USEFULNESS OF SERUM LIPID PEROXIDE AS A DIAGNOSTIC TEST FOR HYPOXIC ISCHEMIC ENCEPHALOPATHY IN FULL TERM NEONATE

By

El Sayed Mohamed El Nagar*, Hasan Ali Hasan*, Kamel Soliman Hammad, Ahmed Ibrahim Semary***

Pediatric* & Clinical Pathology** Departments, Faculty of Medicine, Al-Azhar University

ABSTRACT

Backgrounds: *Hypoxic-ischemic encephalopathy (HIE) significantly increased mortality and morbidity.*

Objectives: *The aim of this study is to evaluate the usefulness of serum lipid peroxide (LPO) as an early prediction of HIE in full-term neonates.*

Methods: *This case control study was conducted on group 1 which included 30 asphyxiated full term infants delivered at Dar Al Shefaa hospital and Sayed Galal university hospital during a period of ten months from November 2018 to August 2019. These cases were compared to group 2 which included 30 age matched apparently healthy term neonates as a control group. Both groups of patients and control were subjected to:*

1. *Full maternal history with special emphasis on medical and obstetric data at delivery, including the mode of delivery, Apgar score at 1 and 5 min and resuscitation data*
2. *Assessment of gestational age, anthropometric measurements (head circumference, weight and length), vital signs and full systemic examination.*
3. *Neurological examination with assessment of severity of hypoxic ischemic encephalopathy using Sarnat and Sarnat staging (1976).*

Results: *There was a highly significant difference in serum lipid peroxide between cases and control groups (P value < 0.001). Lipid peroxide was statistically highly significant in cases delivered by caesarean section rather than by vaginal delivery (p=0.017). There was a significant negative correlation between lipid peroxide level and 5th minute Apgar score & blood pH and a significant positive correlation with base excess, sarnat stage and mortality.*

Conclusion: *LPO level is a useful marker for early detection of HIE in full-term neonate, the grade of hypoxia and the outcome prediction.*

Key words: *Lipid peroxide, Full term neonate, Neonatal encephalopathy.*

INTRODUCTION

Neonatal encephalopathy is a clinical phenotype characterized by a syndrome of disturbed neurologic function in the earliest days of life in term infants, manifested by difficulty in initiating and maintaining respiration, depression of tone and reflexes, subnormal level of consciousness, and often by seizure **(Ramaswamy et al., 2009)**.

The pathogenesis of hypoxic ischemic brain damage is highly complex **(Alonso-Spilsbury et al., 2005)**. Lipid peroxide (LPO) can be a method used for early identification of hypoxic ischemic encephalopathy by measuring the degradation of free radicals that react with unsaturated fatty acids in the lipid membrane, causing lipid peroxidation that produces membrane damage and necrosis in neuronal cells **(Barrera-de León et al., 2013)**.

The aim of this study is to evaluate the value of serum lipid peroxide (LPO) in diagnosis of hypoxic ischemic encephalopathy (HIE) in full-term neonates.

PATIENTS AND MATERIALS

This case control study was conducted on group 1 which included 30 asphyxiated full term infants delivered or admitted shortly after birth to the neonatal intensive care unit (NICU) at Dar Al Shefaa and Sayed Galal hospitals during a period of ten months from November 2018 to August 2019. Group 1 was then compared to group 2 which included 30 age matched apparently healthy term neonates as a control group with no obstetrical problems.

Inclusion criteria:

- Full term neonates (gestational age ≥37 weeks) with perinatal compromise that can be evident by at least two of the following according to American Academy of Pediatrics;
  1. Apgar score ≤ 3 at 1 minute or ≤ 6 at 5 min.
  2. Umbilical cord arterial PH ≤ 7.2 with base deficit ≥10 mmol.
  3. Presence of post natal clinical complications attributed to hypoxia and/or ischemia such as shock, oliguria and convulsion.
- Age matched control group of apparently healthy newborn.

Exclusion Criteria:

Infants will be excluded from the study if they meet any of the following conditions as these factors decrease serum lipid peroxide level:

1. Major congenital anomalies.
2. Inborn error of metabolism.
3. Indirect bilirubin levels >15mg /dl.
4. Neonates whose mothers had history of addiction.
5. Neonates whose mothers had received general anesthesia during birth process.

All cases were subjected to:

1. Full maternal history with special emphasis on medical and obstetric data at delivery, including the mode of delivery, Apgar score at 1 and 5 min and resuscitation data.
2. Assessment of gestational age, anthropometric measurements (head circumference, weight and length), vital signs and full systemic examination.
3. Neurological examination with assessment of severity of hypoxic ischemic encephalopathy using Sarnat and Sarnat staging (1976) and physical criteria for maturity by Expanded New Ballard Score (NBS) **(Ballard et al., 1991)**.

Cord Blood samples were collected as early as possible immediately after birth and analyzed for arterial blood gases. A 1-ml peripheral venous blood sample was taken from all neonates included in the study at 4h after birth for LPO - Assay from the Cayman Chemical Company1 was used for quantitative LPO determination.

Statistical Analysis:

All statistical calculations were done using computer program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 15 for Microsoft Windows. Numerical data were statistically described in terms of mean ± standard deviation (± SD), median and range, or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between the study groups was done using Student t test for independent samples. For comparing categorical data, Chi square (χ^2) test was performed. Exact test was used instead when the expected frequency is less than 5. Correlation between various variables was done using Pearson moment correlation equation for linear relation in normally distributed variables and Spearman rank correlation equation for non-normal variables. Accuracy was represented using the terms sensitivity, and specificity. Receiver operator characteristic (ROC) analysis was used to determine the optimum cut off value for the studied diagnostic markers. P values less than 0.05

was considered statistically significant.

Ethical consideration:

- Written Parent consent for the study was obtained before the study.
- Approval of the local ethical committee in the pediatrics department, college and university were obtained before the study.
- The authors' declared no potential conflict of interest with respect to the research & publication of this article.
- All the data of the patient & results of the study are confidential & the patient has the right to keep it.
- The authors received no financial support for the research & publications of the article.

RESULTS

The demographic data of either asphyxiated or control group was recorded in table (1)

Table (1): Demographic data of studied population

Variable	Asphyxiated group (n=30)	Control group (n=30)
GA (WK)	38.42 ± 1.11	37.98 ± 0.86
Anthropometric measures: WT (KG) L (CM) HC (CM)	 3.33 ± 0.48 49.69 ± 1.49 34.96 ± 0.78	 3.07 ± 0.33 49.50 ± 1.23 34.96 ± 1.66
Duration of hospital stay (day) according to sarnat stage: Grade I (n=12) Grade II (n=10) Grade III (n=8)	 8.11 ± 3.06 7.50 ± 8.59 17.53 ± 8.49	
Gender (M/F)	19(63.4%)/ 11(36.6%)	17(56.6%)/13(43.4%)
Mode of delivery (NVD/CS)	16(53.4%)/ 14(46.6%)	16(53.3%)/ 14 (46.7%)
Outcome (discharged/died)	26(86.7%)/ 4 (grade III) (13.3%)	30(100.0%)/ 0(0.0 %)

Table (2): Clinical picture of asphyxiated group

	N	(percent) %
Abnormal consciousness:		
Hyper alert	12	40%
Lethargic	10	33.3%
Comatosed	8	26.7%
Convulsion	13	43.3%
Apnea	17	56.6%
Shock	13	43.3%
Oliguria	2	6.6%

Table (2) shows that the most frequent manifestations of perinatal asphyxia were Apnea, Convulsion and Shock.

Table (3): HIE Severity according to Sarnat & Sarnat (1976).

Variable	HIE Severity (n = 30 cases)		
	Grade I	Grade II	Grade III
Cases (n, %)	Cases (12, 40%)	Cases (10, 33.3%)	Cases (8, 26.7%)

In Table (3) patients were divided according to Sarnat and Sarnat scoring system into three stages 12 cases (40%) were classified as grade I (HIE), 10 cases (33.3%) as grade II (HIE) and 8 cases (26.7%) as grade III (HIE).

Table (4): Comparison between case and control group as regarding lipid peroxide level

Variable	Case group	Control group	P value
	Mean ± SD		
Lipid peroxide (nmol)	6.9 ± 3.01	1.78 ± 1.09	<0.001

Table (4) shows a highly significant difference between cases and control groups in relation to serum lipid peroxide level.

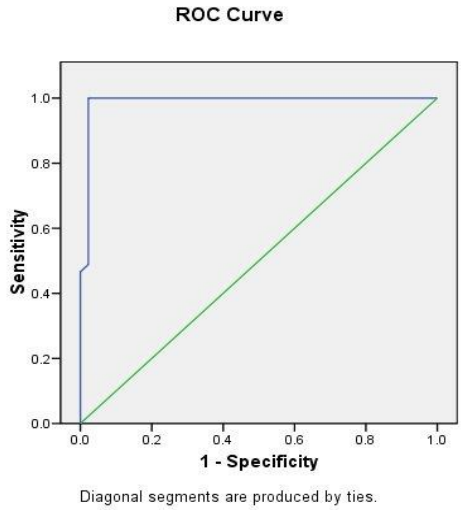
Table (5): To assess cut-off of serum lipid peroxide level in detecting hypoxia.

	AUC	Std error	Asymptomatic 95% confidence		Sensitivity	Specificity	P value
			Upper bound	Lower bound			
Lipid peroxide (nmol)	0.98	0.012	0.965	1.011	100 %	98%	<0.001

P-value <0.001 Highly Significant

In Table (5), the best cut-off of lipid peroxide to detect hypoxia was > 3.2 nmol with a sensitivity of 100 %, specificity 98%, positive predictive value 97.83 %, negative predictive value 100 % and diagnostic accuracy of 98.89%.

Figure (1): Receiver Operating Characteristic (ROC) curve to define the best cut-off of lipid peroxide to detect hypoxia

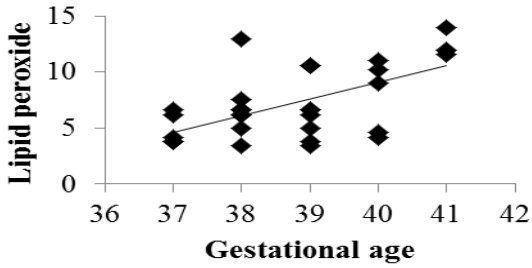


In figure (1), lipid peroxide was reliable to predict hypoxia as P <0.001 and AUC (area under the curve) was 98%.

Table (6): Correlation between Lipid peroxide and gestational age in study cases.

Lipid peroxide (nmol)	Mean ± SD	P value	sig
GA (wks)	7.353 ± 3.114	0.034	HS

Figure (2): Correlation between Lipid peroxide and gestational age among study cases

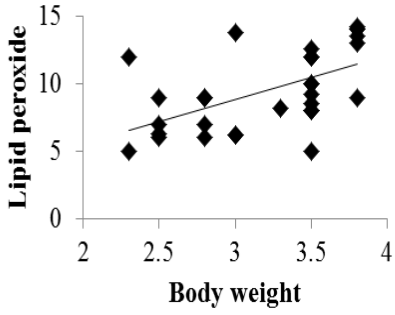


According to Table (6) and Figure (2), there was a statistically significant positive correlation between lipid peroxide and gestational age (p value=0.034).

Table (7): Correlation between Lipid peroxide and body weight among study cases

Lipid peroxide (nmol)	Mean ± SD	P value	sig
BW(kg)	9.451 ± 2.86	0.045	HS

Figure (3): Correlation between Lipid peroxide and body weight among study cases



According to Table (7) and Figure (3), there was a statistically significant positive correlation between lipid peroxide and body weight (p value=0.045).

Table (8): Relation between Lipid peroxide in neonates and mode of delivery

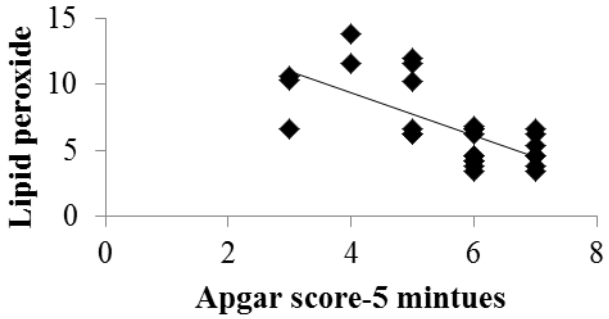
Lipid peroxide (nmol)	Mean	SD	P value
CS (n= 16)	7.991	±2.915	0.017
NVD (n= 14)	5.887	±2.785	

Also in relation to mode of delivery in Table (8), there was statistically significant higher of lipid peroxide in cases with CS (7.991±2.915) than normal vaginal delivery (5.887±2,785) as p value =0.017

Table (9): Correlation between Lipid peroxide and Apgar score in cases

Lipid peroxide (nmol)	R	P value	Sig
Apgar score at 5th minute	-0.532	<0.001	HS

Figure (4): Correlation between Apgar at 5 th min and lipid peroxide among study cases

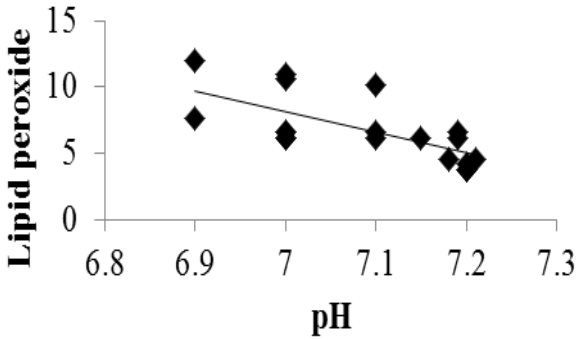


As seen in Table (9) and figure (4), there was a significant negative correlation between lipid peroxide level in studied neonates and Apgar score at 5th minute (R = - 0.532, P value <0.001), which means increased lipid peroxide with decreased Apgar score at the 5th minute.

Table (10): Correlation between Lipid peroxide and blood pH in cases

Lipid peroxide (nmol)	R	P value	Sig
Blood pH	-0.664	<0.001	HS

Figure (5): Correlation between pH and lipid peroxide among study cases

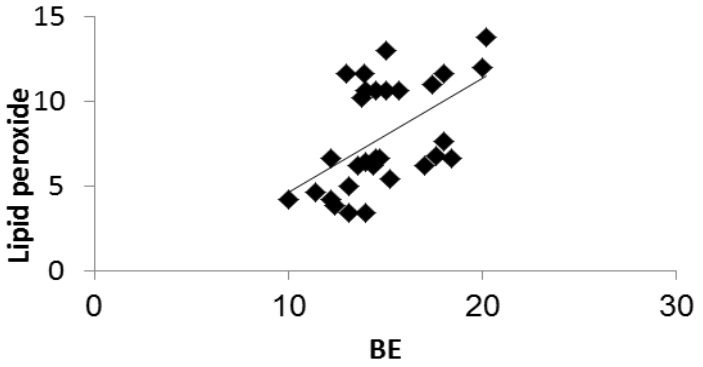


As seen in Table (10) and Figure (5), there was a significant negative correlation between lipid peroxide level in studied neonate and pH of arterial blood gas as (R = - 0.664, P value <0.001), which means increased lipid peroxide level with each decrease in arterial pH.

Table (11): Correlation between lipid peroxide in neonates and BE among study cases

Lipid peroxide (nmol)	R	P value	Sig
Base Excess (BE)	0.635	<0.001	HS

Figure (6): Correlation between lipid peroxide in neonates and BE in cases

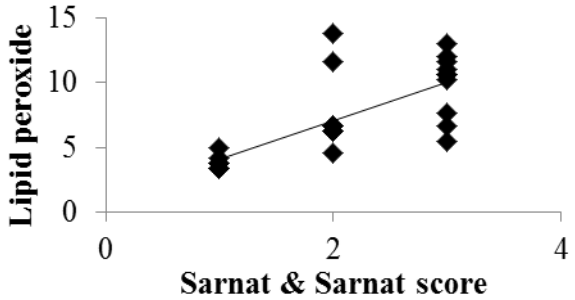


In Table (11) and Figure (6), there was a statistically significant positive correlation between lipid peroxide in studied neonates and base excess which means increased lipid peroxide level with increased base excess.

Table (12): Correlation between Lipid peroxide and sarnat stage in cases

Lipid peroxide (nmol)	R	P value	Sig
Sarrnat & Sarrnat stage	0.943	<0.001	HS

Figure (7): Correlation between Sarnat & Sarnat score and lipid peroxide among study cases

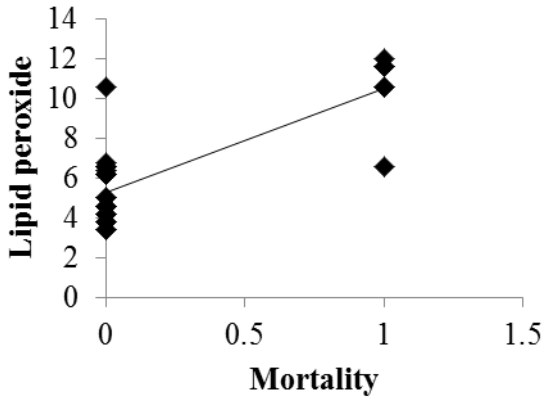


According to Table (12) and Figure (7), there was a statistically significant positive correlation between lipid peroxide in studied neonates and sarnat stage as a higher lipid peroxide level with increasing degree of sarnat.

Table (13): Correlation between Lipid peroxide in neonates and mortality among cases

Lipid peroxide (nmol)	R	P value	Sig
Mortality	0.445	0.002	HS

Figure (8): Correlation between Lipid peroxide in neonates and mortality in cases



In Table (13) and Figure (8), there was a statistically significant positive correlation between lipid peroxide in studied neonates and mortality. There was a higher of lipid peroxide level with died neonates (R = 0.943, P value=0.002).

DISCUSSION

We found no gender difference regarding hypoxic insult; this was in accordance with other authors **(Kirimi et al., 2010**; **Tekgul et al., 2004)**. However, others **(Sitthivuddhi Futrakul, Praisuwanna, & Thaitumyanon, 2006)** found a significant relation between HIE and male gender in their study as a risk factor of HIE. They supposed that the male gender is highly vulnerable to any threatening factors such as increasing the risk of sepsis, bronchial hyperresponsiveness, atopy, and mortality of RDS etc.

Also, regarding the mode of delivery, we found a significant difference between both groups that serum Lipid peroxide is higher in terms delivered by caesarean section than by vaginal delivery; this was in accordance with other authors **(Kaye, 2003**; **White, Zhang, Helvey, & Omojola, 2013)** and **(Kirimi et al., 2010**; **Tekgul et al., 2004**; **Uzodimma et al., 2013)**. However, another author Butt,

Farooqui, and Khan, found that HIE developed in 76.5% of vaginal delivered cases, while caesarean section occurred in 23.5% of cases **(Butt, Farooqui, & Khan, 2008)**. The variation in different studies concerning mode of delivery may be explained by the fact that neonatal encephalopathy may originate early in the antepartum period in some cases of HIE.

The significant difference between both groups regarding gestational age in our study was in accordance with **(Sitthivuddhi Futrakul et al., 2006)**, et al. Stated that gestational age particularly post-term gestation was significantly associated with HIE, this might be related to the uteroplacental insufficiency; but another study found no statistically significant difference between HIE and control groups regarding gestational age **(Vasiljević, Maglajlić-Djukić, Gojnić, & Stanković, 2012)**.

The current study showed statistically significant difference between HIE and control groups regarding birth weight, in addition to the positive correlation between birth weight and risk of HIE which may be explained by increased risk of obstructed labour with increase birth weight; however, Airede, reported that infants with intrauterine growth retardation play a significant role in occurrence of severe asphyxia due to placental insufficiency, this conflict may be due to deprivation of our sample from cases with intrauterine growth retardation **(Airede, 1991)**. However, Ghotbi and Najibi, found no statistically significant difference between HIE and control groups regarding birth weight **(Ghotbi & Najibi, 2010)**.

In the present study, 6 cases (13.3%) died in the first week of life due to multiorgan dysfunction and all of them were classified as grade III hypoxic ischemic encephalopathy. One study showed slightly higher mortalities where 20% of their cases died with grade III HIE **(Singh, Kumar, Majumdar, & Narang, 2004)**.

Also in the present study, the median of Apgar score at 1 and 5 minutes was 2 and 6 respectively and it was significantly lower than the control which had normal Apgar score (6-9 at 1 and 5 minutes). Therefore, cases show statistical significant low Apgar score than control, this in agreement with Karlsson, et al. **(Karlsson et al., 2010)**.

In the present study, serum LPO level showed a sensitivity of 100 % and specificity of 98% with

a diagnostic accuracy of 98.89% which is superior to other markers such as S100B which showed a sensitivity of 71%, and the specificity of 90% **(Nagdyman, Kömen, Ko, Müller, & Obladen, 2001)** and superior to another study by Gazzolo, et al. they demonstrated that S100B concentration had a sensitivity of 91.3% and a specificity of 94.6% for predicting the development of HIE **(Gazzolo et al., 2009)**.

Also, LPO is superior to other markers such as nRBCs (nucleated red blood cells) which showed a sensitivity of 83.4% and specificity of 73.5% **(Boskabadi, Maamouri, Sadeghian, Ghayour-Mobarhan, & Heidarzade, 2010)**.

High LPO sensitivity and specificity value were observed in the present study for identifying HIE in full-term neonates with low Apgar score or the need for cardiopulmonary resuscitation at birth. These data are important for supporting early diagnosis and being able to opportunely initiate therapeutic measures for preventing mortality and long-term neurological consequences.

Schmidt, Grune, Müller, Siems, and Wauer, studied LPO product level in blood plasma from the umbilical cord artery at birth was measured in full-term neonates. LPO elevation was twice as high in the full-term neonate group with acidosis and asphyxia when compared with the healthy full term neonate control group and those results were statistically significant. However, there was no correlation with HIE clinical data. These results are similar to those of the present study in which LPO elevation was higher in the full-term neonates with Apgar score ≤ 6 or need for cardiopulmonary resuscitation **(Schmidt, Grune, Müller, Siems, & Wauer, 1996)**.

In addition, the present study compared LPO results with the different clinical stages of the sarrnat clinical classification for HIE diagnosis and showed that there was positive correlation between HIE stage and LPO level, this in concur with Barrera-de León, et al. **(Barrera-de León et al., 2013)**.

In the present study, serum LPO sample was taken 4 h after birth because some authors **(Gonzalez & Ferriero, 2008)** in their experimental designs have established 6 h as the optimum time for therapy such as hypothermia. For this reason, serum LPO level was studied before initiating any HIE treatment.

CONCLUSION

It can be concluded that determining LPO level could be a useful marker for early HIE diagnosis in full-term neonates and also in determining the grade of hypoxia and hence predicting the outcome.

RECOMMENDATION

1. Prevention of hypoxic ischemic encephalopathy in utero by proper antepartum and intrapartum surveillance of all deliveries especially high risk groups.
2. Usage of early predictors as LPO for diagnosis of hypoxic encephalopathy to start treatment as early as possible.
3. Appling new approaches in treatment specially the neuroprotective techniques as hypothermia and other new modalities of treatment.
4. More studies with large samples are needed to detect the importance of LPO as a marker for predicting and determining the severity of perinatal asphyxia.
5. Long term follow up of the cases to study relation between LPO level and degree of neurodevelopmental disabilities.

REFERENCES

1. **Airede, A. I. (1991):** Birth asphyxia and hypoxic-ischaemic encephalopathy: incidence and severity. Annals of tropical paediatrics, 11(4), 331-335.
2. **Alonso-Spilsbury, M., Mota-Rojas, D., Villanueva-García, D., Martínez-Burnes, J., Orozco, H., Ramírez-Necoechea, R., . . . Trujillo, M. E. (2005):** Perinatal asphyxia pathophysiology in pig and human: A review. Animal reproduction science, 90(1), 1-30. doi:https://doi.org/10.1016/j.anireprosci.2005.01.007
3. **Ballard, J., Khoury, J., Wedig, K., Wang, L., Eilers-Walsman, B., & Lipp, R. (1991):** New Ballard Score, expanded to include extremely premature infants. The Journal of pediatrics, 119(3), 417-423.
4. **Barrera-de León, J., Cervantes-Munguía, R., Vásquez, C., Higareda-Almaraz, M., Bravo-Cuellar, A., & González-López, L. (2013):** Usefulness of serum lipid peroxide as a diagnostic test for hypoxic ischemic encephalopathy in the full-term neonate. Journal of Perinatology, 33(1), 15.
5. **Beken, S., Aydın, B., Dilli, D., Erol, S., Zenciroğlu, A., & Okumuş, N. (2014):** Can biochemical markers predict the severity of hypox-icischemic encephalopathy? Turkish Journal of Pediatrics, 56(1).
6. **Boskabadi, H., Maamouri, G., Sadeghian, M. H., Ghayour-Mobarhan, M., & Heidarzade, M. (2010):** Early diagnosis of perinatal

asphyxia by nucleated red blood cell count: a case-control study. Archives of Iranian medicine, 13(4), 275.

7. **Buonocore, G., Perrone, S., Longini, M., Terzuoli, L., & Bracci, R. (2000):** Total hydroperoxide and advanced oxidation protein products in preterm hypoxic babies. Pediatric research, 47(2), 221.

8. **Butt, T. K., Farooqui, R., & Khan, M. (2008):** Risk factors for hypoxic ischemic encephalopathy in children. J Coll Physicians Surg Pak, 18(7), 428-432.

9. **Gazzolo, D., Frigiola, A., Bashir, M., Iskander, I., Mufeed, H., Aboulgar, H., . . . Frulio, R. (2009):** Diagnostic accuracy of S100B urinary testing at birth in full-term asphyxiated newborns to predict neonatal death. PloS one, 4(2), e4298.

10. **Ghotbi, N., & Najibi, B. (2010):** Measurement of the urinary lactate/creatinine ratio for early diagnosis of the hypoxic–ischemic encephalopathy in newborns. Iranian journal of pediatrics, 20(1), 35.

11. **Gonzalez, F. F., & Ferriero, D. M. (2008): Therapeutics for neonatal brain injury. Pharmacology & therapeutics, 120(1), 43-53.**

12. **Karlsson, M., Wiberg-Itzel, E., Chakkarapani, E., Blennow, M., Winbladh, B., & Thoresen, M. (2010): Lactate dehydrogenase predicts hypoxic ischaemic** encephalopathy in newborn inf ants: a preliminary study. Acta Paediatrica, 99(8), 1139-1144.

13. **Kaye, D. (2003):** Antenatal and intrapartum risk factors for birth asphyxia among emergency obstetric referrals in Mulago Hospital, Kampala, Uganda. East african medical journal, 80(3), 140-143.

14. **Kirimi, E., Peker, E., Tuncer, O., Yapicioglu, H., Narli, N., & Satar, M. (2010):** Increased serum malondialdehyde level in neonates with hypoxic-ischaemic encephalopathy: prediction of disease severity. Journal of International Medical Research, 38(1), 220-226.

15. **Nagdyman, N., Kömen, W., Ko, H.-K., Müller, C., & Obladen, M. (2001):** Early biochemical indicators of hypoxic-ischemic encephalopathy after birth asphyxia. Pediatric research, 49(4), 502.

16. **Ramaswamy, V., Horton, J., Vandermeer, B., Buscemi, N., Miller, S., & Yager, J. (2009):** Systematic review of biomarkers of brain injury in term neonatal encephalopathy. Pediatric neurology, 40(3), 215-226.

17. **Sarnat, H. B., & Sarnat, M. S. (1976):** Neonatal encephalopathy following fetal distress: a clinical and electroencephalographic study. Archives of neurology, 33(10), 696-705.

18. **Schmidt, H., Grune, T., Müller, R., Siems, W. G., & Wauer, R. R. (1996):** Increased levels of lipid peroxidation products malondialdehyde and 4-hydroxynonenal after perinatal hypoxia. Pediatric research, 40(1), 15.

19. **Singh, D., Kumar, P., Majumdar,**

S., & Narang, A. (2004): Effect of phenobarbital on free radicals in neonates with hypoxic ischemic encephalopathy–a randomized controlled trial. Journal of perinatal medicine, 32(3), 278-281.

20. **Sitthivuddhi Futrakul, M., Praisuwanna, P., & Thaitumyanon, P. (2006):** Risk factors for hypoxic-ischemic encephalopathy in asphyxiated newborn infants. J Med Assoc Thai, 89(3), 322-328.
21. **Tekgul, H., Yalaz, M., Kutukculer, N., Ozbek, S., Kose, T., Akisu, M., . . . Gokben, S. (2004):** Value of biochemical markers for outcome in term infants with asphyxia. Pediatric neurology, 31(5), 326-332.
22. **Uzodimma, C., Okoromah, C., Ekure, E., Ezeaka, C., & Njokanma, F. (2013):** Correlation of cardiac troponin T level, clinical parameters and myocardial ischaemia in perinatal asphyxia. Nigerian Journal of Paediatrics, 40(2), 165-168.
23. **Vasiljević, B., Maglajlić-Djukić, S., Gojnić, M., & Stanković, S. (2012):** The role of oxidative stress in perinatal hypoxic-ischemic brain injury. Srp Arh Celok Lek, 140(1-2), 35-41.
24. **White, M. L., Zhang, Y., Helvey, J., & Omojola, M. (2013):** Anatomical patterns and correlated MRI findings of non-perinatal hypoxic–ischaemic encephalopathy. The British journal of radiology, 86(1021), 20120464-20120464.

فائدة استخدام دهون بيروكسيد كفحص تشخيصى لمرض نقص الأكسجين للمخ للإطفال حديثي الولادة كاملى النمو

****احمد ابراهيم سمرى*سيد محمد النجار*حسن على حسن*كامل سليمان حماد**

اقسام الأطفال* والباثولوجيا الإكلينيكة ,كلية الطب ,جامعة الإزهر**

مازال نقص الأكسجين والإعتلال الدماغي الناجم عن الاختناق ما قبل الولادة من أهم الإعاقات المزمنة كالإعاقات العصبية , التخلف العقلي , صعوبة التعلم والصرع.

يعتبر تشخيص الإعتلال الدماغي الناجم عن نقص الأكسجين والتنبؤ بنتائجه التي تحدث علي المدي البعيد من أهم التحديات.

ينجم مرض الإعتلال الدماغي عن أسباب أخري غير نقص الأكسجين وسرعة تحديد هذه الأسباب مهم لإتاحة آي فرصة للتدخل الناجح.

يعتمد تشخيص مرض الإعتلال الدماغي علي الترابط بين الدلالات البيولوجية المقترحة للإختناق الوليدي مع الأعراض المرضية للإعتلال الدماغي بعد الولادة.

الهدف من هذه الدراسة:

تقييم فائدة دهون البيروكسيد للإكتشاف المبكر لمرض الاعتلال الدماغي فى الأطفال حديثى الولادة. .

المرضى وطرق البحث:

دراسة المجموعة الأولى التي شملت ٣٠ رضيعًا مصابًا باسفيكسيا الإختناق أثناء الولادة أو تم إدخالهم عقب الولادة إلى وحدة العناية المركزة للإطفال حديثى الولادة في مستشفى دار الشفاء وسيد جلال الجامعى خلال فترة عشرة أشهر من نوفمبر ٢٠١٨ إلى أغسطس ٢٠١٩ وتمت مقارنتها بالمجموعة الثانية التي شملت ٣٠ حديثي الولادة كمجموعة ضابطة دون أي مشاكل صحية.

وقد خضع جميع المرضى فى هذه الدراسة الى:

- أخد تاريخ ألام بالكامل مع التركيز بشكل خاص على البيانات الطبية بما فى ذلك طريقة الولادة ودرجة أبجر فى الدقيقة ألاولى والخامسة وبيانات الإنعاش.
- تقييم عمر الحمل والقياسات البشرية (محيط الرأس والوزن والطول) والعلامات الحيوية.
- فحص الجهاز العصبي مع تقييم شدة اعتلال الدماغ الإقفاري بنقص الاكسجين باستخدام تدريج سرنات ١٩٧٦.

معايير الإستبعاد:

- العيوب الخلقية.
- أمراض التمثيل الغذائى.
- مستويات البيليروبين غير المباشرة أكبر من ١٥ ملليجرام / ديسيلتر.
- ألاطفال حديثى الولادة الذين تلقت أمهاتهم تخدير عام أثناء الولادة.

وقد أظهرت النتائج أنه لم يكن هناك فرق ذو دلالة احصائية بين المجموعتين التي ينطبق عليها معايير اسفكسيا الاختناق والمجموعة الضابطة بالنسبة لنوع الجنين بينما كان هناك فرق بين المجموعتين بالنسبة للعمر الجنيني والوزن وطريقة الولادة.

كما أظهرت النتائج أن هناك فرق ذو دلالة إحصائية بين المجموعة التي ينطبق عليها معايير اسفكسيا الاختناق والمجموعة الضابطة من حيث متوسط دهون البيروكسيد حيث أظهرت النتائج أنه أعلي في مجموعة اسفكسيا الاختناق عنه في المجموعة الضابطة.

وقد أظهرت النتائج أنه هناك فرق ذو دلالة إحصائية بين المجموعة الأولى والمجموعة الثانية من حيث مستوي دهون البيروكسيد حيث أنه أعلي في المجموعة الثانية.

نتائج الدراسات أثبتت قيمة دهون البيروكسيد كعامل مهم يمكنه المساعدة في التنبؤ المبكر لمرض الإعتلال الدماغي ودرجته .