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Young Maternal Age and the Risk of Neonatal Mortality in Rural Nepal

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Abstract

Objective—To investigate the relationship between adolescent pregnancy and neonatal mortality in a nutritionally deprived population in rural Nepal, and to determine mechanisms through which low maternal age may impact neonatal mortality.

Design—Nested cohort study using data from a population-based, cluster-randomized, placebo-controlled trial of newborn skin and umbilical cord cleansing with chlorhexidine.

Setting—Sarlahi District of Nepal.

Participants—Live-born singleton infants of parity 0 or 1 women under 25 years of age (n=10,745) were included in this analysis.

Main Exposure—Maternal age at birth of offspring.

Outcome Measure—Crude and adjusted odds ratios (OR) of neonatal mortality by maternal age category.

Results—Infants born to women aged 12−15 years were at a higher risk of neonatal mortality than those born to women aged 20−24 years (OR=2.24, 95% CI 1.40−3.59). After adjustment for confounders, there was a 53% excess risk of neonatal mortality among infants born to women in the youngest versus oldest age category (OR=1.53, 95% CI 0.90, 2.60). This association was attenuated upon further adjustment for low birthweight (LBW), preterm birth or small for gestational age (SGA).

Conclusions—The higher risk of neonatal mortality among adolescent women in this setting is partially explained by differences in socioeconomic factors in younger versus older women and is mediated primarily through preterm delivery, SGA and LBW or some interaction of these variables.

INTRODUCTION

Pregnancy during adolescence is a significant problem globally, with the highest incidence rates occurring in developing nations[1]. It is estimated that over 14 million adolescent girls between 15 and 19 years of age give birth each year, and over 90% of these occur in developing countries[2]. While early childbearing has often been regarded as a social issue, there is mounting evidence that young maternal age may be linked to adverse infant outcomes including low birthweight (LBW), preterm birth, and intrauterine growth restriction, as well as neonatal

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mortality[3–8]. Attempts to elucidate the etiology of these poorer pregnancy outcomes among adolescent women have produced conflicting data and considerable debate remains as to whether the excess risks are due to biologic immaturity or are the consequence of deleterious social and environmental factors[3,4,9–11].

Since adolescent mothers are more likely to be poor, less educated, and to have inadequate prenatal care and fewer social supports than older mothers, socioeconomic and lifestyle factors have often been cited as the main explanatory variables for disparities in reproductive outcomes[12]. However, a number of studies have shown strong associations between maternal age and adverse infant outcomes even after controlling for these factors[4, 5]. Thus, investigations in both industrialized and developing nations lend support to an intrinsic biologic risk associated with young maternal age[3, 7, 8].

The adolescent period is a time of significant growth; 45% of adult weight and 15% of adult height is attained during this stage[13]. Continued growth during pregnancy could result in competition between the mother and fetus for important nutrients and may be associated with increased risk of adverse pregnancy outcomes[14, 15]. In countries where chronic malnutrition is prevalent, the consequences of this competition may be even more detrimental to the mother and infant. Moreover, in adolescents, chronic malnourishment is associated with delayed age at menarche and prolonged puberty, and thus may contribute to poor reproductive outcomes[16, 17]. In addition to pubertal growth, postmenarcheal growth may also affect pregnancy outcomes in such populations. Few studies, however, have investigated the risks associated with adolescent pregnancy in areas of high malnutrition.

In Nepal, chronic malnutrition is common and early marriage is customary; more than two-thirds of rural females are married by the age of 20[18]. Rates of adolescent pregnancy are reported to be as high as 89 births per 1000 females aged 15−19, but births among younger girls are also frequent[1]. Early childbearing, however, is considered a successful outcome in Nepal, and the extended family network often helps to care for the infant, thereby eliminating some of the postulated social and environmental problems associated with adolescent pregnancy.

The primary aim of this study was to investigate the relationship between young maternal age and neonatal mortality in a nutritionally deprived population in rural Nepal. We also sought to determine whether preterm birth, LBW and small for gestational age (SGA) are mechanisms through which low maternal age impacts the risk of neonatal mortality in this setting.

METHODS

This study utilized data from the Nepal Newborn Washing Study, a cluster-randomized, placebo-controlled, community-based trial of newborn skin and umbilical cord cleansing with chlorhexidine conducted in the Sarlahi District of Nepal between 2002 and 2005. The details of the methodology and the main results of this trial have been reported elsewhere[19, 20]. Briefly, 413 sectors were randomized for newborn infants to receive one of two skin cleansing regimens by a local female ward distributor (WD) immediately after delivery. The regimens consisted of full body skin cleansing of the infant excluding the eyes and ears with either Pampers baby wipes (Proctor and Gamble Co, Cincinnati, OH) containing 0.25% free chlorhexidine or with baby wipes that lacked chlorhexidine (Placebo).

Women were recruited for participation in the study at approximately 6 months' gestation by WDs who visited women in their area on a weekly basis. All women received weekly vitamin A supplementation, iron-folic acid supplements, albendazole as well as tetanus immunization if deficient, and a clean delivery kit. At recruitment, project workers provided education regarding proper nutrition, hygienic delivery and neonatal care, and collected data on

education, literacy and maternal health. Information regarding socioeconomic status including ethnic group, caste, latrine and cattle ownership, the presence of electricity in the home, and maternal occupation was also obtained at this time.

In this population, most women deliver at home with the assistance of family members or untrained traditional birth attendants. WDs, who were alerted by relatives once labor began, visited the woman's home during or soon after delivery (mean 6.8 hours after delivery) to provide the skin cleansing intervention to the newborn infant. Subsequently, a birth assessment team arrived at the home to collect information regarding the delivery process and condition of the newborn infant and to measure axillary temperature and infant birthweight using a digital infant scale (Seca 727, Hamburg, Germany). Infant vital status was assessed on days 2, 3, 4, 6, 8, 10, 12, 14, 21, and 28 after birth. Infants with specific signs and symptoms were referred for medical care.

Only live-born singleton infants of women less than 25 years of age who were either parity 0 or 1 were included in this analysis. In this population, most adolescents are parity 0 or 1 and almost all women 25 or older are of parity greater than 1. Hence confining the analysis to women of parity 0 or 1 and younger than 25 would reduce confounding of the association between maternal age and survival by parity. Older parity 0 or 1 women were also excluded because their low parity may have been associated with reproductive or other health problems. Maternal age was calculated as the age of the mother at time of delivery based upon information solicited from the mother at the enrollment interview. Gestational age was estimated from two maternal reports of time since last menstrual period; these estimates were provided at enrollment and at first assessment after delivery. Delivery before 37 weeks gestation was defined as preterm. For birthweight data, only those weights measured within 72 hours of delivery were considered in this analysis, and infants were classified as SGA if their weight fell below the tenth percentile for gestational age and sex as defined by the US reference for fetal growth[21].

Verbal informed consent was obtained from all participants. The study received ethical approval from the Nepal Health Research Council and the Committee on Human Research of the Johns Hopkins Bloomberg School of Public Health. The trial was registered at Clinicaltrials.gov (NCT00109616).

Statistical Analysis

Statistical analysis was performed using Stata 9.0 (Stata Corp, College Station, TX). Four maternal age categories (12−15 yrs, 16−17 yrs, 18−19 yrs and 20−24 yrs) were defined a priori based on evidence from similar research studies[3,5]. The distributions of various maternal, socioeconomic and infant characteristics were compared across maternal age groups using chi-squared tests for categorical data or analysis of variance for continuous measurements. To investigate the relationship between maternal age and death during the neonatal period, neonatal mortality rates (NMRs), calculated as the number of deaths within the first 28 days over number of live births, were assessed by maternal age and stratified by parity. Because a number of deaths occurred prior to receiving the assigned intervention, a 2-level “treatment received” variable was created by categorizing infants into those who received chlorhexidine versus those who received either placebo or no treatment. This variable was assessed as a potential confounder and/or effect modifier.

Logistic regression models were constructed to compute crude and adjusted odds ratios (ORs) for neonatal mortality by maternal age category. Potential confounders significantly associated ($p<0.05$) with both maternal age and neonatal mortality were included in the multivariable models. Interactions between maternal age and parity or treatment received were assessed through testing of product terms. The Hosmer-Lemeshow goodness of fit statistic was used to

assess adequacy of model fit. ORs and their 95% confidence intervals were corrected using generalized estimating equations with an independent correlation structure to account for clustering within a subset of participants who contributed more than one pregnancy to the study[22].

Finally, ORs for the risks of other adverse outcomes including LBW, preterm delivery and SGA by maternal age category were obtained using logistic regression. Since these were thought to be in the causal pathway of infant survival, we assessed whether they were mediators of the relationship between maternal age and neonatal mortality by creating models which further adjusted for LBW, preterm delivery, SGA or both preterm delivery and SGA.

RESULTS

Of 23,296 live-born singleton infants, 10,745 mother-infant pairs met the selection criteria (Figure 1). Of these, 9,077 infants were weighed within 72 hours of birth. The disproportionately higher mortality in infants without birthweight data is due to the majority of these deaths occurring soon after delivery, before arrival of the birth assessment team to weigh the infant. Similarly, of the 163 infants that did not receive the assigned intervention (chlorhexidine vs placebo), 106 died prior to the arrival of the WD. In total, the study population consisted of 10,745 infants born to 9,733 unique mothers, with the contribution by 1,012 women of 2 pregnancies each to the study.

Selected characteristics of the 10,745 mother-infant pairs are displayed in Table 1. Approximately 3.2% of infants in this study were born to young women aged 12−15 years, 17.1% to women aged 16−17 years, 31.4% to women aged 18−19 years, and 48.3% to women aged 20−24 years. The distribution of characteristics such as ethnic group, caste, literacy and various socioeconomic attributes differed by age category, with the youngest women more likely to be from the *Madeshi* (people originating from the plains region of Nepal) ethnic group, illiterate and without a latrine or electricity at their home.

Rates of adverse infant outcomes including LBW, preterm birth and SGA were also significantly higher in the younger age groups (Table 1). More than 51% of infants born to women aged 12−15 years were LBW, 24.0% were preterm and 73.5% were classified as SGA compared to 27.8%, 16.4% and 56.3% of infants respectively in the 20−24 years of age category.

There were 371 infant deaths during the neonatal period, corresponding to an overall NMR of 34.5 per 1000 live births. NMRs were greatest in the youngest women, and decreased with increasing maternal age (Table 2). The NMR for infants born to women under the age of 16 was more than double that of infants born to mothers above 20 years of age (61.8 vs 28.5 per 1000 live births). Trends were similar for both parity 0 and parity 1 women, but there was a paucity of parity 1 women in the youngest age categories.

Crude and adjusted ORs for neonatal mortality presented in Table 3 show declining risk of neonatal mortality with increasing maternal age. Infants of mothers 12−15 years of age were at a more than 2-fold greater risk of mortality than those of mothers aged 20−24 years (OR=2.24, 95% CI 1.40, 3.59). After adjusting for treatment received, maternal literacy, ethnic group, caste, latrine and cattle ownership, electricity in the home, maternal occupation, parity and gestational nightblindness, infants were found to have a 53% excess risk of neonatal mortality if born to women in this youngest age group versus the oldest (OR=1.53, 95% CI 0.90, 2.60), although this association was no longer statistically significant. The adjusted ORs for neonatal mortality associated with mothers aged 16−17 years, and 18−19 years compared to the 20−24 year old age category were 1.17 (95% CI 0.87, 1.64) and 1.00 (95% CI 0.76, 1.32) respectively. Treatment received, but not assigned treatment group, was a confounder of the

maternal age neonatal mortality relationship. There was no evidence of interaction between maternal age and parity or treatment received. The reversal of the association between caste and neonatal mortality in the multivariable model was due to strong association between caste and ethnicity. Restricting the analysis to early neonatal deaths that occurred during the first week of life yielded a similar but weaker maternal age effect (data not shown). The maternal age neonatal mortality relationship also held when the analysis was restricted to infants without birthweight data (those infants who had died prior to arrival of the WD or those infants who were not weighed within 72 hours after birth) (data not shown).

Ethnicity was one of the strongest predictors of neonatal mortality. Even after adjustment, *Madeshi* infants were almost twice as likely to die in the first 28 days of life as *Pahadi* infants (OR=2.01, 95% CI 1.43, 2.84). A maternal history of nightblindness during pregnancy was also strongly linked to neonatal death (OR=2.03, 95% CI 1.34, 3.07).

LBW, preterm delivery and SGA were strongly associated with both neonatal mortality (OR=4.27, 95% CI 3.06, 5.97; OR=3.61, 95% CI 2.91, 4.47; and OR=1.77, 95% CI 1.24, 2.53 respectively) and young maternal age (Tables 3, 4). To investigate whether the increased risk of neonatal death observed among younger women after adjustment for socioeconomic and other confounders was linked to the higher rates of LBW, preterm birth and SGA in younger age groups, 5 different models of neonatal mortality which further adjusted for these factors were considered (Table 4). The association between maternal age and neonatal mortality diminished upon adjustment for LBW, preterm delivery or SGA. The addition of both SGA and preterm birth to the model further dampened the effect of maternal age on neonatal mortality. The excess risk of neonatal mortality in infants born to women aged 12−15 years compared to women 20−24 years old, after controlling for these two factors, dropped from 53% to 14% (OR=1.14, 95% CI O.50, 2.61). Thus, the increased risk of neonatal mortality associated with adolescent pregnancy was primarily mediated through elevated rates of preterm births and SGA infants in these younger women.

COMMENT

This study found a strong association between young maternal age and neonatal mortality that was significantly attenuated after controlling for socioeconomic and other confounders. The effect of maternal age on infant survival in the first week of life was no stronger than for the entire neonatal period, thus confirming the greater influence of socioeconomic rather than biologic factors on the survival of infants born to young mothers. Our results are comparable to a population based cohort study in Missouri which found a 1.69 times higher risk of neonatal mortality in younger adolescents aged 12−17 years versus older mothers aged 20−34 years[9]. In that study, adjustment for socioeconomic status, race, education, parity, smoking and prenatal status accounted for most of the increased risk of neonatal death. Similarly, a hospital based study found no association between young maternal age and neonatal mortality once maternal race, prenatal care and other factors had been controlled for[23]. Despite similarities with these results, an alternate explanation for our findings could be that the biologic risk associated with young maternal age was expressed through increased rates of miscarriages and stillbirths. In that event, fetuses surviving to be live-born would be at a lower biologic than environmental risk once born.

A number of investigations in industrialized and developing countries provide evidence supporting a role for biologic factors in poorer pregnancy outcomes of young mothers. A cross-sectional study in Latin America reported a 50% excess risk of early neonatal mortality among adolescent women ≤16 years of age versus women 20−24 years of age after adjustment for 15 different socioeconomic and other confounders[24]. A retrospective cohort study of 3.8 million primiparous pregnant women under 25 years of age yielded increased risks of neonatal

mortality, LBW, and preterm delivery in adolescents even after taking into account deleterious social and environmental factors[3]. A Swedish study found that the increased risk of neonatal mortality among adolescents was explained largely by higher rates of preterm birth which the authors attributed to biologic causes[5].

Preterm birth is one of the leading direct causes of neonatal death[25]. LBW, which may indirectly account for 60−80% of neonatal deaths, arises through preterm birth, intrauterine growth restriction or both[25, 26]. Consistent with other investigations, our study found that LBW, preterm birth and intrauterine growth restriction were strongly associated with both neonatal mortality and low maternal age[3-8]. The excess risk of neonatal mortality in the youngest women, although diminished after controlling for socioeconomic confounders, was further attenuated with adjustment for either LBW, preterm birth, SGA or both preterm birth and SGA. This suggests that while disparities in socioeconomic factors in younger compared to older women partially explain the increased risk of neonatal mortality among adolescent women, the biologic mechanism of this excess mortality is mediated primarily through preterm birth, SGA and LBW or some interaction of these variables.

This study also demonstrated a striking relationship between ethnicity and neonatal mortality. *Madeshi* infants were at much higher survival risk in the neonatal period than *Pahadi* infants even after controlling for confounders. This excess risk may be due to differing behavioral practices related to maternal and infant nutrition as well as antenatal and newborn care. For example, breastfeeding was more likely to be initiated early in *Pahadi* than *Madeshi* infants, and the early initiation was shown to be associated with survival[27]. Given this association, there are likely other behavioral factors that explain the ethnic difference that were not collected.

Our findings help to illuminate the complex links between maternal age and adverse reproductive outcomes in a nutritionally deficient, low resource setting where cultural norms favor adolescent pregnancy. In Nepal, early marriage and childbearing are considered socially acceptable and are in fact, viewed as successful outcomes[28, 29]. There is significant cultural and family pressure to give birth early and in particular to produce a male heir[28, 29]. That the median age at first birth in Nepal has remained consistent at 19.9 years of age over the 2001 and 2006 Demographic and Health Surveys (DHS), and is also similar across all age cohorts, provides evidence that social and cultural attitudes regarding early childbearing continue to be prevalent[30, 31]. Conversely, in many industrialized nations, adolescent pregnancy often occurs outside the sphere of marriage and is linked to social marginalization, low socioeconomic status and inadequate prenatal care[12]. These factors are often cited as the primary causes of poorer birth outcomes among younger women. In our study population, despite the social and cultural acceptance of adolescent pregnancy, the youngest women still appeared to be the most socioeconomically disadvantaged. While poor socioeconomic status may be a more critical influence on adverse reproductive outcomes than social marginalization, preterm birth and SGA are overriding factors in higher mortality of neonates born to adolescent women.

Limitations of this analysis include potential recall bias associated with estimating gestational age based on the self-reported date of the last menstrual period. Although we adjusted for a large number of socioeconomic variables, several well established maternal risk factors such as Body Mass Index (BMI) and mid-upper arm circumference (MUAC), smoking and alcohol use, and weight gain during pregnancy were unavailable. Smoking and alcohol use, however, were low among younger women in a similar population[31].

The age at menarche of the women was also unavailable. Hence, it was not possible to distinguish between risks associated with pubertal versus postmenarcheal growth of these adolescents. Low gynecologic age has been associated with increased rates of adverse

reproductive outcomes in adolescents and may be more closely associated with biologic outcomes than chronologic age[33]. Hediger et al. reported that women who were young but whose gynecologic age exceeded two years were at no greater risk for preterm delivery than older women[7]. In our study population, where chronic malnutrition is prevalent, puberty and age at menarche may have been delayed in a substantial proportion of the adolescents. However, we were unable to ascertain which of the women had low gynecologic age and whether those women in particular were at a higher risk for neonatal mortality.

All women in this study received micronutrient supplementation and educational visits by WDs which may have impacted mortality during the neonatal period. However, the overall NMR in this study was 34.5 deaths per 1000 live births which is comparable to the most recent national NMR estimate of 33 deaths per 1000 live births reported in the 2006 DHS[30]. We do not believe the provision of Vitamin A and iron-folic acid impacted the relationship between maternal age and neonatal mortality since women in all age categories benefited from these interventions. Since albendazole was given to everyone and not randomized, we cannot know whether this affected women of all ages similarly. While women in this study may have been better off with respect to micronutrient status than those not participating in the study, calorie and protein deficiency remained a significant problem among our study population and probably was a primary contributor to low birth weight. We therefore believe our findings are generalizable to populations with similar nutritional deficiencies.

In summary, compared to older women, infants born to adolescents were at an increased risk of neonatal mortality that was ascribed largely to social and environmental factors and was mediated through increased rates of SGA, LBW and preterm delivery among the younger women. Further research is needed to elucidate the complex relationship between adolescent pregnancy and adverse reproductive outcomes, particularly in resource poor settings where delayed age at menarche due to chronic malnourishment may be an important influence. Delaying the age at first pregnancy may be a valuable strategy to promote and improve infant health and survival.

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Joanne Katz had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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Table 1

Household, women and infant characteristics by maternal age category[a]

Variable	All Women	Maternal Age Category (years)				P-value[b]
	12–24	12–15	16–17	18–19	20–24	
Household Characteristics						
Latrine at house[c] (%)	15.5	7.5	10.1	13.1	19.5	<0.001
Electricity in house[d] (%)	29.0	21.9	26.4	27.9	31.2	<0.001
Own ≥ 2 cattle[e] (%)	51.4	44.0	52.4	52.1	51.1	0.021
Maternal Characteristics						
Age (years)	20.0 (2.27)	15.2 (0.84)	17.2 (0.56)	19.0 (0.59)	21.9 (1.34)	
Parity (%)						
0	54.7	93.8	87.2	65.2	33.8	<0.001
1	45.3	6.2	12.8	34.8	66.2	
Ethnicity (%)[f]						
Pahadi	34.8	23.7	27.2	30.7	40.1	<0.001
Madeshi	65.2	76.3	72.8	69.2	59.2	
Caste (%)[g]						
Brahmin	8.6	3.9	4.2	6.2	12.0	<0.001
Chetri	8.5	4.8	5.9	7.0	10.7	
Vaiysha	63.5	59.5	65.5	66.8	61.0	
Shudra	12.7	19.5	16.2	12.6	11.1	
Muslim/Other	6.6	12.3	8.1	7.5	5.2	
Literate[h] (%)	37.1	23.5	32.5	34.9	41.0	<0.001
Work outside house[i] (%)	11.3	6.5	9.0	9.6	13.5	<0.001
Night blindness druing pregnancy[j] (%)	4.1	4.0	4.8	4.4	3.8	0.279
Infant Characteristics						
No. of live births	10745	340	1841	3374	5190	
Treatment Group (%)						
Chlorhexidine	63.5	62.4	62.5	62.6	64.6	0.189
Placebo	36.5	37.6	37.5	37.4	35.4	
Treatment Received (%)						
Chlorhexidine	62.5	61.5	61.8	61.3	63.6	0.143
No Chlorhexidine	37.5	38.5	38.2	38.7	36.4	
Birth weight[k] (g)	2654.0 (418.9)	2471.9 (415.5)	2568.0 (402.2)	2625.8 (416.1)	2716.0 (416.0)	<0.001
Gestational age[l] (weeks)	39.2 (2.46)	38.6 (2.57)	38.9 (2.54)	39.0 (2.50)	39.4 (2.37)	<0.001

Variable	All Women	Maternal Age Category (years)				P-value[b]
	12–24	12–15	16–17	18–19	20–24	
Low birth weight[k] (<2500g)(%)	33.3	51.4	40.4	36.0	27.8	<0.001
Preterm[l] (<37 wks) (%)	18.8	24.0	22.4	20.0	16.4	<0.001
Small for Gestational Age[m] (%)	60.5	73.5	67.7	61.7	56.3	<0.001

[a] Estimates represent mean (SD) for continuous variables and % for categorical variables.

[b] Chi squared tests were used for comparison of categorical variables by age group and analysis of variance for continuous variables.

[c] A total of 9 were missing latrine data

[d] A total of 229 were missing electricity data

[e] A total of 236 were missing cattle ownership data

[f] A total of 229 were missing ethnicity data

[g] A total of 220 were missing caste data

[h] A total of 4 were missing literacy data

[i] A total of 9 were missing occupation data

[j] A total of 598 were missing night blindness data

[k] A total of 1668 were missing birth weight data

[l] A total of 80 were missing gestational age data

[m] A total of 1716 were missing small for gestational age data

Table 2

Neonatal Mortality Rate (per 1000 live births) by maternal age and parity

Age (yrs)	All Women				Parity=0				Parity=1			
	Alive (n)	Died (n)	NMR	OR	Alive (n)	Died (n)	NMR	OR	Alive (n)	Died (n)	NMR	OR
12−15	319	21	61.8	2.24	299	20	62.7	1.92	20	1	47.6	1.88
16−17	1758	83	45.1	1.61	1533	73	45.5	1.37	225	10	42.6	1.67
18−19	3255	119	35.3	1.25	2117	84	38.2	1.14	1138	35	29.8	1.16
≥20	5042	148	28.5	1.00	1697	59	33.6	1.00	3345	89	25.9	1.00

Table 3

Neonatal Mortality Rates (NMR), crude and adjusted Odds Ratios (OR) and their 95% Confidence Intervals, for neonatal mortality by maternal age category and other maternal characteristics

	Alive (n)	Died (n)	NMR (per 1000 live births)	Crude OR	Adjusted OR[a]
Age, yrs					
12−15	319	21	61.8	**2.24 (1.41, 3.58)**	1.53 (0.90, 2.60)
16−17	1758	83	45.1	**1.61 (1.22, 2.12)**	1.17 (0.84, 1.64)
18−19	3255	119	35.3	1.25 (0.97, 1.59)	1.00 (0.76, 1.32)
20−24	5042	148	28.5	1.00	1.00
Treatment Received (%)					
None	3805	225	55.8	1.00	1.00
Chlorhexidine	6569	146	21.7	**0.38 (0.30, 0.47)**	**0.39 (0.31, 0.49)**
Parity					
0	5646	236	40.1	1.00	1.00
1	4728	135	27.8	**0.68 (0.55, 0.84)**	**0.75 (0.58, 0.97)**
Ethnic Group[b]					
Pahadi	3585	76	20.8	1.00	1.00
Madeshi	6576	279	40.7	**2.00 (1.54, 2.60)**	**2.01 (1.43, 2.84)**
Caste[c]					
Brahmin	884	23	25.4	1.00	1.00
Chetri	875	24	26.7	1.05 (0.58, 1.92)	0.83 (0.44, 1.57)
Vaiysha	6453	232	34.7	1.38 (0.88, 2.17)	**0.59 (0.35, 0.99)**
Shudra	1287	49	36.7	1.46 (0.87, 2.45)	**0.54 (0.29, 0.98)**
Muslim/other	670	28	40.1	1.61 (0.90, 2.86)	0.56 (0.29, 1.09)
Maternal Literacy[d]					
Illiterate	6488	272	40.2	1.00	1.00
Literate	3882	99	24.9	**0.61 (0.48, 0.77)**	0.84 (0.61, 1.14)
Latrine at house[e]					
No	8543	325	36.6	1.00	1.00
Yes	1597	29	17.8	**0.48 (0.32, 0.71)**	**0.64 (0.41, 1.00)**
Maternal Occupation[f]					
None	9177	347	36.4	1.00	1.00
Some work outside home	1188	24	19.8	**0.53 (0.35, 0.81)**	0.70 (0.45, 1.09)
Electricity in house[g]					
No	7188	275	36.8	1.00	1.00
Yes	2973	80	26.2	**0.70 (0.54, 0.91)**	0.82 (0.63, 1.08)
Cattle ownership[h]					
0	3441	138	38.6	1.00	1.00
1	1469	56	36.7	0.95 (0.69, 1.30)	0.83 (0.60, 1.16)
2+	5244	161	29.8	**0.77 (0.60, 0.97)**	0.79 (0.62, 1.02)
Maternal Night blindness[j]					
No	9406	322	33.1	1.00	1.00
Yes	390	29	69.2	**2.17 (1.47, 3.22)**	**2.03 (1.34, 3.07)**
Birth Weight[j]					

	Alive (n)	Died (n)	NMR (per 1000 live births)	Crude OR	Adjusted OR[a]
≥2500g	6001	52	8.6	1.00	-
<2500g	2916	108	35.7	**4.27 (3.06, 5.96)**	-
Gestational age[k]					
≥37 wks	8463	201	23.2	1.00	-
<37 wks	1843	158	79.0	**3.61 (2.90, 4.49)**	-
Small for gestational age[l]					
No	3522	42	11.8	1.00	-
Yes	5352	113	20.7	**1.77 (1.24, 2.53)**	-

[i] A total of 598 were missing night blindness data

[a] Adjusted for treatment received, maternal literacy, ethnic group, caste, latrine ownership, maternal occupation, electricity in the home, cattle ownership, parity and maternal nightblindness during pregnancy

[b] A total of 229 were missing ethnicity data

[c] A total of 220 were missing caste data

[d] A total of 4 were missing literacy data

[e] A total of 9 were missing latrine data

[f] A total of 9 were missing occupation data

[g] A total of 229 were missing electricity data

[h] A total of 236 were missing cattle ownership data

[j] A total of 1668 were missing birth weight data

[k] A total of 80 were missing gestational age data

[l] A total of 1716 were missing small for gestational age data

Table 4

Crude and adjusted Odds ratios (OR) and 95% Confidence Intervals for adverse birth outcomes by maternal age category

Outcome	Maternal Age Category (years)			
	12−15	16−17	18−19	20−24
Low birth weight (<2500g)	**2.75 (2.16, 3.49)**	**1.76 (1.56, 1.99)**	**1.46 (1.32, 1.62)**	1.00
Preterm (<37 wks)	**1.62 (1.25, 2.10)**	**1.47 (1.29, 1.68)**	**1.27 (1.14, 1.43)**	1.00
Small for Gestational Age	**2.15 (1.65, 2.82)**	**1.62 (1.44, 1.83)**	**1.25 (1.14, 1.38)**	1.00
Neonatal Mortality	**2.24 (1.40, 3.59)**	**1.61 (1.22, 2.12)**	1.25 (0.97, 1.59)	1.00
Neonatal Mortality[a]	1.53 (0.90, 2.60)	1.17 (0.84, 1.64)	1.00 (0.76, 1.32)	1.00
Neonatal Mortality[b]	1.36 (0.65, 2.84)	1.23 (0.78, 1.94)	0.81 (0.54, 1.22)	1.00
Neonatal Mortality[c]	1.37 (0.79, 2.37)	1.10 (0.78, 1.54)	0.97 (0.73, 1.28)	1.00
Neonatal Mortality[d]	1.19 (0.53, 2.70)	1.30 (0.82, 2.05)	0.84 (0.55, 1.26)	1.00
Neonatal Mortality[e]	1.14 (0.50, 2.61)	1.24 (0.78, 1.97)	0.82 (0.54, 1.25)	1.00
Neonatal Mortality[f]	1.14 (0.50, 2.61)	1.24 (0.78, 1.97)	0.82 (0.54, 1.25)	1.00

[a] Adjusted for treatment received, maternal literacy, ethnic group, caste, latrine ownership, maternal occupation, electricity in the home, cattle ownership, parity and maternal nightblindness during pregnancy

[b] Same as [a] plus adjustment for low birth weight

[c] Same as [a] plus adjustment for preterm birth

[d] Same as [a] plus adjustment for small for gestational age

[e] Same as [a] plus adjustment for preterm and small for gestational age

[f] Same as [a] plus adjustment for 4 preterm birth and small for gestational age categories (term & not SGA, term & SGA, preterm & not SGA, both preterm & SGA)