Original Article

AN AUDIT OF PREGNANCIES COMPLICATED BY INTRAUTERINE GROWTH RESTRICTION (IUGR) AT LAUTOKA HOSPITAL FROM 1st JANUARY 2016 TO 31st DECEMBER 2016

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ABSTRACT

Introduction
IUGR is an obstetrical complication that is difficult to identify in order to allow intervention to lessen morbidity and mortality. The Lancet series on stillbirths highlighted the causes of stillbirths globally, identifying IUGR as one of the five major causes. IUGR has accounted for almost a third of all still births at Lautoka Hospital over the past three years. The aim of this study was to audit all pregnancies complicated by IUGR, the contributing risk factors and their outcomes at Lautoka Hospital from 1st January to 31st December 2016.

Aim
To conduct a retrospective audit of pregnancies complicated by IUGR at Lautoka Hospital from 1st January 2016 to 31st December 2016.

Method
This is a retrospective descriptive audit using clinical notes, conducted on 170 women diagnosed with IUGR in 2016.

Results
There were 4,131 deliveries during the study period; 191 patients of whom were diagnosed with IUGR of which 170 folders were retrieved. The Incidence rate of those diagnosed with IUGR during this period was 4.3%. Seventy percent of women with IUGR had low to normal Body Mass Index (BMI) and booked in the late second to third trimester. The risk of developing IUGR was significantly increased in Fijians of Indian Descent (FID) (RR 4, CI 2.9-5.3, p-value <0.0001); in primigravida (RR 4.1, CI 3.5 – 4.7, p-value <0.0001); and those with previously Low Birth Weight baby (LBW) (< 2500g) (RR 2.3, CI 1.67 – 3.26, p-value <0.0001). Anaemia or hypertension diagnosed during pregnancy significantly increased the risk of developing IUGR (RR 1.7, CI 1.3-2.40, p-value 0.0002) and (RR 2.6, CI 1.75 – 4.05, p-value <0.0001) respectively. Women with IUGR have a 6 times higher chance of having a Still Birth (SB) (RR 6.1, CI 3.78-9.92, p-value <0.0001); higher risk of Induction of Labour (RR 4.2, CI 3.65-5.64, p-value <0.0001) and caesarean section delivery (RR 2.1, CI 1.54-2.85, p-value <0.0001). Seventy eight percent of still births were delivered beyond 37 weeks, a possible delay, which could have been avoided potentially improving the SB rate. SB risk was significantly higher in those diagnosed at or > 37 weeks gestation compared to those with an earlier diagnosis (RR 1.61 95% CI 1.11 – 2.35, p value 0.05).

Conclusion
IUGR contributes significantly to still births. There were delays in diagnosis and appropriate surveillance to allow timely delivery at Lautoka Hospital, which could have reduced the still birth rate.

Key Words: *Pregnancy, Intrauterine Growth Restriction (IUGR), diagnosis, risk factors, outcomes*

INTRODUCTION

Lautoka Hospital is one of three tertiary referral Divisional hospitals that serve five Sub divisional hospitals – Sigatoka, Nadi, Ba Tavua and Rakiraki. There were 4131 deliveries at Lautoka Hospital in 2016 with a 19.4% caesarean section rate.[3] In 2016 Lautoka Hospital recorded a Still Birth (SB) Rate of 14 per 1,000 live births [2] with majority occurring in the antepartum rather than the intrapartum period. IUGR has been highlighted as one of the major contributing factors to SBs' at Lautoka Hospital, with approximately 30% of SBs' in the past 3 years being affected by it [2,6]. A retrospective audit of SBs' at Lautoka Hospital from January 2012 to December

2013 by J.Poulter et al [5] demonstrated that of the 96 SB's which occurred in this period IUGR was the dominating cause of SBs' accounting for 21 SBs' with a reported OR: 168.5 (CI: 105.44-269.47) for an IUGR foetus to have a SB compared to those babies without IUGR.

The definition of IUGR refers to a weight below the 10th percentile for gestational age [7]. At Lautoka Hospital, clinical assessment of foetal size by abdominal palpation and measurement of Symphysiofundal Height (SFH) is used. If there is a lag of three centimetres or more from gestational age in weeks this raises the suspicion of IUGR. Once there is clinical suspicion of IUGR, ultrasonographic techniques are used to try to confirm or exclude the diagnosis by plotting growth parameters on a standard growth curve adopted from National Women's Health Hospital, Auckland, New Zealand. The Growth chart has 3 lines for each of the morphometric parameters which are the 5th, 50th and 95th centiles for the biparietal diameter, head circumference, abdominal circumference and femur length which demonstrates the growth centile of the foetus. The measurements of the head circumference and abdominal circumference and their congruence are used to determine the type of IUGR rather than foetal weight below the 10th centile per se. There currently is no local growth charts for biometry or weight of foetus in utero available in Fiji and the general consensus is a birth weight of <2,500g and < 3,000g at term is low for a Fijian if Indian descent and iTaukei respectively.

Lack of antenatal recognition of foetal growth problems is one of the most frequent causes of avoidable adverse outcome. Lancet Still Birth Series recommends targeting the following key areas to reduce preventable SBs' [8]:

- Childbirth complication
- Maternal infection in pregnancy
- Hypertension and Diabetes
- Intrauterine growth restriction
- Congenital anomalies

The large influence of IUGR to SB risk has been established in a recent population-based study of SBs' in the West Midlands where 44% of SBs' were attributed to IUGR. IUGR increased the threat of SB substantially as highlighted by The Lancet Still Birth Series, and this risk was even higher if IUGR was not identified antenatally [9]. The reduction in SB risk with antenatal diagnosis and timely management has been shown in a recent study from New Zealand [10]. De Onis et al observed incidence of IUGR in Africa, Asia, Latin America, Caribbean and Oceania noting it to affect 24% of newborns (approximately 30 million infants) every year [13]. Majority of cases were found in Asia, which accounts for nearly 75% of all affected infants, 20% in Africa and 5% in Latin America.

A number of social, cultural, and environmental factors seen in developed and developing countries, affect intrauterine growth. In developed countries, 25% of cases are due to cigarette smoking; whereas low weight gain, low body mass index, primiparity, and short stature are implicated for nearly half of the cases [17]. The contribution of cigarette smoking is lower in developing countries, whereas the other causes mentioned above appear to have a greater impact [17]. Poor nutritional status, maternal anaemia, and poor prenatal care and substance abuse were associated with poor foetal growth in women with lower socioeconomic status and residing in developing countries [21, 22].There is a 3.5 fold increased risk of IUGR in smokers compared with non-smokers.[23] Smoking has dose, duration, and trimester-related effect on foetal growth. Up to 19 % of term LBW has been reported to be due to smoking during pregnancy. Smoking throughout pregnancy and heavy smoking (>15 cigarette daily), mainly in the third trimester, is related with low birth weight [23]. In addition, foetal alcohol syndrome is often linked with IUGR [24].

Symmetric IUGR is where the Biparietal Diameter (BPD), Head Circumference (HC), Abdominal Circumference (AC) and Femur Length (FL) are proportionately small. Generally an early insult from infection or foetal abnormality leads to a proportionate reduction of both head and body size [26]. In asymmetric IUGR, the head remains bigger than the abdominal circumference as an indication of head-sparing from the placental insult. Asymmetrical growth restriction is due to a later pregnancy insult causing placental insufficiency, which may be due to hypertension or other placental problems [26]. The genetic growth potential of the foetus can be compromised as a result of maternal, placental, or foetal causes. [26]

Diagnosis and Management of IUGR

A comprehensive history and physical examination is carried out to evaluate maternal disorders that may be associated with IUGR. Precise knowledge of gestational age (GA) is crucial to the diagnosis of IUGR as normal and abnormal foetal measurements are described against gestational age. In addition, obstetrical ultrasound examinations and laboratory evaluations are carried out to observe foetal and placental issues.

Clinical measurement of SFH using a tape measure is a reasonable screening tool for IUGR in low risk pregnancies, as there is no superior approach to better neonatal outcome [27, 28]. This approach works best when all of the serial measurements are done by the same person using the unmarked side of the tape to decrease bias [30] and are plotted to reproduce foetal growth for the individual patient, rather than documenting measurement in folder [31, 32]. Other factors, which affect sensitivity, include maternal weight,

bladder volume, parity, and ethnic group [33-36]. Foetal size by abdominal palpation for detecting IUGR has sensitivities ranging from 30 to 50 percent only [37-39].

There is a general consensus that once the suspicion of IUGR is raised due to risk factors or physical examination, sonographic techniques are to be used to make the diagnosis [40-42]. When foetal growth is affected by placental insufficiency, the foetal abdominal circumference (AC) is smaller than expected because of exhaustion of abdominal adipose tissue and hepatic size decreases due to decreased glycogen storage in the liver. Majority of studies report reduced AC is the most sensitive single biometric parameter of IUGR [43-48].

Estimated foetal weight (EFW) has become an additional method of isolating IUGR where weight below 10th centile of expected is defined as IUGR [49]. The use of birth weight centiles based on customized centiles for prediction of IUGR and perinatal morbidity still remains controversial.

As discussed above, the use of any parameter (e.g. AC, EFW) in the prediction of IUGR is based on accurate assessment of GA. If dates are unknown, serial sonographic examinations at two-week intervals should be performed to evaluate the rate of interval growth (i.e. growth velocity) [52]. Irrespective of GA, there is a significantly lower rate of change of growth over time of AC or EFW in IUGR foetuses when compared with appropriately grown foetuses [51].

The tests available for surveillance of the IUGR foetus vary in terms of the time required to undertake them and personnel required to complete and interpret them. The principle of surveillance is to preempt foetal academia and conduct a timely delivery prior to end–organ damage and intrauterine foetal death. The Royal College of Obstetrics and Gynaecology (RCOG) Guidelines states "use of umbilical artery Doppler has been revealed to decrease perinatal morbidity and mortality" [52]. Umbilical artery Doppler should be the primary surveillance tool in the IUGR foetus" [53]. When umbilical artery Doppler flow indices are normal it should be repeated every two weeks in IUGR foetuses. More frequent Doppler surveillance may be appropriate in a severely growth restricted foetus. When umbilical artery Doppler flow indices are abnormal (pulsatility or resistance index > +2 standard deviations above mean for gestational age) and delivery is not intended; repeat surveillance twice weekly in foetuses with normal end–diastolic flow is recommended [54]. Those foetuses with absent or reversed end–diastolic flow usually need delivery.

Cerebral vasodilatation is a response to increase in diastolic flow, a sign of the 'brain–sparing effect' of chronic hypoxia, and results in reduction in Doppler indices of the middle cerebral artery (MCA). This and Biophysical Profile which has four variables (foetal breathing movement, gross body movement, tone and cardiotocography (CTG – computerized analysis of foetal heart rate variation), and amniotic fluid volume have been shown on systematic reviews of Cochrane database to perform poorly collectively and individually in the prevention of perinatal morbidity and mortality [55-58].

At present there is no effective intervention to alter the course of IUGR except delivery. Planning delivery requires balancing the risks of prematurity against continuing intrauterine stay with risks of death or organ impairment due to placental insufficiency leading to insufficient foetal tissue perfusion [59].

A randomized trial by Boers et al compared the effect of inducing labour to expectant monitoring in women suspected to have IUGR who were 36 weeks of gestation and over [60] in 650 foetuses of which 14 had umbilical artery absent or reversed end diastolic volume. A total of 5.3% infants in the induction group had an adverse outcome (defined as death, umbilical artery pH < 7.05 or admission to intensive care) compared to 6.1% in the expectant monitoring group (difference –0.8%, 95% CI –4.3–3.2). Based on these results; it is reasonable to offer delivery in IUGR infants at 37 weeks of gestation. The Randomized Control Trial Growth Restriction Intervention Trial (GRIT) compared the effect of delivering early (after completion of a steroid course) with waiting for as long as possible (i.e. until the obstetrician was no longer uncertain) [61]. There was no difference in total deaths before discharge (10% versus 9%, OR 1.1, 95% CI 0.6–1.8), deducing obstetricians are delivering sick preterm foetuses at about the correct time to minimize mortality [61]. At 2 years overall rates of death (12% versus 11% respectively) or severe disability (7% versus 4%) were similar (OR 1.1, 95% CI 0.7–1.8) [62].

The optimal approach to management of the pregnancy with suspected growth restriction related to utero-placental insufficiency has not been established; there is very limited evidence from randomized trials [63]. Serial ultrasound evaluation of (1) foetal growth, (2) foetal behaviour, and (3) impedance to blood flow in foetal arterial and venous vessels represent the key elements of foetal assessment and guide pregnancy management decisions. The purpose is to identify those foetuses that are at highest risk of in utero demise and neonatal morbidity and thus may benefit from preterm delivery.

PROBLEM STATEMENT

Still Birth Rate at Lautoka Hospital in 2016 was 14 per 1,000 live births. The Lancet SB Series suggests that for countries with SB Rates of more than 5 per 1,000 births the goal is to reduce it by at least 50% by 2020 [8]. IUGR is a major contributor to Still Births as supported by the Lancet Still Birth Series as well as the local audit done

by J.Poulter [5, 8]. There has never been a study looking at contributing factors, modes of diagnosis, management and outcomes of pregnancies complicated by IUGR in Lautoka hospital. The impact of IUGR burden on the obstetric population in Lautoka has not been determined. This retrospective audit will provide information on the predisposing factors, management and outcomes of pregnancies complicated by IUGR at Lautoka Hospital.

AIM

To conduct a retrospective audit of pregnancies complicated by IUGR at Lautoka Hospital from 1st January 2016 to 31st December 2016.

OBJECTIVES

- To ascertain the socio-demographic characteristics of women diagnosed with IUGR pregnancies at Lautoka Hospital from January 1st 2016 to December 31st 2016.
- To determine diagnostic practices and contributing factors of IUGR at Lautoka Hospital from January 1st 2016 to December 31st 2016.
- To describe the management as well as maternal and foetal outcomes of pregnancies complicated with IUGR at Lautoka Hospital from January 1st 2016 to December 31st 2016.

METHODOLOGY

This is a retrospective audit of the clinical records of women diagnosed with IUGR at Lautoka Hospital from January 1st 2016 to December 31st 2016.

Inclusion Criteria:

- All those classified as IUGR from 28 weeks onwards
- Had SFH lagging >3cm behind Gestational Age
- Ultrasonographic evidence of IUGR
- Birth weight <2,500g and Gestational Age > 37 weeks
- Singleton Pregnancies

Excluded were all those that do not meet the inclusion criteria. All deliveries in the study period (4131 folders) were screened using the above criteria and names were identified from the antenatal ward, labour ward, operating theatre, still birth and neonatal intensive care unit registries for those classified as IUGR. Extracted folders were de identified and coded then entered into an excel spread sheet.

Their records were checked for the following specific variables:

- Demographics and clinical risk factors
- Modes of diagnoses of IUGR
- Management of IUGR during their antenatal and labour periods
- Maternal and foetal outcomes

The data on the following variables were collected:

- Demographics: Age, Ethnicity, Level of Education, Parity
- Booking History and past Obstetric History: Booking gestation, Past history of IUGR, Gestational Diabetes Mellitus, Hypertension, LBW
- Identifiable risk factors: Family history of Diabetes/Hypertension, Pre-existing Medical, Conditions and medications, Smoking, Drug, Alcohol, Body Mass Index (BMI)
- Antenatal risk factors: Hypertension, Syphilis, Anaemia. Diabetes, Placental abnormalities
- Modes of diagnosis: Clinical measurement of Symphysiofundal Height, Ultrasound scan – symmetric vs. asymmetric
- Antenatal management: Monitoring: Foetal movements (Foetal Kick Chart), Measuring Amniotic Fluid Index/Volume, Umbilical Artery Doppler studies, CTG
- Intrapartum: Spontaneous Labour or Induction of Labour, Delivery, CTG in labour
- Foetal outcome: Stillbirth, Neonatal death, Neonatal Intensive Care Unit admission, Birth weight

A total of 4.131 patients were delivered from January 1st 2016 to December 31st 2016. Of these, 191 deliveries were complicated by intrauterine growth restriction. There were 170 folders retrieved and this gave a retrieval rate of 90%.

The population studied includes all pregnancies complicated with IUGR at Lautoka Hospital from January 1st 2016 to December 31st 2016. They were identified from the delivery register at labour ward, antenatal ward admission and induction register, and still birth and neonatal audit records at Lautoka Hospital. Also all deliveries were screened and included if certain criteria were met. The national health number (NHN) was used to ensure there is no duplication of cases. The clinical records was extracted and data entered onto an excel spreadsheet that was password protected. All data was de-identified and coded. These were kept separate to ensure patient's identity was not revealed.

The information on the excel spreadsheet was coded and categorized and transported to Epi-info for statistical analysis. Univariate and bivariate analyses were performed, univariate analysis for frequency

computations and bivariate analysis in computing associations between variables. The Chi-square test was used to measure the strength of associations between categorical variables.

One-way analysis of variance was used for comparison of continuous variables between groups. A 5% significance level was used throughout.

With regards to ethical considerations, approval was obtained from:

- Head of Department for Obstetrics and Gynaecology unit at Lautoka Hospital.
- College Research Committee and Department Research Committee of Fiji National University.
- Fiji National Health Research and Ethics Review Committee from Ministry of Health

All data was managed carefully to ensure security and confidentiality was maintained. The folders were de-identified and given a unique code. This unique code was kept in a separate sheet and only known to the researcher. This allows confidentiality and also allows us to go back and review the folder if we have to. There was no direct contact or interviews with patients in this study. Given this is a retrospective study; patients' current care will not be affected.

RESULTS

The incidence of IUGR was noted to be 4.3% at Lautoka hospital during the period of this study. Fijian of Indian Descent mothers accounted for majority of the cases with a 4 times higher risk of IUGR compared with iTaukei women (RR 4, 95% CI 2.9 – 5.3, p value <0.0001). Fijian of Indian descent women had an ethnic specific incidence of 8.5% compared to 1.8% in iTaukei women. Ninety two percent of mothers received at least secondary education which is similar to the rest of the obstetric population2. Being primiparous is a significant risk factor showing a 4 times higher risk of developing IUGR (RR 4.1, 95% CI 3.5 – 4.7, p value <0.0001) [Table 1].

Seventy percent of women with IUGR had normal to low BMI. [Table 2]

There were no pre-existing medical conditions in 95% of women with IUGR. Only 2% of women were smokers and 6% consumed alcohol [Table 3].

Thirty two percent of women in Table 4 had a previous baby with low birth weight (LBW) i.e. less than 2500g. A history of previous LBW delivery had a 2 fold increased risk of developing IUGR (RR 2.3, 95% CI, 1.67, p value <0.0001) in the subsequent pregnancy [Table 4].

Of the 170 patients with documentation; 52% of pregnancies were planned [Table 5].

Table 1: Demographics of women with pregnancies complicated by IUGR at Lautoka Hospital

Age (years)
<20 – 23 (14%)
>20 to 30 – 109 (64%)
31 to 40 – 36 (21%)
40+ – 2 (1%)

Ethnicity
iTaukei – 61 (36%)
Fijian of Indian Descent (FoID) – 106 (62%)
Others – 3 (2%)

Level of Education
Primary – 13 (8%)
Secondary – 104 (62%)
Tertiary – 15 (30%)

Parity
Primiparous – 99 (58%)
Multiparous – 71 (42%)

Table 2: Body Mass Index of women with IUGR
Body Mass Index (kg/m^2)
<18.5 – 35 (21%)
18.5 to 24.9 – 80 (49%)
25 to 29.9 – 27 (17%)
30 to 34.9 – 17 (10%)
35 to 39.9 – 3 (2%)
40+ – 2 (1%)

Table 3: Pre-existing risk factors of women with IUGR
Pre-existing Medical Conditions
No Pre-existing conditions – 162 (95%)
Asthma – 7 (4%)
Diabetes – 1 (1%)
Smoking – 166 (98%)
Not smoking – 4 (2%)
Alcohol consumption – 159 (94%)
No alcohol consumption – 11 (6%)

Table 4: Frequency of Previous Obstetric History in women with IUGR
Previous Obstetric History
Previous Low Birth Weight – 32 (31%)
No complications – 29 (28%)
Miscarriage – 25 (24%)
Previous Perinatal Mortality – 6 (5%)
Preterm Labour – 4 (4%)
Pregnancy Induced Hypertension – 3 (3%)
Previous Caesarean Section –3 (3%)
Postpartum Haemorrhage – 1 (1%)
Gestational Diabetes Mellitus – 1 (1%)

Table 5: Planned vs. Unplanned Pregnancy
Planned – 88 (52%)
Unplanned – 64 (38%)
Missing data – 18 (10%)

Table 6: Percentage of women with IUGR that were booked locally vs. transfer from other Subdivisions
Source Number (%)
Local (Lautoka) – 84 (49%)
Transfer from Subdivision – 86 (51%)

Table 7: Booking Gestation of women with IUGR
Booking Number (%)
First Trimester (0-13weeks) – 49 (29%)
Second Trimester (14-26weeks) – 104 (61%)
Third Trimester (27-40weeks) – 17 (10%)

Table 8: Clinical Measurement of SFH in women with IUGR
Measurement (cm)
Lagging ≥ 3cm – 132 (78%)
Normal (+/-2cm) – 38 (22%)

Table 9: Gestational Age at Diagnosis of IUGR
Gestation (weeks)
<30 – 12 (7%)
30 to 36 – 78 (46%)
37 to 40 – 69 (40%)
40+ – 8 (5%)
At delivery – 3 (2%)

Table 10: Types of IUGR
Asymmetrical – 142 (87%)
Symmetrical – 22 (13%)

Table 11: Antenatal Risk Factors in women diagnosed with IUGR
Complications
Anaemia – 91 (46%)
No Complications – 49 (25%)
Hypertension – 24 (12%)
Diabetes – 11 (6%)
Rupture of membranes – 10 (5%)
Mal-presentation – 7 (4%)
Syphilis – 2 (1%)
Cholestasis – 2 (1%)
Post Dates – 1 (<1%)
Antepartum Haemorrhage – 1 (<1%)

Table 12: Frequency of Antenatal Foetal Surveillance Noted in IUGR pregnancies
Foetal Movements Present – 155 (91%)
Foetal Movements Decreased – 15 (9%)
Antenatal CTG Reassuring – 160 (96%)
Antenatal CTG Non-reassuring – 6 (4%)
Amniotic Fluid Volume Low – 25 (15%)
Amniotic Fluid Volume Normal – 136 (84%)
Amniotic Fluid Volume High – 2 (1%)
Uterine Artery Doppler Normal – 132 (85%)
Uterine Artery Doppler Abnormal – 23 (15%)

Table 13: Gestational Age at Delivery
Gestation
32 to < 37 weeks – 20 (12%)
37 to 40 weeks – 135 (80%)
40+ weeks – 14 (8%)

Table 14: Onset of Labour
Spontaneous – 48 (34%)
Induced – 94 (66%) [RR 4.2 (95% CI 3.15-5.64) <0.0001]

Table 15: Types of Induction of Labour in women with IUGR
Misoprostol – 33 (37%)
Foleys – 46 (52%)
Both (Misoprostol then Foleys or vice versa) – 6 (7%)
Surgical (Artificial rupture of membranes) – 4 (4%)

Table 16: Cardiotocography in labour
Cardiotocography (CTG) in Labour
Reassuring – 93 (70%)
Non Reassuring – 25 (19%)
Not monitored – 15 (11%)

Table 17: Type of Delivery among IUGR cases
Vaginal delivery – 113 (66%)
Caesarean Section – 57 (34%) [2.09 (95% CI 1.54-2.85) p<0.0001]

Table 18: Birth Weights of IUGR foetuses
Weight (kg)
1.00 to 1.49kg – 3(1%)
1.50 to 1.99kg – 25 (15%)
2.00 to 2.49kg – 63 (37%)
2.50 to 2.99kg – 64 (38%)
3+ kg – 15 (9%)

Table 19: Birth Weight by Ethnicity amongst women with IUGR
(FoID – Fijian of Indian Descent, iT – iTaukei)
Weight (kg)
1.00 to 1.49kg – 2 FoID, 1 iT
1.50 to 1.99kg – 1 Others, 17 FoID, 7 iT
2.00 to 2.49kg – 48 FoID, 15 iT
2.50 to 2.99kg – 2 Others, 36 FoID, 26 iT
3+ kg – 3 FoID, 12 iT

Table 20: Adverse Neonatal Outcome
Stillbirth – 14 [RR 6.1 (95% CI 3.78-9.92) p<0.0001]
Admission to Neonatal Intensive Care Unit – 14 [RR 0.7 (95% CI, 0.43-1.27) p=0.33]
Preterm delivery – 14 [RR 1.23 (95% CI, 0.73-2.05) p=0.38]

Table 21: Summary of Significant Risk Factors Identified for developing of IUGR
Fijian of Indian Descent – RR 4.0 (95% CI, 2.9–5.3) p<0.0001
Primiparous – RR 4.1 (95% CI, 3.5–4.7) p<0.0001
Previous low birth weight – RR 2.3 (95% CI, 1.67–3.26) p<0.0001
Hypertension – RR 2.6 (95% CI, 1.75–4.05) p<0.0001
Anaemia – RR 1.7 (95% CI, 1.3–2.4) p=0.0002

Table 22: Summary of Significant Outcomes
Stillbirth – RR 6.1 (95% CI, 3.78–9.92) p<0.0001
Induced labour – RR 4.2 (95% CI, 3.65–5.64) p<0.0001
Caesarean delivery – RR 2.1 (95% CI, 1.54–2.85) p<0.0001

Fifty one percent were transferred from sub divisional hospitals. Lautoka Hospital serves as a referral centre for 5 subdivisions [Table 6].

Twenty nine percent of women with IUGR booked in the first trimester and 71% in the second and third trimesters [Table 7].

Seventy eight percent of all symphysiofundal heights measured were noted to be lagging by 3 cm or more than gestational age in weeks [Table 8].

Majority of cases were diagnosed in the third trimester [Table 9].

Eighty seven percent of all growth-restricted babies were asymmetrical IUGR [Table 10].

Of all the antenatal risk factors anaemia and hypertension increased the risk of IUGR (RR 1.7, 95% CI 1.7 – 2.4, p value <0.0002) and (RR 2.6, 95% CI 1.75 – 4.05, p value <0.0001) respectively [Table 11].

The recordings in the folder had 15 (9%) cases complaining of reduced movements; 6 (4%) with antenatal non-reassuring CTG, 25 (15%) with low amniotic fluid volume and 23 (15%) with abnormal Umbilical Artery Dopplers [Table 12].

Majority delivered from 37 weeks onwards which may reflect low intervention before 37 weeks or later diagnosis of IUGR [Table 13].

Induction of labour rate in the overall obstetric population at Lautoka was 19% compared to 66% in women with IUGR in the study period; the risk of induction of labour in women with IUGR was four times higher [Table 14].

Foleys Induction of labour was the favoured method of induction amongst women with IUGR [Table 15].

Table 23: Stillbirths Features In The IUGR Group With Classification As Avoidable Vs Unavoidable					
Gestational Age at Diagnosis	**Gestational Age at SB**	**Interval from diagnosis to SB**	**Birth weight**	**Possible Delay**	**Avoidable/ Unavoidable Outcome**
34 weeks	34 weeks 4 days	4 days	1.92kg	Sent home without a scan when SFH was low	Avoidable
34 weeks 5 days	38 weeks	Undiagnosed	1.67kg	Followed up in midwife clinic SFH recorded as normal throughout	Unavoidable
37 weeks	38 weeks	1 week	3.02kg	Scan showing IUGR not recognized	Avoidable
35 weeks	37 weeks	2 weeks	1.98kg	Fundal height lagged unrecognized & scan plotted wrongly	Avoidable
31 weeks	37 weeks 2 days	3 weeks	2.46kg	Breech, low AFI and raised Doppler from 34 weeks managed conservatively till demise prior to planned caesarean section	Avoidable
33 weeks	37 weeks	4 weeks	2.5kg	Patient chose to wait despite abnormal umbilical artery Doppler and oligohydramnios	Unavoidable
33 weeks	38 weeks 5 days	1 day	2.38kg	Growth plotted wrongly so asymmetrical IUGR not recognized developed oligohydramnios and died a day prior to planned induction of labour	Avoidable
31 weeks	40 weeks 4 days	Defaulted	2.1kg	Defaulted from booking clinic	Unavoidable
33 weeks	36 weeks	3 weeks	2.32kg	Was unable to do scan at 33 weeks as requested. At next review asymmetrical IUGR and IUD noted	Avoidable
40 weeks	40 weeks 4 days	4 days	2.7kg	Asymmetrical IUGR at 40weeks managed conservatively	Avoidable
37 weeks 1 day	37 weeks 2 days	1 day	1.9kg	Raised dopplers Abnormal CTG unrecognized booked for C/S next day but died before that	Avoidable
32 weeks	36 weeks	Fresh SB	1.83kg	Misoprostol used in a background of oligohydramnios and abnormal CTG	Avoidable
37 weeks	39 weeks	2 weeks	2.8kg	Abnormal CTG unrecognized and Scan delayed	Avoidable
29 weeks	33 weeks	4 weeks	1.65kg	Abnormal CTG	Avoidable

Of the 25 non-reassuring CTGs during labour there were no intrapartum stillbirths [Table 16].

Caesarean Section Rate in study group was 34% compared with 18.8% in the overall obstetric population during the study period. This was significantly higher in the IUGR group [Table 17].

Fifty three percent of babies born to the women with IUGR were below 2.5kg [Table 18].

Of the 53% that had a birth weight below 2.5kg, 73% were among FID women; whereas 80% of those with a birth weight more than or equal to 3kg were iTaukei women [Table 19].

If a pregnancy was complicated by IUGR the risk of Still Birth was significantly higher with a 6-fold increase (p value <0.0001) [Table 20].

Fijians of Indian Descent, being primiparous and those with previous LBW baby were at increased risk of developing IUGR. During the course of pregnancy if anaemia or hypertension occurred this also increased the risk of developing IUGR [Table 21].

If the pregnancy was complicated by IUGR the risk of still birth is 6 times higher, 4 times more likely to be induced and 2 times more likely to have a caesarean section compared to the rest of the obstetric population [Table 22].

Table 23 illustrates the problems associated with timely diagnosis, failure to recognize ominous signs and delaying delivery. Ten of the 14 stillbirths occurred after 37 weeks gestation. Only 3 of the 14 cases of SB were deemed unavoidable. In the overall group of women with IUGR, SB risk was higher in those diagnosed at or > 37 weeks gestation compared to an earlier diagnosis RR 1.61 95% CI 1.11–2.35, p value 0.05.

DISCUSSION

During the study period, there were 4,131 deliveries at Lautoka hospital. Of these, 196 were classified or identified as pregnancies complicated with IUGR, which accounted for 4.3% of pregnant women at Lautoka hospital. This is much lower than the 10 to 24% rate demonstrated by de Onis in her study from Africa, Asia, Latin America, Caribbean and Oceania. However, if looked at from an ethnic perspective the incidence amongst the FID ethnic group is 8.5% compared to iTaukei, which is 1.8%; the FIDs closer to the lower range described in the literature [13].

Under diagnosis of IUGR in our population is likely as majority of our women book late in pregnancy. This makes gestational age dating with ultrasound inaccurate and hence diagnosis of IUGR difficult, as women often do not remember their last menstrual period as well. Seventy percent of women in this study group booked late either in the second or third trimester similar to the rest of our obstetric population [2]. However relatively more women booked in the first trimester compared to the rest of the obstetric population 29% vs. 16% [2]. This reflects ethnicity, as Fijian of Indian descent women tend to book earlier than iTaukei women and are more likely to plan their pregnancies [2]. The earlier a mum books the more accurate her gestational data. Precise knowledge of gestational age is crucial to the diagnosis of IUGR because weights are compared with other foetuses at the same gestational age. This may be the reason for

the apparent lower prevalence of IUGR in our population as many may have been undiagnosed and unclassified.

Sixty four percent of cases occurred in women between the ages 21 – 30 years which is comparable to the rest of the obstetric population [2].

Fijians of Indian Descent (RR 4 CI 2.9-5.3, p value <0.0001) were at a 4 times higher risk of developing IUGR compared to iTaukei's. Indians have been described as a high risk group in many other studies [18, 19, 64]. In Singapore Indian women were seen to have lower birth weights than Chinese or Malays [65]. Thompson et al. showed that babies of Pacific Islanders, born in New Zealand, were unlikely to have IUGR when compared with Caucasian women.19 Lesley McCowan et al in New Zealand also found the Asian and Indian ethnic group to be more prone to developing IUGR. [64]

Majority of mothers received at least secondary education which again is similar to the rest of the Obstetric Population2 and lack of education is not a contributing factor in our group as was also shown by S Muthayya et al [66] in South Indian mothers with IUGR.

A primiparous mother is significantly more likely to have a growth restricted baby (RR 4.1 CI 3.5-5.7, p value < 0.0001) compared to multiparous women. The role of parity in the epidemiology of IUGR was not well understood. Ilana Vardi Shoham et al [67] demonstrated the association between primiparous women and IUGR in 25,614 women from the southern part of Israel and found it to be an independent risk factor (RR 1.99, 95% CI 1.69 – 2.35, p value <0.0001). This has also been demonstrated in Brazil by Kramer et al [17], (RR 1.55, 95% CI 1.25 – 1.91, p value <0.0001). Lesley McCowan et al also demonstrated primiparity to be a risk factor in women with IUGR in New Zealand [64].

In this study 95% of mothers had no significant pre-existing medical conditions. In an unpublished paper, by V. Sema on "Modes of diagnosis, management and outcomes of diabetes in pregnancy at Lautoka Hospital from 2013 to 2015" she demonstrated an increased risk of IUGR (OR 1.59 [95% CI 1.05 – 2.42], p value 0.02) in pre-existing Diabetes compared with gestational Diabetes [68].

Fewer women (2%) smoked in our group compared with the general population as shown in WHO FIJI STEPS survey 2002 and 201169 , which demonstrated that 3.9% of females between 15 and 64 years smoke daily. Only 6% of mothers consumed alcohol, which is similar to the general female population's consumption 5.5% [69]. Given the low prevalence of these risk factors in our study and the unavailability of overall rates of smoking and alcohol consumption in the obstetric population in the study period, meaningful comparisons could not be evaluated.

Seventy percent of cases were in the low to normal BMI. Small women typically have smaller newborns; Simpson et al demonstrated that if a woman begins pregnancy weighing less than 45kg the risk of delivering an IUGR infant is increased at least 2 fold.70 Unfortunately, there are no preconception clinics in Fiji and majority booked in the second and third trimester hence maternal weight gain and pre-gravid weights were not assessed. V. Cati et al [71] in an unpublished paper "Impact of Obesity on pregnancy outcomes at Lautoka Hospital in 2012" demonstrated overall more women in our pregnant women tend to be overweight and obese with an average BMI of 34.9kg/m2. This is not the case in women with IUGR in this study where majority had low to normal BMI. This potentially identifies low to normal BMI as a significant predictor for IUGR as has previously been noted by Drysdale et al18 for women in Australia, McCowan et al [64] for women in New Zealand and Simpson et al [70] for women in America.

Fifty one percent of IUGR cases were transferred from the periphery for various reasons the highest amongst which were patients' preference to deliver at Lautoka hospital and uncontrolled hypertension. Less than half of these referred cases were identified as IUGR in the periphery. Despite the prior knowledge of at risk groups in our population, these cases remained unrecognized. This could be due to lack of adequately trained personnel as well as well as adequate ultrasound services in these subdivisional or peripheral hospitals. Late booking and lack of adequate gestational aging would be another underlying factor rendering diagnosis of IUGR difficult.

A history of previous low birth weight babies (less than 2500g) had a 2 fold increased risk (RR 2.3 95% CI 1.67 – 3.26, p value <0.0001) of developing IUGR in this study. Kleijer reported a fourfold increased risk of IUGR in women with previous LBW infants in the Northern suburbs of Metropolitan Adelaide [72]. Surkan PJ et al [20] in a nationwide Swedish study of 410,021 demonstrated in women who delivered an IUGR baby at term or preterm previously were at increased risk of a stillbirth (RR 2.1, 95% CI 1.6 – 2.8) and (RR 3.4, 95% CI 2.1 – 5.6) respectively.

Eighty eight percent of women with IUGR were diagnosed in the third trimester which reflects the type of growth restriction i.e. Asymmetrical IUGR which is consistent with the literature25. This is mainly due to placental insufficiency due to chronic hypertension or abnormal placentation [26].

Identifying the subset of pregnancies with growth restriction in our setting is a challenge. It appears that clinical measurement of SFH proved to be of value as 78% of women with IUGR had measurements which were lagging more or equal to 3cm behind gestational age. With the challenges of accurate gestational dating in our setting it is difficult to estimate how many cases

may have not been picked up with this clinical measure as IUGR apart from the 22% that were not recognized clinically in the study group.

If a mother developed anaemia in the course of her pregnancy she was at increased risk (RR 1.7 05% CI 1.3–2.35, p value <0.0001) of developing IUGR. Naoko Kozuki et al [73] in her systematic review and meta-analysis demonstrated an odds ratio of 1.53 (95% CI: 1.24–1.87); P < 0.001 for a woman with anaemia to develop IUGR.

As expected, hypertension in pregnancy significantly increased the risk of IUGR (RR 2.6 95% CI 1.75 – 4.05, p value <0.0001). This has been reported in many studies [19, 74]. Groom et al looked at a large cohort of primiparous pregnancies (n = 1847) and determined the risk of IUGR according to the gestation at delivery in women with pre-eclampsia and gestational hypertension. The risk of IUGR was higher in women with pre-eclampsia needing preterm delivery (<37 weeks) compared with those delivered at term (57.1% IUGR at <34 weeks (RR: 3.1 (95% CI: 2.3–4.2)), 31.7% IUGR at 34–36 ± 6 weeks (RR: 1.7 (95% CI: 1.2–2.5)) and 18.3% IUGR at term (RR: 1.0). Gestational hypertension also demonstrated a similar pattern of increasing IUGR (57.6% IUGR at <34 weeks (RR: 4.8 (95% CI: 3.4–6.6)), 30.5% IUGR at 34–36 ± 6 weeks (RR: 2.5 (95% CI: 1.8–3.5)) and 12.1% IUGR at term (RR: 1.0)).74 Thompson et al also demonstrated developing hypertension increased the risk of IUGR for women in New Zealand (OR 2.42; 95% CI 1.08–5.40).19

During antenatal foetal surveillance if there was reduced foetal movements, non-reassuring cardiotocography, reduced amniotic fluid volume and abnormal umbilical artery dopplers, there is increased rate of Still Births and therefore any of these features should lead to a consideration of delivery [75, 76]. These features are ominous and the Royal College of Australia and New Zealand Obstetricians and Gynaecologists (RANZCOG), RCOG, and Society of Obstetrics and Gynaecology Canada (SOGC) recommend to either to increase surveillance with supplementary use of steroids less than 34 weeks gestation if immediate delivery is not desired, or to terminate the pregnancy [52, 77, 78]. The former is usually a reserved option in an under resourced Neonatal Intensive care unit.

Eighty eight percent of women with IUGR were delivered at 37 to 40 weeks, which may suggest a later diagnosis of IUGR or a low intervention before 37 weeks at Lautoka Hospital. Having IUGR significantly increased the risk of being induced (RR 4.2 95% CI 3.15 – 5.64, p value <0.0001) and delivering by caesarean section (RR 2.09 95% CI 1.54 – 2.85, p value <0.0001) compared to the rest of the obstetric population where induction and caesarean rates were lower, 66% vs. 19% and 34% vs. 18.8% respectively. Of those induced, 51% had foleys catheter induction of labour rather than misoprostol. In fear of still births these pregnancies are usually induced resulting in an increase operative delivery rate. This is one of the controversies revolving around timing and decision of delivery [60, 61]. Other obstetricians prefer a more conservative approach in order to avoid morbidity associated with an operative delivery and its implication on the subsequent pregnancy and thus advocate continuous foetal and maternal monitoring till spontaneous labour occurs or ominous features arise [60, 61]. Unfortunately little evidence is available to inform best practice about the optimum management of the suspected growth restricted foetus near term. Labour itself is a stressful time for foetuses; an IUGR pregnancy with poor or no placental reserve increases the chance of foetal distress and chance of caesarean section [79].

Of those that had CTG surveillance in labour 19% were non-reassuring and did not result in intrapartum deaths signifying timely intervention during labour. 11% were not monitored, either presenting late in advanced labour or not recognized in labour while being induced or awaiting labour in antenatal ward. This is probably due to the skeletal staff on the ground.

Fifty three percent of the foetuses delivered were less than 2.5 kg. For infants weighing 2,000–2,499 grams at birth, the risk of neonatal death is 4 times higher than for infants weighing 2,500–2,999 grams and 10 times higher than for infants weighing 3,000–3,499 grams [80].

Having IUGR increased the risk of still birth (RR 6.1 95% CI 3.78 – 9.92, p value <0.0001). This has been demonstrated in many studies [5, 8]. It is this significant contribution to perinatal mortality that has made it an important issue the world over more so in developing countries where the burden is heaviest [13]. Even though 89% of babies survived there were 14 still births. Of the 14 still births 10 were delivered over 37 weeks suggesting a conservative approach in the management of IUGR at Lautoka Hospital and a later diagnosis. Sill birth was found to be higher in those diagnosed at or more than 37 weeks gestation compared to an earlier diagnosis RR 1.61 95% CI 1.11 – 2.35, p value 0.05. The average interval between diagnosis and still birth were 15.5 days. Further review revealed 11 had avoidable factors that if addressed appropriately and in a timely manner may have resulted in favourable outcomes thus reducing still births.

LIMITATIONS OF STUDY

It is a retrospective audit so data had to be extracted from clinical records. Illegible writing and missing data made this challenging. Some data were not captured in annual reports making statistical computations for comparisons impossible.

CONCLUSION

IUGR imposes a challenge at Lautoka Hospital because of delays in diagnosis, institution of appropriate surveillance in order to schedule delivery that may prevent still births. Identifying at risk groups will help to actively screen and diagnose pregnancies at risk of developing IUGR. This study has identified being Fijian of Indian descent, primiparous and having low to normal BMI increases the risk of IUGR among our population. Furthermore, if there was previous history of deliveries with birthweight less than 2,500g, the risk increases. Development of anaemia and hypertension during the course of the pregnancy also shows a similar pattern in increasing IUGR risk.

Once an IUGR pregnancy is noted to have signs such as reduced foetal movements, oligohydramnios, abnormal dopplers; close monitoring with CTG and ultrasound scan is required and timely delivery undertaken. Diagnosis and timely delivery of IUGR women at Lautoka Hospital could be improved to reduce the Still Birth rate.

RECOMMENDATIONS

- Independent risk factors should be used for triaging women for closer antenatal surveillance.
- Fijian of Indian Descent, Primiparous and low BMI women need to have at least one third trimester scan around 34 weeks.
- Advocate early booking for early diagnosis, appropriate surveillance and timely intervention.
- Midwifery and Medical Staff need to be upskilled in surveillance in monitoring growth velocity and foetal wellbeing.
- Protocol of management of IUGR should be developed based on local and international data.
- Develop Birth Weight and Growth Charts for the 2 major local ethnic populations.

REFERENCES

1. 2007 Census of Population, Fiji Islands Bureau of Statistics
2. *PHIS and Hospital, HIU, MOHMS 2016 Stats*
3. Lautoka Hospital Maternity Annual Stats 2016, Dr Jimi Taria
4. Garite TJ, Clark R, Thorp JA. Intrauterine growth restriction increases morbidity and 310 mortality among premature neonates. Am J Obstet Gynecol. 2004; 191:481-7
5. J Poulter Unpublished Retrospective Audit of Still births at Lautoka Hospital from January 2012 to December 2013
6. Still Birth Registry Lautoka Hospital
7. Battaglia FC, Lubchenco LO. A practical classification of newborn infants by weight and gestational age. J Pediatr 1967; 71:159.
8. R Horton et al. The Lancet Stillbirth Series April 2011
9. Gardosi J, Madurasinghe V, Williams M et al (2013a). Maternal and foetal risk factors for stillbirth: population based study. BMJ 346(7893):15.
10. Stacey T, Thompson J, Mitchell A et al (2012). Antenatal care, identification of suboptimal foetal growth and risk of late stillbirth: findings from the Auckland Stillbirth Study. Australian and New Zealand Journal of Obstetrics and Gynaecology 52(3):242-7.
11. Gardosi J, Kady S, McGeown P et al (2005). Classification of stillbirth by relevant condition at death (ReCoDe): population based cohort study. BMJ 331(7893):1113-17.
12. Manning FA. Intrauterine growth retardation. In: Foetal Medicine: Principles and Practice, Appleton and Lange, Norwalk, CT 1995. p.307
13. De onis M, Blossner M, Villar J: Levels and patterns of intrauterine growth retardation in developing countries. Eur J Clin Nutr. 1998, 52: S83-S93.PubMedGoogle Scholar
14. Director General World Health Organization: Bridging the gaps. The World Health Report 1995. [http://www.who.int/whr/1995/en/index.html]
15. Antonisamy B, Sivaram M, Richard J, Rao PSS: Trends in Intra-uterine Growth of Single Live Births in Southern India. J Trop Pediatr. 1996, 339-341. 42CrossRefPubMedGoogle Scholar
16. Pinheiro A, David A, Joseph B: Pregnancy weight gain and its correlation to birth weight. Indian J Med Sci. 2001, 55: 266-270.PubMedGoogle Scholar
17. Kramer MS, Séguin L, Lydon J, Goulet L 2000 Socio-economic disparities in pregnancy outcome: why do the poor fare so poorly? Paediatr Perinat Epidemiol 14:194–210
18. Drysdale et al Ethnicity and the risk of late pregnancy still birth. MJA. 2012 September;197(5): 278 – 281
19. Thompson Jm et al Risk factors for small-for-gestational-age babies: The Auckland Birthweight Collaborative Study. J Paediatr Child Health. 2001 Aug; 37(4):369-75.
20. Surkan PJ et al Previous Preterm and Small-for-Gestation-Age Births and the Subsequent Risk of Stillbirth. NEMJ 2004 Feb 350 (8): 777-785
21. Wen SW, Goldenberg RL, Cutter GR, et al. Intrauterine growth retardation and preterm delivery: prenatal risk factors in an indigent population. Am J Obstet Gynecol. 1990; 162:213–8
22. Berghella V. Prevention of recurrent foetal growth restriction. Obstet Gynecol. 2007; 110(4):904–12.
23. Lieberman E, Gremy I, Lang JM, Cohen AP. Low birth weight at term and the timing of foetal exposure to maternal smoking. Am J Public Health. 1994; 84(7):1127–31.
24. Shu XO, Hatch MC, Mills J, et al. Maternal smoking, alcohol drinking, caffeine consumption, and foetal growth: results from a prospective study. Epidemiology. 1995; 6:115–20.
25. Campbell S, Wilkin D. Ultrasonic measurement of foetal abdomen circumference in the estimation of foetal weight. Br J Obstet Gynaecol 1975; 82:689.
26. Severi FM, Rizzo G, Bocchi C, et al. Intrauterine growth retardation and foetal cardiac function. Foetal Diagn Ther. 2000 Jan-Feb. 15(1):8-19.
27. Harkness UF, Mari G. Diagnosis and management of intrauterine growth restriction. Clin Perinatol 2004; 31:743.
28. Duff GB. A randomized controlled trial in a hospital population of ultrasound measurement screening for the small for dates baby. Aust N Z J Obstet Gynaecol 1993; 33:374.
29. Belizán JM, Villar J, Nardin JC, et al. Diagnosis of intrauterine growth retardation by a simple clinical method: measurement of uterine height. Am J Obstet Gynecol 1978; 131:643
30. Jelks A, Cifuentes R, Ross MG. Clinician bias in fundal height measurement. Obstet Gynecol 2007; 110:892.
31. Bailey SM, Sarmandal P, Grant JM. A comparison of three methods of assessing inter-observer variation applied to measurement of the symphysis-fundal height. Br J Obstet Gynaecol 1989; 96:1266.
32. Roex A, Nikpoor P, van Eerd E, et al. Serial plotting on customized fundal height charts results in doubling of the antenatal detection of small for gestational age foetuses in nulliparous women. Aust N Z J Obstet Gynaecol 2012; 52:78.
33. Mongelli M, Gardosi J. Symphysis-fundus height and pregnancy characteristics in ultrasound-dated pregnancies. Obstet Gynecol 1999; 94:591.
34. Rosenberg K, Grant JM, Hepburn M. Antenatal detection of growth retardation: actual practice in a large maternity hospital. Br J Obstet Gynaecol 1982; 89:12.
35. Engstrom JL, Ostrenga KG, Plass RV, Work BA. The effect of maternal bladder volume on fundal height measurements. Br J Obstet Gynaecol 1989; 96:987.
36. Hall MH, Chng PK, MacGillivray I. Is routine antenatal care worthwhile? Lancet 1980; 2:78.
37. Bais JM, Eskes M, Pel M, et al. Effectiveness of detection of intrauterine growth retardation by abdominal palpation as screening test in a low risk population: an observational study. Eur J Obstet Gynecol Reprod Biol 2004; 116:164.
38. Harding K, Evans S, Newnham J. Screening for the small foetus: a study of the relative efficacies of ultrasound biometry and symphysiofundal height. Aust N Z J Obstet Gynaecol 1995; 35:160.
39. Duncan KR, Issa B, Moore R, et al. A comparison of foetal organ measurements by echo-planar magnetic resonance imaging and ultrasound. BJOG 2005; 112:43.
40. American College of Obstetricians and Gynecologists. ACOG Practice bulletin no. 134: foetal growth restriction. Obstet Gynecol 2013; 121:1122.

41. Goetzinger KR, Tuuli MG, Odibo AO, et al. Screening for foetal growth disorders by clinical exam in the era of obesity. J Perinatol 2013; 33:352.
42. Sparks TN, Cheng YW, McLaughlin B, et al. Fundal height: a useful screening tool for foetal growth? J Matern Foetal Neonatal Med 2011; 24:708.
43. Engstrom JL, Ostrenga KG, Plass RV, Work BA. The effect of maternal bladder volume on fundal height measurements. Br J Obstet Gynaecol 1989; 96:987.
44. Snijders RJ, Nicolaides KH. Foetal biometry at 14-40 weeks' gestation. Ultrasound Obstet Gynecol 1994; 4:34.
45. Brown HL, Miller JM Jr, Gabert HA, Kissling G. Ultrasonic recognition of the small-for-gestational-age foetus. Obstet Gynecol 1987; 69:631.
46. Chang TC, Robson SC, Boys RJ, Spencer JA. Prediction of the small for gestational age infant: which ultrasonic measurement is best? Obstet Gynecol 1992; 80:1030.
47. Owen P, Khan KS, Howie P. Single and serial estimates of amniotic fluid volume and umbilical artery resistance in the prediction of intrauterine growth restriction. Ultrasound Obstet Gynecol 1999; 13:415.
48. Warsof SL, Cooper DJ, Little D, Campbell S. Routine ultrasound screening for antenatal detection of intrauterine growth retardation. Obstet Gynecol 1986; 67:33.
49. Papageorghiou AT, Ohuma EO, Altman DG, et al. International standards for foetal growth based on serial ultrasound measurements: the Foetal Growth Longitudinal Study of the INTERGROWTH-21st Project. Lancet 2014; 384:869.
50. Kiserud T, Piaggio G, Carroli G, et al. The World Health Organization Foetal Growth Charts: A Multinational Longitudinal Study of Ultrasound Biometric Measurements and Estimated Foetal Weight. PLoS Med 2017; 14:e1002220.
51. De Jong CL, Francis A, van Geijn HP, Gardosi J. Foetal growth rate and adverse perinatal events. Ultrasound Obstet Gynecol 1999; 13:86.
52. Green–top Guideline No. 31 2nd Edition The Investigation and Management of the Small–for–Gestational–Age Foetus February 2013
53. Morris RK, Malin G, Robson SC, Kleijnen J, Zamora J, Khan KS. Foetal umbilical artery Doppler to predict compromise of foetal/ neonatal wellbeing in high–risk popualtion: systematic review and bivariate meta–analysis. Ultrasound Obstet Gynecol 2011; 37: 135–42.
54. Alfirevic Z, Stampalija T, Gyte GL. Foetal and umbilical Doppler ultrasound in high–risk pregnancies. Cochrane Database Syst Rev 2010;(1):CD007529.pub2
55. Grivell RM, Alfirevic Z, Gyte GML, Devane D. Antenatal cardiotocography for foetal assessment. Cochrane Database Syst Rev 2010; (1): CD007863.
56. Nabhan AF, Abdelmoula YA. Amniotic fluid index versus single deepest vertical pocket as a screening test for preventing adverse pregnany outcome. Cochrane Database Syst Rev 2008; (3): CD006593.
57. Lalor JG, Fawole B, Alfirevic Z, Devane D. Biophysical profile for foetal assessment in high risk pregnancies. Cochrane Database Syst Rev 2008; (1): CD007529.
58. Morris RK, Say R, Robson SC, Kleijen J, Khan KS. Systematic review of middle cerebral artery Doppler to predict foetal growth restriction/compromise of foetal wellbeing. Arch Dis Child Foetal Neonatal Ed 2008; 93(Suppl 1):31–6.
59. Baschat AA. Doppler application in the delivery timing of the preterm growth–restricted foetus; another step in the right direction. Ultrasound Obstet Gynecol 2004; 23: 111–8
60. Boers KE, Vijgen SM, Bijlenga D, van der Post JA, Bekedam DJ, Kwee A, et al. Induction versus expectant monitoring for intrauterine growth restriction at term: randomised equivalence trial (DIGITAT). BMJ 2010; 341.
61. GRIT Study Group. A randomised trial of timed delivery for the compromised preterm foetus: short term outcomes and Bayesian interpretation. BJOG 2003; 110:27–32.
62. Chernausek SD. Update: consequences of abnormal foetal growth. J Clin Endocrin Metab 2012; 97(3):689–695.
63. Grivell RM, Wong L, Bhatia V. Regimens of foetal surveillance for impaired foetal growth. Cochrane Database Syst Rev 2012; CD007113.
64. Lesly McCowan et al Risk Factors for small for gestational age. Best Practice & Research Clinical Obstetrics and Gynaecology 2009 Vol. 23 (6): 779 – 793.
65. K. Hughes, N.R. Tan, K.C. Lun Low birthweight of live singletons in Singapore, 1967–1974 Int J Epidemiol, 13 (1984), pp. 465-471
66. S Muthayya et al. Low maternal vitamin B12 status is associated with intrauterine growth retardation in urban South Indians. European Journal of Clinical Nutrition (2006) 60, 791–801.
67. Shoham-Vardi Ilana et al The association of primiparity with intrauterine growth retardation. Eur J Obstet Gynecol Reprod Biol. 1994 Feb; 53(2):95-101.
68. Virisila Sema et al. Unpublished Modes of diagnosis, management and outcomes of diabetes in pregnancy at Lautoka Hospital from January 2013 to January 2015.
69. WHO STEPs Chronic Disease Risk Factor surveillance Fact Sheet; www.who.int/chp/steps (Accessed 13/7/17)
70. Simpson JW et al: Responsibility of the obstetrician to the foetus, II. Influence of prepregnancy weight and pregnancy weight gain on birth weight. Obstet Gynecol 45(4):481, 1975
71. Vasitia Cati Unpublished The impact of Obesity on pregnancy outcomes at Lautoka Hospital from Jan-Dec 2012.
72. M.E. Kleijer, G.A. Dekker, A.R. Heard Risk factors for intrauterine growth restriction in a socio-economically disadvantaged region J Matern Foetal Neonatal Med, 18 (2005), pp. 23-30
73. Naoko Kozuki et al Moderate to Severe, but Not Mild, Maternal Anaemia Is Associated with Increased Risk of Small-for-Gestational-Age Outcomes. J. Nutr. February 1, 2012 vol. 142 no. 2 358-362
74. K.M. Groom, R.A. North, K.K. Poppe, et al. The association between customized small for gestational age infants and pre-eclampsia or gestational hypertension varies with gestation at delivery Br J Obstet Gynaecol, 114 (2007), pp. 478-484
75. Giuliano, N et al IUGR Management: New Perspectives. Journal of Pregnancy; New York 2014
76. Amer Imdad et al. Screening and triage of intrauterine growth restriction (IUGR) in general population and high risk pregnancies: a systematic review with a focus on reduction of IUGR related stillbirths. BMC Public Health201111(Suppl 3):S1
77. SOGC Clinical Practice Guideline (https://sogc.org/wp-content/uploads/2013/08/August2013-CPG295-ENG-Revised.pdf) Accessed 3/6/17.
78. RANZCOG Clinical Guideline (https://www.ranzcog.edu.au/RANZCOG_SITE/media/RANZCOG-MEDIA/Women%27s%20Health/Statement%20and%20guidelines/Clinical-Obstetrics/Algorithm-May-2014-(1).pdf?ext=.pdf) Accessed 14/6/17.
79. Gunyeli, Ilker. Histopathological analysis of the placental lesions in pregnancies complicated with IUGR and stillbirths in comparison with noncomplicated pregnancies. Journal of the Turkish German Gynecological Association. 12(2) 75-79.
80. Mercedes de Onis 2020 Focus 5 (Health and Nutrition Emerging and Reemerging Issues in Developing Countries), Brief 6 of 11, February 2001.