



RESEARCH ARTICLE

CORRELATION OF COMPLETE BLOOD COUNT PARAMETERS WITH SERUM UREA AND CREATININE IN ACUTE RENAL FAILURE PATIENTS

Arpita Chakraborty and *Dr. Ravindra Maradi

Department of Biochemistry, Kasturba Medical College, Manipal, Manipal University, Manipal, India- 571604

ARTICLE INFO

Article History:

Received 13th August, 2017
Received in revised form
28th September, 2017
Accepted 06th October, 2017
Published online 30th November, 2017

Key words:

Hemoglobin, Lymphocyte,
Neutrophil, Basophil,
Acute renal failure,
Urea, Creatinine.

ABSTRACT

Acute renal failure (ARF) is a sudden, sharp decline in the function of kidneys as a result of an acute toxic or inadequate oxygen supply to the kidneys. Abnormality in complete blood count parameters occur in kidney failure. The aim of the present study is to correlate blood count parameters with serum urea and creatinine in acute renal failure patients. Spearman's rho correlation was used to correlate hemoglobin, neutrophils, lymphocyte and basophil levels with serum urea and creatinine in which hemoglobin was estimated by photometric method, lymphocytes, neutrophils and basophils by VCS Technology by Beckman Coulter and urea and creatinine by the Roche Cobas 6000 autoanalyser. This study included 102 diagnosed ARF cases taken from nephrology ward of Kasturba Hospital, Manipal. Our study showed decline in hemoglobin levels in ARF patients. This decline in hemoglobin levels may be due to decreased production of erythropoietin in ARF cases. A positive correlation of neutrophils with urea in ARF cases may be due to ischemia and inflammation in the kidneys leading to enhanced retention of toxic substances. As a result we can conclude that complete blood count parameters play a significant role in the pathophysiology of ARF cases.

Copyright © 2017, Arpita Chakraborty and Dr. Ravindra Maradi. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Citation: Arpita Chakraborty and Dr. Ravindra Maradi, 2017. "Correlation of complete blood count parameters with serum urea and creatinine in acute renal failure patients", *International Journal of Current Research*, 9, (11), 61030-61033.

INTRODUCTION

The vital role of kidney is to eliminate the waste materials from the body. Renal failure can result into diminished glomerular filtration rate (GFR). The plasma clearance of different glomerular filtration markers such as creatinine, urea, inulin etc can be determined by GFR (Barai *et al.*, 2012; Russell *et al.*, 1984). Homeostasis is responsible for normal functioning of the kidney. Renal failure is classified into two categories namely chronic and acute. ARF is characterized by sudden decrease in filtration function of the kidney, intrinsic damage to the kidney and obstruction to urine flow (Haller and Schelling, 2000). ARF is marked by elevation in blood urea nitrogen (BUN), serum creatinine and diminished production of urine (Thadhani *et al.*, 1996). It usually takes place when the GFR is reduced to less than 10ml/min. When excretory function of the kidneys are found to decline over hours or days ARF is diagnosed. The incidence of ARF increases with age and co-morbidity. ARF develops in a rapid manner which is basically due to rapid electrolyte, acid base and fluid imbalances which becomes often uncontrollable. The biochemical evaluations which lead to the development of ARF include rapid elevation in urea and creatinine levels.

Hyperkalemia and metabolic acidosis are the severe and life threatening metabolic derangements. Agents that unexpectedly lead to Acute Kidney Injury (AKI) may impair dilation of afferent arterioles or efferent vasoconstriction of inhibitors like angiotensin- converting enzyme [ACE] and angiotensin receptor blockers [ARBs] (Johnson *et al.*). Mostly all forms of acute glomerulonephritis can be present as AKI. Acute Kidney Injury Network criteria stated increase of serum creatinine up to a level of 3mg/dl in acute renal failure cases within 48 hours. The causes of ARF are divided into pre-renal, renal and post-renal. Blood flow is limited to the kidney in pre renal failure. The capacity to restore blood flow of kidney fails during the functional pre-renal stage leading to ischemic acute tubular necrosis (ATN) and tubular cell injury. It is caused by hemorrhage, major surgery, cardiogenic shock, septicemia, severe diarrhea and vomiting. In the earlier stages, kidney function is normal with kidneys attempting to replenish water and electrolyte loss. The stimulation of rennin angiotensin aldosterone system and vasopressin secretion due to volume depletion result in the increased reabsorption of sodium and water by the kidneys. As a result, urine volume decreases and urine sodium concentration is lower. Because of volume contraction and tubular reabsorption, plasma urea levels are increased. Renal ARF may result from damage to interstitium, glomeruli or kidney tubules. Post renal ARF is associated with urinary tract obstruction such as prostate enlargement, and urinary calculi (Aror). Kidney also serves as an important

*Corresponding author: Dr. Ravindra Maradi

Department of Biochemistry, Kasturba Medical College, Manipal, Manipal University, Manipal, India- 571604.

endocrine organ which synthesizes the essential hormones required for performing the vital activities in the body. One of the essential hormones is erythropoietin (EPO) which produces red blood cells by stimulating bone marrow. Diminished levels of RBCs take place inside the body as the affected kidney fails to produce EPO as in normal conditions. Low hemoglobin levels occur in kidney failure as the amount of hemoglobin varies according to the number of RBCs in blood. Moreover increased toxicity in the body due to abnormal functioning of kidneys can shorten the life span of RBCs which can lead to kidney failure (Huaxia Kidney Disease Hospital, 2013). When biopsy of kidney is carried out neutrophils and mononuclear cells are seen in peritubular capillaries (Johnson *et al.*).

A number of studies have also demonstrated that cells of the adaptive immune system, including B and T lymphocytes contribute to renal injury in models of acute tubular necrosis (ATN). Ischemic reperfusion injury which has been experimented may be improved by deficiency of T or B cells. It is not known whether these responses are antigen specific. Furthermore, some B and T cell subsets such as T regulatory cells, help to limit renal injury. Basophils are generally present in the blood of peripheral tissues, spleen and bone marrow. They migrate into lymph nodes and tissues during inflammation. The aim of the present study is to correlate blood count parameters with serum urea and creatinine in acute renal failure patients. This study helps to know the changes and possible causes for the alteration of blood count parameters in acute renal failure cases.

MATERIALS AND METHODS

This is a retrospective study, in which 102 diagnosed ARF cases were taken from nephrology ward of Kasturba Hospital, Manipal after Institutional ethical committee clearance was taken. Hemoglobin, basophil, lymphocyte and neutrophil counts along with urea and creatinine tests were conducted in all the cases. Hemoglobin was estimated by photometric method. Other parameters like neutrophils, lymphocytes and basophils were estimated by VCS Technology/ light microscopy by Beckman Coulter. Urea and creatinine were estimated by the Roche cobas 6000 autoanalyser. Spearman's rho correlation was used to correlate hemoglobin levels with serum urea and creatinine.

RESULTS

Urea showed negative correlation with hemoglobin ($p=.015$, $r=-.24$), lymphocyte ($p=.012$, $r=-.247$) and basophil ($p=.003$, $r=-.292$) and a positive correlation with neutrophil ($p=.024$, $r=.223$) in ARF cases. However creatinine did not show any significant correlation with any of the parameters in the ARF cases.

Table 1. Correlation of complete blood count parameters with urea

| | Urea (mg/dl) | |
|----------------------------------|--------------|------------|
| Total Number of Blood Parameters | P value | R value |
| Hemoglobin (g/dl) | P=0.015 | $r=-0.24$ |
| Neutrophil (%) | P=0.024 | $r=0.223$ |
| Lymphocyte (%) | P=0.012 | $r=-0.247$ |
| Basophil (%) | P=0.003 | $r=-0.292$ |

P*- Spearman's rho significant correlation

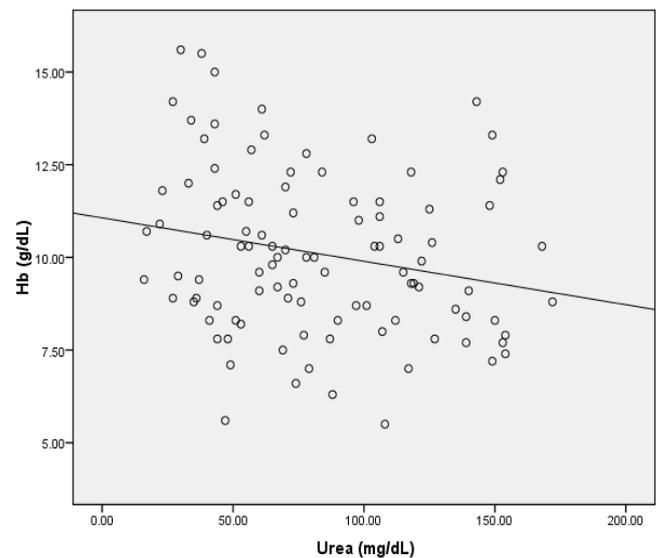


Fig. 1. Correlation of hemoglobin with urea in acute renal failure patients

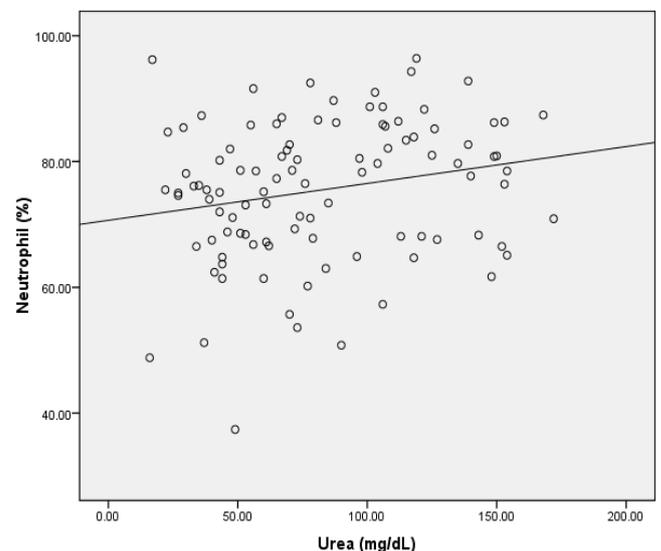


Fig. 2. Correlation of neutrophils with urea in acute renal failure patients

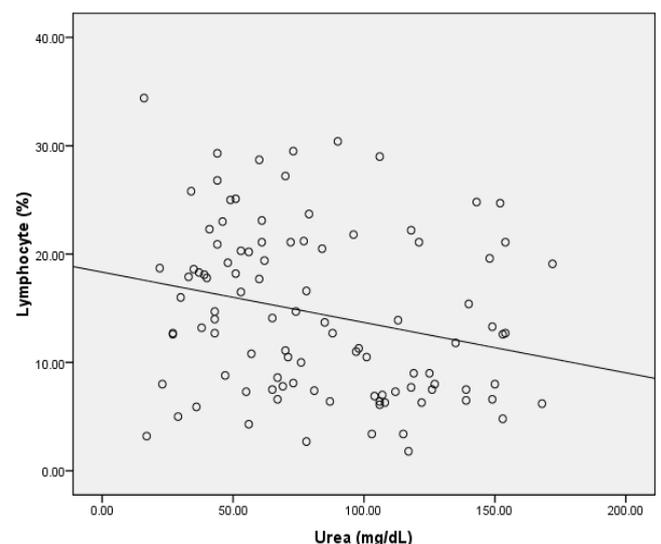


Fig. 3. Correlation of lymphocytes with urea in acute renal failure patients

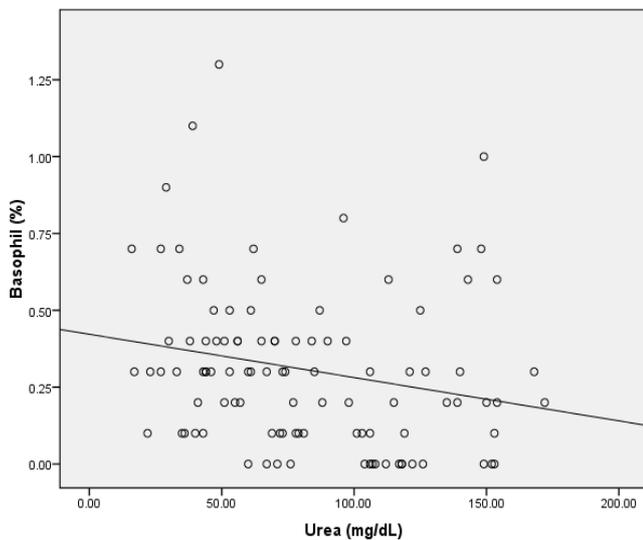


Fig. 4. Correlation of basophils with urea in acute renal failure patients

DISCUSSION

In acute renal failure, there will be damage to the kidneys leading to increase in the blood urea and creatinine levels. Blood urea level varies with dietary intake of proteins. When there is inadequate intake of food, body utilizes stored carbohydrates, fats and proteins as the energy source. Excess breakdown of body protein leads to excess catabolism of amino acids and excess production of ammonia and urea. Study of Akbar Dorgalaleh et al revealed that patients with acute renal failure were considerably detected with anemia (Akbar Dorgalaleh *et al.*, 2013). Our study also showed the decrease in hemoglobin level in acute renal failure patients. This decrease in hemoglobin may be due to the decreased production of erythropoietin in acute renal failure cases. As a result the bone marrow makes fewer red blood cells leading to anemia. The weak negative correlation between serum creatinine and hemoglobin compared to serum urea and hemoglobin may be due to constant production of creatinine from the muscle and urea production depends on dietary intake and break down of protein. The other reason for weak negative correlation between creatinine and hemoglobin may be due to the ARF patients undergoing dialysis which we did not consider in our study. Controversies had been there regarding the role of neutrophils in ARF. Experimental studies have shown that activation of neutrophils and release of proteases and reactive oxygen species can increase the severity of injury (Johnson *et al.*). Although the chance of acute renal failure develops due to the partial activation of primed neutrophils and ischemic organs studies have not shown any interaction between PMN and ischemic organs in any biological system. Study of Linas et al showed that when attacked by polymorphonuclear (PMN) cells normal kidneys usually show resistant behavior whereas partially ischemic kidneys show resistant behavior to PMN which remains untreated. ARF is associated with decreased blood supply to the kidney which activates PMN by the release of oxidants or proteases (Linas *et al.*, 1992). Neutrophils are associated with the activation of complement and decreased recovery from ARF. In conclusion, in ischemic acute renal failure neutrophils play a major contribution. It is still unclear whether decline in neutrophil function advances recovery in ARF (Lauriat *et al.*, 1998). Our study showed a positive correlation with neutrophil with urea

in acute renal failure cases which may be due to the ischemia and inflammation in the kidney leading to the enhanced retention of toxic substances. To illustrate the destructive effects of infiltration or activation of T cells in kidney disease several pharmacological and genetic methods have been used to manage immune cell function in experimental animals. Many studies have been done in recent times about the effector mechanisms of infiltrating cells to describe subtypes of T cells responsible for these effects.

The article by Hyodo et al demonstrates that development of anti-glomerular basement membrane glomerulonephritis (anti-GBM GN) is basically due to a vital role of effector memory T lymphocytes (TEM cells) and macrophages (Hyodo *et al.*, 201). A significant correlation between influx of mast cells and serum creatinine concentration has been revealed by a past study (Hiromura *et al.*, 1998). In renal disorders such as chronic glomerulonephritis, IgA nephritis and, rapidly progressive glomerulonephritis and renal transplantation (Holdsworth *et al.*, 2008; Mengel *et al.*, 2008) basophils has sought a major attention in the development of tissue fibrosis. No experimentally studies have suggested or proved the role of mast cells or basophils leading to renal deterioration in glomerular disease by making interstitial fibrosis to take place. However, the specific role of mast cells in the advancement of kidney disease and fibrosis have remained determined. For detection and functional analysis of basophils in kidney disease data on the role of basophils and mast cells are limited because of the unavailability of advanced techniques. Basophils and mast cells could have played a vital role in the inflammation of kidney based on observational and experimental evidences from human and animal studies. Therefore, in future improved techniques should be devised to examine the prominent role of these cells in kidney diseases. Our study showed negative correlation of lymphocytes and basophils with urea which may be due to ARF patients undergoing dialysis and antibiotic treatments.

Conclusion

Complete blood count parameters play a significant role in the pathophysiology of ARF. By taking care of these parameters can help in reducing the damage caused to the kidneys and as well as the overall health of the patients.

Limitations

We have not considered patients undergoing dialysis and other treatments.

Acknowledgement

I would like to express my deepest gratitude to the Medical Superintendent and Medical Records Department of Kasturba Hospital Manipal to identify the patients with acute renal failure and my guide for helping me throughout the project.

REFERENCES

- Akbar Dorgalaleh, Mohammad Mahmudi, Shadi Tabibian, Zahra Kashani Khatib, GholamHossein Tamaddon, Esmaeil Sanei Moghaddam, Taregh Bamedi, Shaban Alizadeh, and Eshagh Moradi, 2013. Anemia and Thrombocytopenia in Acute and Chronic Renal Failure. *Int J HematolOncol Stem Cell Res.*, 7(4): 34–39.

- Aroor, A. R. Medical Biochemistry 5th Edition, Acute Renal Failure, pgnos 723-724.
- Barai, S., Parasar, D., Gupta, A., Prasad, N., Gambhir, S., Ora, M. and Sharma, R. 2012. Comparison of glomerular filtration rate estimated by plasma clearance method with modification of diet in renal disease prediction equation and Gates method. *Indian Journal of Nephrology*, 22(2), p.103.
- Haller, M. and Schelling, G. 2000. Akutes Nierenversagen. *Der Anaesthetist*, 49(4), pp.349-352.
- Hirumura, K., Kurosawa, M., Yano, S. and Naruse, T. 1998. Tubulointerstitial mast cell infiltration in glomerulonephritis. *American Journal of Kidney Diseases*, 32(4), pp.593-599.
- Holdsworth, S. and Summers, S. 2008. Role of Mast Cells in Progressive Renal Diseases. *Journal of the American Society of Nephrology*, 19(12), pp.2254-2261.
- Huaxia Kidney Disease Hospital. Low Hemoglobin in Kidney Failure. <http://www.kidneyabc.com/kidney-failure-symptoms/2089.html>. 2013-08-26.
- Hyodo, T., Oda, T., Kikuchi, Y., Higashi, K., Kushiyama, T., Yamamoto, K., Yamada, M., Suzuki, S., Hokari, R., Kinoshita, M., Seki, S., Fujinaka, H., Yamamoto, T., Miura, S. and Kumagai, H. 2010. Voltage-gated potassium channel Kv1.3 blocker as a potential treatment for rat anti-glomerular basement membrane glomerulonephritis. *AJP: Renal Physiology*, 299(6), pp.F1258-F1269.
- Johnson J. Richard, Feehally John, Floege Jurgen, Comprehensive Clinical Nephrology 5th Edition, Pathophysiology and Etiology of Acute Kidney Injury, pg no 808.
- Lauriat S, Linas SL. 1998. The role of neutrophils in acute renal failure. *Semin Nephrol.*, 18(5):498-504.
- Linas, S., Whittenburg, D., Parsons, P. and Repine, J. 1992. Mild renal ischemia activates primed neutrophils to cause acute renal failure. *Kidney International*, 42(3), pp.610-616.
- Mengel, M., Reeve, J., Bunnag, S., Einecke, G., Sis, B., Mueller, T., Kaplan, B. and Halloran, P. 2008. Molecular Correlates of Scarring in Kidney Transplants: The Emergence of Mast Cell Transcripts. *American Journal of Transplantation*, 9(1), pp.169-178.
- Russell, C., Bischoff, P., Kontzen, F., Rowell, K., Yester, M., Lloyd, L., Tauxe, W. and Dubovsky, E. 1984. Measurement of glomerular filtration rate by the single injection method without urine collection: a comparison of single sample, double sample, and multiple sample methods using ytterbium-DTPA. *Clinical Nuclear Medicine*, 9(Supplement), p.P62.
- Thadhani, R., Pascual, M. and Bonventre, J. 1996. Acute Renal Failure. *New England Journal of Medicine*, 334(22), pp.1448-1460.
