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ORIGINAL ARTICLE

CHANGES IN BLOOD BIOCHEMISTRY IN ACUTE RENAL FAILURE

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Abstract

Acute renal failure, currently referred to as acute kidney injury (AKI), is a clinical syndrome characterized by the rapid decline of renal function over hours to days, resulting in impaired excretion of metabolic waste products, disturbances of electrolyte homeostasis, acid-base imbalance, and accumulation of uremic toxins. Blood biochemical abnormalities develop early during the disease process and serve as essential indicators for diagnosis, assessment of disease severity, therapeutic monitoring, and prediction of clinical outcomes. The present study aimed to evaluate biochemical alterations occurring in patients with acute renal failure and to determine their clinical significance during diagnosis and management. Comprehensive analysis demonstrated that elevations of serum creatinine, blood urea nitrogen, potassium, phosphorus, inflammatory biomarkers, and metabolic acids are accompanied by reductions in glomerular filtration rate, serum bicarbonate, calcium, hemoglobin, and albumin concentrations. Recognition of these biochemical changes enables early intervention, optimization of treatment strategies, and reduction of complications associated with acute kidney injury

Keywords: acute kidney injury, acute renal failure, serum creatinine, blood urea nitrogen, electrolytes, metabolic acidosis, biochemical markers, renal dysfunction, nephrology, laboratory diagnosis.

1. Introduction

Acute renal failure remains one of the most serious medical emergencies encountered in nephrology and intensive care medicine. It is characterized by a sudden deterioration of kidney function resulting in reduced glomerular filtration, impaired regulation of body fluids, electrolyte disturbances, accumulation of nitrogenous waste products, and disruption of acid-base homeostasis. The condition develops within a relatively short period and may occur in hospitalized patients, critically ill individuals, postoperative patients, and those suffering from severe infections, trauma, cardiovascular disorders, or nephrotoxic drug exposure.

The incidence of acute kidney injury has increased considerably during recent decades because of aging populations, greater prevalence of diabetes mellitus, hypertension, cardiovascular disease, sepsis, and increasing use of complex surgical procedures. Despite significant improvements in intensive care management, acute kidney injury continues to be associated with high morbidity,

prolonged hospitalization, increased healthcare costs, and substantial mortality.

The kidneys perform numerous physiological functions extending beyond filtration of metabolic waste. They regulate extracellular fluid volume, maintain electrolyte balance, participate in acid-base regulation, produce erythropoietin, activate vitamin D, and contribute to endocrine and metabolic homeostasis. Acute impairment of these functions rapidly produces measurable biochemical abnormalities in blood, making laboratory evaluation fundamental for diagnosis and clinical monitoring.

Serum creatinine remains the most widely utilized biomarker for assessment of renal function. Progressive elevation of serum creatinine reflects declining glomerular filtration rate and remains an essential criterion in the diagnosis and staging of acute kidney injury. Nevertheless, creatinine concentration alone may not accurately represent the earliest stages of renal injury because measurable elevation frequently occurs after substantial nephron dysfunction has already developed.

Blood urea nitrogen also increases as renal excretory capacity declines. Although influenced by hydration status, protein intake, gastrointestinal bleeding, and catabolic state, elevated blood urea nitrogen provides valuable information regarding the severity of renal dysfunction when interpreted together with serum creatinine and clinical findings.

Electrolyte disturbances constitute another characteristic biochemical feature of acute renal failure. Hyperkalemia develops because of impaired renal potassium excretion and represents one of the most life-threatening complications owing to its association with fatal cardiac arrhythmias. Simultaneously, phosphorus accumulates, calcium concentrations frequently decline, sodium balance becomes disturbed, and magnesium abnormalities may occur depending on the underlying etiology and disease severity.

Metabolic acidosis develops as impaired renal function reduces hydrogen ion excretion and bicarbonate regeneration. Progressive reduction in serum bicarbonate concentration contributes to cardiovascular instability, impaired myocardial contractility, respiratory compensation, protein catabolism, and systemic inflammatory activation. Persistent metabolic acidosis is considered an important indicator of severe renal dysfunction requiring urgent therapeutic intervention.

Recent advances in laboratory medicine have introduced novel biomarkers including cystatin C, neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), interleukin-18, liver-type fatty acid-binding protein, and tissue inhibitor of metalloproteinases. These biomarkers allow earlier detection of tubular injury before substantial elevation of serum creatinine becomes evident, thereby facilitating prompt diagnosis and initiation of nephroprotective therapy.

In addition to renal-specific biomarkers, inflammatory mediators such as C-reactive protein, procalcitonin, and various cytokines frequently increase during acute kidney injury, particularly when associated with sepsis or systemic inflammatory disorders. These biochemical alterations provide additional information regarding disease severity, prognosis, and therapeutic response.

The present study aimed to investigate changes in blood biochemistry occurring during acute renal failure and to evaluate the clinical significance of biochemical markers in diagnosis, disease monitoring, therapeutic decision-making, and prediction of patient outcomes.

2. Materials and Methods

This prospective observational study was performed between January 2023 and April 2025 at the departments of nephrology, emergency medicine, and intensive care of tertiary referral hospitals. The objective of the investigation was to evaluate alterations in blood biochemical parameters among patients with acute renal failure and determine their diagnostic and prognostic significance during clinical management.

A total of 214 adult patients diagnosed with acute kidney injury were included in the study. Diagnosis was established according to internationally accepted clinical criteria based on rapid elevation of serum creatinine, reduction in urine output, and decline in estimated glomerular filtration rate. Patients were enrolled within the first forty-eight hours after diagnosis to ensure evaluation of early biochemical alterations.

Participants were classified according to disease severity into three groups. Group I included patients with mild acute kidney injury, Group II consisted of individuals with moderate renal dysfunction, and Group III included patients with severe acute kidney injury requiring intensive monitoring or renal replacement therapy.

Patients with pre-existing end-stage chronic kidney disease, previous kidney transplantation, active

malignant disease, pregnancy, or incomplete laboratory data were excluded to minimize confounding variables.

Comprehensive clinical evaluation included demographic characteristics, underlying causes of acute kidney injury, blood pressure, body temperature, heart rate, fluid balance, urine output, previous medical history, medication exposure, diabetes mellitus, hypertension, cardiovascular disease, and septic complications.

Venous blood samples were collected immediately after hospital admission and subsequently at regular intervals during hospitalization. Laboratory analysis included serum creatinine, blood urea nitrogen, cystatin C, sodium, potassium, chloride, calcium, phosphorus, magnesium, bicarbonate, uric acid, glucose, total protein, albumin, complete blood count, liver enzymes, C-reactive protein, procalcitonin, lactate, coagulation profile, and arterial blood gas analysis.

Estimated glomerular filtration rate was calculated using internationally validated equations.

Urinalysis included urine microscopy, urinary sodium concentration, urinary protein excretion, urine osmolality, and urinary sediment examination to differentiate prerenal, intrinsic renal, and postrenal causes of acute kidney injury.

Renal ultrasonography was performed in all patients to exclude urinary tract obstruction and evaluate renal morphology. Doppler ultrasonography was additionally performed when renal vascular abnormalities were suspected.

Therapeutic management consisted of individualized fluid resuscitation, optimization of hemodynamic status, discontinuation of nephrotoxic medications, correction of electrolyte disturbances, antimicrobial therapy when infection was present, nutritional support, and renal replacement therapy for patients fulfilling accepted clinical indications.

3. Results

Clinical evaluation demonstrated progressive deterioration of blood biochemical parameters with increasing severity of acute kidney injury. Serum creatinine increased significantly during the initial stages of disease and remained the principal laboratory indicator of declining glomerular filtration. Patients with severe acute kidney injury exhibited serum creatinine concentrations several times higher than baseline values.

Blood urea nitrogen also increased markedly throughout disease progression. Higher concentrations were particularly evident among patients with dehydration, septic shock, gastrointestinal bleeding, and severe catabolic states. Combined elevation of serum creatinine and blood urea nitrogen strongly correlated with reduced renal filtration capacity.

Cystatin C concentrations increased earlier than serum creatinine in many patients, indicating that this biomarker may provide earlier recognition of renal dysfunction before conventional laboratory abnormalities become pronounced. Elevated cystatin C was associated with prolonged hospitalization and greater likelihood of intensive care admission.

Electrolyte disturbances represented one of the most significant biochemical abnormalities observed during hospitalization. Hyperkalemia developed frequently among patients with advanced renal dysfunction and was associated with electrocardiographic abnormalities requiring urgent therapeutic intervention. Progressive potassium accumulation increased the risk of life-threatening cardiac arrhythmias.

Serum phosphorus concentrations increased significantly as renal excretory function deteriorated, whereas total serum calcium gradually decreased because of impaired vitamin D activation and phosphate retention. These abnormalities contributed to neuromuscular dysfunction and metabolic complications during severe acute kidney injury.

Metabolic acidosis became increasingly pronounced with disease progression. Declining serum bicarbonate concentrations and reduced arterial blood pH reflected impaired renal acid excretion. Patients with severe metabolic acidosis demonstrated greater hemodynamic instability, respiratory compensation, and higher mortality risk compared with individuals maintaining near-normal acid-base balance.

Inflammatory biomarkers including C-reactive protein and procalcitonin were significantly elevated among patients with septic acute kidney injury. These biochemical changes reflected activation of systemic inflammatory pathways and correlated with disease severity, multiorgan dysfunction, and prolonged intensive care treatment.

Serum albumin concentrations gradually declined in critically ill patients because of inflammation,

increased vascular permeability, and impaired protein synthesis. Hypoalbuminemia was associated with longer hospitalization, delayed renal recovery, and poorer overall prognosis.
Anemia developed progressively during hospitalization, particularly among patients requiring prolonged intensive care or renal replacement therapy. Reduced hemoglobin concentrations contributed to impaired tissue oxygen delivery and delayed recovery of renal function.

4. Discussion

The present investigation confirms that blood biochemical abnormalities provide essential information regarding diagnosis, severity assessment, therapeutic monitoring, and prognosis in acute kidney injury. Laboratory alterations occur rapidly following renal dysfunction and reflect the loss of filtration, excretory, metabolic, and endocrine functions of the kidneys.
Elevation of serum creatinine remains the most widely accepted diagnostic indicator of acute kidney injury. Nevertheless, reliance on creatinine alone may delay diagnosis because significant nephron dysfunction often precedes measurable increases. Early biomarkers such as cystatin C may therefore improve recognition of renal injury and facilitate prompt therapeutic intervention.
Hyperkalemia remains one of the most dangerous biochemical complications of acute kidney injury because of its direct effects on cardiac conduction and myocardial excitability. Immediate identification and correction are essential to prevent fatal ventricular arrhythmias and sudden cardiac death.
Metabolic acidosis contributes significantly to clinical deterioration through impaired cardiovascular performance, increased protein catabolism, inflammatory activation, and reduced responsiveness to vasoactive medications. Early correction of acid-base disturbances improves hemodynamic stability and supports recovery of organ function.
The observed increase in inflammatory biomarkers emphasizes the important interaction between systemic inflammation and acute kidney injury. In septic patients, inflammatory cytokines aggravate endothelial dysfunction, microvascular injury, and tubular cell apoptosis, thereby accelerating deterioration of renal function.
Contemporary management increasingly combines traditional laboratory parameters with novel biomarkers capable of detecting renal injury before irreversible structural damage occurs. Integration of these biomarkers into routine clinical practice may allow earlier diagnosis, more accurate risk stratification, and individualized therapeutic decision-making.
Renal replacement therapy remains a lifesaving intervention for patients with severe biochemical abnormalities that cannot be corrected by conservative management. Indications include refractory hyperkalemia, severe metabolic acidosis, progressive azotemia accompanied by symptomatic uremia, pulmonary edema due to fluid overload, and persistent oliguria or anuria. Appropriate timing of dialysis significantly influences patient survival and recovery.
Future research should focus on precision diagnostic biomarkers, metabolomic profiling, artificial intelligence-assisted prediction models, and individualized therapeutic strategies capable of identifying high-risk patients before irreversible renal injury develops.

5. Conclusion

Acute renal failure is characterized by profound biochemical disturbances reflecting rapid deterioration of renal function. Elevation of serum creatinine, blood urea nitrogen, cystatin C, potassium, phosphorus, inflammatory markers, and metabolic acids, together with reductions in bicarbonate, calcium, albumin, and hemoglobin, provides valuable information regarding disease severity and prognosis.
Routine biochemical monitoring is indispensable for early diagnosis, evaluation of therapeutic response, detection of life-threatening complications, and determination of indications for renal replacement therapy. Early recognition of abnormal laboratory findings, combined with individualized clinical management, significantly improves renal recovery, reduces complications, shortens hospitalization, and enhances overall patient survival.
Continued development of sensitive biomarkers and multidisciplinary management strategies will further improve the diagnosis and treatment of acute kidney injury while reducing the burden of this serious clinical syndrome.

References

[1] Kidney Disease: Improving Global Outcomes (KDIGO). KDIGO 2012 Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl. 2012;2(1):1–138.

[2] Kidney Disease: Improving Global Outcomes (KDIGO). KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 2024;105(Suppl 4):S1–S150.

[3] Ostermann M, Joannidis M. Acute kidney injury 2016: diagnosis and diagnostic workup. Crit Care. 2016;20:299.

[4] Ronco C, Bellomo R, Kellum JA. Acute kidney injury. N Engl J Med. 2019;380(8):756–766.

[5] Hoste EAJ, Kellum JA, Selby NM, et al. Global epidemiology and outcomes of acute kidney injury. Nat Rev Nephrol. 2018;14(10):607–625.

[6] Makris K, Spanou L. Acute kidney injury: definition, pathophysiology and clinical phenotypes. Clin Biochem Rev. 2016;37(2):85–98.

[7] Mehta RL, Cerdá J, Burdmann EA, et al. International Society of Nephrology's 0by25 initiative for acute kidney injury. Lancet. 2015;385(9987):2616–2643.

[8] Endre ZH, Pickering JW, Walker RJ. Clearance and beyond: the complementary roles of biomarkers in acute kidney injury. Nat Rev Nephrol. 2011;7(12):721–731.

[9] Haase M, Bellomo R, Devarajan P, et al. Novel biomarkers early predict the severity of acute kidney injury. Kidney Int. 2009;75(10):1118–1125.

[10] Devarajan P. Biomarkers for the early detection of acute kidney injury. Curr Opin Pediatr. 2011;23(2):194–200.

[11] Kellum JA, Romagnani P, Ashuntantang G, et al. Acute kidney injury. Nat Rev Dis Primers. 2021;7(1):52.

[12] National Kidney Foundation. KDOQI Clinical Practice Guideline for Hemodialysis Adequacy: 2019 Update. Am J Kidney Dis. 2020;75(4 Suppl 2):S1–S164.

[13] Johnson RJ, Feehally J, Floege J, Tonelli M. Comprehensive Clinical Nephrology. 7th ed. Elsevier; 2024.

[14] Brenner BM, Rector FC. Brenner and Rector's The Kidney. 12th ed. Elsevier; 2024.

[15] International Society of Nephrology. Global Kidney Health Atlas. 3rd ed. ISN; 2023.

[16] World Health Organization. Chronic Kidney Disease: Global Health Perspectives. Geneva: WHO; 2024.

[17] World Health Organization. Global Health Observatory: Kidney Diseases. Geneva: WHO; 2025.

[18] Palevsky PM, Liu KD, Brophy PD, et al. KDOQI US commentary on the 2012 KDIGO Clinical Practice Guideline for Acute Kidney Injury. Am J Kidney Dis. 2013;61(5):649–672.

[19] Med1.uz. O‘tkir buyrak shikastlanishi: etiologiyasi, patogenezi va diagnostikasi. Available from: https://med1.uz/articles/nefrologiya/otk ir-buyrak-shikastlanishi

[20] Med1.uz. O‘tkir buyrak yetishmovchiligi: zamonaviy davolash tamoyillari. Available from: https://med1.uz/articles/nefrologiya/otk ir-buyrak-yetishmovchiligi

[21] Med1.uz. Buyrak kasalliklarida qonning biokimyoviy tekshiruvlari. Available from: https://med1.uz/articles/laboratoriya/bi okimyoviy-tekshiruvlar

[22] Med1.uz. Elektrolitlar almashinuvi buzilishlari va buyrak kasalliklari. Available from: https://med1.uz/articles/nefrologiya/ele ktrolitlar

[23] Med1.uz. Kislota-ishqor muvozanati buzilishlari. Available from: https://med1.uz/articles/laboratoriya/ki slota-ishqor-muvozanati

[24] Med1.uz. Gemodializ va buyrak yetishmovchiligini davolash. Available from: https://med1.uz/articles/nefrologiya/gem odializ

[25] Med1.uz. Buyrak kasalliklarida laborator diagnostikaning zamonaviy usullari. Available from: https://med1.uz/articles/laboratoriya/la borator-diagnostika

[26] Med1.uz. O‘tkir buyrak shikastlanishining profilaktikasi va monitoringi. Available from: https://med1.uz/articles/nefrologiya/pro filaktika

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