Original Article

Screening, investigation and follow-up of neonates with small for gestational age in a metropolitan hospital in Australia—observational retrospective study

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Background: Infants with small for gestational age (SGA) have an increased risk of short and long-term health outcomes, with potentially modifiable risk factors. This study aims to determine the prenatal risk factors associated SGA and evaluate the clinical management of affected infants.

Methods: An observational retrospective study of medical records of infants born at Nepean Hospital and discharged with a diagnosis of SGA over 5 years (1st January 2015 to 31st December 2019). Data included demographic details, antenatal care, maternal risk factors and clinical management of the infants.

Results: Six hundred and seven infants had a discharge diagnosis of SGA, from 20,392 infants born. Of the 607 infants identified, 487 (80%) had SGA, 97 (16%) had asymmetrical SGA, 175 (29%) had symmetrical SGA, and 50 (8%) were incorrectly diagnosed with SGA based on growth measurements taken at birth. The most prevalent maternal risk factors were the presence of chronic disease (n=402, 66.23%), current smoking (n=159, 26.19%), social work input (n=108, 17.79%), gestational diabetes mellitus (n=96, 15.82%) and Aboriginal background (n=73, 12.03%). Prenatal genetic testing was conducted in 89.62% (n=544); 58.81% (n=357) had placental abnormalities; 36.57% (n=222) were recommended follow-up with a general practitioner (GP) and paediatrician, and 21.09% (n=128) were recommended a combination of midwifery in the home (MITH), GP, and paediatric follow-up. Two infants were recorded with no follow-up.

Conclusions: Diagnostic inaccuracies were found in infants with SGA. More intensive antenatal care for women with risk factors for SGA might improve the health of those with chronic disease; support for smoking cessation could also be offered.

Keywords: Small for gestational age (SGA); neonate; risk factor; screening; intrauterine growth restriction (IUGR)

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Introduction

All infants with a birth weight (BW) below the tenth percentile are referred to as small for gestational age (SGA) (1). A proportion of SGA infants may be constitutionally small or may have pathological restriction, called intrauterine growth restriction (IUGR). Identifying SGA infants is important as SGA carries an increased risk of perinatal morbidity and mortality.

Infants who are born with a lower weight than would be expected for their sex and gestational age are at a higher risk of neurodevelopmental delay and other pathologies, including cardiovascular disease, diabetes mellitus and coagulopathies (2). Long-term follow-up of these infants is particularly important so that they can receive appropriate intervention, both in the "catch-up" phase and afterwards, on account of their increased risk of morbidity and mortality (3).

Risk factors for SGA can be divided into maternal and placental factors (1). Maternal factors include smoking, drug and alcohol use, maternal chronic health conditions, in particular, metabolic and vascular disease and previous stillbirths or babies with SGA (4). Maternal and foetal genetic composition and epigenetic changes associated with many of these risk factors play a significant role in the outcome of SGA/IUGR (5).

Highlight box

Key findings

- This study found that a significant proportion of small for gestational age (SGA) infants are missed in the discharge diagnosis. The most prevalent maternal risk factors were the presence of chronic disease (n=402, 66.23%), current smoking (n=159, 26.19%), social work input (n=108, 17.79%), gestational diabetes mellitus (n=96, 15.82%) and Aboriginal background (n=73, 12.03%). 58.81% (n=357) had placental abnormalities.

What is known and what is new?

- Infants with SGA are at a higher risk of perinatal morbidity and mortality, with risk factors ranging from maternal and foetal genetics, to potentially modifiable risks.
- The most significant maternal risk factors for SGA in this study were Aboriginal and Torres Strait Islander background and maternal diabetes. SGA was more prevalent in term infants than pre-term infants.

What is the implication, and what should change now?

- Routine documentation of birth weight percentile at time of discharge is recommended to ensure SGA infants are not missed, and therefore ensure appropriate follow-up is arranged.

Importance of screening and follow-up

The importance of this research lies in the short and long-term health outcomes of infants with SGA. The sequelae are quite extensive, with research demonstrating that IUGR and SGA can affect many physiological systems into adulthood. In the short term, infants with SGA are likely to have a longer period of neonatal intensive care compared to gestational age-matched infants (6). Short-term complications can include perinatal asphyxia, hypothermia, hypoglycaemia, and polycythaemia (1). Anecdotally, some infants are lost to follow-up, and so careful monitoring of longer-term outcomes is often not recorded.

Terminology

The terms IUGR and SGA are often used interchangeably, and definitions vary across the literature. Within this study, the inclusion criteria were a discharge diagnosis of IUGR, fetal growth restriction (FGR) or SGA. Our cohort was then assessed for SGA, defined as a BW of less than the 10th percentile corrected for sex and gestational age (1).

IUGR is a clinical definition of pathological growth restriction, regardless of percentiles (4). SGA infants can be classified as symmetric or asymmetric. Symmetric SGA refers to a proportionately small neonate, with weight, head circumference (HC) and length (L) all less than the 10th percentile. Infants with asymmetric SGA have a BW less than the 10th percentile, and at least one other growth parameter being normal. This may occur with fetal weight loss late in the pregnancy due to placental dysfunction. Generally, asymmetric SGA babies have a better prognosis than symmetrical SGA babies (4).

This study will examine infants with a discharge diagnosis of SGA, IUGR and FGR at Nepean Blue Mountains Local Health District (NBMLHD). The scope of this study is to identify risk factors and assess the screening and follow-up, with a view to improve perinatal outcomes. We present this article in accordance with the STROBE reporting checklist (available at https://tp.amegroups.com/article/view/10.21037/tp-24-83/rc).

Methods

This was a retrospective observational study that examined the medical records of infants born at Nepean Hospital who also had received antenatal care within the NBMLHD.

The study period spanned 5 years (1st January 2015 to 31st December 2019).

Population and sample

The study cohort comprised all infants born 1st January 2015 to 31st December 2019 at the Nepean Hospital who had a discharge diagnosis of SGA, IUGR and FGR. Nepean Hospital is a metropolitan teaching hospital in New South Wales (NSW), Australia. The NBMLHD consists of five hospitals and nine community health centres. It has a population of 361,656 (7), which includes higher proportions of residents identifying as Aboriginal and Torres Strait Islander [3.6% *vs.* NSW state average of 2.9% (7)] and higher rates of teen pregnancy (2.9% *vs.* NSW average of 1.9%) (7).

Data source

The study cohort was identified by a search of the electronic obstetric database ('Obstetrix') for infants with a discharge diagnosis of IUGR, SGA or FGR. Each individual's electronic medical record was retrieved for relevant clinical information. The study participants' parameters of gestational BW, L, and HC were recorded, and percentiles were ascertained to assess diagnostic accuracy. Percentiles were based on the in-built clinical growth chart from the Centre for Disease Control (CDC) (8). Gestational age was recorded and defined by the clinical estimate from early pregnancy ultrasounds or the first day of the last menstrual period. BW percentiles for the entire cohort were recalculated using the Fenton size at birth calculator which corrects for sex and gestational age (9).

Study participants were categorised into four groups: SGA, asymmetrical, symmetrical or misdiagnosed. SGA was defined as a BW <10th centile, asymmetrical as only BW <10th centile, symmetrical as all parameters <10th centile, and misdiagnosed infants refers to an infant with a discharge diagnosis of SGA, however parameters were all within the normal range. Four infants were excluded due to a lack of available data. Ethics approval for this study was obtained from the Human Research Ethics Committee of Nepean Blue Mountains Local Health District (No. 2020/ETH01646). Individual consent for this retrospective analysis was waived. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013).

The prenatal variables investigated include postcode, aboriginality, social work input, smoking status, alcohol and drug use during pregnancy, chronic health conditions, body mass index (BMI), pre-eclampsia and placental abnormalities and when available, TORCH screening (toxoplasmosis, rubella, cytomegalovirus, and herpes simplex virus) and prenatal genetic testing. Maternal chronic conditions were identified by the presence of neurological, musculoskeletal, cardiovascular, respiratory, gastrointestinal, renal, genitourinary, vascular, haematological, endocrine, psychiatric, immunological conditions. Placental histopathology reports of our cohort were analysed, and the pathological terms (e.g., small placenta, chorioamnionitis) were collated.

Postnatal diagnostic investigations carried include genetic testing [comparative genomic hybridization (CGH) and single nucleotide polymorphism (SNP) array] and neonatal cranial ultrasound. Finally, the type of follow-up that was arranged (paediatrician, GP, or child health nurse) for babies with SGA was reviewed.

Statistical analysis

All the statistical analyses were performed using SPSS version 29.0.1.0 (IBM Corp., Armonk, NY, USA). Categorical data were presented as numbers and comparisons between categorical variables were performed using the Chi-squared tests. Such analyses are demonstrated in Table S1.

Results

During the study period, 607 babies (3%), out of a total of 20,392 had a discharge diagnosis of SGA. Based on the sampled data, only 2% (n=487) of infants born at NBMLHD would qualify for being SGA (BW <10th centile). Given that this is substantially less than the expected 10% of the birth cohort, we calculated the percentage of the study cohort at each weight percentile. If the cohort with SGA had precisely included the lowest 10% for BW corrected for sex and GA and that the reference population matched our local population, we would expect each of the percentiles from 0 to 9 to equate to 10% of our study cohort. *Figure 1* demonstrates under-representation of infants at the 5th to 9th percentiles. The majority (76%) of the term infants qualified SGA but only slightly more than half (57%) of the preterm infants were correctly identified SGA.

SGA distribution in pre-term and term babies in a cohort of 607 infants

Figure 2 illustrates SGA distribution according to BW, HC

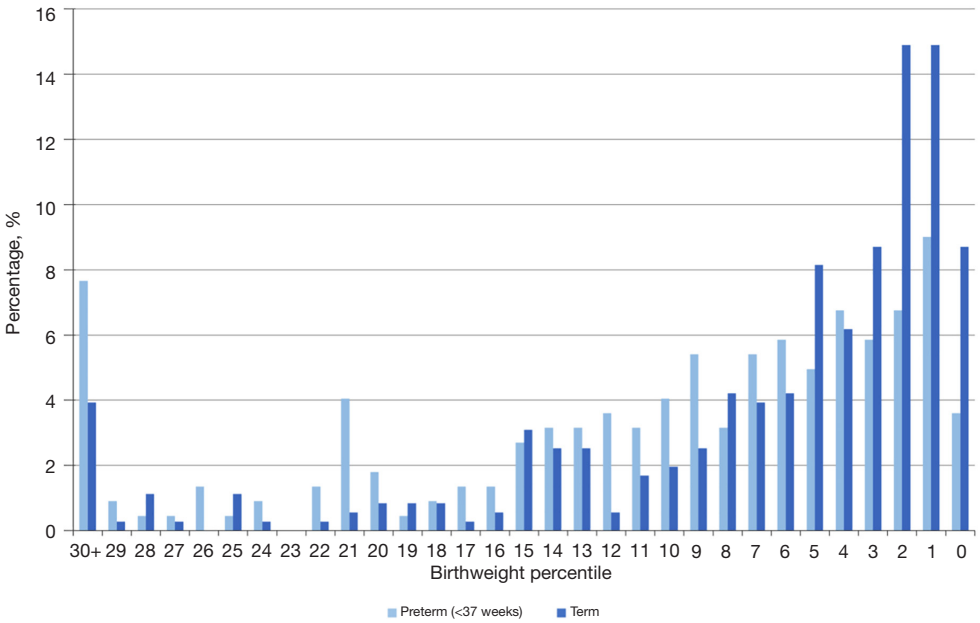

Figure 1 Bar chart of the percentage of preterm and term infants at each birth weight percentile corrected for sex and GA in a cohort of 607 infants (chart created using Fenton 2013 preterm growth chart) (9). GA, gestational age.

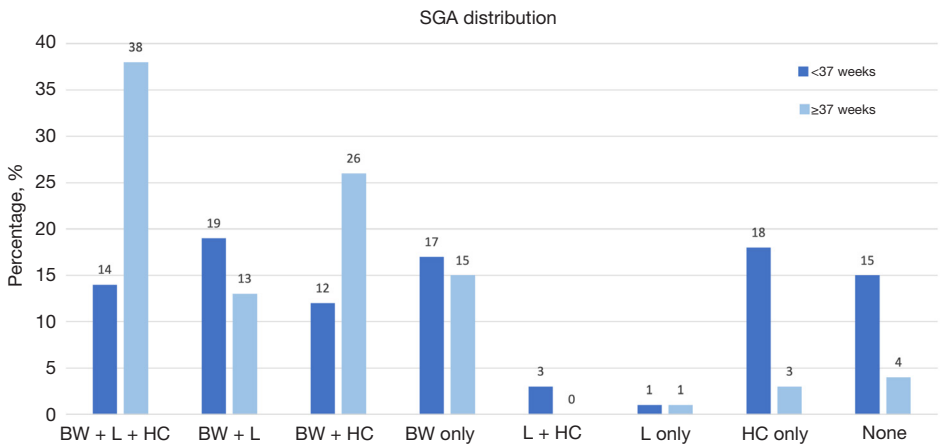

Figure 2 Bar graph illustrating small for gestational age distribution according to BW, HC and L in 607 infants. SGA, small for gestational age; BW, birth weight; L, length; HC, head circumference.

and L. Symmetrical SGA affected term babies more than pre-term babies (38% and 14% respectively). For pre-term babies, the proportions of BW + L, HC only and BW only were comparable (19%, 18% and 17% respectively). By contrast, in term babies, the highest proportion was symmetrical SGA with all three parameters affected (38%), followed by BW + HC (26%). A greater proportion of pre-term babies had a discharge diagnosis of SGA but did not have SGA based on measurements made (15% *vs.* 4%).

Maternal risk factors

Table 1 provides an overview of infants born with SGA for each identified maternal risk factor in the NBMLHD. The most significant risk factors were chronic disease (n=402, 66.23%), smoking (n=159, 26.19%), social work input (n=108, 17.79%), gestational diabetes mellitus (n=96, 15.82%) and Aboriginal background (n=73, 12.03%). The most prevalent chronic diseases were the presence of a

Table 1 Maternal risk factors identified in cohort of 607 infants

Maternal risk factors	Infants with SGA (n=607), n (%)
Aboriginal and Torres Strait Islander	75 (12.36)
Aboriginal	73 (12.03)
Torres Strait Islander	2 (0.33)
Social work input	108 (17.79)
Smoking	
Ex-smoker	20 (3.29)
Current smoker	159 (26.19)
Urine drug screen: positive	27 (4.45)
Chronic disease	402 (66.23)
Diabetes	
T1DM	3 (0.49)
GDM	96 (15.82)
Alcohol and drug use	47 (7.74)
BMI, kg/m^2	
<18.5	40 (6.59)
35–39.9	22 (3.62)
>40	23 (3.79)

Chronic disease: neurological, musculoskeletal, cardiovascular, respiratory, gastrointestinal, renal, genitourinary, vascular, haematological, endocrine, psychiatric, immunological. SGA, small for gestational age; T1DM, type 1 diabetes mellitus; GDM, gestational diabetes mellitus; BMI, body mass index.

psychiatric condition, gestational diabetes mellitus and respiratory disease (*Figure 3*).

Antenatal risk factors

Six hundred and seven infants were reviewed for possible antenatal risk factors of SGA; 93.74% of TORCH screening results were negative (*Table 2*). In the positive 5.93%, hepatitis was the most common (2.64%, n=16), followed by herpes (1.98%, n=12) and cytomegalovirus (CMV; 0.82%, n=5) (*Table 2*). During this study period, 0.16% (n=1) of SGA infants had toxoplasmosis, rubella, HIV or rubella, and no SGA infants had syphilis or Epstein-Barr virus.

Notably, only 8.73% of infants (n=53) underwent prenatal genetic testing, either by amniocentesis, chorionic villus sampling (CVS) or non-invasive prenatal testing (NIPT). Of the data gained from those who underwent prenatal testing, abnormal results were found in 5 out of 23 (21.74%) amniocentesis results, 2 out of 5 (40%) CVS results and 16 out of 25 (64%) NIPT results (*Table 3*). More individuals underwent NIPT (n=25) as it is used as a screening test prior to CVS or amniocentesis (10).

Pre-eclampsia was the most common hypertensive condition at 13.51% (n=82), followed by new-onset hypertension (HTN) at 3.95% (n=24).

Placental risk factors

Out of the 607 SGA infants, 357 (58.81%) had placental

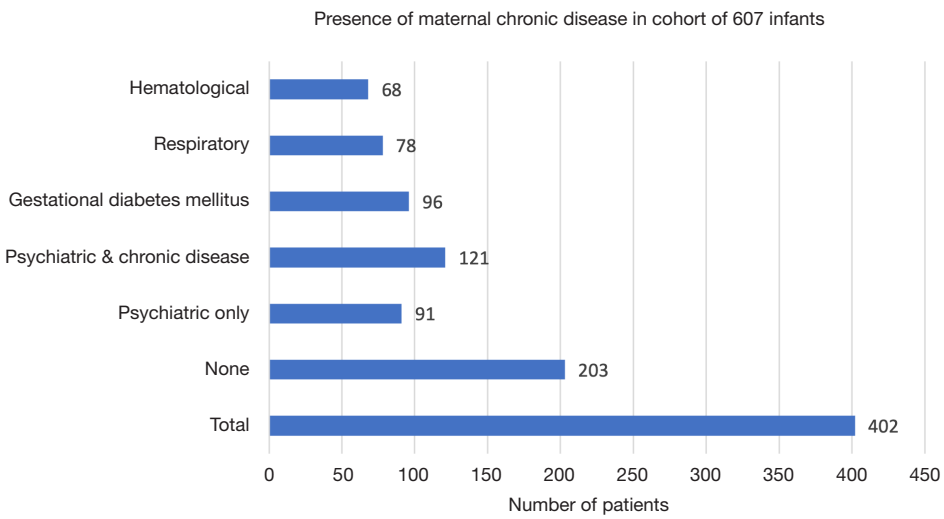

Figure 3 Bar chart of maternal chronic disease in cohort of 607 infants. Note that patients were likely to have >1 diagnosis.

Table 2 Antenatal risk factors in the cohort of 607 infants

Antenatal risk factors	Infants with SGA (n=607), n (%)
TORCH screening	
Negative	569 (93.74)
Positive	36 (5.93)
Toxoplasmosis	1 (0.16)
Rubella	1 (0.16)
Cytomegalovirus	5 (0.82)
Herpes simplex virus	12 (1.98)
HIV	1 (0.16)
Syphilis	0
Epstein-Barr virus	0
Varicella	1 (0.16)
Hepatitis	16 (2.64)
Abnormal prenatal genetic testing	
None performed	544 (89.62)
Amniocentesis	5 (0.82)
CVS	2 (0.33)
NIPT	16 (2.64)
Hypertensive disorder	
Pre-eclampsia	82 (13.51)
Pre-existing HTN	10 (1.65)
New HTN	24 (3.95)
HELLP syndrome	2 (0.33)
Eclampsia	1 (0.16)
Placental abnormalities	356 (58.65)

SGA, small for gestational age; TORCH, toxoplasmosis, rubella, cytomegalovirus, and herpes simplex virus; HIV, human immunodeficiency virus; CVS, chorionic villus sampling; NIPT, non-invasive prenatal testing; HTN, hypertension; HELLP, haemolysis, elevated liver enzymes, low platelets.

abnormalities (Table S2). The two most common placental anomalies found were small placenta (20.1%) and placental infarcts (19.1%) (*Figure 4*).

Postnatal screening

Post-natal screening of SGA infants consisted of a head ultrasound and genetic screening; 20% of IUGR/SGA infants had an abnormal head ultrasound, and 47.45%

Table 3 Prenatal genetic testing in the cohort of 607 infants

Prenatal genetic testing	Infants with SGA (n=607), n (%)	
	Normal result	Abnormal result
None performed	544 (89.62)	
Amniocentesis	18 (2.97)	5 (0.82)
CVS	3 (0.49)	2 (0.33)
NIPT	9 (1.48)	16 (2.64)

SGA, small for gestational age; CVS, chorionic villus sampling; NIPT, non-invasive prenatal testing.

(n=288) did not receive a head ultrasound scan (USS) (*Table 4*). A large majority (83.86%) did not receive post-natal genetic testing. Proportionately, there were more abnormal CGH results (5.93%) compared to SNP results (0.33%) (*Table 4*).

Six hundred and seven patients with an SGA diagnosis were evaluated for post-natal follow-up. Recommended follow-up included midwifery in the home (MITH), general practitioner (GP), paediatrician, or a combination of these. The most common recommended follow-up included GP and paediatrician (222, 36.57%), followed by MITH + GP + paediatrician (128, 21.09%) and then MITH + GP (121, 19.93%) (*Table 5*). GPs were noted to be the main follow-up (94.56%), either on their own or in combination with other types of follow-ups. Two infants were not recorded to receive follow-up (*Table 5*).

Discussion

Summary of principal findings

During the study period, 607 babies (3%), out of a total of 20,392 had a discharge diagnosis of SGA/IUGR/FGR; 2% (n=487) of infants from our study cohort had SGA based on BW <10th centile; 57% of the preterm infants were correctly identified SGA.

The most common maternal risk factors were chronic disease (n=402, 66.23%), smoking (n=159, 26.19%), social work input (n=108, 17.79%), gestational diabetes mellitus (n=96, 15.82%) and Aboriginal background (n=73, 12.03%).

Fifty-two infants (8.6%) were identified as twins, which is a known risk factor for SGA (11). Additionally, 58.94% (n=356) of infants had associated placental abnormalities, with the two most common being small placenta (20.1%) and placental infarcts (19.1%) (Table S2).

Infants (n=53, 8.73%) received prenatal genetic testing,

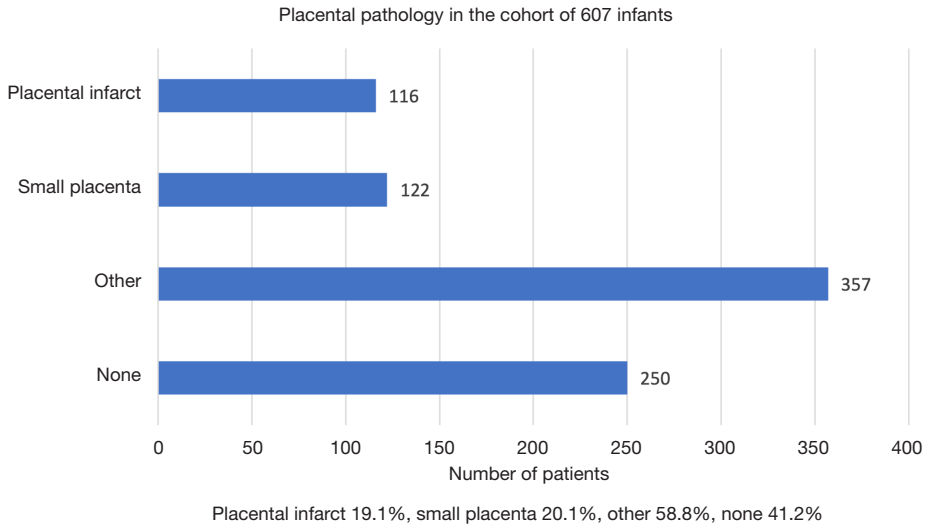

Figure 4 Bar chart of placental pathology in the cohort of 607 infants.

Table 4 Postnatal screening of the cohort of 607 infants

Post-natal screening	No investigation (n=607), n (%)	Normal result (n=607), n (%)	Abnormal result (n=607), n (%)
Head USS	288 (47.45)	197 (32.45)	122 (20.10)
Genetic screening			
None	509 (83.86)	–	–
CGH	–	60 (9.88)	36 (5.93)
SNP	–	0	2 (0.33)

USS, ultrasound scan; CGH, comparative genomic hybridisation; SNP, single-nucleotide polymorphism.

Table 5 Recommended outpatient follow-up of cohort of 607 infants

Recommended outpatient follow-up	SGA infant with follow-up, N (%)
None	2 (0.33)
MITH	1 (0.16)
GP	103 (16.97)
Paediatrician	25 (4.12)
MITH + GP	121 (19.93)
MITH + paediatrician	5 (0.82)
GP + paediatrician	222 (36.57)
MITH + GP + paediatrician	128 (21.09)

SGA, small for gestational age; MITH, midwifery in the home; GP, general practitioner.

either by amniocentesis, CVS or NIPT.

The most common recommended follow-up included GP and paediatrician (n=222, 36.57%), with only two infants having no recorded follow-up recommendation.

We found that 8% of infants (15% pre-term and 4% term) had a discharge diagnosis of SGA/FGR/IUGR but all parameters (BW, HC, L) were >10th centile. Overall, 43% of pre-term and 24% of term infants had a BW >10th centile and therefore did not meet the definition of SGA.

Maternal risk factors

Due to ongoing socio-economic disadvantage in the NBMLHD, high rates of poor health literacy and low socio-economic status may lead to a larger number of

pregnant women having ongoing chronic diseases and engaging in high-risk health behaviours. As such, it can be hypothesised that higher rates of IUGR/SGA are seen as a result of the poorer health status of our mothers. The maternal risk factors of chronic disease, smoking, social work input, gestational diabetes mellitus and Aboriginal background strongly correlated with SGA (Table S1), which is consistent with current literature. In this study, chronic disease and smoking were the most common risk factors, whilst Aboriginal and Torres Strait Islander background and diabetes were of significant correlation (Table S1). The most significant risk factors identified in Royal College of Obstetricians and Gynaecologists (RCOG) 2014 were maternal diabetes [odds ratio (OR) 6], renal impairment (OR 5.3), chronic HTN [absolute risk reduction (ARR) 2.5], maternal age >40 years (OR 3.2), cocaine use (OR 3.23) and smoking >11 cigarettes/day (OR 2.21). A comparison of maternal age could not be identified in this study as this variable was not measured.

Other significant risk factors identified in the RCOG 2014 that were not assessed in our study included antiphospholipid syndrome [risk ratio (RR) 6.22], previous SGA baby (OR 3.9), stillbirth (OR 6.4), pregnancy-associated plasma protein A (PAPP-A) <0.4 (OR 2.6) and maternal (OR 2.64) or paternal (OR 3.47) history of SGA.

Thekkedathu's (12) prospective study reviewed 60 singleton pregnancies born over 28 weeks' gestation who were diagnosed with IUGR. The study found that statistically significant maternal risk factors for IUGR included chronic HTN, pre-eclampsia, low socioeconomic status of the mother, diabetes, anaemia, gestational diabetes mellitus and hypothyroidism, which all had a P value of <0.001. This is consistent with data within the retrospective audit. Maternal factors that were not significantly associated with IUGR were extremes of maternal age (P=0.56), previous pregnancy with IUGR (P=0.31), antiphospholipid syndrome (P=0.16), assisted reproductive techniques (P=0.73), anticonvulsants (P=0.16), and renal disease (P=0.32).

Placental factors were consistent with Thekkedathu's study (12). Statistically significant placental factors include chorangiomatosis, increased syncytial knotting, villous infarction, increased peri-villous fibrinoid deposition, accelerated villous maturation, retroplacental haemorrhage and acute chorioamnionitis (Table S2). Notably, a placental factor of single umbilical artery had no significant association with IUGR. A large number of IUGR/SGA infants are caused by placental insufficiency, which leads to foetal haemodynamic adaptations (6). Additionally, our study identified 52 twins (8.6%) with a discharge diagnosis of SGA. FGR accounts for approximately 25–47% of twin pregnancies, with placental insufficiency being the most common cause (11).

Antenatal screening/antenatal risk factors

Screening of IUGR/SGA babies was minimal, with no antenatal genetic screening performed on 89.62% of IUGR/SGA infants. NIPT is not available through the Medicare Benefits Schedule (MBS) nor covered by private health insurance (10) and may correlate to low rates of prenatal genetic testing in the NBMLHD; 94.05% of TORCH screening results were negative. The most significant neonatal risk factors were hepatitis viruses and herpes, and surveillance of such in this district is recommended.

Post-natal follow-up

The short- and long-term morbidities associated with IUGR/SGA ascertain the importance of post-natal follow-up. Current NSW recommendations suggest a surveillance head ultrasound for infants with severe IUGR, congenital infections (CMV, meningitis), antenatally diagnosed intracranial abnormalities, large/small HC and seizures (13). Premature infants are at a higher risk for intraventricular haemorrhages; 20% of SGA infants had an abnormal head ultrasound, and 47.45% (n=288) did not receive a head USS. This is consistent in Roufaeil *et al.*'s systematic review, noting 19.4% to 33% of pre-term infants with cranial ultrasound abnormalities (14).

A large majority (83.96%) did not receive genetic testing to determine the cause of IUGR/SGA. Only two infants had SNP, whilst 96 had CGH testing. CGH offers increased sensitivity, higher resolution, and cost-effectiveness (15), which may indicate the disparity between testings. Current literature recommendations for SNP/CGH testing were found to be scarce. Stalman's study found 19% (4 out of 21 infants) had a genetic abnormality on CGH (16). However, the small cohort size of 21 SGA infants makes it difficult to draw conclusions (16). Further, Paz *et al.*'s study identified 274 (0.75%) of extreme low BW [<2.5 standard deviation (SD)] in 36,405 term infants (17). Seven (11%) reported a postnatal diagnosis of a genetic syndrome, and 18 (29%) agreed to undergo both exome sequencing and neurodevelopmental testing. Six (33%) of these participants who underwent developmental testing

showed poor neurodevelopment. In Australia, it is currently recommended that all newborns receive a bloodspot screening test, which screens for particular genetic and metabolic conditions.

IUGR/SGA babies (94.56%) received follow-up with a GP. GPs are an integral follow-up tool for monitoring postnatal catch-up growth. The long-term complications can involve growth retardation and poor neurodevelopmental outcomes, and it is estimated that catch-up growth for term IUGR infants is completed by 2 years of age. Endorsement of health promotion through the primary care provider should be maintained.

Strengths

The study provides a sequential assessment from prenatal screening to postnatal follow-up, offering a comprehensive view of antenatal care in the NBMLHD network. This can inform areas for further development and reform policy. The study was conducted over 5 years, allowing a large cohort (607 infants), increasing the validity of the data.

Limitations

A limitation of our study is that infants were found by discharge diagnosis of SGA/FGA/IUGR, and not by birth parameters (BW, HC, L). As a result, some babies with parameters <10th centile were missed. The frequency distribution of BW percentiles in *Figure 1* provides a descriptive estimate of infants likely missed in our study.

Another limitation is in the definition of the maternal risk factor of chronic disease. Chronic disease was defined as a broad term encompassing various systems (neurological, cardiovascular, etc.). The generalisability of this data is similar to the placental risk factors where findings such as 'small placenta' were determined by pathology reports as opposed to numerical definitions.

Our study found that 15% of pre-term babies had a discharge diagnosis of SGA but did not have SGA based on BW. There is an existing discrepancy between the definitions of IUGR and SGA, with variations noted within the literature. This can lead to false diagnoses and, therefore, inaccurate data interpretation.

Data on maternal risk factors predisposing to symmetric and asymmetric SGA was not analysed. As risk factors for SGA infants are vast, and in addition to logistical barriers of the database used, not all factors could be assessed. Risk factors omitted include maternal age, use of assisted reproductive technologies, previous history of IUGR/SGA and level of antenatal care (1). RCOG 2014 identified these risk factors as OR <2 [in vitro fertilization (IVF) singleton pregnancy—OR 1.6, pregnancy interval <6 months—adjusted OR (aOR) 1.26]. Such risk factors were unavailable in the NSW eMaternity database, preventing inclusion in this study.

Similarly, the prevalence of placental conditions such as a single umbilical artery, placental haemangiomas, placenta previa, low-lying placenta and chronic placental abruption were not examined. Such conditions may contribute towards IUGR/SGA to varying degrees (1), which can be studied in a future audit.

Implications of the study

Our study has found the prevalence of SGA within NBMLHD and the diagnostic inaccuracies in the sampled data. The study highlights modifiable risk factors of SGA and values the importance of follow-up in long-term health outcomes.

Our recommendation is at the time of discharge, the doctor should formally check the percentile of BW, as we are missing infants in our discharge diagnosis. It can be hypothesised that the infants missing from our cohort are closer to the 10th centile. Further discussion is required to determine the clinical impact of increased SGA identification. For example, pre-term babies are more likely to be misclassified as SGA as opposed to term babies. Further, there is a risk of increased morbidity and parental concern for constitutionally small infants.

We recommend increased educational programs to inform clinicians of the differences between SGA, FGR and IUGR, with clear and well-defined guidelines to prevent diagnostic errors. Teaching guidelines can be obtained from the Perinatal Society of Australia and New Zealand (18) and the NSW Health Guideline on Fetal Growth Restriction (19). This study will also enable early neonatal detection and education on the importance of long-term follow-up. Hospital practices can restructure priorities for screening and follow-up. Routine prenatal surveillance of SGA risk factors specific to the health district is recommended to identify modifiable risk factors and decrease the likelihood of SGA.

For health professionals involved in follow-up at a primary care level, long-term follow-up as a preventative health measure may reduce potential adverse outcomes. This includes health promotion and literacy on known

risk factors for SGA. The endorsement of an Aboriginal and Torres Strait Islander health practitioner may aid the communication of health information within the NBMLHD.

Conclusions

We conclude that SGA is complex, with definition disparities, multifactorial risk factors and low levels of prediction, hence requiring a coordinated approach. Our retrospective audit provides local data that can inform decision-making within the NBMLHD. Targeted health promotion and improving health literacy to parents and health professionals during antenatal and postnatal periods can decrease adverse outcomes. Improved antenatal surveillance of identified risk factors and appropriate postnatal screening and follow-up are critical to reducing neonatal mortality and morbidity. Further research collaborating with other hospitals situated in lower socioeconomic areas is encouraged to establish a correlation between results and geographical location.

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Footnote

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Supplementary

Table S1 Relationship between risk factors and gestational age in the cohort of 607 infants

Risk factor	Pre-term (<37 weeks)	Term (≥37 weeks)	Total	P value
Maternal risk factors				
Aboriginal	34	39	73	
Torres Strait	1	1	2	
Aboriginal and Torres Strait Islander				0.08
Social issues	41	67	108	0.84
Ex-smoker	6	14	20	
Current smoker	60	99	159	
Smoking history				0.82
Positive drug urine	9	18	27	0.46
Alcohol & drug use	14	33	47	0.24
Chronic disease	155	247	402	0.36
Type 1 diabetes	3	0	3	0.02
Type 2 diabetes	0	0	0	0
Gestational diabetes mellitus	40	56	96	0.23
BMI <18.5 kg/m^2	10	30	40	0.13
BMI 35–39.9 kg/m^2	6	16	22	0.66
BMI >40 kg/m^2	11	12	23	0.24
Antenatal risk factors				
Torch screen	11	25	36	0.67
Toxoplasmosis	0	1	1	
Rubella	0	1	1	
CMV	2	3	5	
Herpes	5	5	10	
HIV	0	1	1	
Syphilis	0	0	0	
Hepatitis	4	12	16	
Epstein-Barr virus	0	0	0	
Varicella	0	1	1	
Genetic screening (antenatal)				
None	199	350	549	
Normal amniocentesis	7	12	19	
Abnormal amniocentesis	1	3	4	
Normal CVS	2	1	3	
Abnormal CVS	1	1	2	
Normal NIPT	12	12	24	
Abnormal NIPT	1	0	1	
Hypertensive conditions				
None	156	328	484	
Pre-existing HTN	7	2	9	0.009
New HTN	13	11	24	0.06
Pre-eclampsia	46	36	82	<0.001
HELLP	1	0	1	0.18
Eclampsia	1	0	1	0.18
Placental abnormalities				
No placental abnormality	47	201	248	
Placental abnormality	178	178	356	<0.001
Head USS				
Normal USS	95	101	196	
Abnormal USS	67	55	122	<0.001
Genetic screening (postnatal)				
None	174	333	507	
CGH				
CGH normal	36	14	69	
CGH abnormal	33	13	27	0.36
Total CGH	50	46	96	
SNP				
SNP normal	0	0	0	
SNP abnormal	0	2	2	0.29
Total SNP	0	2	2	
Recommended post-natal follow-up				
None	2	0	2	0.06
Midwifery in the home	2	0	2	0.18
GP	2	64	66	0.77
Paediatrician	2	11	13	0.09
Midwifery in the home + GP	2	94	96	<0.001
Midwifery in the home + paediatrician	2	2	4	0.27
GP + paediatrician	2	117	119	<0.001
All 3	2	93	95	0.02

BMI, body mass index; CMV, cytomegalovirus; HIV, human immunodeficiency virus; CVS, chorionic villus sampling; NIPT, non-invasive prenatal testing; HTN, hypertension; HELLP, haemolysis, elevated liver enzymes, low platelets USS, ultrasound; CGH, comparative genomic hybridisation; SNP, single-nucleotide polymorphism; GP, general practitioner.

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Table S2 Placental abnormalities identified in the cohort of 607 infants

Placental pathology	Number (n=607), N (%)
None	250 (41.19)
Small placenta	122 (20.10)
Placental infarcts	116 (19.11)
Chorangiosis	1 (0.16)
Placenta praevia	6 (0.99)
Immature	28 (4.61)
Calcifications	2 (0.33)
Placental insufficiency	1 (0.16)
Chorioamnionitis	27 (4.45)
Subchorionic thrombi	8 (1.32)
Fibrin deposition	6 (0.99)
Umbilical cord vasculitis	5 (0.82)
Over-coiled cord	16 (2.64)
Chorionitis	6 (0.99)
Placental vasculitis	4 (0.66)
Haemorrhage	3 (0.49)
Thin umbilical cord	1 (0.16)
Foetal thrombotic vasculopathy	2 (0.33)
Increased syncytial knots	21 (3.46)
Intervillous thrombus	24 (3.95)
Villitis	16 (2.64)
Total	665

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