EVALUATION OF HAEMATOLOGICAL PROFILE IN PATIENTS WITH ACUTE KIDNEY INJURY (AKI) - A HOSPITAL BASED STUDY

Kallol Bhattacharjee[1], Dwijen Das[2], Abu Nasar Mohd. Masroor[3], Chandra Prakash Thakur[4]

[1]Associate Professor, Department of Medicine, Silchar Medical College and Hospital, Silchar, Assam.
[2]Associate Professor, Department of Medicine, Silchar Medical College and Hospital, Silchar, Assam.
[3]Postgraduate Trainee, Department of Medicine, Silchar Medical College and Hospital, Silchar, Assam.
[4]Postgraduate Trainee, Department of Medicine, Silchar Medical College and Hospital, Silchar, Assam.

ABSTRACT

BACKGROUND

Acute kidney injury (AKI) is characterised by sudden impairment of kidney function resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys as well as impairment of acid-base balance, and water homeostasis. AKI is not a single disease but, rather, a designation for a heterogeneous group of conditions that share common diagnostic features. When this impairment is prolonged, erythropoietin secretion by this organ is decreased and toxic metabolites accumulate and cause haematological changes including decrease in Hb, HCT, MCH, MCV, RBC and platelet counts. This study evaluates haematological profile in patients with acute kidney injury.

MATERIALS AND METHODS

This study was conducted on 100 patients with acute renal impairment and equal number of age and sex matched healthy individuals. Initially patients with renal impairment were tested and after confirmation of AKI, complete blood count was performed for each patient and compared with control group and finally obtained data were analysed.

RESULTS

Comparison between patients with AKI and controls revealed that there is significant haematological changes (RBC, HCT, Hb and MCHC) in case group in comparison with control group.

CONCLUSION

It was inferred that AKI results not only in anaemia but also a number of other haematological abnormalities and is a new field for future researches as there is a dearth of literature in this arena.

KEYWORDS

Acute Kidney Injury, Haematological Profile, Anaemia.

HOW TO CITE THIS ARTICLE: Bhattacharjee K, Das D, Masroor ANM, et al. Evaluation of haematological profile in patients with acute kidney injury (AKI) - a hospital based study. J. Evid. Based Med. Healthc. 2018; 5(13), 1136-1139. DOI: 10.18410/jebmh/2018/235

BACKGROUND

Acute Kidney Injury (AKI) is characterized by sudden impairment of kidney function resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. It is typically marked by increase in BUN and serum creatinine and decreased urine production. It is described as a decrease in Glomerular Filtration Rate (GFR) and can be determined by measuring the plasma clearance of different glomerular filtration markers like inulin, ethylene-diamine-tetra-acetic-acid etc.[1,2] AKI complicates 5-7% of acute care hospital admissions and up to 30% of admissions to the intensive care unit[3]. For simplicity, the cause of AKI is divided according to sources of renal injury such as pre-renal, intrinsic-renal and post-renal. Pre-renal AKI is the most common form of AKI. Intrinsic AKI can be conceptualized anatomically according to major site of renal parenchymal damage: glomeruli, tubule-interstitium and vessels. Post renal AKI occurs when the normally unidirectional flow of urine is acutely blocked either partially or totally leading to increased retrograde hydrostatic pressure and interference with glomerular filtration.[3] AKI is a devastating clinical problem with grave prognosis if intervention is not done at the earliest. The rate of mortality depends on underlying disease and it is much more in intensive care unit setting. Approximately 50% to 80% of patients in ICU died from AKI.[4-6]

Anaemia is seen in patients with acute kidney injury but the exact relationship between them remains unclear.[7] Anaemia associated with AKI has substantial clinical and public health importance in terms of morbidity, mortality and quality of life. In addition to anaemia, AKI is also associated with bleeding tendency attributed to platelet dysfunction due to abnormal platelet aggregation and adhesiveness.[8] The exact pattern of platelet count in patients with renal failure is controversial but several

Financial or Other, Competing Interest: None.
Submission 17-02-2018, Peer Review 06-03-2018,
Acceptance 13-03-2018, Published 21-03-2018.
Corresponding Author:
Dr. Kallol Bhattacharjee,
Shivalik Park, Meherpur, Silchar,
Assam-788015.
E-mail: kbsilchar64@gmail.com
DOI: 10.18410/jebmh/2018/235

studies revealed the decrease in platelet count in renal failure.[9]

The purpose of this study is to evaluate the derangements in the haematological profile of patients with acute kidney injury and to correlate the same with the duration and severity of renal injury.

Aims and Objectives

1. To study prevalence of anaemia in Acute Kidney Injury
2. To evaluate any other haematological abnormalities in Acute Kidney Injury.

MATERIALS AND METHODS

This case-control observational study was conducted in Silchar Medical College & Hospital on 100 patients with acute kidney injury from 1st August 2016 to 31st July, 2017 with equal number of age and sex matched controls. All patients with age >18 years and acute kidney injury based on KDIGO criteria were included in the study. Patients with established kidney diseases, anaemia and also patients with use of drugs causing anaemia were excluded from the study.

KDIGO Defines AKI as Any One of the Followings

- Increase serum creatinine by 0.3 mg/dl within 48 hours OR
- Increase in serum creatinine to 1.5 times of baseline or more within the last 7 days OR
- Urine output less than 0.5 ml/kg/hr for 6 hours

Inclusion Criteria

Acute Kidney Injury based on KDIGO

Exclusion Criteria

Established kidney diseases, anaemia, patients on known nephrotoxic drugs, age below 18 years, other comorbid conditions, patients on haematinics or erythropoietin, renal transplant recipients, prostatomegaly, pregnant women.

During the study, no patients were transfused with blood or blood components such as fresh frozen plasma (FFP), platelet etc. Initially two separate blood samples were taken from each patient, 2 ml uncoagulated sample harvest for biochemical assay and EDTA anti coagulated whole blood sample for complete blood cell count. Serum were used to determine level of blood urine nitrogen (BUN) and creatinine, and then complete blood count were done with EDTA anti coagulated samples by Sysmex (kx 21 Japan).

Statistical analysis was done by SPSS software (version 22.0). P value <0.05 was considered as significant change.

RESULTS

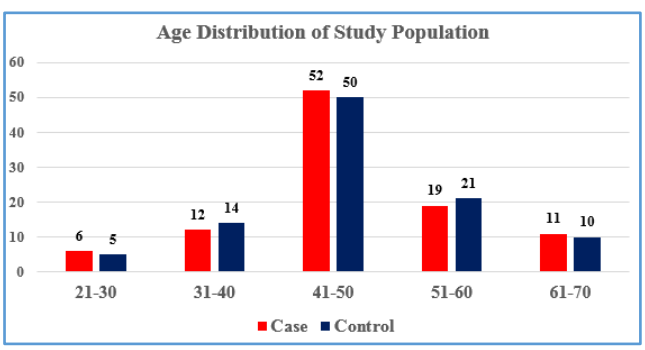
In case group (46% male, 54% female), mean age was 56±13 years. In control group (48% male, 52% female) mean age was 57±13.5 years.

The average levels of BUN and creatinine in patients with acute kidney injury were 85 ± 19 mg/dl and 2.9 ± 0.8 mg/dl respectively, and in control groups they were 19 ± 6.1 mg/dl and 1.1 ± 0.4 mg/dl.

Comparison between patients with acute kidney injury and the control group revealed that RBC count, haemoglobin and haematocrit level were significantly lower in the patient group than control group (P<0.001) (Table 2).

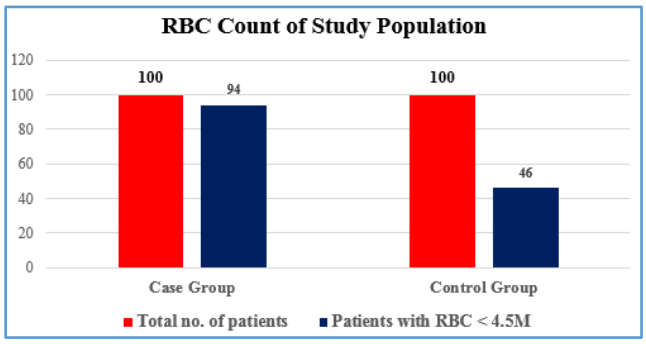
		Case	Control
Age	21-30	6	5
	31-40	12	14
	41-50	52	50
	51-60	19	21
	61-70	11	10
Sex	Male	46	48
	Female	54	52
BMI	<18.5	9	7
	18.5-24.9	66	71
	25-29.9	17	16
	>30	8	6
Co-morbidities	Hypertension	17	7
	Diabetes Mellitus	11	4
	Coronary Artery Disease	9	5
	Dyslipidaemia	12	8
Previous history of AKI		7	1

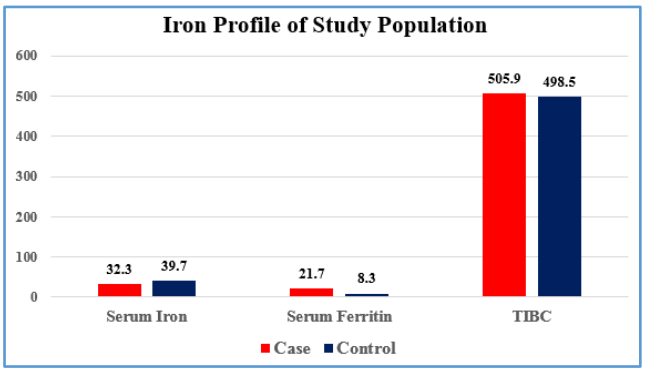
Table 1. Demographic Profile of Study Population



Index		Number	Mean	Std. Deviation	P. value
RBC (×10^9/µL)	Case	100	3.57	0.71	<0.0001
	Control	100	4.38	0.33	
Hb (g/dL)	Case	100	9.28	2.25	<0.001
	Control	100	12.88	2.17	
HCT (%)	Case	100	33.04	3.51	<0.0001
	Control	100	42.72	1.5	
MCV (fl)	Case	100	84.35	7.12	0.169
	Control	100	86.24	6.47	
MCH (pg)	Case	100	28.02	2.56	0.058
	Control	100	27.09	2.27	
MCHC (g/dl)	Case	100	31.92	2.21	0.0078
	Control	100	33.25	2.66	
RDW (%)	Case	100	14.64	1.82	0.0002
	Control	100	12.5	1.11	
PLT (×10^3/µl)	Case	100	188.42	77.59	0.171
	Control	100	208.6	68.74	
WBC (×10^3/µl)	Case	100	6.4	4.03	0.341
	Control	100	9.46	3.63	

Table 2. Comparison of Haematological Indices Between Patients with Acute Kidney Injury and Healthy Individuals





Index		Number	Mean	Std. Deviation	p. value
BUN(mg/dl)	Case Control	100 100	56.45 13.26	3.63 3.98	0.00001
Creatinine(mg/dl)	Case Control	100 100	2.44 0.78	0.72 0.31	0.00001

Table 3. BUN & Serum Creatinine in Study Population

Signs & Symptoms	Total No. (n=100)	Male (n=46)	Female (n=54)	Chi- Square	P- Value	Significance
Oliguria	82	38	44	0.711	0.39	NS
Fever	70	32	38	0.08	0.76	NS
Vomiting	90	39	51	1.82	0.17	NS
Loose Motions	30	14	16	0.08	0.76	NS
Oedema	27	11	16	0.16	0.68	NS
Fatigue	74	30	44	0.02	0.86	NS
Haematuria	17	8	9	0.56	0.45	NS
Jaundice	26	11	15	0.97	0.32	NS
Hypotension	29	12	17	0.15	0.69	NS

Table 4. Clinical Manifestations of AKI

Iron Profile	Case	Control	P-value
Serum Iron (µg/dL)			
Mean ± SD	32.3 ± 7.9	39.7 ± 9.4	0.07
Median	31	40	
Serum Ferritin (µg/dL)			
Mean ± SD	21.7 ± 13.9	8.3 ± 4.9	0.001
Median	15.6	7.3	
TIBC (µg/dL)			
Mean ± SD	505.9 ± 95.6	498.5 ± 97.5	0.82
Median	490	473.5	
Transferrin Saturation (%)			
Mean ± SD	6.9 ± 4.01	8.2 ± 2.3	0.68
Median	5.9	8.2	

Table 5. Serum Iron Profile of Patients with AKI and Healthy Individuals

DISCUSSION

Renal failure is a condition where there is inadequate removal of toxins and waste products by kidneys from the blood. Anaemia is common in AKI. In summary anaemia of AKI is a multifactorial process due to relative EPO deficiency, uremic induced inhibitors of erythropoiesis, shortened erythrocyte survival, disordered iron homeostasis, and hepcidin excess. In established AKI, there is an increased risk of bleeding and spontaneous gastrointestinal haemorrhage due to the uraemia[10]. In addition to anaemia, several studies showed the decrease of platelet count in renal failure.

Oliguria, vomiting, fever and loose motion are the predominant symptoms in AKI in our study. It is observed that clinical features are almost in accordance with studies conducted earlier (Maulita P Kapadia et. al.)[11]

Our study revealed that some red blood cell indices including RBC count, Hb, HCT and MCHC levels were significantly lower in-patient group in comparison with healthy individual (P<0.05).

In the study of Michele Hales et al, anaemia was present in 91% of patients with AKI as a result of increase in urea and presence of oliguria.[7] In the present study 87% had anaemia during their hospital stay. In the study of Michele Hales, forty-three of the patients had a haematocrit below 30%, but this finding was not observed in our patients and only 12.5 percent of patients had a haematocrit lower than 30 percent. The probable cause of this discrepancy is the serum urea level. Because Michele Hales et al., found a significant correlation between maximum serum urea and severity of anaemia thus a higher serum urea in their patients in comparison with our patients can be the reason of a higher percent of patients with lower haematocrit level.

In another study by Mishra S K et al[12] anaemia was present in 60% of the patients with malarial AKI. Although anaemia is commonly associated finding, Powell-Tuck et al.[13] in their study in critically ill patients with AKI stage 1 observed that anaemia was not associated with an increased risk of progression to more severe AKI.

In our study, it is noticed that serum iron, serum ferritin are low in patients with AKI which is significant for serum ferritin (p<0.01), which is not corroborative with study by Mavromatidis K, et al[14]. where they observed a higher level of ferritin in AKI and they further compared the same with another group of cases with infectious aetiology without AKI or CKD which is not the modus operandi in our series.

Our study also revealed that acute kidney injury did not cause significant thrombocytopenia. (p=0.171**)** which is non-corroborative with the study by Hassanein AA et al. Due to the scarcity of researches in this field, the findings observed on meticulous analysis of our series could not be compared with related works in this arena.

CONCLUSION

Haematological abnormalities in AKI is a relatively untrodden path for the researchers and very few studies pertaining to the subject are available in the world literature to put forward a universally acceptable concluding remark. However, the inference derived from the study that anaemia is a major manifestation/association of AKI and the accompanying haematological abnormalities in AKI observed in the study may go a long way in pursuit of future research in this field.

Limitation of the Study

This study was conducted in a limited number of patients for a short duration in a single centre. It is recommended that multi-centric studies involving greater number of patients for a longer duration will throw more light in this important aspect where there is enormous scope for future researchers.

REFERENCES

[1] Prasad N, Barai S, Gambhir S, et al. Comparison of glomerular filtration rate estimated by plasma clearance method with modification of diet in renal disease prediction equation and gates method. Indian J Nephrol 2012;22(2):103-107.

[2] Russell CD, Bischoff PG, Kontzen FN, et al. Measurement of glomerular filtration rate: single injection plasma clearance method without urine collection. J Nucl Med 1985;26(11):1243-1247.

[3] Waikar SS, Bonventre JV. Acute Kidney Injury. In: Kasper D, Fauci A, Hauser S, et al, eds. Harrison's principles of internal medicine. Vol. 2. 19th edn. McGraw-Hill Education 2015:1799-1811.

[4] Haller M, Schelling G. Acute kidney failure. Physiopathology--clinical diagnosis--therapy. Anaesthesist 2000;49(4):349-352.

[5] Star RA. Treatment of acute kidney injury. Kidney Int 1998;54(6):1817-1831.

[6] Thadhani R, Pascual M, Bonventre JV. Acute renal failure. N Engl J Med 1996;334(22):1448-1460.

[7] Hales M, Solez K, Kjellstrand C. The anemia of acute renal failure: association with oliguria and elevated blood urea. Ren Fail 1994;16(1):125-131.

[8] Hassanein AA, McNicol GP, Douglass AS. Relationship between platelet function tests in normal and uremic subjects. J Clin Path 1970;23:402-406.

[9] Gafter U, Bessler H, Malachi T, et al. Platelet count and thrombopoietic activity in patients with chronic renal failure. Nephron 1987;45(3):207-210.

[10] Goddard J, Turner AN. Acute kidney injury. In: Walker B, Colledge NR, Ralston S, et al, eds. Davidson's principles and practice of medicine. 22nd edn. Churchill Livingstone 2014: p. 479.

[11] Kapadia MP, Kamdar PK, Jha PR. A study of clinical profile of patients with acute kidney injury in a tertiary care centre. International Journal of Advances in Scientific Research 2016;2(8):160-166.

[12] Mishra SK, Mohanty S, Mohanty A, et al. Management of severe and complicated malaria. J Postgrad Med 2006;52(4):281-287.

[13] Powell-Tuck J, Crichton S, Raimundo M, et al. Anaemia is not a risk factor for progression of acute kidney injury: a retrospective analysis. Crit Care 2016;20:52.

[14] Mavromatidis K, Fytil C, Kynigopoulou P, et al. Serum ferritin levels are increased in patients with acute renal failure. Clin Nephrol 1998;49(5):296-298.