**Acute kidney injury in intensive care unit in AL-hussain teaching hospital**

This study was submitted in partial fulfillment of the requirement for the M.B.CH.B

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**Abstract**

Background: Acute Kidney Injury (AKI) is an unsolved clinical problem in critical care patients with a high mortality rate, increasing incidence, and no definitive therapy.

We studied the AKI in ICU patients and we collected the data retrospectively from intensive care unit at AL hussain teaching hospital in al Nasiriya-Iraq for three month from dcember 2018 to march 2018 .and collected the investigations of the patients –serum creatinine and blood urea,age ,sex, past history,chief complaint.

The total numbers of the patients was **148** And the percent of AKI is (**28.38 %**)

**Introduction**

Acute kidney injury (AKI) has been long recognized as a common complication in intensive care unit (ICU) patients. However, it has not been until recently that intensivists have shown a growing interest in and awareness of its real impact on costs and outcomes,. At present, it is widely accepted that AKI is a common clinical syndrome in ICUs[1–3](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0395) and has a central role as a systemic disease causing multiple systemic sequels and lesions in extra-renal organs. Also, AKI exerts a negative effect on the course and short- and long-term prognosis of the disease[4](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0410) not only for the patient but also for the kidney. These findings have led to a change in the definition of AKI,[5](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0415) with special emphasis on early detection and prevention. Considering these facts and the emerging role that extracorporeal depuration therapies have in intensive care units, our workgroup resolved to develop a comprehensive up-to-date review of all these topics, which will be presented in this journal in a series of six reviews, being this paper the one that starts the series.

**Pathophysiology of AKI**

The main causes of AKI are ischemia, hypoxia and nephrotoxicity. The mechanisms involved in kidney injury and repair are complex. The kidney is particularly susceptible to ischemia and toxins, resulting in vasoconstriction, endothelial damage, and activation of inflammatory processes. This susceptibility arises in part from the vascular-tubular relationships in the outer medulla of the kidney, where the partial pressure of oxygen is low, even at baseline, making them more vulnerable to a decreased renal blood flow.[6](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0420) In the presence of a decreased glomerular filtration rate (GFR) secondary to hypoperfusion,[7](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0425) the normal response of the kidney is to maximally concentrate urine and reabsorb sodium avidly in an effort to maintain/increase intravascular volume and normalize renal perfusion. However, a prolonged decrease in renal perfusion can result in irreversible ischemic damage, leading to ischemic AKI or acute tubular necrosis (ATN), which it is the most severe form of AKI. ATN is characterized by sub-lethal and lethal injury to the tubular cells, mainly in distal regions of the proximal tubule and thick ascending limb of Henle's loop.[7](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0425)

Historically, AKI has been divided into well-characterized and sequential phases, namely, initiation, maintenance and recovery[8](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0430) and, more recently, Sutton et al.[9](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0435) added a prerenal and an extension phase, establishing five pathophysiological stages during ischemic ATN.

* **(1)** *Prerenal*: continuous with the next stage, occurs when renal blood flow (RBF) decreases but cellular integrity is yet maintained.
* **(2)** *Initiation*: characterized by a decrease in GFR due to a decrease in net ultrafiltration pressure. Ischemic injury is higher in the S3 segment of proximal tubule and thick ascending limb due to the high consumption of ATP in these areas, located in the outer medulla where partial pressure of oxygen is lower. Ischemia causes ATP depletion, inhibition of active sodium transport, formation of reactive oxygen species, alterations in the cytoskeletal structure and loss of cell polarity (relocalization of Na/K ATPase), tight junctions between cells (E-cadherin) and attachment of cells to the basement membrane (integrins). The accumulation of detached cells and necrotic debris in the lumen of the tubule contribute to occlusion and back-leak of glomerular filtration . This damage can be repaired if blood flow restores early.
* **(3)** *Extension*: morphological and functional changes appear in vascular endothelial cells and renal tubular epithelium, resulting in the recruitment of circulating inflammatory cells such as neutrophils, lymphocytes and macrophages, and the expression of adhesion molecules and chemokines. Cells of the S3 segment produce interferon regulatory factor 1 (IRF-1), which activates transcription of proinflammatory genes. Proximal tubule cells produce cytokines (TNF-α, TGF-β, interleukins) and in addition IL-18 and IL-6 are also released into the tubular lumen and can be used as early biomarkers of kidney damage. Therefore, this injury induces the production of inflammatory mediators by endothelial and tubular cells, contributing to the recruitment of leukocytes. We can say that inflammation plays an important role in both the initiation and the extension of kidney damage).
* **(4)** *Maintenance*: lasts 1 or 2 weeks and during this phase GFR is stabilized at its lowest level and now oliguria and uremic complications can occur. GFR is kept low by dysregulation of possible release of vasoactive mediators from endothelial cells, the congestion of medullary blood vessels and damage by reactive oxygen species and inflammatory mediators produced by leukocytes and renal cells after reperfusion. During this clinical phase, cells undergo repair, migration, apoptosis and proliferation in an attempt to reestablish and maintain cellular and tubule integrity.
* **(5)** *Recovery*: characterized by the repair and regeneration of tubular epithelium, and the gradual return of GFR. During this phase, differentiation continues, epithelial polarity is reestablished and normal cellular and organ function returns. Surviving cells are quiescent and undergo a process of de-differentiation and migration to enter the cell cycle and repopulate the basement membrane, regenerating damaged epithelium. For this to occur successfully, there must be a parallel process to clear the accumulation of tubular cells. The successful recovery from AKI depends on the degree to which these repair processes ensue and may be compromised in elderly or chronic kidney disease (CKD) patients.[7–9](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0425) Recovery takes 1–2 weeks after normalization of renal perfusion, requiring repair and regeneration of tubular cells.[8](http://medintensiva.org/es-acute-kidney-injury-renal-disease-articulo-S0210569116300948#bib0430) This phase may be complicated by a diuretic phase due to lack of functionality of the cells of the proximal tubule to reabsorb water and solutes.

**Aim to study**

**To study acute kidney injury in intensive care unit at Al hussain teaching hospital by using the database of patients to know how many pattients developed acute kidney injury in intensive care unit.**

**Methodology**

In retrospective study was conducted at al hussain teaching hospital it is a general teaching hospital for populations in al Nasiriya –Thi-Qar which is located in the south of Iraq

.This research collected for three month during the period from 1 december 2018 to 1 march 2019.the database was collected from intensive care unit about every week in the room of statistics of ICU and the data reviewed retrospectively.

The data collection include name ,age chief complaint,past history,sex,investigation include serum creatinine and blood urea

**Result**

A total 148 who were collected from our study for three month :42 developed acute kidney injury ..27 with high s.cr and 15 with high B.U while 106 did not develop acute kidney injury in intensive care unit and the most common chief complain is DLOC (disturb level of consciousness),, 17 patient with normal investigation and the other patients without investigation

Table (1) this table show the frequency and percent of AKI ,Non AKI and patients without investigations which is more in patient without investigations

|  |  |  |
| --- | --- | --- |
| PERCENT | FREQUENCY |  |
| 28.38 % | 42 | AKI |
| 11.49 % | 17 | NON AKI with normal investigations |
| 60.14 % | 89 | Patients without investigations |
| 100 % | 148 | TOTAL |

**Table (2) this table show AKI is more frequent of the patients related to chief complaint presented with disturb level of consciousness**

|  |  |  |
| --- | --- | --- |
| PERCENT | FREQUENCY |  |
| 35.71 % | 15 | AKI with DLOC |
| 23.81 % | 10 | AKI with SOB |
| 9.52 % | 4 | AKI with confusion |
| 7.14 % | 3 | AKI with RTA |
| 4.76 % | 2 | AKI with Fit |
| 2.38 % | 1 | AKI with stoke |
| 2.38 % | 1 | AKI with fever |
| 2.38 % | 1 | AKI with abdominal distension |
| 2.38 % | 1 | AKI with Bullet injury |
| 2.38 % | 1 | AKI with ca bladder |
| 2.38 % | 1 | AKI with FBM |
| 2.38 % | 1 | AKI with dizziness |
| 2.38 % | 1 | AKI (chief complaint non available) |
| 100 % | 42 | TOTAL |

**Table (3) show the patient wih AKI related to age which is more in age 45- 65 year**

|  |  |  |
| --- | --- | --- |
| **Percent** | **Patients with AKI** | **AGE** |
| **4.76 %** | **2** | **<18** |
| **16.67 %** | **9** | **18-44** |
| **28.57 %** | **15** | **45-65** |
| **38.01 %** | **14** | **>65** |
| **4.76 %** | **2** | **Age not available** |
| **100 %** | **42** | **TOTAL** |

**Table (4) show the patient with AKI related to sex which is more in males**

|  |  |  |
| --- | --- | --- |
| **percent** | **AKI** | **Sex** |
| **57.14 %** | **24** | **Male** |
| **42.86 %** | **18** | **Female** |

**Table (5) show the numbers of the patients that have acute kidney injury in intensive care unitin AL hussain teaching hospital**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Blood Urea(Mg) | Serum creatinine(MMOL) | Past history | Chief complain | Age | Patients |
|  | 1.71 | HT,CVA | Abdominal distention | 67 | Patient 1 |
|  | 4.55 | NEGATIVE | DLOC | 55 | Patient 2 |
|  | 2.50 | NEGATIVE | DLOC | 19 | Patient 3 |
|  | 1.74 | NEGATIVE | SOB | 1 DAY | Patient 4 |
|  | 1.45 | NEGATIVE | CONFUSION | 68 | Patient 5 |
|  | 3.6 | HT,DM | SOB | 63 | Patient 6 |
|  | 1.15 | HT,DM | FBM | 70 | Patient 7 |
|  | 3.45 | HT,DM | DLOC | 65 | Patient 8 |
|  | 2.41 | NEGATIVE | DLOC | 45 | Patient 9 |
|  | 5.14 | HT,DM,CVA | DLOC | 60 | Patient 10 |
|  | 2.6 | NEGATIVE | DLOC | 50 | Patient 11 |
|  | 5.5 | NEGATIVE | FIT | 50 | Patient 12 |
|  | 2.14 | CA BLADDER,DM | CA BLADDER | 50 | Patient 13 |
|  | 5.29 | HT,DM | DLOC | 90 | Patient 14 |
|  | 2.96 | HT,DM | CONFUSION | 68 | Patient 15 |
|  | 2.17 | NEGATIVE | SOB | 70 | Patient 16 |
|  | 3.93 | NEGATIVE | STROKE | 21 | Patient 17 |
|  | 2.64 | NEGATIVE | SOB | 74 | Patient 18 |
|  | 2.77 | DM | SOB | 15 | Patient 19 |
|  | 3.5 | HT | DLOC |  | Patient 20 |
|  | 1.6 | NEGATIVE | RTA | 38 | Patient 21 |
|  | 3 | NEGATIVE |  |  | Patient 22 |
| 98 |  | NEGATIVE | CONFUSION | 60 | Patient 23 |
| 140 |  | NEGATIVE | DIZZINESS | 77 | Patient 24 |
| 50 |  | HT,DM | RTA | 43 | Patient 25 |
| 100 |  | NEGATIVE | BULLET INJURY | 19 | Patient 26 |
| 72 |  | DM | DLOC | 80 | Patient 27 |
| 233 |  | NEGATIVE | DLOC | 65 | Patient 28 |
| 49 |  |  | FIT | 57 | Patient 29 |
| 53 |  | NEGATIVE | SOB | 70 | Patient 30 |
| 49 |  | NEGATIVE | CONFUSION | 42 | Patient 31 |
| 72 |  | NEGATIVE | DLOC | 80 | Patient 32 |
|  | 1.96 | NEGATIVE | SOB | 56 | Patient 33 |
|  | 1.05 | NEGATIVE | SOB | 32 | Patient 34 |
| 55 |  | NEGATIVE | SOB | 60 | Patient 35 |
| 124 |  | NEGATIVE | FEVER | 20 | Patient 36 |
| 81 |  | BRAIN TUMOR | DLOC | 49 | Patient 37 |
| 47 |  | NEGATIVE | RTA | 25 | Patient 38 |
| 68 |  | COPD | SOB | 75 | Patient 39 |
|  | 1.78 | NEGATIVE | DLOC | 58 | Patient 40 |
|  | 1.91 | NEGATIVE | DLOC | 75 | Patient 41 |
|  | 4.06 | NEGATIVE | DLOC | 90 | Patient 42 |

**Conclusion**

We conclude that AKI is common among patient with disturb level of consciousness is and shortness of breath who admitted in ICU and this need aggressive treatment and should alarm our staff for any procedure or action to minimize AKI and prevent it if possible also other measure to treat AKI to minimize conversion to CKD this study alarm us for further more expanded study to deal with subject

**Discussion**

This research was conducted in A l hussain teaching hospital Thi-Qar Iraq. The age distribution of the AKI patients in our setting ranged from one day to 90 years the result was 28.38 % .AKI develop in males more than females .when we compare our research with another research in Iran which is collected from 235 patients the result was 31% {10} and we found it is a a high percent that need action to found the cause of AKI .and the patient presented with different chief complaint such as DLOC,SOB ,RTA and the most common is Dloc

This study has several limitations , This study used the retrospective study which only relied on the medical record of patients. Some of the records were incomplete which is without investigations ,

And in first days of this research we went to room of statistics of the hospital to collect database of patients, but they refuse so we collect it in statistics of ICU after the patients are discharged

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