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Laboratory Indicators of AKI in the Setting of HELLP Syndrome in Resource-constrained Backgrounds

Collins Amadi[1, 2, *], Bright Amadi[1, 3]

[1]Department of Chemical Pathology, Rivers State University, Port Harcourt, Nigeria

[2]Department of Chemical Pathology, PAMO University of Medical Sciences, Port Harcourt, Nigeria

[3]Department of Chemical Pathology, University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria

Email address:

collins338@yahoo.com (C. Amadi)

[*]Corresponding author

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Abstract: Background: Acute Kidney Injury (AKI) in the setting of HELLPs (HELLP syndrome) brings in a high rate of maternal and perinatal morbidity/mortality. Its diagnosis depends on proper evaluation of relevant laboratory indices; a factor that is very limited in resource-constrained environs. Hence, the current study evaluated some routine laboratory indices of AKI in the setting of HELLPs in resource-constrained backgrounds. Methods: The study was conducted retrospectively at a tertiary hospital in Nigeria among 198 pregnant women diagnosed/managed for HELLPs from 2011-2020. Relevant variables and data were extracted from laboratory and other medical files using a well-designed research pro forma and analyzed using standard guidelines. Results: Among those evaluated, 57.1% (n=115) developed AKI. The AKI cohorts had higher mean plasma creatinine (PCr) and plasma uric acid (PUA) levels but lower 24-hour urine volume (UV) compared to the non-AKI cohorts ($p<0.05$). PCr and PUA levels increased while 24-hour urine volume (UV) decreased with advancing AKI stages ($p<0.05$). The PCr, PUA, and 24-hour UV predicted AKI on univariate logistic regression analysis (LRA) ($p<0.05$). However, on multivariate LRA, PUA level and the 24-hour UV lost their statistical significance while that of PCr level was significantly amplified (OR: 9.440; 95%CI: 6.733-11.202). At a cut-off value of 106.7 µmol/L, PCr level maintained a robust predictive potential (AUC: 0.938; 95%CI: 0.859-1.000; $p<0.001$) for AKI. Conclusion: PCr had a robust predictive potential of AKI among the studied population. Hence, timely measurement of PCr level should be considered during the management of HELLPs to reduce the burden of AKI among this at-risk group, especially in resource-constrained settings.

Keywords: HELLP, HELLPs, HELLP Syndrome, AKI

1. Introduction

Hemolysis, Elevated Liver enzymes, and Low Platelet (HELLP) syndrome is a dreaded but rare pregnancy-associated disorder [1, 2]. The syndrome is adjudged a variant of severe preeclampsia as both share similar pathophysiologic features [3]. However, the syndrome has been reported among pregnant/postpartum women without clinical/laboratory evidence of severe preeclampsia [1-3]. HELLP syndrome occurs in 0.2% - 0.8% of all pregnancies but at a higher rate when associated with preeclampsia (10-20%) or following eclampsia (27.6%) [4-6].

The syndrome is associated with varying degrees of adverse maternal and perinatal/neonatal consequences [7-9]. Several reports abound in the literature which suggests that Acute Kidney Injury (AKI) is a relatively frequent complication of HELLP syndrome [10-11]. Several investigators of AKI in the setting of the syndrome have conclusively surmised that AKI significantly heightens the adverse consequences in pregnancy when associated with the syndrome [12-13].

AKI, including HELLP syndrome, is a biochemical disorder and its definitive diagnosis is hinged on evaluation of laboratory parameters which are very limited in resource-constrained backgrounds including many Nigerian centers. Hence, the current study evaluated the predictive potentials of some routine laboratory indices of AKI in the setting of

HELLP syndrome among Nigeria women.

2. Materials and Methods

The study was carried out at one of the tertiary hospitals in Nigeria: the University of Port Harcourt Teaching Hospital (UPTH), Nigeria. Data of eligible pregnant women who developed HELLP syndrome over 10 years (2011-2020) was used as study tools.

The criteria for exclusion from the study included existing liver/hepatobiliary/gallbladder diseases, diabetes mellitus, thyroid disorders, chronic renal diseases, hemoglobinopathies, inherited/acquired thrombotic microangiopathies, chronic and gestational hypertension, acute fatty liver disease of pregnancy, HIV/AIDS cases, preeclampsia/eclampsia superimposed on chronic hypertension, renal transplant recipients, those diagnosed with drug-induced liver injury, and those who are markedly edematous, those with incomplete, data and those diagnosed outside the study period.

Data was acquired anonymously without any distinguishing identifiers using trained research assistants. Key variables of which data was collected included the number of deliveries within the study period and the number of cases of HELLP syndrome diagnosed within the study period. For each eligible case, all the relevant socio-demographic, clinical, anthropometric, obstetric, biochemical, and hematological data were abstracted from laboratory records and medical files.

The diagnosis of HELLP syndrome was defined as previously described, including the following laboratory findings: microangiopathic hemolytic anemia based on detection in a peripheral blood smear; elevated lactate dehydrogenase (LDH) ≥600U/L or total bilirubin level of ≥20.5 µmol/L, respectively; liver dysfunction characterized by elevated aspartate transaminase (AST) and alanine transaminase (ALT) activities >70 U/L; and platelet count of $<150 \times 10^9$ cell/L [12].

The diagnosis of AKI was defined by the Kidney Disease Improving Global Outcomes (KDIGO) guidelines, including an elevation of plasma creatinine level of >26.5 µmol/L within a 48-hour window or more than 1.5 times higher than the baseline level known or presumed to have occurred during the prior 7 days; a urine volume less than 0.5 ml/kg/hour for 6 hours [14].

AKI was further staged according to the KDIGO guidelines as follows [14]:

Stage 1: defined as an increase in plasma creatinine level of >26.5 µmol/L or 1.5–1.9 times the 7-day baseline level or a urine output less than 0.5 ml/kg/h for 6–12 hours.

Stage 2: defined as an increase in plasma creatinine level 2.0–2.9 times the 7- day baseline level or a urine output of <0.5 ml/kg/hour for more than 12 hours.

Stage 3: defined as an increase in plasma creatinine level of >353.6 µmol/L or more than three times the 7-day baseline level, or the initiation of renal replacement therapy, or a urine output less than 0.3 ml/kg/hour for over 24 hours or anuria for more than 12 hours.

Acquisition of specimens for all laboratory analyses was done using standard protocols. Laboratory analysis was carried out using fully automated chemistry and hematological systems by well-experienced laboratory technologists. During analytical processes, at least two levels of commercially-produced quality control materials were introduced to monitor analytical precision and accuracy.

Data were managed using Statistical Package for Social Science version 25 software. The distribution of continuous data was explored using the Shapiro-Wilk test. Data with non-Gaussian distribution were all logarithmically transformed before analysis and presented as mean ± standard deviation; comparison explored using the independent-samples t-test or analysis of variance where necessary. Categorical data were presented as proportions in numbers/percentages; the comparison was made using the Chi-square test or Fisher's exact test and Yate's continuity correction was applied when necessary. Univariate and multivariate logistic regression was used to explore the predictive potentials of AKI at 95% confidence intervals (CI). A p-value <0.05 was set as statistically significant.

3. Results

During the observation period, a total of 298 cases of HELLP syndrome were diagnosed and managed in the study center. However, 198 met the eligibility criteria and were included in the analysis while 100 did not meet the eligibility criteria and were subsequently excluded.

Among these 198 HELLP syndrome cases evaluated in the present study, 57.1% (n=115) developed AKI while others did not (non-AKI, n=83; 41.9%) (Table 1).

Table 1 also depicts results from the comparative analyses of age, obstetric data, and laboratory variables obtained at the point of HELLP syndrome diagnosis between the non-AKI and the AKI populations. From the comparative analysis, the only significant finding was the higher mean plasma creatinine and uric acid levels but lower 24-hour urine volume observed among the AKI versus the non-AKI populations (p<0.05) (Table 1).

Illustrated in Table 2, following the comparative analyses of the three significant variables obtained from the analyses in Table 1 by different AKI stages, it was observed that plasma levels of creatinine and uric acid tended to increase with increasing AKI stages while that of 24-hour urine volume tended to decrease with advancing AKI stages (p<0.05) (Table 2).

Table 3 depicts results from the logistic regression analyses of the plasma creatinine and uric acid including urine volume to predict AKI among the studied population. Results of the univariate logistic regression analysis show the three variables under evaluation significantly predicted AKI with plasma creatinine levels (OR: 6.766; 95%CI: 4.134-7.988; p<0.001) having a higher univariate predictive potential compared to plasma uric acid and 24 urine volume (Table 3). However, on

multivariate logistic regression analysis, the statistical significance of plasma uric acid level and 24-hour urine to predict AKI was lost while that of plasma creatinine level (OR: 9.440; 95%CI: 6.733-11.202) was significantly amplified (Table 3).

Table 4 shows the results of the receiver operating characteristics curve analysis (ROC) on the predictive potential of plasma creatinine levels for AKI diagnosis among the studied population. Following the ROC analysis, at a cut-off value of 106.7 µ mol/L, plasma creatinine level maintained a robust predictive potential (AUC: 0.938; 95%CI: 0.859-1.000) over AKI diagnosis among the studied HELLP syndrome population (Table 4).

Table 1. *Comparison of variables obtained at diagnosis among different HELLP categories.*

Variables	All HELLP n=198 (100%) M ± SD	Non-AKI n=83 (41.9) M ± SD	AKI n=115 (57.1%) M ± SD	Non-AKI vs. AKI p-value
Age, years	30.67 ± 2.90	30.18 ± 3.01	29.44 ± 2.94	0.084
Gravidity	2.28 ± 0.59	2.33 ± 0.52	2.42 ± 0.74	0.063
Parity	1.76 ± 0.23	1.65 ± 0.31	1.71 ± 0.34	0.112
GA at presentation, weeks	35.66 ± 3.43	34.93 ± 3.17	35.07 ± 3.16	0.154
SBP, mmHg	153. 67 ± 6.90	155.08 ± 6.73	156.41 ± 7.03	0.378
DBP, mmHg	109. 51 ± 5.07	110. 02 ± 5.04	109.58 ± 5.112	0.079
Plasma sodium, mmol/L	136.73 ± 7.56	136.14 ± 8.04	135.99 ± 8.66	0.064
Plasma potassium, mmol/L	4.64 ± 1.61	4.09 ± 0.98	4.13 ± 1.80	0.721
Plasma bicarbonate, mmol/L	22.61 ± 2.96	23.73 ± 3.03	22.71 ± 2.84	0.263
Plasma urea, mmol/L	3.74 ± 1.04	3.07 ± 1.14	3.24 ± 1.24	0.411
Plasma creatinine, µmol/L	108.92 ± 4.73	88.65 ± 4.69	195.05 ± 8.02	<0.001*
Plasma uric acid, mmol/L	1.78 ± 0.61	1.06 ± 0.11	3.03 ± 0.55	<0.001*
ALT, U/L	209.71 ± 8.13	211.64 ± 8.06	210.64 ± 7.81	0.187
AST, U/L	165.41 ± 5.44	164.70 ± 6.08	167.01 ± 5.39	0.201
LDH, U/L	716.75 ± 16.63	714.91 ± 15.89	713.62 ± 15.66	0.560
Plasma albumin, g/L	33.65 ± 3.90	35.07 ± 4.05	34.73 ± 4.10	0.076
Total bilirubin, µmol/L	57.91 ± 4.12	56.13 ± 5.02	57.84 ± 5.17	0.434
Platelet count, x 10^9 cell/L	87.83 ± 6.74	88.65 ± 5.97	85.75 ± 5.64	0.607
PCV, %	29.72 ± 3.81	28.05 ± 3.64	27.94 ± 3.95	0.584
QUP, +	2.34 ± 0.62	2.21 ± 0.73	2.38 ± 0.90	0.073
UV, liters/24 hours	1.82 ± 0.22	1.90 ± 0.34	0.49 ± 0.04	<0.001*

*Statistically significant; AKI: acute kidney injury; M ± SD: mean ± standard deviation; SBP: systolic blood pressure; DBP: diastolic blood pressure; ALT: alanine aminotransferase; AST: aspartate aminotransferase; LDH: lactate dehydrogenase; PCV: packed cell volume; QUP: qualitative urine protein; UV: urine volume.

Table 2. *Comparison of variables obtained at diagnosis by different AKI stages.*

Variables	Stage 1 AKI n=80 M ± SD	Stage 2 AKI n=25 M ± SD	Stage 3 AKI n=10 M ± SD	p-value
Plasma creatinine level, µmol/L	167.09 ± 8.86	189.68 ± 9.67	207.65 ± 10.05	<0.001*
Plasma uric acid level, mmol/L	2.04 ± 0.63	2.93 ± 1.08	3.56 ± 1.14	<0.001*
Urine volume, liters/24 hours	0.76 ± 0.15	0.58 ± 0.09	0.34 ± 0.03	<0.001*

*statistically significant; AKI: acute kidney disease; M ± SD: mean ± standard deviation.

Table 3. *Logistic regression of potential biochemical predictors of AKI.*

Variables	Univariate Logistic Regression		Multivariate Logistic Regression	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Plasma creatinine level µmol/L	6.766 (4.134-7.988)	<0.001*	9.440 (6.733-11.202)	<0.001*
Plasma uric acid level, mmol/L	1.414 (0.637-2.269)	0.021*	-----	0.061
Urine volume, liters/24 hours	1.178 (0.565-2.173)	0.036*	-----	0.057

*Statistically significant; OR: odd ratio; CI: confidence interval.

Table 4. *ROC characteristics of the predictive potential of plasma creatinine over AKI.*

Variable under ROC evaluation	Plasma creatinine level, µmol/L
Cut-off value, µmol/L	106.7
Sensitivity	94.4%
Specificity	88.9%
ROC AUC	0.938
AUC 95% CI	Lower border: 0.859 Upper border: 1.000
p-value	<0.001*

*Statistically significant; ROC: receiver operating characteristics curve;
AUC: area under the curve; CI: confidence interval.

4. Discussion

4.1. Key Findings

The current study evaluated the likely laboratory indicators of AKI among cases of HELLP syndrome among women of Nigerian origin. Among those evaluated, 57.1% developed AKI. The AKI cohorts had higher mean plasma creatinine and plasma uric acid levels but lower 24-hour urine volume compared to the non-AKI cohorts. Plasma creatinine and uric acid levels increased while the 24-hour urine volume decreased with advancing AKI stages. The plasma creatinine, plasma uric acid, and 24-hour urine volume predicted AKI on univariate logistic regression analyses. However, on multivariate logistic regression analyses, plasma uric acid level and 24-hour urine volume lost their statistical significance while that of plasma creatinine level was significantly amplified. At a cut-off value of 106.7 μmol/L, plasma creatinine level maintained a robust predictive potential for AKI diagnosis.

4.2. Relationship to Previous Studies

Several investigators from the western populations have consistently maintained in a large number of reports of the very high rate of AKI among cases of HELLP syndrome globally [10-13]. Numerous researchers have conclusively surmised also that HELLP syndrome is a substantial etiologic factor of pregnancy-related AKI [10, 11]. However, few, if any, of those reports have emanated from the developing populations with virtually known from Nigeria.

In this current study, the observed rate of AKI (57.1%) among the studied HELLP syndrome population was very high. Though the AKI rate was very high, the rate remains within the rates documented in the literature. Ye et al. 2019 and Huang et al. 2017 reported 48.1% and 60% rates, respectively from two similar studies among Chinese populations [11, 12]. Gedik et al. 2017 reported a 25% rate of AKI among Turkish women with HELLP syndrome. [10] A recent study reported by Novotny et al. 2020 among HELLP syndrome patients of United States origin found AKI rate of 14.4% [15].

The higher mean plasma creatinine and uric acid levels but lower 24-hour urinary volume observed in the current study among the HELLP syndrome patients with AKI are all related to the pathophysiologic processes in the ensuing AKI [12]. In addition, the rising plasma creatinine and uric acid levels and the decreasing 24-hour urinary volume with advancing AKI stages are related to the worsening of the AKI [13]. These patterns of biochemical alterations have also been observed in previous studies and underscore the clinical significance of these indicators in the evaluation of at-risk HELLP syndrome cases for AKI [11, 13, 16].

Rapid changes in plasma creatinine level are useful in the detection of incident AKI in HELLP syndrome and have been associated with outcomes in HELLP syndrome [17]. Findings in this current study corroborate this scientific evidence. Though other AKI biochemical indices of AKI abound in the more affluent societies, plasma creatinine as an indicator of AKI could be valuable in resource-constrained backgrounds. In the current study, plasma creatinine level had a more robust predictive potential of AKI detection relative to the plasma uric acid level and the 24-hour urinary volume. Similar findings have been documented in the literature [12, 16].

4.3. Mechanism of HELLP Syndrome-associated AKI

From existing literature, the mechanism of HELLP syndrome-induced AKI remains ill-defined. [13] However, some studies have previously noted that most of the kidney pathological biopsy is acute tubular necrosis (ATN) with a minority being acute cortical necrosis (ACN) [13, 17-19]. These pathological features of ATN and the ACN both show widespread microvascular injury and microthrombosis [17-19]. In addition, HELLP syndrome induces intravascular platelet activation and widespread microvascular endothelial damage, which may well be the cardinal basis of intravascular microthrombosis [13, 18-20]. The evolution of endothelial injury and the thrombotic microangiopathies in patients with HELLP syndrome leading to renal tubular necrosis is a pathogenic process that may induce AKI [20].

4.4. Clinical Implications

The monitoring of plasma creatinine levels should be ascertained as early as possible during the evolution of the syndrome. This will enable early evaluation of renal status, enhance clinical management, and improve outcomes during HELLP syndrome.

4.5. Limitations

The study was limited by some factors worthy of note. First, its retrospective design may have led to the under-reporting of the actual number of cases of HELLP syndrome identified during the study. Secondly, as a hospital-based study, its conclusions may lack generalizability to the entire larger population within the sampled location. Hence, the study conclusions should be applied with caution during its interpretation and clinical implementation.

5. Conclusion

The study evaluated the likely laboratory indicators of AKI among cases of HELLP syndrome. Among the population evaluated, most of them developed AKI. The plasma creatinine, plasma uric acid, and 24-hour urine volume predicted AKI on univariate logistic regression analyses. However, on multivariate logistic regression analyses, plasma uric acid level and 24-hour urine volume lost their statistical significance while that of plasma creatinine level was significantly amplified and maintained a robust predictive potential for AKI diagnosis compared to the other parameters.

Statement of Ethics

The ethical approval of the study was obtained from the Research Ethics Committee of UPTH following the review of the study protocols and the study was executed in compliance with the principles embodied in the Helsinki Declaration.

Disclosure Statement

All the authors do not have any possible conflict of interests.

Author Contributions

All the authors were involved substantially in the concept and design of the study, data acquisition, analysis and interpretation of the data, drafting the article, revising the article critically for its intellectual content, and in the final approval of the version to be published.

Data Availability

The data that support the findings of this study are not publicly available due to their containing information that could compromise the privacy of research participants but are available from the corresponding author (CA) upon reasonable request.

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