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


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.1-3 AKI complicates 5-7% of acute care hospital admissions and up to 30% of admissions to the intensive care unit.4 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.5 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.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
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.

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 samples by Sysmex (kx 21 Japan). Serum were used to determine level of blood urine nitrogen (BUN) and creatinine in patients for biochemical assay and EDTA anti coagulated whole blood for complete blood cell count. Serum were used to determine level of blood urine nitrogen (BUN) and creatinine in patients for biochemical assay and EDTA anti coagulated samples by Sysmex (kx 21 Japan). Serum were used to determine level of blood urine nitrogen (BUN) and creatinine in patients for biochemical assay and EDTA anti coagulated samples by Sysmex (kx 21 Japan). Serum were used to determine level of blood urine nitrogen (BUN) and creatinine in patients for biochemical assay and EDTA anti coagulated samples by Sysmex (kx 21 Japan).

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).
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, uraemic 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. 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.)

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. 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 anaemia was present in 60% of patients with malarial AKI. Although anaemia is commonly associated finding, Powell-Tuck et al. 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 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**


