Philadelphia: Elsevier Saunders; p. 1205- 1221.

5. Abitbol CL, DeFreitas MJ and Strauss J (2016): Assessment of kidney function in preterm infants: lifelong implications. **Pediatr Nephrol**; 31 (12) 2213- 2222.
6. American Academy of Pediatrics, Task Force on Neonatal Encephalopathy and Cerebral Palsy, American College of Obstetrics and Gynecology (2003): **Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology**. Edited by Washington, DC, American College of Obstetricians and Gynecologists.
7. Ballard JL, Khoury JC, Wedig K, et.al., (1991): New Ballard score: expanded to include extremely premature infants. **J Pediatr**; 119:417- 23.
8. Sarnat HB and Sarnat MS (1976): Neonatal encephalopathy following fetal distress. A clinical and electroencephalographic study. **Arch Neurol.** 1976;33:696- 705.
9. Schwartz GJ, Munoz A, Schneider MF, et.al. (2009): New equations to estimate GFR in children with CKD. **J Am Soc Nephrol**; 20: 629- 637.
10. Pitsawong C and Panichkul P (2012): Risk factors associated with birth asphyxia in Phramongkutklao Hospital. **Thai J. Obstet Gynaecol**; 19(4): 165- 171.
11. Solayman, M, Hoque, S, Akber, T, (2017): Prevalence of Perinatal Asphyxia with Evaluation of Associated Risk Factors in a Rural Tertiary Level Hospital. **KYAMC Journal**, 8(1), 43- 48.
12. Durkan AM and Alexander RT (2011): Acute Kidney Injury Post Neonatal Asphyxia. **J Pediatr**; 158 (2 suppl) e29- 33.
13. Singbartl K and Kellum JA (2012): AKI in the ICU: definition, epidemiology, risk stratification, and outcomes, **Kidney International**, vol. 81, no. 9, pp. 819- 825.
14. Zhang Y, Zhang B, Wang D, et.al. (2020): Evaluation of Novel Biomarkers for Early Diagnosis of Acute Kidney Injury in Asphyxiated Full- Term Newborns: A Case- Control Study. **Med PrincPract** 2020; 29:285- 291.
15. Bengur AR, Beekman RH, Rocchini AP, et.al. (2019): Acute hemodynamic effects of captopril in children with a congestive or restrictive cardiomyopathy. Circulation; 83:523- 527.
16. Killian LR (2016): Neutrophil gelatinase associated lipocalin (NGAL): a new marker of kidney disease. **Scand j Clin Lab Invest**. 68:89- 94.
17. Guo A, Stoesz SP, Buckley P, et.al., (2016): Neutrophil gelatinase associated lipocalin in normal and neoplastic human tissues. cell type- specific pattern of expression. **Histochem** J; 31: 433- 441.
18. Woday A, Muluneh A and St Denis C (2019): Birth asphyxia and its associated factors among newborns in public hospital, northeast Amhara, Ethiopia. **PLoS ONE** 14(12): e0226891.
19. Alaro D, Bashir A, Musoke R, et.al., (2014): Prevalence and outcomes of acute kidney injury in term neonates with perinatal asphyxia. **African Health Sciences**; Vol. 14 No. 3.
20. Medani S, Kheir AE and Mohamed MB (2014): Acute kidney injury in asphyxiated neonates admitted to a tertiary neonatal unit in Sudan. **Sudanese Journal of Pediatrics**. Vol 14, Issue No. 2.
21. Morris MG and Adoppa RD (2012): Nonoliguric and oliguric acute renal failure in asphyxiated term neonates. **Pediatr Nephrol**. 9(6): 718- 722.
22. Engle DC, Holliday MA, Dahwi A, et.al. (2011): Expressing glumerular filtreation rate in children. **Pediatr**; 5:5- 11.
23. Nymo J, Ma Q, Kelly C, et.al., (2012): NGAL is a novel early marker of acute kidney injury following transplantation. **Pediatr Nephrol**; 21: 856- 863.
24. Kumar D, Singh MV, Kumar N, et.al. (2020): Renal Functions in Relation to Severity of Perinatal Asphyxia in Term Neonates. **Indian Journal of Neonatal Medicine and Research**. Vol- 8(3): PO07- PO11.
25. Jayaswal A, Chaurasiya OS and Sethi RS (2016): Renal dysfunction in perinatal asphyxia& its correlation with apgar score and hypoxic ischemic encephalopathy stage. **People's Journal of Scientific Research**. 9(2): 56- 60.
26. Saini R, Sehra RN, Verma S, et.al. (2017): A study of renal functions in asphyxiated term newborns. Pediatric **Review**: **International Journal of Pediatric Research**, 4(6), 363- 369.
27. Girish G (2014): Acute Kidney Injury (AKI) in perinatal asphyxia. **Indian J. Pharm**. Biol. Res. 2(2): 60- 65.
28. Mohan K, Mishra PC and Singh DK (2013): Clinical Profile Of Birth Asphyxia In Newborn. **International Journal of Science& Technology**. Vol. 3, Issue 1.
29. Rafique A, Akram M, Khan RA, et.al. (2017): Birth Asphyxia- Clinical Experience and Immediate Outcome. **Journal of Rawalpindi Medical College (JRMC)**; 21(1): 20- 22.
30. Bruckmann EK and Velaphi S. (2015): Intrapartum asphyxia and hypoxic ischemic encephalopathy in a public hospital. **S Afr Med J**; 105(4):298-303.

In the current study, there was no statistically significant difference found between patients group and control group regarding gestational age, sex, birth weight and birth length and that was in agreement with that reported by[14] who reported no significant difference observed among the studied groups in terms of gender, gestational age, birth weight and birth length (p- value >0.05).

The Apgar score of control group at 5 and 10 minute showed a mean of 7.90± 0.76 at 5 minute and 9.23± 0.68 at 10 minutes, while in the patient group the mean of Apgar score at 5 and 10 minutes was 2.97± 1.04 and 4.43± 1.00, respectively. This result agreed with that of[15] who made a study on 38 newborns with hypoxic ischemic encephalopathy and showed a significantly low Apgar score at 1, 5, and 10 minutes (0.47, 3.15 and 6.1) in comparison to the control group which had Apgar score of 6.63, 9.0 and 9.56 at 1, 5 and 10 minutes, respectively. It was also agreed upon by,[16] that further emphasized that low Apgar score was associated with an increased risk for HIE.

The results of our study showed that the incidence of HIE grading of severity among the studied patients was mild in (55%) of cases, moderate in (25%) of cases, and severe in (20%) of cases in patient group. These result was concordant with (17) and (18) who reported that HIE stage I was the commonest in 60% and 77% respectively. While that was disconcordant to what was reported by (19) and (20) who reported that, the commonest HIE stage was stage II in 50% and 54.1% respectively.

Moreover on comparison of resuscitative measures needed, there was statistically significant relation found between HIE severity and neurologic sequelae and outcome of patients group.

It was found that 13 cases of mild HIE and 14 cases of moderate HIE were resuscitated, compared to all cases of severe HIE and that agreed with what said by (21) who found that those suffered from HIE are liable to complications such as intraventricular hemorrhage and neurologic sequelae and concordant to that reported by (22) who added that, those with severe HIE are at higher risk of intraventricular hemorrhage and death.

Our study showed that there was statistically significant increase in creatinine level at 24 hours, at 48 hours and reduction of GFR at 48 hours in patients group than control group with p- value= 0.029, <0.001 and <0.001 This result was concordant with (23) and was disconcordant with (14) who found no difference in serum creatinine levels 24 hours after birth between control group and neonates with severe and moderate asphyxia with p- value >0.05.

In the present study there was significant increase in serum creatinine levels 24 and 48 hours after birth while serum urea levels were statistically insignificant as HIE staging of neonates progressed from HIE I to HIE stage III and this results is concordant to that reported by (24) found significant increase in serum creatinine on day 1 and 3, while serum urea levels were statistically significant on day 1 and insignificant on day 3 with different stages of HIE and our results were also similar to the study by (25) regarding serum creatinine that increase with increasing HIE severity but discordant regarding serum urea level that increased in their study.

Urine output assessment in our study was decreased on day 1 as the HIE severity increased and the results on day 2 were statistically significant between different HIE stages and that was in agreement with that was reported by (24), but in our study the overall assessment of urine output was insignificant in patients with perinatal asphyxia as 41.5% of the cases had normal urine output and 55% had oliguria and that was disconcordant with (26) who reported that 76% of the cases had oliguria (transient/ persistent) and 24% had a normal urine output, Persistent oliguria was highest (24.44%) in severe asphyxia followed by moderate and mild asphyxia (11.43% and 5.00% respectively). Statistically, urine output was found significantly associated with grade of asphyxia (p value <0.05) in their study.

In the current study glomerular filtration rate (GFR) was significantly decreased in asphyxiated neonates as compared to healthy controls and that was concordant with that reported by (27), who added that GFR values correlated well with the severity of HIE.

In the present study neurological sequelae were statistically significant with different stages of HIE, convulsions were observed in (46.6%) of HIE stage II and 25% of HIE stage III. This was concordant with (28) who reported various neurological manifestations among which encephalopathy was the most common manifestation and found in all term neonates. Seizures were noted in 47% of the babies within the first week. These included tonic clonic and subtle seizures.

The outcome of our patients, 38 (63.3%) patients improved and discharged, 15 (25%) were under treatment and 7 (11.6%) died, This figure was similar to a study by (29) who reported forty eight (78.7) babies were discharged and 13 (21.3%) died. A significant association was found between hypoxic ischemic encephalopathy stage and outcome of the patients (p value- 0.000) that was concordant with that reported by (30) who reported increasing morbidity and mortality with increasing severity of HIE.

Conclusion:

Serum creatinine and GFR are reliable markers for renal function assessment in neonates with perinatal asphyxia and have to be assessed to reduce morbidity and mortality.

References:

1. Antonucci R, Porcella A, Pilloni MD (2014): Perinatal asphyxia in the term newborn. **J Pediatr Neonatal Individual Med**. 3:2.
2. Diaz- Rosello J, Gisore P, Niermeyer S, et.al. (2012): **Guidelines on Basic Newborn Resuscitation 2012**. Geneva: World Health Organization.
3. Bhatnagar A, Bairwa AL and Meena KC (2014): **Incidence of Acute Kidney Injury in Perinatal Asphyxia and its Correlation with Hypoxic Ischemic Encephalopathy (HIE) Staging**. Volume: 3; Issue: 3.
4. Askenazi D, Smith LB, Furth S, et.al. (2012): **Acute kidney injury and chronic kidney disease**. In: Avery's Diseases of the Newborn. 9 ed.

Table (2) Frequency distribution of some clinical parameters among different grades of HIE

		Mild HIE		Moderate HIE		Severe HIE		Test Value*	P- Value	Sig.
		No.	%	No.	%	No.	%			
Neurologic Sequelae	Hypotonia	0	0.0%	6	40.0%	0	0.0%	100.898	0.000	HS
	Irritable	21	63.6%	1	6.7%	0	0.0%			
	Convulsions	2	6.1%	5	33.3%	0	0.0%			
	Hypertonia	9	27.3%	1	6.7%	0	0.0%			
	Coma	0	0.0%	0	0.0%	7	58.3%			
	Flaccid	0	0.0%	0	0.0%	2	16.7%			
	Hypotonia+ Convulsions	0	0.0%	2	13.3%	0	0.0%			
	Convulsions+ Coma	0	0.0%	0	0.0%	3	25.0%			
	Irritable+ Hypertonia	1	3.0%	0	0.0%	0	0.0%			
Outcome	Improved and Discharged	32	97.0%	6	40.0%	0	0.0%	48.258	0.000	HS
	Under Treatment	1	3.0%	8	53.3%	6	50.0%			
	Died	0	0.0%	1	6.7%	6	50.0%			

P- value >0.05: Non significant (NS); P- value <0.05: Significant (S); P- value<0.01: highly significant (HS), *: Chi- Square Test

There was statistically significant relation between HIE severity and neurologic sequelae and outcome of patients group.

Table (3) Levels of creatinine at 24 hours and 48 hours and GFR at 48 hours

		Patients Group	Control Group	Test Value ǂ	P- Value	Sig.
		No. = 60	No. = 30			
1ST Creatinine at 24H (mg/dl)	Median (IQR)	0.5 (0.4- 0.7)	0.46 (0.46- 0.5)	- 2.177	0.029	S
	Range	0.3- 1.2	0.34- 0.58			
2ND Creatinine at 48H (mg/dl)	Median (IQR)	0.9 (0.6- 1.2)	0.46 (0.38- 0.49)	- 7.077	0.000	HS
	Range	0.4- 4	0.37- 0.6			
GFR at 48 Hour	Mean± SD	26.70 ± 12.30	49.39 ± 6.22	9.500	0.000	HS
	Range	5.2- 59	37.5- 58.3			

P- value >0.05: Non significant (NS); P- value <0.05: Significant (S); P- value<0.01: highly significant (HS), ‡: Mann Whitney Test

There was statistically significant increase in creatinine level and significant reduction of GFR in patients group than control group.

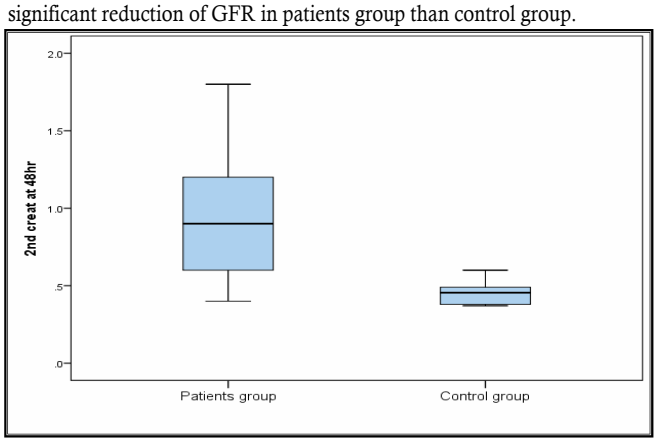

Figure (1) Median creatinine level at 48 hours in patients group and control group.

Table (4) Urine output and laboratory data among different grades of HIE

		Mild HIE	Moderate HIE	Severe HIE	Test Value	P- Value	Sig.
		No. = 33	No. = 15	No. = 12			
UOP at 24hr (Ml/kg/hour)	Median (IQR)	0.8 (0.7- 2.4)	0.7 (0.7- 2.3)	0.5 (0.45- 1.7)	6.572ǂ	0.037	S
	Range	0.4- 4.1	0.5- 3.1	0.3- 3.2			
UOP at 48hr (Ml/Kg/hour)	Median (IQR)	0.4 (0.4- 3.3)	0.4 (0.4- 3.3)	0.35 (0.2- 1.65)	6.072ǂ	0.048	S
	Range	0.3- 4.3	0.3- 4	0- 3.2			
UOP Assessment	Normal	16 (48.5%)	6 (40.0%)	3 (25.0%)	6.215*	0.132	NS
	Oliguric	17 (51.5%)	9 (60.0%)	7 (58.3%)			
	Anuric	0 (0.0%)	0 (0.0%)	2 (16.7%)			
1ST Creatinine at 24H (ml/kg/hour)	Median (IQR)	0.5 (0.4- 0.7)	0.5 (0.4- 0.8)	0.85 (0.5- 1.05)	10.670ǂ	0.005	HS
2ND Creatinine at 48H(ml/kg/hour)	Median (IQR)	0.8 (0.6- 1)	0.8 (0.6- 1.3)	2.2 (0.9- 3.3)	8.556ǂ	0.014	S
	Range	0.4- 2.5	0.5- 1.8	0.4- 4			
Urea(mg/dl)	Mean ± SD	34.30 ± 16.13	36.20 ± 13.80	47.00 ± 18.78	2.766•	0.071	NS
	Range	15- 63	17- 53	18- 70			

P-value >0.05: Non significant (NS); P- value <0.05: Significant (S); P- value< 0.01: highly significant (HS).

Kruskal Wallis Test: Urine output at 24 and 48 hours, serum creatinine level at 24 and 48 hours correlated significantly with severity of HIE. While non significant correlation was found between HIE severity and urea in the studied patients.

Disucsssion:

Perinatal asphyxia is a serious clinical problem worldwide and contributes greatly to neonatal mortality and morbidity.[10] It is a condition of the fetus or newborn due to failure of breathing leading to decrease oxygen perfusion to various organs. It happens in 2 to 10 cases per 1000 newborns that are born at term, and more of those that are born prematurely. It is one of the leading causes of neonatal deaths within first week of life.[11]

Asphyxia can lead to multi- organ dysfunction and a redistribution of cardiac output to maintain cerebral, cardiac, and adrenal perfusion while potentially compromising renal, gastrointestinal, and skin perfusion.[12]

Acute kidney injury (AKI) is defined by an acute and reversible increment in serum creatinine (SCr) levels associated or not with a reduction in urine output (UO) oliguria/ anuria.[13]

Introduction:

Perinatal asphyxia is a condition characterized by an impairment of exchange of the respiratory gases resulting in hypoxemia and hypercapnia, accompanied by metabolic acidosis.[1] It is defined by the World Health Organization as "the failure to initiate and sustain breathing at birth".[2]

Overall incidence of asphyxia is reported to vary from 1 to 1.5% at all various centers and is related to birth weight and gestational age of the baby. Perinatal asphyxia leads to multi- organ dysfunction. Virtually any organ can be affected. Many of these complications are potentially fatal. In term infants with asphyxia, renal, brain, cardiac and lung dysfunction may occur.[3]

As kidneys are very sensitive to hypoxia, renal insufficiency can occur within 24 h of a hypoxic insult, which if prolonged, may lead to irreversible injury. AKI is characterized by a sudden impairment of renal function that results in altered fluid, electrolyte, and acid□ base balance.[4]

Renal function assessment in neonates is of the utmost importance in predicting drug dosing, ensuring safe drug therapy, and detecting acute kidney injuries early. However, the extreme vulnerability, unique body composition, and rapid growth and maturation of young infants make this a rather challenging task. Premature infants present additional difficulties in assessing kidney function because nephrogenesis, which normally would continue until the 36th week of gestation in utero, is interrupted at preterm birth.[5] This study aimed to assess renal functions in neonates with perinatal asphyxia.

Patients and Methods:

This study is a case control study that was carried out in neonatal intensive care unit of Faculty of Medicine, Ain Shams University. The study included sixty neonates with perinatal asphyxia based on the criteria of American Academy of Pediatrics. Blood samples for serum creatinine, blood urea nitrogen levels were assessed. Thirty age/ sex matched healthy controls served as reference.

Ethical consideration and approval were obtained from the research ethics committee of the Faculty of Postgraduate Childhood studies and National Research Centre.

1. Inclusion criteria for the patient group: Neonates with criteria of perinatal asphyxia, based on the criteria of American Academy of Pediatrics: Profound metabolic or mixed acidemia (pH <7.0 in umbilical cord blood) , persistence of low Apgar scores less than 3 for more than 5 minutes, signs of neonatal neurologic dysfunction (e.g., seizures, encephalopathy, tone abnormalities) and evidence of multiple organ involvement (such as that of kidneys, lungs, liver, heart and intestine).[6]
2. Exclusion Criteria: Neonates with major congenital malformations, chromosomal abnormalities, suspected IEM, sepsis, maternal DM and pre- eclampsia and those of mothers who received nephrotoxic drugs.

All patients were subjected to the following:

1. Full history taking including: Antenatal maternal and obstetric history, natal and postnatal history, resuscitation measure and Apgar score.
2. Thorough Clinical Examination Including.
3. Anthropometric measures (weight, length and head circumference) .
4. Assessment of gestational age using the criteria of the new Ballard score.[7]
5. Systemic examination (CVS, chest and abdomen).
6. Neurological assessment and classification of hypoxic ischemic encephalopathy.[8]
7. Urine output assessment as oliguria or anuria, through daily assessment of urine output at age of 24 hours and 48 hours after birth.
8. Laboratory investigations: Blood samples were done including: serum creatinine and blood urea nitrogen.
9. Calculation of GFR using Schwartz formula.[9]

Statistical Analysis:

Data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) version 23. The quantitative data with parametric distribution were presented as mean, standard deviations and ranges while with non parametric distribution were presented as median with inter- quartile range (IQR). Also qualitative variables were presented as number and percentages.

Results:

Table (1): Descriptive data of patients and controls.

		Patients Group No. = 60	Control Group No. = 30	Test Value	P-Value	Sig.
Gestational Age (Weeks)	Mean ± SD	38.18 ± 1.23	38.53 ± 2.27	0.951•	0.344	NS
	Range	37- 40	37- 40			
Delivery Mode	Normal	18 (30.0%)	4 (13.3%)	3.008*	0.083	NS
	C. S.	42 (70.0%)	26 (86.7%)			
Sex	Female	22 (36.7%)	14 (46.7%)	0.833*	0.361	NS
	Male	38 (63.3%)	16 (53.3%)			
Resuscitation	Smooth	21 (35.0%)	30 (100.0%)	34.412	0.000	HS
	Extensive	39 (65.0%)	0 (0.0%)			
Apgar 5 Min	Mean ± SD	2.97 ± 1.04	7.90 ±0.76	23.048•	0.000	HS
	Range	1- 5	6- 9			
Apgar 10 Min	Mean± SD	4.43 ± 1.00	9.23 ±0.68	23.715•	0.000	HS
	Range	2- 6	8- 10			
Birth Weight (Kg)	Mean± SD	3.21 ±0.48	3.04 ±0.38	- 1.663•	0.100	NS
	Range	2.5- 4.2	2.5- 4.1			
Length (Cm)	Mean ± SD	48.13 ± 1.65	48.23 ± 1.30	0.289•	0.773	NS
	Range	45- 52	46- 51			

P- value >0.05: Non significant (NS); P- value <0.05: Significant (S); P- value<0.01: highly significant (HS), *: Chi- square test; •: Independent t- test; ‡: Mann Whitney test.

Apgar scores at 5 and 10 minutes and resuscitation measures showed highly significant statistical difference. Patients were categorized according to severity of HIE into mild 33 patients (55%), moderate 15 patients (25%) and severe HIE 12 patients (20%).

Assessment Of Acute Kidney Injury Among Asphyxiated Neonates

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Abstract

Background: Perinatal asphyxia is characterized by an impairment of respiratory gases exchange resulting in hypoxemia and hypercapnia, accompanied by metabolic acidosis. Early recognition of acute kidney injury is important in asphyxiated neonates as the kidneys are extremely sensitive to hypoxia which if prolonged may lead to irreversible cortical necrosis.

Aim: This study aimed to assess renal functions in neonates with perinatal asphyxia.

Design& setting: The study is a case control study that was conducted in neonatal intensive care unit of Faculty of Medicine, Ain Shams University and Wadi El- Nil hospital in the period from June 2014 to June 2015.

Patients& Methods: The study included 60 neonates with perinatal asphyxia based on the criteria of American Academy of Pediatrics. Thirty age/sex matched apparently healthy neonates as a control group. Blood samples for serum creatinine, blood urea levels were assessed, estimation of GFR and urine output assessment were performed.

Results: There was an increase in creatinine level at 24 hours and at 48 hours and a reduction of GFR at 48 hours in patients group than control group with highly significant p- values0.029, <0.001 and <0.001 respectively. HIE severity correlated significantly with urine output at 24 and 48 hours. HIE severity correlated significantly with and creatinine level at 24 and 48 hours, while no statistically significant relation was found between HIE severity and blood urea.

Conclusion: Serum creatinine and GFR are reliable markers for renal function assessment in neonates with perinatal asphyxia and have to be assessed to reduce morbidity and mortality.

Key Words: perinatal asphyxia; Hypoxic ischemic encephalopathy; Creatinine and Glomerular filtration rate.

تقييم إصابات الكلى الحادة فى الأطفال المصابين باختناق ما حول الولادة

الخلفية: يحدث الاختناق فى الفتره ماحول الولادة نتيجة ضعف تبادل غازات الجهاز التنفسى مما يؤدى إلى نقص الاكسجين وفرط ثانى أكسيد الكربون فى الدم، مصحوبا بزيادة الحموضه بالدم. يعد الكشف المبكر على إصابة الكلى الحادة أمرا مهما عند الأطفال حديثى الولادة المصابين بالاختناق فى فتره ماحول الولادة وذلك لحساسية الكلى لنقص الأكسجين الذى قد يؤدى استمراره لفترة طويلة إلى نخر قشرى لا رجعة فيه.

الاهداف: وقد كان الهدف من هذه الدراسة التى أجريت فى محضن كل من كلية الطب بجامعة عين شمس ومستشفى وادى النيل فى الفترة من يونيو ٢٠١٤ إلى يونيو ٢٠١٥ إلى تقييم وظائف الكلى عند الأطفال حديثى الولادة المصابين بالاختناق فى فترة ماحول الولادة. وقد تم عمل الدراسة على ٦٠ طفل مكتملى النمو تم تقسيمهم حسب إصابة المخ بنقص الأكسجين إلى بسيط ومتوسط وشديد، كما شملت الدراسة ٣٠ من حديثى الولادة الطبيعيين مكتملى النمو (كمجموعة ضابطة). تم اخضاع المرضى الى اخذ التاريخ المرضى الكامل والفحص الشامل وعمل الفحوصات والاختبارات المعملية مثل كمية البول عند عمر ٢٤ ساعه و٤٨ ساعه ونسبة اليوريا والكرياتينين بالدم.

النتائج: وكانت نتائج البحث كالتالي: كانت هناك زيادة فى مستوى الكرياتينين بعد ٢٤ ساعة و٤٨ ساعة وانخفاض معدل الترشيح الكلوى فى ٤٨ ساعة فى مجموعة المرضى مقارنة بمجموعة الضابطه مع قيم p ذات دلالة عالية (٠,٠٢٩ و٠,٠٠١> و٠,٠٠١>) على التوالي. وقد ارتبطت شدة نقص الاكسجين بشكل كبير بإخراج البول فى ٢٤ و٤٨ ساعة وبمستوى الكرياتينين فى ٢٤ و٤٨ ساعة، بينما لم توجد دلالة إحصائية بين شدة نقص الاكسجين بالمخ ومستوى اليوريا فى الدم.

الخلاصة: نسبة الكرياتينين فى الدم ومعدل الترشيح الكلوى هى علامات موثوقة لتقييم وظائف الكلى عند االاطفال المصابين بالاختناق فى الفترة ماحول الولاده ويجب تقييمها لتقليل المرض والوفاه.