence of macrosomia was 3.4\%. Main risk factors for macrosomia were previous history of macrosomia, male sex, hypertensive disorders of pregnancy, body mass index ≥ 25, para 1 and 2, diabetes and higher gestational age at delivery. Mother and neonate are at increased risk of complications.

\textbf{4.3 Recommendations}

1. Long term follow up of macrosomic infants are recommended
2. Management of suspected macrosomia should be individualized with the aim to minimize maternal and fetal complications
3. Regular obstetric drills should be conducted
4. A study comparing the incidence of macrosomia of our diverse population in our setting is needed
Chapter 5: References


52. Nassar AH, Usta IM, Khalil AM, Melhem ZI, Nakad TI, Musa AAA. Fetal macrosomia (≥4500 g): perinatal outcome of 231 cases according to the mode of delivery. J Perinatol 2003; 23:136–141.


105. El Hak MS. Macrosomiefoetale. Médecine, Casablanca,Université Hassan II, Thèse N°20.84; 2006(English abstract)


Chapter 6: Appendices

Appendix 1: Data Sheet

1. Study No: 
2. Date Of Delivery: 
3. Maternal BMI: 
4. Age: 
5. Parity: 
6. Gravidity: 
7. Booked : ( Y=1, N=2) 
8. Booking Gestational Age: 
9. Gestational Age at delivery: 
10. Number of Antenatal visits: 
11. Ethnic Group: ( African=1, Indian=2, Coloured=3, white=4) 
12. RH: 
13. RPR: (+ve=1, -ve=2) 
14. HB: 
15. HIV Status(neg=1, pos=2) 
16. Maternal obstetric condition: ( Overt diabetic=1, gestational diabetic=2, Other=3) 

17. Socioeconomic

17.1 Employed: (Y=1, N=2) 
17.2 Cigarette smoke: (Y=1, N=2) 
17.3 Alcohol use: (Y=1, N=2) 
17.4 Marital Status: ( single=1, married=2, divorced=3, engaged=4) 

44
18. **Past Obstetric History**

18.1 Previous big baby: (Y=1, N=2)  
18.2 Gest Age at delivery:  
18.3 Outcome: (1=alive, 2=SB, 3=ENND, 4=LNND)

19. **Past medical history**

( Diabetes mellitus=1, Thyroid disorder Anaemia=3, Hypertension=4,  
Cardiac disease=5, Epilepsy=6)

20. **Maternal Outcomes**

20.1 Induction of labour: (Y=1, N=2)  
20.2 Prolonged second stage: (Y=1, N=2)  
20.3 Elective caesarean delivery  
20.4 Emergency caesarean delivery  
20.5 Instrumental vaginal delivery  
20.6 Shoulder dystocia  
20.7 Second and third degree perineal tear  
20.8 Fourth degree perineal tear  
20.9 Post-partum haemorrhage  
20.10 Length of stay > 3 days

45
21. **Foetal outcome**

21.1 Male gender

21.2 Birth weight

21.3 Apgar’s:

21.3.1 1\textsuperscript{st} min

21.3.2 5\textsuperscript{th} min

21.4 Outcome:( 1=alive, 2=SB, 3= ENND, 4=LNND)

21.5 Congenital abnormality:( Y=1, N=2)

21.6 Resuscitation

21.7 Intensive care unit/nursery

21.8 Neonatal complications:( neonatal seizures=1, Erb’s palsy=2

( birth trauma=3, other=4)

21.9 Number of days in nursery:

46