Free Radicals and Antioxidants., 2025; 15(1):35-41.
https://www.antiox.org

Original Article

Cord Blood Oxidant and Antioxidant Profiles in Term Nigerian Newborns with Perinatal Asphyxia

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ABSTRACT

Background: Perinatal Asphyxia (PA) often leads to the generation of free radicals and oxidants which affect the newborns antioxidant status. Little is known about the oxidant and antioxidant status of babies with various severity of asphyxia in developing countries with huge burden of the disease, hence the aim of this study. **Materials and Methods:** Babies delivered at term at the Wesley Guild Hospital, Ilesa, Nigeria were consecutively recruited into a comparative cross-sectional study design over a nine-month period. Those with 5-min Apgar score <7 were classified as PA, while the comparative group had Apgar score ≥7. 5 mL of cord blood from the babies were analysed for Total Oxidants (TOS) and Antioxidants Status (TAS) and their components using Liquid Chromatography. (Water Incorporate, California, U.S.A) Data obtained were analysed using SPSS version 21. **Results:** 84 babies were recruited for each group with male preponderance 1.2:1. Sixty-one (72.6%) of the PA group had moderate (score 4-6); 23 (27.4%) had severe (score ≤3) PA, and 66 (78.6%) had features of Hypoxic Ischaemic Encephalopathy (HIE). The cord blood TOS [0.4 (0.2-5.7) vs. 0.1(0.02-0.4) ng/dL; $p<0.001$] was higher in PA group. Conversely, cord blood TAS [114.2 (97.7-137.9) vs. 249.9 (111.8-340.6) ng/dL; $p<0.001$] was lower in the babies with PA. However, TOS and TAS were not related to the severity of PA and HIE. **Conclusion:** Increased oxidative stress was demonstrated in term babies with PA, which was not associated with severity. Cord blood TOS and TAS may be good biomarkers of PA in term Nigerian babies.

Keywords: Asphyxia, Antioxidants, Free radicals, Hypoxic-ischaemic encephalopathy, Oxidative stress.

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Received: 09-06-2025;
Revised: 17-07-2025;
Accepted: 18-08-2025.

INTRODUCTION

Perinatal Asphyxia (PA) in the newborn is characterised by hypoxia, ischaemia, acidosis, hypercapnia and multi-organ dysfunction.[1,2] It is often caused by antepartum, intrapartum and postpartum events resulting in inability of the newborn to initiate or maintain breathing at birth.[1-3] It is a significant cause of neonatal morbidity and mortality with the majority (>90%) of deaths due to PA occurring in developing countries.[4,5]

Babies with PA often suffer hypoxic and ischaemic injuries which trigger cellular anaerobic glycolysis, with consequent reduction in Adenosine Triphosphate (ATP) production and failure of the sodium-potassium ATPase pump (primary energy failure).[6] With primary energy failure, there is disruption of ion exchange across the cell membrane leading to intracellular influx of sodium, calcium and water.[2,6] This results in a cascade of events that induce the production of oxygen free radicals and oxidants through the activation of nitric oxide synthase, increased prostaglandin synthesis and activation of xanthine oxidase.[2,6]

Free radicals are unstable molecules produced during normal cellular metabolism while oxidants are reactive molecules produced either within the body or in the environment that can oxidize and damage cellular molecules such as protein, DNA and lipids.[7] Free radicals are oxidants which are either Reactive Oxygen Species (ROS) such as superoxide anion radical (O_2^-), Hydrogen Peroxide (H_2O_2), Hydroxyl Radical (OH^-) or Reactive Nitrogen Species (RNS) such as Nitric Oxide radical (NO^-).[8] These oxidants are often assayed together as Total Oxidant Status (TOS) which is the sum total of the oxidants in the body.[7,8] Free radical mediated injuries play a major role in the multi-organ dysfunction that occurs in babies with PA.

Under physiologic condition, antioxidants are produced to mop up free radicals to maintain a balance in the oxidant/antioxidant status.[9,10] However in PA, oxidative stress induces a wide range of cellular injuries due to an imbalance caused either by excessive generation of free radicals or diminished antioxidant capacity.[10,11] Antioxidants can be enzymatic like Superoxide Dismutase (SOD),

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DOI: 10.5530/fra.2025.1.5

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Catalase (CAT) and glutathione peroxidase or non-enzymatic like bilirubin, selenium, flavonoids, vitamin A, C, E.[10] The sum total of the serum non-enzymatic antioxidants is known as the Total Antioxidant Status (TAS).[11]

Previous published works on oxidants and antioxidants profile in babies with PA used different parameters as a measure of oxidants and antioxidant status with variable results as related to severity and outcomes of PA.[12-15] Some of these studies have very small sample size, and did not assess for summation of total oxidant and antioxidants.[13-15] This study therefore hopes to compare the cord blood oxidants and antioxidant status of Nigerian babies with and without PA and determine the relationship with oxidant and antioxidant levels with severity of PA.

MATERIALS AND METHODS

A hospital-based comparative cross-sectional study was carried out over a period of nine months at the neonatal and labour wards of the Wesley Guild Hospital (WGH), Ilesa, Nigeria- a tertiary unit of the Obafemi Awolowo University Teaching Hospitals Complex (OAUTHC), Ile-Ife, Nigeria.

Term babies delivered at the WGH during the nine-month study period were consecutively recruited after mothers or caregiver had given consent during the antenatal periods. Babies with low 5-min Apgar score (<7) were defined to have PA, and those with normal Apgar score (≥7) at one and 5 min were recruited as the comparative group. Babies with PA were further classified into mild-moderate (Apgar scores 4-6) and severe PA (Apgar scores ≤3).[16] Hypoxic Ischaemic Encephalopathy (HIE) for this study was defined and classified using Levene's criteria.[17] Babies with major congenital malformations (such as neural tube defect, chest and abdominal wall defect) as well as babies of mothers with preeclampsia/eclampsia who received medications like magnesium sulphate, diazepam within 4 hr of delivery were excluded.

Recruited babies were thoroughly examined and appropriately resuscitated. 5 mL of cord blood were collected into a plain bottle from the foetal end of the separated umbilical cord after delivery. The babies' anthropometric parameters were then taken using standard protocol and recorded. The baby's birth weight for gestational age was plotted on the intergrowth chart to classify the babies into Appropriate for Gestational Age (AGA), Small for Gestational Age (SGA) or Large for Gestational Age (LGA). The vital signs (temperature, heart rate, respiratory rate) were documented at recruitment. For the babies with PA, important perinatal events such as cord accidents, cord round the neck, prolonged labour, instrumental delivery were documented. These babies were managed based on the neonatal unit protocol. All the babies were examined at discharge and any sequelae/outcome were duly documented. The duration of hospitalisation was noted. All the information was recorded into a data proforma specifically designed for the study.

The collected cord blood samples were allowed to clot and serum separated and analysed for TOS and TAS and their components at the Analytical Services Laboratories, International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria. These oxidants and antioxidants were assayed using High Performance Liquid Chromatography (HPLC) methods[18] via an automated Waters 616/626 HPLC machine (Waters Incorporate, California, USA) following standard protocol.

Sample Size determination

This was estimated using open Epi(R) sample size software. At 5% significance (alpha) level, 80% study power and 95% confidence interval, using a mean difference in Serum mean (SD) TAS in Neonates with and with PA as 2.15 (0.26) μmol/L and 2.00 (0.06) μmol/L respectively from a study by Aydemir *et al.*,[14] and ratio of cases to control being 1. The calculated sample size was approximately 84 each for Neonate with PA and comparative group.

This study was approved by the Institution Research Board of the OAUTHC, Ile-Ife with protocol number IRB/IEC/0004553. Written informed consent was obtained from the parents or caregivers of these babies before enrolment.

Data analysis

Data obtained were analysed using Statistical Product Service Solutions version 21. (IBM Armonk, NY: IBM Corp). Continuous variables such as mother's age, cord blood TAS, cord blood TOS and length of hospital stay were tested for normality using Kolmogorov-Smirnov statistics and by plotting the frequency distribution chart. The mother's age and length of hospitalisation found to be normally distributed were summarised using mean and Standard Deviations (SD) while the TOS, TAS and other individual oxidants as well as antioxidants found to be non-parametric were summarised using median and Interquartile Ranges (IQR). Categorical data like sex, presence or absence of low 5-min Apgar scores and HIE severity as well as socioeconomic class categories were summarised using percentages and proportions. Differences between categorical variables were determined using Chi square or Fisher's exact tests as appropriate, while the differences between continuous variables like TOS and TAS of cases and control were assessed using Mann-Whitney U test. A p-value <0.05 was considered statistically significant at 95% Confidence Interval (CI).

RESULTS

A total of 168 babies (84 with PA and 84 without PA) were recruited for the study. There were ninety (53.6%) males with M: F ratio of 1.2:1. Sixty-one (72.6%) of the cases had moderate (score 4-6) and 23 (27.4%) had severe (score ≤3) PA. Also, 66 (78.6%) had HIE. Mean (SD) maternal age was 28.9 (6.1) years. Mothers of babies with PA were significantly younger than that

of non-asphyxiated babies. [28.3 (6.3) years vs. 29.6 (5.8) years; p=0.044] (Taable 1). The birth weight, head circumference and full length of the babies were similar in both groups, however the axillary temperature of babies with PA at presentation was significantly lower. [36.2 (0.7) vs. 36.5 (0.1) ^{o}C; p<0.001].

The cord blood TOS of the babies ranged from 0.003 to 15.32 ng/dL with a median (IQR) of 0.3 (0.02-0.52) ng/dL. The median (IQR) TOS was significantly higher in babies PA than in those with normal Apgar scores [0.4 (0.2-5.7) vs. 0.03 (0.02-0.4) ng/dL; p<0.001]. In addition, babies with PA had significantly higher median levels of specific oxidants such as hydrogen peroxide [3.8 (2.6-5.5) vs. 2.6 (0.1-3.9) ng/dL; p<0.001] and nitric oxide [7.2 (4.1-9.3) vs. 5.9 (0.2-8.5) ng/dL; p=0.038] than those without PA (Table 1).

The cord blood TAS of the study cohort ranged from 26.0 to 535.9 ng/dL, with a median (IQR) of 125.3 (104.4-295.7) ng/dL. The median (IQR) TAS was significantly lower in babies with PA [114.2 (97.7-137.9) vs. 249.9 (111.8 -340.6) ng/dL; p<0.001]. Other antioxidants like flavonoids, vitamin A, carotenoids and enzymatic antioxidants like glutathione, glutathione peroxidase, glutathione transferase and superoxide dismutase were significantly lower in babies with PA than in the comparative group. (Table 2) Cord blood oxidants and antioxidants were however not significantly related to severity of PA and presence or absence of HIE (Tables 3 and 4).

DISCUSSION

This study highlighted significantly higher cord blood TOS and lower TAS in babies with PA compared to non-asphyxiated babies. It also highlighted no significant relationship between TOS and TAS as well as some of their components with severity of PA using Apgar score and presence of HIE. The findings of significantly higher cord blood levels of oxidants like TOS, hydrogen peroxide and nitric oxide in babies with PA are consistent with findings of Aydemir *et al.*,[12] Gunes *et al.*,[13] and Ergenekon *et al.*,[14] all from Turkey. The elevated oxidants observed may be related to hypoxia and ischaemia in babies with PA which results in influx of calcium into the cell via the N-Methyl-D-Aspartate (NMDA) channels. Increased intracellular calcium generates free radicals including reactive nitrogen species like nitric oxide (from activation of nitric oxide synthase) and reactive oxygen species like hydrogen peroxide (from activation of super oxides and catalytic action of free Fe^{2+}).[2,6] Since the process of asphyxiation often starts in utero around the time of delivery, the cord blood oxidants inevitably became elevated as observed in this study and similar ones.[12-15]

Table 1: Baseline characteristics of the study participants and mothers.

Socio-demographic features	Babies with Asphyxia *n*=84 (%)	Babies with no asphyxia *n*=84 (%)	Total *n*=168	*p*-value
Sex Male female	50 (59.5%) 34 (40.5%)	40 (47.6%) 44 (52.4%)	90 78	0.122
Mother's age (years) ≤19 20-35 >35	7 (8.3%) 64 (76.2%) 13 (15.5%)	1 (1.2%) 68 (81.0%) 15 (17.8%)	8 132 28	0.070*
Mother's level of education Primary Secondary Post-secondary	14 (16.7%) 29 (34.5%) 41(48.8%)	1 (1.2%) 25 (29.8%) 58 (69.0%)	15 54 99	0.001*
Socio-economic class Upper Middle Lower	5 (6.0%) 67 (79.8%) 12 (14.2%)	9 (10.7%) 74 (88.1%) 1 (1.2%)	14 141 13	0.002*
Mother's parity Primiparous multiparous	49 (58.3%) 35 (41.7%)	32 (39.5%) 52 (61.9%)	81 87	0.003
Mode of delivery Vaginal Caesarean section	51 (60.7%) 33 (39.3%)	47 (55.9%) 37 (44.1%)	98 70	0.003

*Fischer's exact test applied.

Table 2: Comparison of the cord blood oxidant and antioxidant profile of the study participants.

Oxidants and antioxidants	Babies with asphyxia	Babies with no asphyxia	*p*-value*
Oxidants	Median (IQR)	Median (IQR)	
Total oxidant status (ng/dL)	0.4 (0.2-5.7)	0.03 (0.02-0.4)	<0.001
Hydrogen peroxide (ng/dL)	3.8 (2.6-5.5)	2.5 (0.1-3.9)	<0.001
Nitric oxide (ng/dL)	7.2 (4.1-9.3)	5.9 (0.2-8.5)	0.038
Malondialdehyde (ng/dL)	3.1 (2.02-7.1)	4.5 (0.9-7.2)	0.165
Antioxidants	Median (IQR)	Median (IQR)	
Total antioxidant status (ng/dL)	114.2 (97.7-137.9)	249.9 (111.8-340.6)	<0.001
Total Carotenoids (ng/dL)	3.9 (1.5-7.7)	8.3 (3.2-17.2)	0.001
Total Flavonoids (ng/dL)	5.6 (2.4-8.8)	7.4 (2.7-52.2)	0.019
Vitamin A (µg/dL)	14.8 (10.7-19.6)	19.7 (12.4-88.1)	0.004
Vitamin E (ng/dL)	2.5 (1.3-6.8)	0.1 (0.1-2.3)	<0.001
Superoxide dismutase (ng/dL)	7.3 (4.1-10.4)	56.6 (7.9-72.8)	<0.001
Glutathione peroxidase (ng/dL)	3.7 (2.7-6.7)	21.1 (5.4-29.0)	<0.001
Glutathione transferase (ng/dL)	4.8 (3.3-9.9)	12.8 (7.7-54.6)	<0.001
Glutathione (ng/dL)	9.1 (5.1-14.2)	11.9 (4.4-31.2)	0.033
Catalase (ng/dL)	2.4 (1.3-4.5)	0.1 (0.1-5.9)	0.001

IQR interquartile range; Bold figures denote statistical significance; * Mann Whitney U test applied.

Table 3: Association between Cord Blood oxidants and antioxidant and severity of Perinatal Asphyxia.

Oxidants and antioxidants	Babies with severe asphyxia *n*=23	Babies with mild-moderate asphyxia *n*=61	Mann- Whitney U test	*p*-value
Oxidants	Median (IQR)	Median (IQR)		
TOS (ng/dL)	0.4 (0.2-0.6)	0.4 (0.2-5.8)	673.0	0.775
Malondialdehyde (ng/dL)	4.5 (2.4-8.7)	2.9 (1.8-7.0)	571.0	0.191
Hydrogen peroxide (ng/dL)	4.0 (2.5-5.4)	3.7 (2.7 -5.7)	701.0	0.996
Nitric oxide (ng/dL)	7.7 (4.4-9.7)	7.1 (4.1-9.2)	670.0	0.752
Antioxidants	Median (IQR)	Median (IQR)		
TAS (ng/dL)	113.5 (9.1 - 138.8)	114.9 (9.6 - 13.4)	695.0	0.948
Total Carotenoids (ng/dL)	3.7 (0.4-7.2)	3.9 (1.8-7.7)	604.0	0.328
Total Flavonoids (ng/dL)	4.1(2.5-8.9)	5.6 (0.1-8.6)	689.0	0.900
Vitamin A (µg/dL)	14.5 (7.9-17.1)	15.0 (10.8-22.2)	580.0	0.223
Vitamin C (mg/dL)	3.1 (2.3-10.5)	2.8 (1.6-10.7)	684.0	0.861
Vitamin E (ng/dL)	2.7 (1.1-3.3)	2.4 (1.4-7.2)	652.0	0.620
Glutathione (ng/dL)	6.5 (3.9-14.4)	9.4 (5.9-13.9)	613.0	0.375
Enzymatic antioxidants				
Superoxide dismutase (ng/dL)	7.9 (3.2-12.0)	7.0 (4.2-9.9)	681.0	0.837
Glutathione peroxidase (ng/dL)	3.5 (2.8-7.5)	3.8 (2.6-6.4)	678.0	0.814
Glutathione transferase (ng/dL)	5.6 (4.2-9.5)	4.4 (2.8-10.5)	552.0	0.134
Catalase (ng/dL)	3.1 (1.2-5.4)	2.1 (1.3-3.3)	526.0	0.078

IQR interquartile range; TOS total oxidant status; TAS total antioxidant status.

Table 4: Relationship between Cord Blood oxidants and antioxidant and Hypoxic-Ischaemic Encephalopathy.

Oxidants and antioxidants	Babies with HIE *n*=66	Babies without HIE *n*=18	Mann- Whitney U test	*p*-value
Oxidants	Median (IQR)	Median (IQR)		
TOS (ng/dL)	0.5 (0.2-6.0)	0.4 (0.02-0.5)	475.0	0.195
Malondialdehyde (ng/dL)	3.1 (2.1 -7.0)	2.7 (1.5-7.5)	525.0	0.452
Hydrogen peroxide (ng/dL)	4.0 (2.7-5.5)	3.6 (1.7-6.6)	550.0	0.631
Nitric oxide (ng/dL)	7.2 (4.3-9.5)	7.2 (0.1-8.9)	494.0	0.276
Antioxidants	Median (IQR)	Median (IQR)		
TAS (ng/dL)			481.0	0.218
Total Carotenoids (ng/dL)	3.81 (1.40-7.23)	5.85 (1.27-9.30)	496.0	0.285
Total Flavonoids (ng/dL)	5.30 (2.39-8.83)	6.63 (0.08-9.33)	564.0	0.744
Vitamin A (µg/dL)	15.1 (10.7-19.4)	14.4 (7.8-23.3)	582.0	0.896
Vitamin C (mg/dL)	2.92 (1.68-10.89)	2.51 (1.20-10.37)	535.0	0.520
Vitamin E (ng/dL)	2.75 (1.42-6.95)	2.12 (0.03-2.99)	437.0	0.087
Glutathione (ng/dL)	8.99 (5.25-14.21)	10.67 (4.82-15.01)	562.0	0.727
Enzymatic antioxidants				
Superoxide dismutase (ng/dL)	7.29 (3.95-9.28)	7.99 (4.55-63.52)	534.0	0.513
Glutathione peroxidase (ng/dL)	3.54 (2.70-6.18)	4.48 (2.48-23.22)	545.0	0.593
Glutathione transferase (ng/dL)	4.57 (3.17-9.15)	5.77 (3.54-12.33)	480.0	0.214
Catalase (ng/dL)	2.49 (1.63-5.01)	2.19 (0.16-3.81)	494.0	0.276

IQR interquartile range; TOS total oxidant status; TAS total antioxidant status.

In the present study, another oxidant Malondialdehyde (MDA) which is an end-product of lipid peroxidation was not significantly higher in cases than in controls. This contrasts with reports by Mondal *et al.*,[15] Kumar *et al.*,[19] and Saini *et al.*,[20] all from India who reported significantly higher serum MDA in babies with PA than in controls. The possible reasons for this difference may be related to the definition of perinatal asphyxia used in the aforementioned studies compared to this present study. For instance, Mondal *et al.*,[15] defined PA using arterial blood gas parameters with cord blood pH < 7.1, Apgar score <6 at 5-min with or without meconium-stained liquor and clinical evidence of HIE. Kumar *et al.*,[19] recruited only babies with severe asphyxia (defined as need for positive pressure ventilation for >1 min after birth or Apgar score ≤3 at 1 min) and Saini *et al.*,[20] recruited babies with clinical evidence of HIE and/or need for assisted ventilation for >3 min after birth. These definitions were more stringent than Apgar score <7 at 5 min used in the present study, hence the aforementioned studies recruited more severe cases compared to the present study.

Concerning antioxidants, there was significantly lower non-enzymatic antioxidants (like TAS, total carotenoids, flavonoids and vitamin A) and enzymatic antioxidants (SOD, glutathione, glutathione peroxidase and glutathione transferase) in babies with PA than in controls. These findings were in keeping with reports by Sonowal *et al.*,[21] from India who also reported lower enzymatic and non-enzymatic antioxidants in 37 term babies with PA than in controls. In contrast to the findings in this study, Kumar *et al.*,[19] reported higher cord blood enzymatic antioxidants (glutathione peroxidase, SOD) in babies with PA than in controls. The difference in the reports by Kumar *et al.*,[19] compared to the present study may be related to the fact that Kumar *et al.*,[19] studied very few controls (8 non-asphyxiated babies), hence the study may not be adequately powered to detect a valid result. Also, Kumar *et al.*,[19] used ELISA kit to assay for the enzymatic antioxidants reported in their study in contrast to the more accurate and reliable HPLC method used in the present study.[18]

Antioxidants-both enzymatic and non-enzymatic are produced to mop up excess free radicals and oxidants generated during the process of PA in order to reduce oxidative stress and maintain oxidative balance.[22,23] For instance, SOD and glutathione peroxidase detoxify superoxide anion and hydrogen peroxide respectively thereby eliminating these free radicals.[22-24] Non-enzymatic antioxidants like Vitamins A, C and E act by terminating free radical chain reactions thereby converting them to harmless metabolites.[22-24] Hence with increased generation of

free radicals and oxidants, more antioxidants are used to mop up these free radicals. This may explain the lower levels of most antioxidants among babies with PA observed in this study and similar ones.[14, 21] Also of note in the present study, is that cord blood vitamin E and catalase were higher in babies with PA than controls. These findings agreed with the study of Kumar *et al.*,[19] who reported higher cord blood catalase among 50 babies with PA than in controls. Elevated catalase may be in response to elevated hydrogen peroxide observed in the babies, since catalase is needed to metabolise hydrogen peroxide to water and oxygen.[6,22-24] Sonowal *et al.*,[21] reported lower cord blood vitamin E among 37 babies with PA in contrast to the finding in the present study. These differences in the findings may be explained by relatively low sample size studied by Sonowal *et al.*, which may affect the power of their study to detect valid results.

In this study, the cord blood oxidants and antioxidants of babies with PA were not related to the severity of the disease and the presence of HIE. There is paucity of study that looked at oxidants and antioxidants in babies with various severity of asphyxia using Apgar score alone as a yardstick. The non-significant difference in the cord blood oxidants and antioxidants observed among babies with severe compared to mild/moderate depression in this study may be because the babies recruited for this study were all inborn whose mothers received prompt in-hospital interventions like bed rest, oxygen therapy, hydration, etc., before delivery to ameliorate the effect of perinatal asphyxia and hypoxia in the babies in-utero.[25] Hence the impact of hypoxia and ischaemia on the babies are limited.

This study recruited babies delivered at a tertiary health facility in a resource-poor country and Apgar score were assessed by trained personnels, this coupled with robust sample size and the use of quality-assured HPLC method to assay for cord blood oxidants and antioxidants are strength of this study. We however appreciate that this study was limited in its ability to exactly identify babies with PA using arterial blood gas parameters (blood pH, partial pressures of CO_2 and O_2). Although blood gas analysis is an objective means of making diagnosis of PA, it is not without certain limitations such as cost, dearth of blood gas analysers in most centres in developing countries as well as variation of blood gas parameters with some maternal and foetal factors.[25,26]

CONCLUSION

In conclusion, term babies with PA showed evidence of increased oxidative stress with significantly higher cord blood oxidants and lower antioxidants. Prompt interventions to neutralise the oxidants through appropriate antioxidants may hold prospect as adjunct therapy in the management of babies with PA.

ACKNOWLEDGEMENT

The authors acknowledge with thanks the roles of clinicians and nurses working at the Labour ward and neonatal units of the WGH, Ilesa during the study period for their roles in the management of the study participants and Mr. Basil of IITA, Ibadan Nigeria for the analysis of the oxidants and antioxidants.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

TOS: Total Oxidant Status; **HPLC:** High Performance Liquid Chromatography; **TAS:** Total Antioxidant Status; **PA:** Perinatal Asphyxia; **HIE:** Hypoxic-Ischaemic Encephalopathy; **SOD:** Superoxide Dismutase; **CAT:** Catalase.

SUMMARY

Perinatal asphyxia is a leading cause of neonatal ill-health and death particularly in developing countries. It is often related to oxidative stress with elaboration of free radicals, oxidants and consequent increased demand for antioxidants. This study highlights significant higher oxidants (and lower antioxidants) in the cord blood samples of term babies with PA compared to those without PA. These oxidants and antioxidants were however not related to severity of PA. Cord blood oxidants and antioxidants therefore may be good biomarkers of PA and may hold prospects for diagnostic and therapeutic relevance in babies with PA.

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Cite this article: Kuti DK, Ogundele T, Kuti BP, Oyelami OA, Adejuyigbe EA. Cord Blood Oxidant and Antioxidant Profiles in Term Nigerian Newborns with Perinatal Asphyxia. Free Radicals and Antioxidants. 2025;15(1):35-41.