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Acute kidney injury in patients with respiratory infections in Al-Nasiriyah teaching hospital

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

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Content

Abstract

Background: Acute kidney injury (AKI) is a frequent complication of community acquired pneumonia (CAP).

previously reported patients with AKI coexisted with pneumo-

nia were worse than patients with either pneumonia or AKI alone.

We studied the AKI in patients with respiratory infections

and we collected the data retrospectively from intensive care unit at AL Nasiriyah teaching hospital in Al Nasiriyah-Iraq for three month from November 2021 to February 2022. And collected the investigations of the patients _ serum creatinine and blood Urea, age, sex, past history.

The Total number of patients was 100 And the percent of AKI is 23% .

Introduction

Pneumonia is a common cause of hospitalization and can be complicated by the development of acute kidney injury. Acute kidney injury is associated with major adverse kidney events (death, dialysis, and durable loss of renal function (chronic kidney disease).

Acute kidney injury is defined as an abrupt (within 48 hours) reduction in kidney function based on an elevation in serum creatinine level, a reduction in urine output, the need for renal replacement therapy (dialysis), or a combination of these factors. The term acute kidney injury should replace terms such as acute renal failure and acute renal insufficiency, which previously have been used to describe the same clinical condition.

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 In the presence of a decreased glomerular

filtration rate (GFR) secondary to hypoperfusion,7 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.

Historically, AKI has been divided into well-characterized and sequential phases, namely, initiation, maintenance and recovery8 and, more recently, Sutton et al.9 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 Recovery takes 1–2 weeks after normalization of renal perfusion, requiring repair and regeneration of tubular cells.8 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 patients with community acquired pneumonia at Al Nasiriyah 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 nasiriyah 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 november 2021 to 1 february 2022 . 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, past history, sex, investigation include serum creatinine and blood urea.

Result

A total 100 who were collected from our study for three month : 23 developed acute kidney injury ..15 with high s.cr and 8 with high B.U while 77 did not develop acute kidney injury in intensive care unit. 31 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

	Frequency	Percent
AKI	23	23%
Non AKI with Normal investigations	31	31%
Patients without investigations	46	46%
TOTAL	100	100%

Table (2) show the patient wih AKI related to age which is more in age > 65

Age	patients with AKI	percent
< 18	2	8.69%
18 _44	5	21.73%
45 _65	7	30.43%
> 65	9	39.13%
TOTAL	23	100%

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

Sex	AKI	percent
Male	14	60.86%
Female	9	39.13%

Table (4) show the numbers of the patients that have acute kidney injury in AL Nasiriyah teaching hospital

Patients	Age	Sex	Past history	Serum creatinine (mg)	Blood urea (mg)
Patient 1	70	male	HTN ,DM , CVA	2.3	
Patient 2	22	male	DM		60
Patient 3	45	female	Asthma , DM		79
Patient 4	68	female	Negative	3.45	
Patient 5	38	male	DM	1.6	
Patient 6	85	female	DM, HTN		49
Patient 7	73	female	DM, HTN, Diabetic foot		68
Patient 8	75	female	COPD		53
Patient 9	55	male	Negative		49
Patient 10	25	male	DM, Myasthenia gravis	4.06	
Patient 11	80	male	CVA, HTN		72
Patient 12	60	male	Negative	1.96	

Patient 13	63	male	DM, HTN	3.6	
Patient 14	45	female	DM	3.94	
Patient 15	1 day	male	Negative	1.74	
Patient 16	35	female	DM, HTN	2.3	
Patient 17	42	female	DM	1.6	
Patient 18	90	male	HF, HTN	2.14	
Patient 19	81	male	HF, HTN, DM		110
Patient 20	74	male	HTN , DM	2.64	
Patient 21	49	female	HTN, DM	1.78	
Patient 22	43	female	HTN, DM	1.91	
Patient 23	65	male	Negative	2.4	

Conclusion

We conclude that AKI is common among patient with CAP .Patients with CAP who developed AKI had worse in-hospital outcomes. 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 at AL-Nasiriyah teaching hospital in Thi-Qar Iraq. The age distribution of the AKI patients in our setting ranged from one day to 90 years the result was 23 % .AKI develop in males more than females . Moreover, we found that advanced age, hypertension, cardiac dysfunction, diabe- tes were independent risk factors for AKI in patients with CAP .

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.

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