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Association between Serum Level of Oncostatin M and Development of Acute Kidney Injury among Critically Hospitalized Patients and its Role as a Predictive Biomarker

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Abstract

Background: More than one out of every twenty patients needing critical care unit care develop severe AKI (Acute Kidney Injury). Oncostatin M (OSM), a member of the IL-6 family of cytokines, plays an important roles in renal diseases as they have been found to be elevated in the renal tissue of patients with kidney diseases.

Aim of Study: To study the role of Oncostatin M as an early biomarker of AKI among critically ill hospitalized patients and for the prediction of mortality or requirement for renal replacement therapy in patients with AKI.

Patients and Methods: A case-control study was done on 180 patients admitted to ICU within Kasr El-Aini Hospital Cairo University. They were eligible to participate in the study. Those patients were divided into 2 groups. Group 1: 90 patients admitted to ICU with sepsis and developed AKI. Group 2: 90 patients admitted to ICU without AKI. Serum concentration of Oncostatin M was performed for all patients and compared between participating groups. Serum level of Oncostatin M was measured by Enzyme linked immunosorbant assay (ELSA).

Results: Although there was Oncostatin M serum levels and urea, Potassium, Calcium, and phosphrous serum levels had a marginally negative correlation, none of these (p=0.163, p=0.240, p=0.669, p=0.978) were statistically significant. With a sensitivity of 83.8% and a specificity of 61.4%, we discovered that Oncostatin M is a useful tool for predicting death among patients admitted to the ICU with sepsis and developing AKI (AUC=0.673, 95% CI: 0.532-0.814).

Conclusion: It was found that patients admitted to the ICU frequently had AKI. It was linked to a higher incidence of morbidity and mortality. It is essential to predict AKI in ICU patients early. Lower serum levels of OSM can be a major predictor of acute kidney injury in ICU patients if combined with patients' clinical examination and general hemodynamic status. In this way, patients' outcome can be improved.

Key Words: *AKI – Oncostatin M – Prediction – Mortality – Renal replacement therapy.*

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Introduction

ACUTE kidney injury (AKI) is characterized by abrupt deterioration in kidney function, manifested by an increase in serum creatinine level with or without reduced urine output. The spectrum of injury ranges from mild to advanced, sometimes requiring renal replacement therapy **[1]**. In critically ill patients, AKI employs about 30-60% of them and is accompanied by acute morbidity and mortality **[2]**.

Severe AKI is present in more than one every twenty patients who need care in an intensive care unit and is associated with death rates of 50% to 70%. Maintaining nutrition, preventing or treating electrolyte and acid-base imbalances, adjusting the dosage of medications that are excreted by the kidney, and avoiding secondary hemodynamic and nephrotoxic renal injury in the absence of effective pharmacologic therapies are all important components of supportive care in the management of AKI. Although all these conservative precautions, many of AKI patients require several dialysis and hemofiltration methods as renal replacement therapy (RRT) **[3]**.

It is possible to think of rescue therapy when considering the standard list of RRT indications for AKI, which also includes overt uremic manifestations like pericarditis and encephalopathy, volume overload unresponsive to diuretic therapy, and electrolyte and acid-base disturbances unresponsive to medical treatment, especially severe hyperkalemia **[4]**.

Kidney cells that express and secrete members of the IL-6 family include podocytes, endothelial cells, mesangial cells, and tubular epithelial cells. By encouraging cell proliferation, the signaling of

IL-6 cytokine family members can affect a variety of cell types and either promote or worsen tubulointerstitial fibrosis [5].

Several studies have shown that serum Oncostatin M (OSM) was a useful early marker for renal disorders [6,7,8], but; up to our knowledge; there was no evidence concerning its role in diagnosis of AKI among hospitalized patients.

So, we conducted our study to evaluate the association between the risk of development of AKI with measurement of the serum level of OSM as an early biomarker of AKI among critically ill patients. Also, to predict mortality and need for renal replacement therapy.

Patients and Methods

This case-control study was conducted on 180 Subjects admitted to ICU within Kasr El-Aini University Hospitals from 2016 2018. Those patients were divided into 2 groups: Group 1: 90 patients admitted to ICU with sepsis and developed AKI. Group 2: 90 patients admitted to ICU without AKI.

Inclusion criteria: Adult Patients with AKI as defined in accordance with criteria established by the Acute Kidney Injury Network: An abrupt increase in serum creatinine ≥0.3mg/dl within 48 hours or a ≥50% increase in serum creatinine or decrease in urine output <0.5ml/kg per hour for more than 6 hours [2].

Exclusion criteria: Patients with CKD, recent therapy with elemental vitamin D, history of parathyroid disease and history of fat malabsorption or duodenal resection.

Detailed history was taken from all patients with special emphasis on: Age, Gender, Weight, Height and BMI.

Full clinical examination was performed for all patients with special emphasis on: General examination, GIT examination, chest examination and neurological examination.

Routine labs Laboratory analysis was carried out in the Clinical and Chemical Pathology Department, Cairo University for all patients including: Complete blood count, kidney functions: Urea, Creatinine and serum electrolytes: Na+, K+, Ca, $PO4^{-}$.

Analysis of serum Oncostatin M (OSM):

All participants were subjected to:

Three milliliter (3ml) of blood were collected in a plain sterile vacutainer. Blood samples were left to clot at room temperature and then centrifuged for 5 minutes for serum separation and kept at 20ºC until time of assay of OSM. Oncostatin M was measured by Human Oncostatin M ELISA kit supplied by Sun Red technical service. Catalog No. 201-12-1664. (eMail: sunredbio@msn. cn) which employs a double-antibody sandwich enzyme-linked immunosorbent assay.

Statistical analysis: To analyze the data, we utilized SPSS version 24 for Windows, the statistical tool for social sciences. Frequencies and percentages will be used to express qualitative data. If quantitative data is normally distributed, it will be expressed in terms of means and standard deviations; if it is not, it will be expressed in terms of median and interquartile ranges. In order to examine the relationship between categorical variables, the chi square test will be utilized. Fisher exact test will be used to test the violation of assumptions. Student *t*-test will be used to test the difference of numerical variables between the 2 study groups. In case of non-parametric data, Mann Whitney test will be used. p-value <0.05 will be considered statistically significant.

Results

180 patients were recruited in the study. 61.4% of them (1 10 patients) were males. Their mean age was 39.81±16.81 years old. Diabetes mellitus was the commonest co-morbidity found in those patients. 63.9% of patients (115 patients) were diabetic. 20.8% of patients (37 patients) were hypertensive. 8.4% of patients (15 patients) suffered from SLE. Pneumonia was the least found co-morbidity among patients that was prevalent among 4.5% of patients (8 patients) (Table 1).

Table (1): The socio-demographic and associated co-morbidities among included patients (n=180).

Variable	N (%)
Gender:	
Male	110 (61.4)
Female	70 (38.6)
Diabetics	115 (63.9)
Hypertensive	37 (20.8)
SLE patients	15 (8.4)
Pneumonia patients	5 (4.5)
Others	16 (8.9)

* Mean ± SD.

Results of lab findings in the studied patients are provided on Table (2).

Table (2): The laboratory findings of included patients (n=180).

Lab test	Results
Urea (mg/dl)	66 (32-182)*
Creatinine (mg/dl)	2.45±0.8**
Na (mmol/L)	137.4±5.8**
K (mmol/L)	4.93±0.89**
Ca (mg/dl)	8.56±0.83 **
PO4 (mg/dl)	3.59± 1.15**
Oncostatin M (ng/L)	30 (2-45)*

*Median (25th - 75th percentile). **Mean ± SD.

Participants in the study were divided into two groups: 90 sepsis patients hospitalised to the ICU with acute kidney injury (AKI) were in Group 1; 90 sepsis patients admitted to the ICU without AKI were in Group 2. The patients' and control group's varying ages were noticeably greater (p 0.001). It was found that while both men and women were equally distributed among cases and controls, men made up the majority of the cases. Significant differences existed here (p 0.001). Additionally, we discovered that, compared to other patients, patients who had AKI had a considerably greater mortality rate. (5% of the control group vs. 43.6% of the case group) According to Table (3), this was statistically significant (p 0.001).

Table (3): The difference between both groups concerning Socio-demographic characteristics and associated co-morbidities.

Variable	Case group (n=90)	Control group (n=90)	p-value
Age (years)	48.6± 15.7	31.02± 12.8	<0.001 T
Gender:			
Male	66 (73.3%)	46 (49.5%)	0.001 C
Female	24 (26.7%)	44 (50.5%)	
Diabetics	49 (54.7%)	65 (73.3%)	0.008 C
Hypertensive	37 (41.6%)	0	<0.001 C
SLE patients	5 (5.9%)	9 (10.9%)	0.311 C
Pneumonia patients	8 (8.9%)	0	0.003 F
Mortality	39 (43.6%)	4 (5%)	<0.001 C

T: Independent sample *t*-test.
C: Chi square test.
F: Fissure exact test.

We examined the laboratory results between the two groups and discovered that the median urea levels were considerably higher in the case group when compared to the control group (p 0.001). Similar to this, patients had significantly higher mean creatinine serum levels than controls (p 0.001) Additionally, we discovered that cases had considerably higher mean K serum levels than controls (p 0.001), which is another finding. Similarly, mean Na serum levels were significantly higher among cases when compared to controls (p=0.024). We also found that mean PO4 serum levels were higher among cases when compared to controls (p=0.044). In contrast, mean Ca serum levels were significantly lower among cases when compared to controls (p<0.001). We found median Oncostatin M serum levels were significantly lower among cases when compared to controls (p<0.001) as shown in Table (4).

Table (4): The difference between both groups concerning laboratory findings.

Variable	Case group (n=90)	Control group (n=90)	p-value
Urea (mg/dl)	180 (100-202)	33 (28-40)	<0.001 M
Creatinine (mg/dl)	3.73± 1.67	1.16±0.67	<0.001 T
Na (mmol/L)	138.36±5.91	136.5±5.58	0.024 T
K (mmol/L)	5.2±0.76	4.65±0.93	<0.001 T
Ca (mg/dl)	8.15±0.84	8.96±0.59	<0.001 T
PO4 (mg/dl)	3.75± 1.31	3.43±0.95	0.044 T
Oncostatin M (ng/L)	2 (2-38)	37 (30-46)	<0.001 M

M: Mann Whitney U test. T: Independent sample *t*-test.

We examined the laboratory results of Oncostatin M, and we found that median oncostatin m serum levels were significantly higher among those who improved clinically when compared to those with poor outcome (death, RRT); p=0.048 as shown in Table (5).

Table (5): The association between Oncostatin M serum levels and outcome among cases group (n=90).

Outcome	Oncostatin M	p-value
Die (n=39)	29 (2-40)	0.048
Improve (n=2)	38 (36-38)	
RRT (n=49)	2 (2-35)	

Kruskal Wallis Test. RRT (Renal Replacement Therapy).

It was also found that Oncostatin M is an effective predictor of mortality in patients admitted to the intensive care unit (ICU) with sepsis and developing AKI (AUC=0.673, 95% CI: 0.532-0.814), with a sensitivity of 83.8% and a specificity of 61.4%. (Table 6), (Fig. 1).

Table (6): The diagnostic accuracy of Oncostatin M in prediction of mortality among patients with sepsis & AKI (n=90).

Specificity	Sensitivity	95% Confidence interval (95% CI)		p-value	AUC
		Upper	Lower		
61.4%	83.8%	0.814	0.532	0.019	0.673

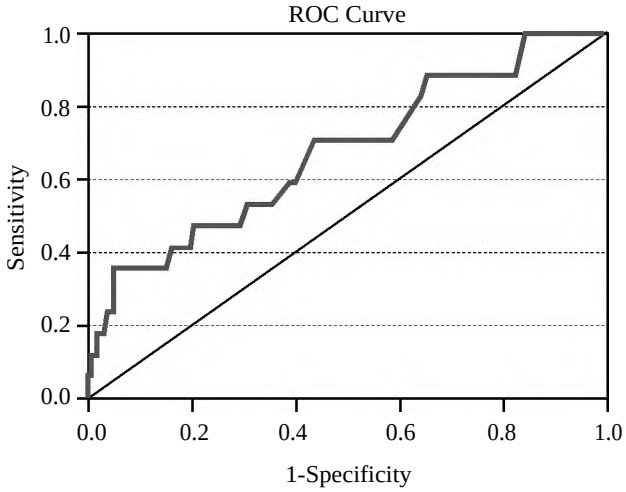

Fig. (1): ROC curve for prediction of mortality among ICU patients with sepsis & AKI.

Discussion

In 2018, Hoste et al., estimated that one third to two thirds of patients admitted to ICU experience AKI and nearly 10%-15% of those patients require renal replacement therapy in the form of either hemodialysis or renal transplantation [9].

Unfortunately, AKI is associated with poor outcomes; increased rates of morbidity and mortality worldwide.

Saran et al., [10] via the United States Renal Data System (USRDS) 2018 report; reported the mortality rate among patients hospitalized due to AKI reached 8.2% in contrast to only 1.8% among those with Non-AKI. Untreated AKI can be a significant risk factor for developing CKD and even increased mortality. Rosner et al., reported that AKI that necessitate hemodialysis have high mortality rates varying between 30% and 80%. In addition, more than 30% of patients who survive AKI don't recover kidney functions and require long life hemodialysis [11].

In our study, we found that mortality rate was high reaching 43.6% of patients among cases group. This was significantly higher than those within the control group. This was slightly higher than Saxena et al., who studied 229 patients admitted to ICU in a tertiary center in India. They reported that the mortality rate reached 28.4% of patients being significantly more in patients with higher degrees of injury [12].

In order for decreasing the mortality rate associated with AKI and improving the patients' outcome, clinicians should early predict the diagnosis through laboratory results [13]. In response to injury, both kidneys and liver secrete acute phase reactants as a mechanism of protection. These proteins serve as protein molecules interacting through interleukin cytokines [14].

In our study, we found that Oncostatin m were significantly lower among patients with AKI when compared to control group. We also found that the lower serum levels, the poorer outcomes the patients get.

Concerning gender, it is reported that great difference between genders is present concerning development of kidney diseases wether acute or chronic. In addition, Haghighi et al., stated that this difference is related to a difference in the glomerular structure. In addition sex hormones have a significant effect on renin angiotensin system which in turn affects both structure and function of the kidneys [15].

In our study, we found a significant difference between males and females concerning developing AKI. 73.3 % of patients with AKI were males while 26.7% of them were females. In contrast, this was similar to what was reported by Güzel et al., [16]. Who studied 1190 patients admitted to ICU between 2015 and 2018 Bezmialem Vakıf University Hospital.

Males under the age of 65 were found to have significantly greater rates of AKI. However, compared to males, females had a considerably higher incidence of AKI over 65. This could be explained by how ageing affects the structure and function of the kidneys [16].

We also found that our results were slightly lower than Garzotto et al., who recruited 601 patients admitted to 10 different ICU centers in Italy. They reported there was a male predominance among patients who developed AKI with a prevalence of 62.5% [17].

In our study we found that mean age for patients who developed AKI was significantly higher than control group (48.6±15.7 vs 31.02±12.8) years old. This was much less than Güzel et al., who reported that mean age of patients with AKI was 66.65 ± 16.86 years old. This may be explained by the great medical evolution in turkey that increased the life expectancy of individuals to reach nearly eighty years [18].

Concerning hypertension, BP variability is known to be a risk factor for many negative outcomes; cerebrovascular events, cardiovascular

diseases and renal affection. Mulè et al. **[19]** reported that short term variability in blood pressure can lead to some subclinical changes in both kidneys like microalbuminuria or decrease in the e GFR between 60 and 30mL/min/1.73m^2 especially in essentially hypertensive patients.

In our review, we observed that hypertension was essentially higher among patients who created AKI. This matches what was accounted for by Garzotto et al., **[13]** who saw that as 52.2% of patients who created AKI were hypertensive. Cartin-Ceba et al., **[20]** played out a precise survey in a preliminary to distinguish risk factors for getting AKI among patients confessed to ICU and found that subsequent to performing responsiveness examination, hypertension showed a critical job in creating AKI with a general gamble of 1.43-95% CI 1.08-1.89).

There is no doubt that serum creatinine levels are significant for the finding and organizing of AKI, as per the "Kidney sickness working on worldwide results" (KDIGO) rules, which characterized AKI in view of the greatest creatinine levels and pee yield. As per their discoveries, AKI would be emphatically recommended by an ascent in serum creatinine of >0.3mg/dl or >50% from pattern. This considers the division of AKI into 4 phases, going from stage 0 to arrange 3 **[21]**.

Also, Bhatraju et al., **[22]** could implement a new method for staging of AKI into resolving and non-resolving ones based on the change in serum creatinine within 72 hours of admission in this way, clinicians could early predict mortality among those patients and help patients reach a better outcome.

In our study we found that there was a significant elevation in serum creatinine among patients who developed AKI when compared to others (3.73±1.67 vs 1.16±0.67). This was much higher than what was reported by Samimagham et al., **[23]** who recruited 263 patients admitted to Shahid Mohamadi Hospital in Iran. The average serum creatinine was 1.27-1.06mg/dl, they discovered. This can be explained by the fact that the patients they studied had smaller age ranges, with mean ages of 39.51-21.22 years.

Conclusion:

We found that AKI was prevalent among patients admitted to ICU. A higher incidence of illness and mortality was associated with it. Early AKI prediction in ICU patients is crucial. Assessment of serum OSM can play an important role to reach the diagnosis in those patients. Lower serum levels of OSM can be a major predictor of acute kidney injury in ICU patients if combined with patients' clinical examination and general hemodynamic status. In this way, patients' outcome can be improved.

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الارتباط بين مستوى مصل اوتكوستاتين وتطور إصابة الكلى الحادة بين مرضى المستشفيات الحرجة ودورها كمؤشر بيولوجى تنبؤى

المقدمة : تحدث أمراض القصور الكلوى الحاد فى أكثر من مريض واحد من كل عشرين مريضاً يحتاجون إلى وحدج العناية المركزة، وقد ارتبطت بمعدلات وفيات تتراوح من ٥٠٪ إلى أكثر من ٧٠٪ فى غياب أى علاجات دوائية فعالة لـ (Oncostatin M OSM, AKI)، أحد أعضاء عائلة السيتوكينات IL-6، له أدوار مهمة فى أمراض الكلى، حيث وجد أن أفراد عائلة IL-6 سيتوكين مرتفع فى الأنسجة الكلوية للمرضى المصابين بأمراض الكلى.

أجرينا دراسة حالة شواهد تهدف إلى الكشف عن القدرة التنبؤية لأونكوستاتين لإصابة الكلى الدراسة هدف الحادة بين المرضى المصابين بأمراض خطيرة.

طرق البحث : أجريت هذه الدراسة على مائة وثمانين مريضاً تم قبولهم فى وحدة العناية المركزة داخل مستشفيات جامعة قصر العينى، وكانوا مؤهلين للمشاركة فى الدراسة، تم تقسيم هؤلاء المرضى إلى مجموعتين. المجموعة ١: تم قبول ٩٠ مريضاً فى وحدة العناية المركزة مصابين بالتسمم وتطور لديهم القصور الكلوى الحاد. المجموعة الثانية : ٩٠ مريضاً تم قبولهم فى وحدة العناية المركزة بدون أمراض القصور الكلوى الحاد. تم قياس مصل (Oncostatin M OSM) لجميع المرضى ومقارنته بين المجموعات المشاركة. تم قياس مستوى بروتين الانكوستاتين م فى الدم بعمل تحليل الاليزا.

النتائج : أظهرت نتائجنا أن متوسط عمر المرضى المشمولين كان ٣٩.٨١±16.81 سنة. كان داء السكرى أكثر حالات الاعتلال المشترك شيوعاً فى هؤلاء المرضى، ووجدنا أن متوسط مستويات مصل Oncostatin كانت أقل بشكل ملحوظ بين الحالات عند مقارنتها بمجموعة التحكم (p<0.001) كما هو موضح فى الجدول ٤ كان هناك ارتباط سلبى ضعيف بين مستويات مصل Oncostatin M ومستويات مصل اليوريا وK وPO4 Ca. ومع ذلك، لم يكن أى منها ذا دلالة إحصائية (p=0.978, p=0.669, p=0.240, p=0.163) على التوالى، وجدنا أن متوسط مستويات مصل أونكوستاتين م كانت أعلى بشكل ملحوظ بين أولئك الذين تحسنوا سريرياً عند مقارنتها مع أولئك الذين لديهم نتائج سيئة الموت (RRT, p=0.048) وجدنا أن Oncostatin أداة تنبؤية جيدة للتنبؤ بالوفيات بين المرضى الذين تم قبولهم فى وحدة العناية المركزة مع تعفن الدم AKI (AUC=0.673, 95% CI: 0.532-0.814) مع حساسية 83.8% ونوعية 61.4 AKI.

الخلاصة : توصلنا أخيراً إلى أن القصور الكلوى الحاد كان منتشراً بين المرضى الذين تم إدخالهم إلى وحدة العناية المركزة كان مرتبطاً مع زيادة معدل المراضة والوفيات. إن التنبؤ المبكر بمرض القصور الكلوى الحاد بين دوراً مهماً للوصول إلى Oncostatin M مرضى وحدة العناية المركزة أمر لابد منه. يمكن أن يلعب تقييم مصل التشخيص لدى هولاء المرضى. يمكن أن تكون مستويات المصل المنخفضة من أونكوستاتين م مؤشراً رئيسياً لإصابة الكلى الحادة لدى مرضى وحدة العناية المركزة إذا تم دمجها مع الفحص السريرى للمرضى والحالة الديناميكية الدموية العامة. بهذه الطريقة، يمكن تحسين نتائج المرضى.