Morbidity during the first year of life in small for gestational age infants

Torstein Vik, Lars Vatten, Trond Markestad, Gunnar Ahlsten, Geir Jacobsen, Leiv S Bakketeig

Abstract

Postneonatal morbidity during infancy was studied in 284 small for gestational age (SGA) and 359 non-SGA term infants. None of these babies had congenital malformations and they were born to para 1 and para 2 mothers. SGA infants had an increased risk (OR: 1.7, 95% confidence interval: 1.1-2.6) of being admitted to hospital compared with non-SGA infants. The principal cause was respiratory tract infections. Increased hospitalisation among SGA infants was a factor only if the mother was a smoker—that is, smoked cigarettes at the time of conception. Among subgroups of SGA babies, there was an increased risk for infants of non-repeaters (women without a previous SGA child) (OR: 2.4, 95% CI: 1.4-3.8) and for infants with symmetric (OR: 2.0, 95% CI: 1.2-3.3) body proportions compared with non-SGA infants.

The results suggest that, beginning in early pregnancy, growth retardation may have long term consequences for subsequent infant morbidity, particularly if the mother is smoker.

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mothers with a previous normal birthweight child (non-repeaters).^{8 16 18}

SGA infants may also be classified as symmetric or asymmetric according to body proportions.^{1 3 8 19-21} Symmetric SGA neonates have normal body proportions; those that are asymmetric have a lower weight in relation to length.^{1 3 19 21} Symmetric SGA infants may be constitutional or the result of intrauterine environmental exposure starting in early pregnancy. Conversely, asymmetric SGA infants may be the result of placental insufficiency in late pregnancy.^{1-3 19 20} Some investigators have found higher neonatal morbidity in asymmetric than in symmetric SGA infants,^{1-3 22 23 29} while others have failed to demonstrate such differences.^{1 24}

It is important to exclude preterm SGA and infants with congenital malformations from the analysis. Otherwise, SGA morbidity may be related to disorders associated with prematurity and congenital malformations, and not to the possible adverse effects of being born small for gestational age.

We have reported that newborn SGA infants with asymmetric body proportions were more often transferred to the neonatal intensive care unit than non-SGA infants.²³ In this study we now present a further follow up of these infants, the aim being to compare morbidity during the first year of life between SGA and non-SGA infants born at term who had no congenital malformations.

Methods

This study is part of a population based prospective multicentre study. Eligible for enrolment were para 1 and para 2 women of Caucasian origin in Trondheim and Bergen, Norway, and in Uppsala, Sweden, who had a singleton pregnancy, and were registered by one of the study centres before the 20th gestational week. Women were enrolled between January 1986 and March 1988. Further details have been reported before.²⁵

SMALL FOR GESTATIONAL AGE (SGA) INFANTS

Among a total of 6354 women referred to the study, 5722 were eligible to participate. Overall, 541 infants born at term (from 37 to 42 weeks of gestation) satisfied the criteria of being SGA, defined as a birthweight below the 10th percentile for gestational age, corrected for sex and parity.²⁵ Twenty nine SGA infants were excluded due to congenital malformations (n = 22), stillbirth (n = 3), and death

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vs; 1 Small for gestational age (SGA) infants have higher rates of perinatal morbidity and mortality than appropriate for gestational age infants.¹⁻³ Most follow up studies of SGA infants have focused on growth and mental and motor development.^{4 5} It has been reported that SGA infants are at risk for the development of cerebral palsy^{6 7} and minor neurological diseases including minimal brain dysfunction and speech and learning impairment.^{4 5} Associations between SGA and sudden infant death syndrome (SIDS)^{8 9} and early onset of insulin dependent diabetes mellitus have been reported.¹⁰ Low birthweight has also been associated with increased risk of cardiovascular disease in adult life.¹¹ Few studies, however, have reported information on infant morbidity after the neonatal period.¹²⁻¹⁴

Some SGA infants are born to mothers who repeatedly give birth to low birthweight infants (repeaters),^{8 15-17} and epidemiological studies have indicated that these infants have a lower perinatal mortality than SGA infants born to

Department of Pediatrics, University Hospital, University of Trondheim, Norway
T Vik

Department of Community Medicine and General Practice
L Vatten
G Jacobsen
L S Bakketeig

Department of Pediatrics, University Hospital, University of Bergen, Norway
T Markestad

Department of Pediatrics, Uppsala University, Sweden
G Ahlsten

Correspondence to:
Dr Torstein Vik,
Department of Community Medicine and General Practice, University Medical Center, N-7005 Trondheim, Norway.

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Table 1 Comparison between infants included in follow up from birth to 13 month examination and infants not included (Numbers are mean and standard deviation)

	Included	Not included	P value
Non-SGA infants:			
Number of infants	359	102	
Birthweight (g)	3773 (468)	3734 (421)	0.4
Length at birth (cm)	51.3 (1.9)	51.5 (1.8)	0.4
Head circumference at birth (cm)	35.5 (1.2)	35.3 (1.6)	0.2
Ponderal index	2.79 (0.23)	2.74 (0.23)	0.03
SGA infants:			
Number of infants	284	82	
Birthweight (g)	2888 (248)	2858 (323)	0.5
Length at birth (cm)	48.4 (1.6)	48.6 (2.1)	0.4
Head circumference at birth (cm)	33.9 (1.1)	33.7 (1.5)	0.4
Ponderal index	2.54 (0.20)	2.48 (0.26)	0.06

Table 2 SGA, non-SGA, and subgroups of SGA infants admitted to hospital during infancy (excluding first week of life) and during first week of life (Odds ratio (OR) and 95% confidence interval (CI) are calculated for comparison with non-SGA infants)

	No	Yes	OR (95% CI)
Admitted during infancy			
SGA:	232	52	1.7 (1.1-2.6)
Repeaters	99	14	1.1 (0.6-2.0)
Non-repeaters	119	37	2.4 (1.4-3.8)
Symmetric	144	38	2.0 (1.2-3.3)
Asymmetric	85	14	1.3 (0.7-2.4)
Non-SGA	317	42	1.0
Admitted during first week of life			
SGA:	326	32	1.7 (1.0-2.9)
Repeaters	126	16	2.1 (1.1-4.2)
Non-repeaters	177	14	1.3 (0.7-2.6)
Symmetric	209	11	1.1 (0.5-2.2)
Asymmetric	114	21	3.2 (1.7-6.0)
Non-SGA	427	25	1.0

during infancy ($n = 4$, sudden infant death syndrome). In all, 146 SGA infants were identified in retrospect, and were not included in this follow up. Infants who were identified as being SGA at birth were examined by a paediatrician allocated to this project. A total of 366 SGA infants were identified and registered for follow up during infancy. None of these infants had a diagnosis of congenital virus infection or toxoplasmosis.

An SGA infant whose mother had previously given birth to a low birthweight infant was classified as the child of a repeater.²⁵ If the mother had not previously given birth to a low birthweight infant the child was classified as born to a non-repeater.

The ponderal index ((weight (grams)/length(cm)³) $\times$ 100) was used to divide the SGA infants into *asymmetric* or *symmetric* categories.²⁶ SGA infants with a sex specific ponderal index below the 10th percentile (boys: 2.40, girls: 2.51) were defined as asymmetric, and those with a ponderal index at the 10th percentile or above were defined as symmetric SGA infants.

CONTROL INFANTS

Between week 17 and 20 of pregnancy, a 10% random sample was drawn from the population of 5722 women. Among a total of 561 women in this random sample, 532 delivered at term and 58 (11%) of these infants were classified as SGA. Four of these were excluded because of congenital malformations or death, as stated above. The remaining 54 infants were included in the group of SGA infants. Of the remaining non-SGA infants, 15 were excluded due to congenital malformations ($n = 12$), perinatal ($n = 1$), or infant ($n = 2$, sudden

infant death syndrome) death, leaving a total of 459 infants to be followed up in the control group.

Data on early neonatal morbidity of the SGA and non-SGA infants have been reported before.²³ To compare infant morbidity and neonatal morbidity, some data from the neonatal study are also presented in this paper.

FOLLOW UP

During their first year of life, all the children were examined by a public health nurse as part of the routine health service offered to all infants in Norway and Sweden. Data on health, growth, nutrition, development and behaviour were recorded at 6 weeks and at 3, 6, and 9 months of age. At each visit, the mother was asked if the infant had had any kind of disease or if the child had been admitted to hospital since the previous visit. The mother was asked to specify any disease, and if the child had been admitted to hospital she was asked to state the reason. After each visit the records were sent to the project organiser for data registration.

At 13 months, the infants were examined by a project paediatrician. Height, weight, head circumference and other anthropometric measurements were recorded and a general paediatric examination was performed. Data on growth and mental development will be published shortly.²⁷ The mother was asked to complete a questionnaire on her child's health. She was asked if the infant had had respiratory or other infections, any other disorder of the respiratory system, disorders of the urinary and gastrointestinal tract, neurological, genital, or skin disorders, or any trauma during the first year of life. If the answer was positive, the mother was asked to give more information and to indicate how many times a physician had been consulted for each condition. Furthermore, the mother was asked if her child had been admitted to hospital during the preceding year, how many times, and for what reason(s).

If a mother had reported to the public health nurse that her infant had been admitted to hospital, or if she reported it in the 13 month questionnaire, the infant was classified as having been *hospitalised during infancy*. Admissions during the early neonatal period (the first week of life) were not recorded as infant morbidity.

Infants with data from at least two of the five examinations between 6 weeks and 13 months were included in the study. Complete follow up data were available for 284 SGA (77.6%) and 359 (78.2%) control infants. In all, 473 (74%) had attended all visits to the public health nurse and the paediatric examination at 13 months, and another 138 infants (21%) had attended all but one. In less than 5%, two (24 infants) or three (8 infants) examination data were missing. There was no difference in missing data between SGA and non-SGA infants.

Of the 284 SGA infants who were included in the follow up, 113 (40%) were born to repeaters and 156 to non-repeaters, and there was no information on previous outcome for 15 mothers. Among the SGA infants who

Table 3 Bivariate analysis of admission to hospital of SGA and non-SGA infants during infancy stratified for SGA risk factors (Odds ratio (OR) and 95% confidence interval are calculated for comparison of SGA with non-SGA infants)

	SGA		Non-SGA		OR (95% CI)
	Admission to hospital				
Risk factors	No	Yes	No	Yes	
Smoking:					
Yes	129	40	102	16	2.0 (1.0-3.7)
No	102	12	214	26	1.0 (0.5-2.1)
Pregnancy weight:					
< 50 kg	36	8	13	4	0.7 (0.2-2.0)
≥50 kg	196	44	304	37	1.8 (1.1-3.0)
Pregnancy body mass index (kg/m ²):					
< 21	134	34	125	20	1.6 (0.9-2.9)
≥21	98	18	191	20	1.8 (0.9-3.5)
Mother's education:					
≤9 years	82	23	135	17	2.2 (1.1-4.4)
> 9 years	62	17	181	25	2.0 (1.3-3.9)

Table 4 SGA and non-SGA infants admitted to hospital during infancy (excluding first week of life), and in subgroups of SGA infants stratified for smoking (Odds ratio (OR) and 95% confidence interval (CI) are calculated for comparison with non-SGA infants)

	Admitted to hospital		OR (95% CI)
	No	Yes	
Non-smokers:			
SGA:	102	12	1.0 (0.5-2.1)
Repeaters	43	4	0.8 (0.3-2.3)
Non-repeaters	57	8	1.1 (0.5-2.6)
Symmetric	61	8	1.1 (0.5-2.6)
Asymmetric	40	4	0.8 (0.3-2.6)
Non-SGA	214	26	1.0
Smokers:			
SGA:	129	40	2.0 (1.0-3.7)
Repeaters	56	10	1.4 (0.6-3.2)
Non-repeaters	62	29	3.1 (1.6-6.1)
Symmetric	83	30	2.3 (1.2-4.5)
Asymmetric	45	10	1.4 (0.7-3.1)
Non-SGA	102	16	1.0

completed follow up, 99 (35%) were classified as asymmetric and 182 infants as having symmetric body proportions, while the ponderal index could not be calculated for three SGA infants.

In table 1 we have compared the perinatal data of infants with and without complete follow up information. There was a small difference in ponderal index between the groups, but there were no differences in the proportions of repeaters/non-repeaters or in symmetric/asymmetric SGA infants (data not shown).

Gestational age was estimated from the first day of the last menstrual period if this was accurately recalled by the mother within more than three days at the first visit. Ultrasound estimates were used for dating in about 17% of the cases, either due to a discrepancy between the date of the last menstrual period and ultrasound based gestational age of more than 14

days, or if the last menstrual period could not be recalled accurately.²⁵

Mothers who reported smoking daily at the time of conception were classified as smokers. Other potential risk factors for SGA which were considered included a prepregnancy weight below 50 kg, a body mass index below the median value of 21 kg/m², and less than 10 years of education.

The odds ratio was used to estimate the relative risk of morbidity among SGA compared with non-SGA infants. Precision of the odds ratio was estimated by 95% confidence intervals, using Mantel-Haenszel χ^2 statistics.

Results

A total of 94 infants were admitted to hospital during the first year of life, excluding the first week, and SGA infants were more often admitted than non-SGA infants (OR: 1.7; 95% CI: 1.1-2.6) (table 2). Nine mothers, all in the SGA group, reported that their child had been admitted to hospital twice. After excluding readmissions of infants who had been transferred to the intensive care unit during the neonatal period, the increased risk of being admitted to hospital persisted in SGA infants (OR: 1.6, 95% CI: 1.0-2.5).

The higher admission rate of SGA infants was mainly found among SGA infants of mothers who were non-repeaters and in SGA infants with symmetric body proportions at birth (table 2).

For comparison, admissions during the first week of life are shown in table 2. Newborn SGA infants had a higher risk of being admitted to hospital than non-SGA infants, but the higher admission rate in the early neonatal period was mainly caused by infants of repeaters and in infants with asymmetric body proportions.

Table 3 shows the odds ratios of being admitted to hospital in SGA and non-SGA infants stratified by maternal risk factors. Among infants of mothers who smoked, there was a twofold increased risk of being admitted to hospital for SGA compared with non-SGA infants. Among infants of non-smokers, there was no such difference between SGA and non-SGA infants.

Among infants of mothers with a prepregnancy weight below 50 kg, being SGA was not associated with an increased risk of being admitted to hospital (table 3). The relative risk for SGA infants was, however, increased if the mother had a prepregnancy weight above 50 kg.

The higher admission rate among SGA infants of smokers was caused by infants of non-repeaters and by infants with symmetric body proportions (table 4). Among non-smokers, no evidence of increased risk was found within subgroups of SGA (table 4).

Table 5 shows that infections more often led to admission to hospital among SGA than non-SGA infants (OR: 2.0, 95% CI: 1.1-3.8). This was mainly due to a higher incidence of respiratory tract infections (OR: 2.2, 95% CI: 1.1-4.4).

Table 5 Causes of admission to hospital during first year of life (1 week to 13 months) in SGA and non-SGA infants (Odds ratio (OR) and 95% confidence interval (CI) are calculated for comparison of SGA with non-SGA infants)

	SGA	Non-SGA	OR (95% CI)
Infections:	32	21	2.0 (1.1-3.8)
Respiratory tract infections	21	12	2.2 (1.1-4.4)
Other infections	11	9	1.6 (0.6-4.2)
Surgery	8	4	2.6 (0.7-10.3)
Poor weight gain/feeding difficulties	2	3	0.8 (0.1-6.2)
Other diagnoses	10	14	0.9 (0.4-2.2)
Total	52	42	1.7 (1.1-2.6)

Upper respiratory tract infections (common cold, pharyngitis, and otitis media) were reported during the first year of life by 91.5% of the SGA and 91.4% of the non-SGA mothers. However, exanthema subitum and varicella were reported significantly more often in the non-SGA (70 infants; 20%) than in the SGA (35 infants; 12%) group (OR: 1.7, 95% CI: 1.1–2.7). In both groups 55% of the mothers reported that they had consulted a physician during the preceding year (mean: 1.3 contacts per infant and year in both groups).

At the clinical examination at 13 months, the paediatricians reported pathological findings in more than 10 infants only for the examination of the ears (25 infants), lungs (12 infants), and skin (55 infants). For SGA compared with non-SGA infants the odds ratios for pathological findings of the ears and lungs (combined) were 1.7 (95% CI: 0.8–3.4) and for pathology of the skin 0.7 (95% CI: 0.4–1.2).

Discussion

We found that SGA infants had an increased risk of being admitted to hospital during infancy compared with non-SGA infants. Our study was restricted to singletons born at term with no congenital malformations and therefore the results were not caused by diseases related to prematurity, congenital malformations, or multiple births. Nor was the higher admission rate caused by a higher morbidity of infants who had been admitted to the neonatal intensive care unit during the newborn period.

The 10th percentile definition of SGA is crude, and many constitutionally small, but healthy, babies will be classified as SGA.²⁸ None the less we found that SGA infants had a 70% increased risk of being admitted to hospital compared with non-SGA infants. A more narrow definition of SGA would most probably have resulted in a higher risk.

The outcome measure, admission to hospital, is likely to have been correctly recalled by the mother. The infants were admitted by many different general practitioners and it is unlikely that they were influenced by the child's SGA status when deciding on admission.

Most follow up studies of term SGA infants have not reported infant morbidity, except for persistent deficits such as cerebral palsy and neurological 'soft signs'.^{3–5} One study found no differences in postneonatal morbidity between SGA and non-SGA infants,¹³ whereas Villar *et al* reported an increased incidence of diarrhoea in symmetric SGA infants compared with non-SGA infants during the first two months of life.¹² In a population based study from Brazil, hospital admissions due to diarrhoea and pneumonia occurred more often during the first two years in children who were growth retarded at birth than in children with appropriate birthweights.¹⁴

Among subgroups of SGA infants, we found an increased risk of hospital admission for infants of non-repeater mothers. The rationale for comparing repeaters and non-repeaters was the hypothesis that an "unexpected" adverse event or exposure during pregnancy may have

caused the birth of an SGA infant to a non-repeater.⁶ SGA infants of repeaters may more often be constitutionally small,^{8 16 18} but may also be the result of an unhealthy lifestyle in consecutive pregnancies.¹⁶ Our results are consistent with those of studies which reported increased perinatal morbidity and mortality among SGA infants of non-repeaters.^{8 16 18}

By using the 10th percentile of the ponderal index as a cutoff between symmetric and asymmetric SGA, we found that symmetric SGA infants had a higher risk of being admitted to hospital. Symmetric SGA infants may either be constitutional, or the result of retarded intrauterine growth which may be caused by early environmental exposure.^{2 3 5 19 20} Thus our results suggest that infant morbidity might be more affected in SGA infants who have been exposed to environmental factors in early pregnancy.

We stratified the infants according to risk factors for SGA and found no difference in the risk of being admitted to hospital between SGA and non-SGA infants of mothers with a low prepregnancy weight. However, the total number of non-SGA infants in this group was low ($n = 17$).

We analysed infants of smokers and non-smokers separately and found that SGA infants of women who smoked at the beginning of pregnancy had an increased risk of being admitted to hospital. Among non-smokers there was no difference between SGA and non-SGA infants. Thus the increased risk of SGA infants of being admitted to hospital in this study was solely observed among SGA infants of smokers.

Smoking may lead to symmetric growth retardation,²⁹ and for SGA infants of smokers we found a threefold increased risk of being admitted to hospital if the mothers were non-repeaters, compared with non-SGA infants. There was no increased risk for SGA infants of non-repeaters who did not smoke.

We found that infections more often led to hospitalisation of SGA than non-SGA infants. This was caused by a higher admission rate for respiratory tract infections. However, we found no increase in morbidity as a result of minor illnesses among SGA infants, and the reported prevalence of respiratory tract infections not leading to hospitalisation did not differ between the groups, suggesting that infections may lead to more severe disease in SGA than in non-SGA infants. This may be mediated through maternal smoking which has been shown to increase the risk of respiratory complications in infancy.^{30–33}

Compared with our results from the early neonatal period, there was a similar increased risk of being admitted to hospital among SGA infants as a whole,²³ but there were different patterns of hospitalisation among subgroups of SGA infants. Asymmetric SGA infants and infants of repeaters had an increased risk in the early neonatal period; later in infancy the increased risk was found among symmetric infants and among infants of non-repeaters.

The different patterns may be related to different reasons for admission. In the early

neonatal period acute complications—for example, hypoglycaemia, hyperviscosity—were the principal causes of admission,²³ and this is consistent with other reports.^{1-3 5} These causes are related to poor placental function late in pregnancy and to asymmetric body proportions at birth. After the neonatal period the asymmetric SGA infant may have recovered. The higher risk of being admitted to hospital in symmetric infants of non-repeating mothers, lasting for the remaining part of infancy, may be caused by earlier exposure with permanent effects on the infant's health.

In conclusion, we have found a higher prevalence of being admitted to hospital among SGA infants compared with non-SGA infants during infancy, which was restricted to symmetric SGA infants of non-repeating mothers who smoked. The results may suggest that an exposure which influences living conditions in utero may have acted from early pregnancy and resulted in long term consequences for infant morbidity, particularly if the mother smokes cigarettes.

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