EARLY NEONATAL OUTCOMES AMONG MOTHERS RECEIVING VARIABLE DOSES OF DEXAMETHASONE FOR PRETERM PREMATURE RUPTURE OF MEMBRANES AT KENYATTA NATIONAL HOSPITAL: A RETROSPECTIVE COHORT STUDY

Authors

Odhiambo SA[1] ; Qureshi ZP[1] ; Ndavi PM[1]; Kosgei RJ[1]; Kihara AB[1]; Ayieko P[2]; Koigi PK [3]; Osoti A[1]; Odawa FX[1]; Gwako GN[1]; Kilonzo MK[1]; Kireki O[1]; Ogutu O[1]

Institutional Affiliations:

1. University of Nairobi, College of Health Sciences, Department of Obstetrics and Gynaecology,
2. London School of Hygiene and Tropical Medicine
3. Department of Obstetrics and Gynaecology, The Nairobi Hospital

Correspondence: scolanick08@gmail.com

Key words: antenatal corticosteroids, complete dose; Incomplete dose, dexamethasone

ABSTRACT

Background: Antenatal corticosteroids have been shown to reduce complications that arise from preterm birth. Globally, the prevalence of preterm birth is 11.1%, with 60% of neonatal mortality in developing countries being due to preterm births. In Sub-Saharan Africa and Kenya, the preterm birth rate is 18% and 12% respectively. Currently, there is no consensus on the optimal dosing of antenatal corticosteroids. However, authors agree that they should be administered even when delivery is anticipated within 12 hours. Therefore, does incomplete dosing of dexamethasone confer any benefit to premature neonates?

Objective: To compare the early neonatal outcomes among mothers who had preterm premature rupture of membranes (PPROM) and received two 12 mg doses of dexamethasone to those who received one 12-mg dose of dexamethasone between 28 to 34 weeks of gestation at KNH

Methods: A retrospective cohort study involving neonates of mothers who had preterm premature rupture of membranes at 28 to 34 weeks gestation in KNH in the period between January 1, 2011, and December 31, 2015 and received either two 12 mg doses of dexamethasone (exposed group) or one 12-mg dose (unexposed group). The groups were compared for early neonatal outcomes. Sample size of 328 neonates was calculated, with 164 neonates in each arm.

Results: There were no differences in the early neonatal outcomes (Apgar score <7 at 5 minutes, RDS, NEC, mortality and duration of hospital stay) apart from neonatal septicemia which was higher in the two 12-mg dexamethasone cohort (RR 0.78, 95%CI 0.62 to 0.99; p=0.039). Subgroup analysis by gestational ages showed increased neonatal mortality in the single 12-mg dose group (RR 2.09 95%CI 1.11-3.93; p=0.023).

Conclusion: The incidence of early neonatal outcomes of mothers with preterm PROM at 28 to 24 weeks gestation at KNH in 2011 to 2015 were similar for mothers who received two doses of 12 mg dexamethasone and those who received single dose dexamethasone dose apart from early neonatal septicemia which was increased in the two 12 mg dexamethasone group.

INTRODUCTION

Globally, 15 million live births are preterm, giving a prevalence of 11% (1). Over 1 million of these babies die, especially in the lower- and middle-income countries (LMIC). (1). In Sub-Saharan Africa and in Kenya, the preterm birth rate is 18% and 12% respectively. Preterm premature rupture of membranes contributes to 1 out of 4 preterm births (2). Numerous severe morbidities are associated with preterm birth, many do result in mortality. However, this can be reduced by administration of antenatal corticosteroids. (3). Many studies have demonstrated the benefit of single course antenatal corticosteroids (24 mg of betamethasone or dexamethasone) (3). In Kenyatta National Hospital (KNH), dexamethasone is the antenatal corticosteroid used and is administered intramuscularly as 12-mg twice over 24 hours (4). There is variability in the administration of dexamethasone since the optimal time when benefits accrue is not known and time of delivery cannot be predicted (3). Few studies have been conducted in the developed world on the benefits of incomplete antenatal corticosteroids (5-6). This is significant because it is not always possible to complete the course of antenatal corticosteroids since many babies are delivered before completion. Studies have shown the completion rate to be 3-10 % and 30% in developing and developed countries respectively (4). However, systematic review and guidelines emphasize the need to administer antenatal corticosteroids even when delivery is anticipated in 12 hours (3, 7-9).

This study aimed to compare the early neonatal outcomes among mothers who had preterm premature rupture of membranes (PPROM) and received two 12mg doses of dexamethasone to those who received one 12mg dose of dexamethasone between 28 to 34 weeks gestation at KNH.

METHODOLOGY

Study design: A retrospective cohort study involving neonates of mothers who had preterm premature rupture of membranes at 28 to 34 weeks gestation and received dexamethasone

Study setting: Kenyatta National Hospital, the largest teaching and referral hospital in Kenya.

Study context: Labor ward has >1000 admissions per month with an average nurse/patient ratio of 1:7 per shift. Mothers are admitted at ± 20 weeks gestation in labor ward where initial management of PPROM, such as dexamethasone administration, is instituted. In event of preterm delivery, the midwife assesses the neonate for poor outcomes like Respiratory distress syndrome (RDS) and transfers the neonate to the new born unit (NBU), where the pediatrics resident takes over management in consultation with the neonatologists on call. NBU has with seven sections with a 50-bed total capacity and has 1000 admissions per month.

Study population

Neonates of mothers who had PPROM between 28 to 34 weeks gestation in KNH during the five-year period between January 1, 2011, and December 31, 2015, and received variable doses of dexamethasone antenatally. The inclusion and exclusion criteria used are shown in Box 1.

Sampling procedure

Purposeful sampling was used to get records of neonates whose mothers had preterm PROM between 28 to 34 weeks gestation. Simple random stratified sampling based on exposure of interest (two 12 mg dosing) or non-exposure (single 12-mg dosing) status was used to select two groups of 164 mothers.

Sample size

The sample size was calculated using Fleiss' formula and sample size of 328 neonates was used.

Inclusion criteria	Exclusion criteria
1. A neonate whose mother had preterm PROM between 28 to 34 weeks gestation 2. Singleton or multiple pregnancies with a live fetus (fetuses). 3. Antenatal exposure to either one or two 12-mg doses of dexamethasone	A neonate whose mother had preterm PROM at 28 to 34 weeks gestation with; 1. Chorioamnionitis 2. Active phase of labor 3. Pregnancies complicated by co-morbidities 4. Intrauterine fetal death 5. Congenital malformations 6. Previously treated with corticosteroids 7. Contraindication to corticosteroids 8. Indication for immediate delivery 9. Anticipated delivery > 7 days

Key: PROM – Premature rupture of membranes

Box 1: Inclusion and exclusion criteria

Exposure variables	Outcome variables
Number of neonates with RDS	Incidence of respiratory distress syndrome
Dexamethasone dose	Incidence of necrotizing enterocolitis
Number of neonates with NEC	Incidence of neonatal septicemia
Number of neonates with neonatal septicemia	Incidence of neonatal mortality
Number of neonatal deaths	Number of days of admission in NBU/NICU
Number of days of admission	

Key: NBU/ NICU: Newborn unit/ Neonatal intensive care unit

Box 2: Exposure and outcome variables

Data variables

The exposure and outcome variables are shown in Box 2. The potential confounders in this study were gestational age, maternal age, duration of preterm PROM and birth weight.

Data management

Data on the study variables were extracted from the patients' files and filled into data abstraction forms by trained research assistants. Data were inspected for completeness, anonymized and transcribed into a Microsoft Excel spread sheet. Data were then analyzed using Statistical Package for Social Science (SPSS) version 21. Univariate and Multivariate analysis was undertaken and relevant tests of statistical significance were applied.

Ethics

Approval to conduct this study was obtained from the University of Nairobi/ Kenyatta National Hospital Ethics Review Committee.

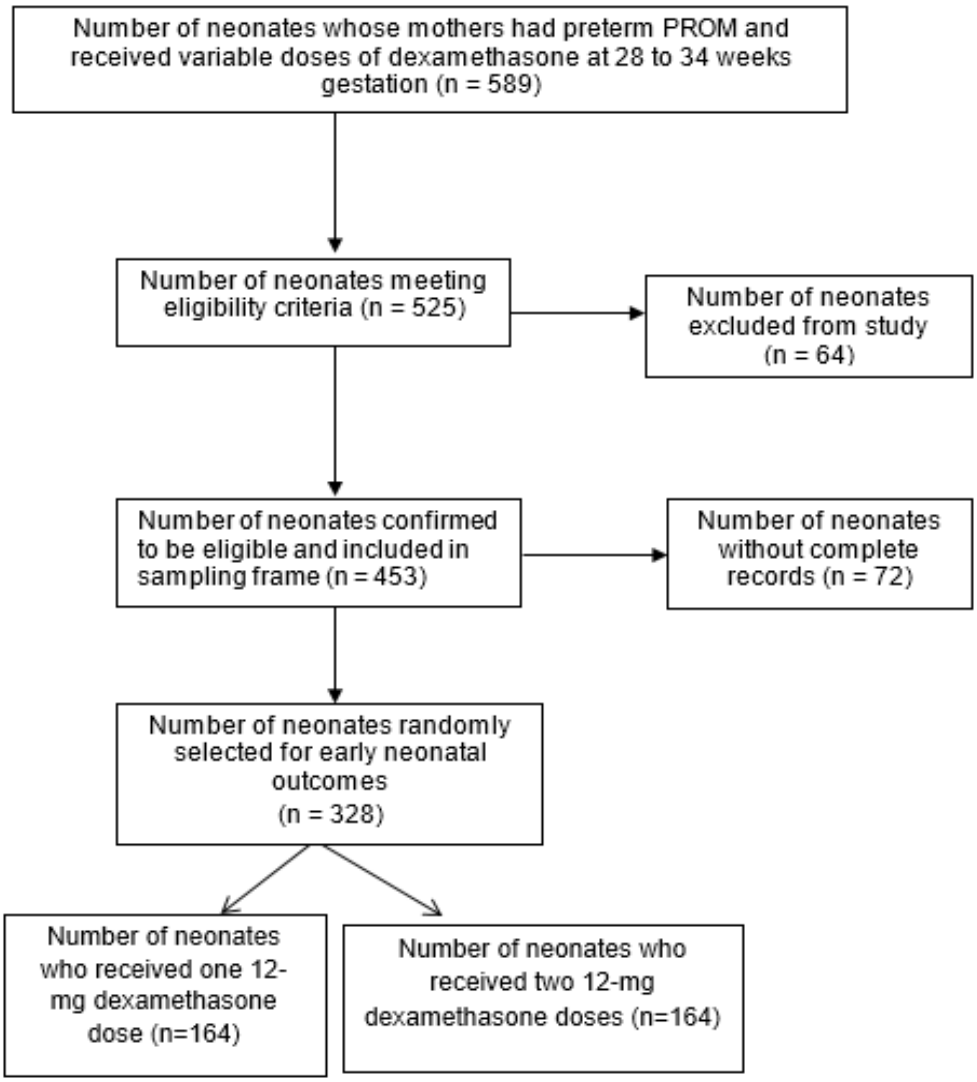


Figure 1: Flow chart showing recruitment of participants in the study

RESULTS

This study recruited a total of 328 files out of a total of 589 records that were screened for eligibility as shown in Figure 1.

The socio-demographic and reproductive age characteristics of the study population are shown in Table 1. The mean maternal age was 27 years. Most of the mothers were married (81% versus 85%) and some had secondary level of education (38% versus 48%). The gestational age of the neonates whose mothers received two 12-mg doses of dexamethasone was 32-34 weeks compared to 30-31 weeks for the one 12-mg dexamethasone group. The mean birth weight was also comparable (1737.3±314.9g and 1745.8±380.8g), for the single 12-mg and two 12-mg dexamethasone doses respectively.

There were no differences in the incidences of APGAR <7 at 5 minutes scores, RDS, NEC and neonatal mortality between the two groups. However, the two 12-mg dexamethasone dose had significantly higher risk of neonatal septicemia compared to the single dose (RR 0.78, 95%CI 0.62 – 0.99; p=0.039).

The subgroup analysis of early neonatal outcomes by gestational age is shown in Table 3. There was reduced risk of RDS at 32-34 weeks with two 12 mg dexamethasone doses (RR 0.66 95%CI 0.53 to 0.83; p=0.001). Single dose dexamethasone was associated with increased mortality at 28-29 weeks gestation (RR 2.52 95%CI 1.27 to 5.01; p=0.008) but at 30-31 weeks gestation, mortality was decreased (RR 0.48 95%CI 0.25 to 0.90; p=0.023) with single dose dexamethasone.

On multivariate logistic regression analysis, women aged >30 yrs had 3-fold higher risk of neonatal mortality (RR 3.34, 95%CI 1.74 to 6.41; p<0.001), table 4.

Table 1: Socio-demographic and reproductive characteristics of mothers who had preterm PROM and received variable doses of dexamethasone between 2011 and 2015 at KNH

Variable	Two 12-mg doses dexamethasone (N = 164) n (%)	One 12-mg dose dexamethasone (N = 164) n (%)	p value
Maternal age (years)			
< 18	5(3)	5(3)	1.0
18-24	50(30)	66(40)	0.065
25-29	60(37)	38(23)	0.008
30-34	30(18)	28(17)	0.772
> 35	19(12)	27(16)	0.203
Marital status			
Single	23(14)	18(11)	0.404
Married	133(81)	139(85)	0.379
Divorced	8(5)	7(4)	0.792
Education level			
None	5(3)	3(2)	0.474
Primary	40(24)	32(20)	0.286
Secondary	63(38)	78(48)	0.094
Tertiary	56(34)	51(31)	0.556
Occupation			
Unemployed	84(51)	71(43)	0.15
Employed	54(33)	66(40)	0.169
Other	26(16)	27(16)	0.881
Parity			
Nullipara	63(38)	88(54)	0.006
Multipara	97(59)	69(42)	0.002
Gestational age (weeks)			
28-29	24(15)	37(23)	0.065
30-31	40(24)	66(40)	0.002
32-34	100(61)	61(37)	<0.001
Birth weight (± SD) (g)	1745.8(±380.8)	1737.3(±314.9)	0.825

Table 2: Early neonatal outcomes of mothers receiving one or two 12mg doses of dexamethasone for preterm PROM at 28 to 34 weeks gestation between 2011 and 2015 at KNH

Variable	One 12mg dose	Two 12mg dose	RR (95% CI)	p value
APGAR <7 at 5 min				
Yes	18(11.0)	11(6.7)	1.00	
No	146(89.0)	153(93.3)	0.79(0.58-1.07)	0.127
RDS				
Absent	52(31.7)	40(24.4)	1.00	
Present	112(68.3)	124(75.6)	0.84(0.67-1.05)	0.127
NEC				
Absent	160(97.6)	162(98.8)	1.00	
Present	4(2.4)	2(1.2)	1.34(0.75-2.39)	0.318
Neonatal sepsis				
Absent	105(64.0)	86(52.4)	1.00	
Present	59(36.0)	78(47.6)	0.78(0.62-0.99)	0.039
Length of stay				
< 3 days	31(18.9)	38(23.2)	1.00	
< 7 days	43(26.2)	49(29.9)	1.04(0.74-1.46)	0.82
> 7 days	90(54.9)	77(47.0)	1.20(0.89-1.61)	0.23
Neonatal outcome				
Alive	124(75.6)	114(69.5)	1.00	
Dead	40(24.4)	50(30.5)	0.85(0.66-1.11)	0.234

Key: RDS: Respiratory Distress Syndrome; NEC: Necrotizing Enterocolitis

Table 3: Early neonatal outcomes of mothers receiving variable doses of dexamethasone for preterm PROM at different gestational ages between 2011 and 2015 at KNH

Variable		Single 12-mg dose	Two 12-mg dose	RR (95% CI)	P value
Age 28-29 weeks					
RDS	Absent	5(20.8)	10(27.0)	1.00	
	Present	19(79.2)	27(73.0)	1.24(0.56-2.76)	0.6
Neonatal sepsis	Absent	10(41.7)	11(29.7)	1.00	
	Present	14(58.3)	26(70.3)	0.73(0.39-1.37)	0.331
Neonatal outcome	Alive	8(33.3)	26(70.3)	1.00	
	Dead	16(66.7)	11(29.7)	2.52(1.27-5.01)	0.008
Age 30-31 weeks					
RDS	Absent	8(20.0)	21(31.8)	1.00	
	Present	32(80.0)	45(68.2)	1.51(0.79-2.88)	0.216
Neonatal sepsis	Absent	24(60.0)	37(56.1)	1.00	
	Present	16(40.0)	29(43.9)	0.90(0.55-1.50)	0.694
Neonatal outcome	Alive	31(77.5)	35(53.0)	1.00	
	Dead	9(22.5)	31(47.0)	0.48(0.25-0.90)	0.023
Age 32-34 weeks					
RDS	Absent	39(39.0)	9(14.8)	1.00	
Neonatal sepsis	Present	61(61.0)	52(85.2)	0.66(0.53-0.83)	0.001
	Absent	71(71.0)	38(62.3)	1.00	
	Present	29(29.0)	23(37.7)	0.86(0.65-1.13)	0.276
Neonatal outcome	Alive	85(85.0)	53(86.9)	1.00	
	Dead	15(15.0)	8(13.1)	1.06(0.76-1.47)	0.732

Key: Respiratory Distress syndrome

Table 4: Multivariate logistic regression analysis of factors associated with early neonatal mortality following delivery after preterm premature rupture of membranes at 28 to 34 weeks gestation between 2011 and 2015 at the Kenyatta National Hospital

Variable	RR	95% CI		p value
Dexamethasone dose				
Two 12-mg dose	1.0			
Single 12-mg dose	1.03	0.70	1.52	0.879
Maternal age				
18-24	1.0			
25-29	0.98	0.50	1.88	0.94
30-34	3.50	2.00	6.11	<0.001
More than 35	3.34	1.74	6.41	<0.001
Parity				
Primigravida	1.0			
Multipara	0.63	0.36	1.09	0.098
Gestation age				
28-29 weeks	1.0			
30-31 weeks	0.95	0.63	1.43	0.81
32-34 weeks	0.59	0.33	1.07	0.082
Duration of PROM				
Less than 12 hours				
12-48 hours	0.65	0.38	1.11	0.119
More than 48 hours	0.97	0.64	1.47	0.895
Birth weight				
<1500 g				
1500 g and above	0.65	0.41	1.03	0.067

DISCUSSION

This study showed no differences in the incidences of APGAR scores< 7 at 5 minutes, RDS, NEC and mortality in the early neonatal period among mothers who had preterm premature rupture of membranes and received single 12-mg dose dexamethasone compared to those who received two 12-mg doses of dexamethasone. These findings are corroborated by other studies evaluating similar outcomes among women treated with betamethasone (5) (6) but contradicted findings of another study that demonstrated decreased mortality and NEC only with complete course of betamethasone (10).

There was decreased neonatal sepsis in the cohort that received a single 12-mg dose of dexamethasone. This probably is due to the immunosuppressive effect of corticosteroids and shorter time-to-delivery interval in this cohort. Two other studies showed increased risk of sepsis with steroid exposure (10-11).

Logistic regression analysis showed no difference in adverse neonatal outcomes based on the dose of dexamethasone administered. However, increased maternal age was significantly predictive of neonatal mortality in this study. Subgroup analysis by gestational age showed variable neonatal mortality, with an increase at 28-29 weeks, a decrease at 30-31weeks and no difference at 32-34 weeks. This may have been caused by the lower gestational age at delivery of the neonates. Another study showed conflicting findings in the different gestational age groups (6)

The limitation of this study was missing data secondary to its retrospective nature. However, this study adds contributes to the existing body of knowledge on the local impact of variable doses of dexamethasone on neonatal mortality. Overall, our results imply that incomplete course dexamethasone may confer similar benefits as complete course and clinicians should not hesitate to administer it in patients with imminent preterm birth.

CONCLUSION

In conclusion, incomplete course dexamethasone may confer similar benefits as complete course and clinicians should not hesitate to administer it in patients with imminent preterm birth. It is therefore recommended that clinicians should administer it to patients with imminent preterm birth.

Acknowledgements: The research leading to this publication was conducted through an adaptation of the Structured Operational Research and Training Initiative (SORT IT), a global partnership led by the UNICEF/UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases (WHO/TDR). The model is based on a course developed jointly by the International Union against Tuberculosis and Lung Disease (The Union) and Médecins sans Frontières.

The specific SORT IT programme which resulted in this publication was developed and implemented by the University of Nairobi, Department of Obstetrics and Gynaecology, Nairobi, Kenya with financial support from WHO/TDR; the staff in the Reproductive health department of Kenyatta National Hospital, Staff working in the records department at KNH, My research assistants (Shirleen Lang'at, E.Hans Odhiambo, Jackline W. Ngigi and Becky W. Njuguna); Special thanks to Nicholas Airo, Kristina Amor, Justin Hawi and Solidard Jennifer.

Conflict of interest: None declared

Funding: Study was conducted using the principal investigator's own resources

Authors: All authors made significant contributions towards development of this scientific manuscript.

REFERENCES

1. March of Dimes, PMNCH, Save the Children, WHO. Born Too Soon: The Global Action Report on Preterm Birth. Eds CP Howson, MV Kinney, JE Lawn World Health Organization. Geneva,2012
2. Goldenberg RL, Culhane JF, Iams JD, Romero R, Slattery M, Morrison J, et al. Epidemiology and causes of preterm birth. Lancet (London, England) [Internet]. Elsevier; 2008 Jan 5 [cited 2016 Aug 8]; 371(9606):75–84. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18177778
3. Roberts, D., Brown, J., Medley, N., & Dalziel, S. R. (2017, March 21). Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. Cochrane Database of Systematic Reviews. John Wiley and Sons Ltd. https://doi.org/10.1002/14651858.CD004454.pub3
4. Gwako G, Qureshi Z, Kudoyi W, Were F. Antenatal corticosteroid use in preterm birth at Kenyatta National Hospital [Internet]. Journal of Obstetrics and Gynaecology of Eastern and Central Africa. Kenya Obstetrical and Gynaecological Society (KOGS); 2016 [cited 2016 May 14]. p. 3–9. Available from: http://www.ajol.info/index.php/jogeca/article/view/130771
5. Elimian, A., Figueroa, R., Spitzer, A. R., Ogburn, P. L., Wiencek, V., & Quirk, J. G. (2003). Antenatal corticosteroids: Are incomplete courses beneficial? Obstetrics and Gynecology, 102(2), 352–355. https://doi.org/10.1016/S0029-7844(03)00485-X
6. Costa S, Zecca E, De Luca D, De Carolis MP, Romagnoli C. Efficacy of a single dose of antenatal corticosteroids on morbidity and mortality of preterm infants. Eur J Obstet Gynecol Reprod Biol. 2007;131(2):154–7.
7. American Congress of Obstetricians and Gynaecologists Committee on Obstetric Practice. ACOG Committee Opinion No. 475: antenatal corticosteroid therapy for fetal maturation. Obstet Gynecol [Internet]. 2011;117(2 Pt 1):422–4. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21252775
8. Royal College of Obstetricians and Gynaecologists. Antenatal Corticosteroids to Reduce Neonatal Morbidity and Mortality. Green-top Guide No 7. 2010;(7):1–13
9. Crane, J., Armson, A., Brunner, M., De La Ronde, S., Farine, D., Keenan-Lindsay, L., Executive Committee of the Society of Obstetricians and Gynaecologists of Canada. (2003). Antenatal corticosteroid therapy for fetal maturation. Journal of Obstetrics and Gynaecology Canada (Journal d'obstetrique et Gynecologie Du Canada), JOGC: 25(1): 45–52. https://doi.org/10.1097/AOG.0b013e31820eee00

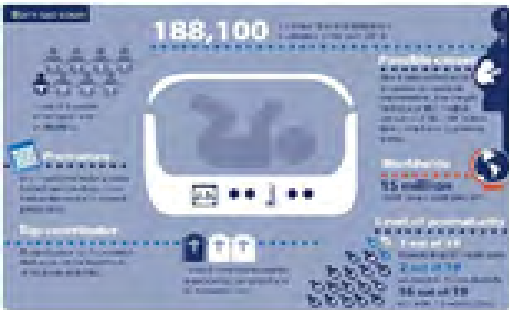
10. Wong D, Abdel-Latif M, Kent A. Antenatal steroid exposure and outcomes of very premature infants: a regional cohort study. Arch Dis Child Fetal Neonatal Ed [Internet]. 2014; 99: F12-20. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24142624

11. Althabe, F., Belizán, J. M., McClure, E. M., Hemingway-Foday, J., Berrueta, M., Mazzoni, A., Buekens, P. M. (2015). A population-based, multifaceted strategy to implement antenatal corticosteroid treatment versus standard care for the reduction of neonatal mortality due to preterm birth in low-income and middle-income countries: The ACT cluster-randomized trial. The Lancet, 385(9968), 629–639. https://doi.org/10.1016/S0140-6736(14)61651-2

****

POLICY BRIEF

BORN TOO SOON: PROVIDE CORTICOSTEROIDS AT THE EARLIEST OPPORTUNITY EVEN IF DOSE IS NOT COMPLETED



THE IMPACT OF BABIES BEING BORN TOO SOON

Preterm birth affects 11% of live births globally (1) . In Sub-Saharan Africa and in Kenya, it accounts for 18% and 12% of live births respectively (1). This accounts for over 188,000 preterm births in a year. Of these, 14,700 die due to complications of prematurity in Kenya. Of those who survive, 4,900 incur delayed developmental milestone achievement (2).

Kenya is ranked 13th highest in a global survey on deaths resulting from preterm births (2). The three main causes of preterm birth are early labor; medical conditions that increase risk of continuing the pregnancy and breakage of water before term and before labor. (1) The good news is that these deaths are preventable if appropriate measures are taken. Use of antenatal corticosteroids has been shown to improve lung maturity, thereby enhancing the chances of survival after preterm birth (3).

PRIORITY ACTIONS

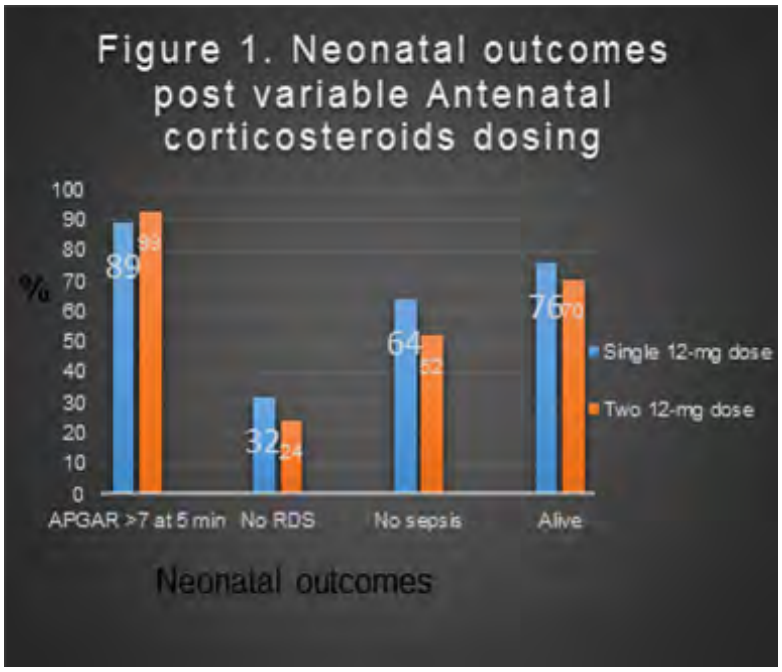
1. Educate health workers on the importance of administering antenatal corticosteroids at the earliest opportunity to mothers at risk of preterm birth

2. Empowerment of pregnant women to understand danger signs and present to hospitals as soon as they have premature rupture of membranes

3. Provide antenatal corticosteroids at points of care for pregnant women at risk of early delivery

PROVISION OF ANTENATAL CORTICOSTEROIDS AT THE EARLIEST OPPORTUNITY

Antenatal corticosteroids are given twice over 24 hours for completion of dosage. However, delivery may occur prior to completion of the two doses. Even a single dose of antenatal corticosteroids given to mothers at risk of preterm delivery may confer some benefit (4). Early administration of antenatal corticosteroids would ensure longer effects of the drugs on the unborn baby before delivery and it could also increase chances of administration of the second dose. Lack of timely administration of antenatal corticosteroids has been a hindrance towards preventing preterm birth complications. Antenatal corticosteroids are best given at the earliest possible opportunity.



KEY FINDINGS

1. **Incomplete dose of antenatal corticosteroids offered some benefits to babies born too soon.**
2. **Babies who were born at lower gestations showed varied risks of neonatal deaths with different dosages of antenatal corticosteroids**
3. **There were various factors that independently affected the neonatal outcomes in the different dosing regimens.**
4. **The risk of babies having complications of preterm births was dependent on time to administration of antenatal corticosteroids**

AUTHORS:
Odhiambo SA, Qureshi ZP, Ndavi PM, Kosgei RJ, Kihara AB, Koigi PK, Ayieko P, Osoti A, Odawa FX, Gwako GN, Kilonzo MK, Kireki O, Ogutu O
Correspondence: Scolanick08@gmail.com

Acknowledgements: The research leading to this publication was conducted through an adaptation of the Structured Operational Research and Training Initiative (SORT IT), a global partnership led by the UNICEF/UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases (WHO/TDR). The model is based on a course developed jointly by the International Union against Tuberculosis and Lung Disease (The Union) and Médecins sans Frontières. The specific SORT IT programme which resulted in this publication was developed and implemented by the University of Nairobi, Department of Obstetrics and Gynaecology, Nairobi, Kenya with financial support from WHO/TDR.

WHAT SHOULD BE DONE?

1. **Ensure availability of corticosteroids in every health facility so that every mother at risk of preterm birth is afforded an opportunity to have a higher chance of their baby surviving**
2. **Ensure that all mothers who are admitted with preterm labor or with preterm breakage of water receive corticosteroids immediately they are admitted, even if delivery is imminent**
3. **Ensure that all mothers are aware that there is treatment available if they go in to premature labor**

REFERENCES

1. **March of Dimes, PMNCH, Save the Children, WHO. Born Too Soon: The Global Action Report on Preterm Birth. Eds CP Howson, MV Kinney, JE Lawn World Health Organization. Geneva,2012**
2. **Liu, L., Oza, S., Hogan, D., Perin, J., Rudan, I., Lawn, J. E., Black, R. E. (2015). Global, regional, and national causes of child mortality in 2000-13, with projections to inform post-2015 priorities: An updated systematic analysis. The Lancet, 385(9966), 430–440. https://doi.org/10.1016/S0140-6736(14)61698-6**
3. **Roberts, D., Brown, J., Medley, N., & Dalziel, S. R. (2017, March 21). Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. Cochrane Database of Systematic Reviews. John Wiley and Sons Ltd. https://doi.org/10.1002/14651858.CD004454.pub3**
4. **Odhiambo SA, Qureshi ZP, Ndavi PM, Kosgei RJ, Kihara AB, Koigi PK, et al. Early neonatal outcomes among mothers receiving variable doses of antenatal corticosteroids for preterm premature rupture of membranes at Kenyatta National Hospital. J Obstet Gynecol E Centr Afr, 2018; 30(2): 54-61**