Anion Gap Value As A Predictor of Short-term Mortality in Critically Ill Patients with Acute Kidney Injury

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Introduction

Acute Kidney Injury (AKI) is a common and potentially reversible condition marked by a sudden decline in renal function, often detected through reduced glomerular filtration rate (GFR). According to KDIGO, AKI is diagnosed by a rise in serum creatinine or reduced urine output. It is classified into prerenal, intrinsic, and postrenal causes. Sepsis remains the most frequent trigger, with AKI pathophysiology involving both structural damage and functional impairment.

Effective management requires close monitoring of renal function and fluid balance, withdrawal of nephrotoxins, dietary adjustments, and, when needed, dialysis. Metabolic acidosis is a frequent complication, especially in critically ill patients, and may present with a high or normal anion gap. The anion gap (AG), a key diagnostic tool, also has prognostic value. Correcting AG for hypoalbuminemia improves accuracy. Management of high AG acidosis targets the underlying cause, with alkalinization reserved for severe acidemia (pH < 7.1).

Aim of the work

This study investigated the prognostic significance of admission anion gap levels in predicting mortality among critically ill patients with acute kidney injury.

Patients and Methods

This prospective study included 230 adult patients diagnosed with AKI, admitted to the Emergency Department of Alexandria Main University Hospital over six months. Sample size was calculated using NCSS 2004 and PASS 2000 by the Biostatistics Department. Inclusion criteria followed KDIGO guidelines. Patients under 18 or with trauma were excluded. Ethics approval was obtained, and informed consent was secured. Data were collected prospectively using standardized forms, including detailed history, clinical examination, and laboratory investigations. Assessments covered vital signs, Glasgow Coma Scale, oxygenation, circulation, and potential infection sources. Laboratory tests included CBC, CRP, electrolytes, creatinine, albumin, and ABG. The anion gap and its corrected value for hypoalbuminemia were calculated. Statistical analysis was performed using SPSS v20. Tests included Chi-square, t-test, Mann-Whitney, and Spearman correlation. Quantitative data were expressed as mean, median, and standard deviation. Significance was considered at $p<0.05$. Primary outcomes were ICU mortality; secondary outcomes included ICU stay and renal recovery.

Results

The study included 230 patients (91 males, 39.6%; 139 females, 60.4%) aged 32–92 years (mean 62.1 ± 13.3). Renal causes accounted for 49.6% of AKI, followed by prerenal (36.5%) and postrenal (13.9%) etiologies. The mortality rate was 45.2%. RRT was required in 24% of cases; among survivors, 32.2% had AKI resolution.Mortality was higher in females (73.1%) and older patients. Comorbidities and prerenal causes were more common among non-survivors. Key mortality predictors included low blood pressure, low GCS, and hypoxia. Lab findings associated with death included low pH, low bicarbonate, high lactate, and elevated corrected anion gap (AG).Non-survivors had significantly higher corrected AG and were less likely to receive RRT. Only 7.7% of non-survivors received RRT vs. 37.3% of survivors ($p < 0.001$). Survivors had longer hospital stays. Higher corrected AG was linked to shorter stays and unresolved AKI, suggesting it reflects greater illness severity and worse outcomes.

Table (1): Vital Signs and Laboratory Parameters on Admission

Parameter	Median (IQR)	Mean ± SD
Vital Signs		
Mean BP (mmHg)	78 (53 – 90)	77.71 ± 27.60
Heart Rate (bpm)	91 (80 – 100)	95.23 ± 21.93
Respiratory Rate (breaths/min)	28 (22 – 30)	27.79 ± 6.80
SpO_2 (%)	97 (93 – 98)	94.56 ± 6.39
Temperature (°C)	37.0 (37.0 – 38.0)	37.47 ± 0.77
Glasgow Coma Scale (GCS)	15 (14 – 15)	13.85 ± 2.70
Urine Output (ml/hr)	30 (10 – 100)	54.26 ± 64.44
Lab Investigations		
Urea (mg/dl)	213 (153 – 300)	238.6 ± 110.5
Creatinine (mg/dl)	6.30 (4.0 – 12.0)	8.35 ± 5.49
Sodium (mmol/L)	134 (130 – 135)	133.43 ± 6.04
Potassium (mmol/L)	5.0 (4.0 – 6.2)	5.15 ± 1.30
Chloride (mmol/L)	100 (99 – 101)	99.46 ± 3.70
Albumin (g/dl)	3.0 (2.5 – 3.5)	2.92 ± 0.51
CRP (mg/L)	90.0 (50.0 – 135.0)	105.25 ± 75.22
ABG Parameters		
pH	7.30 (7.23 – 7.35)	7.27 ± 0.23
HCO_3 (mmol/L)	13.0 (10.0 – 14.0)	12.35 ± 4.19
Lactate (mmol/L)	2.0 (1.0 – 5.0)	3.44 ± 3.59
Anion Gap (mEq/L)	19.0 (18.0 – 24.0)	21.62 ± 7.25
Corrected Anion Gap (mEq/L)	19.63 (18.50 – 24.48)	22.01 ± 7.28

Table (2): Outcomes and Associations with Mortality

Outcome/Association	Value
Primary Outcome	Survived: 126 (54.8%) Died: 104 (45.2%)
Secondary Outcomes	RRT: 55 (24.0%) AKI Resolved: 74 (32.2%) Unresolved AKI: 55 (23.9%)
ICU Length of Stay (days)	Range: 1 – 60 Mean ± SD: 7.96 ± 7.45 Median (IQR): 7.0 (5.0 – 10.0)
Mortality Predictors	Low BP, low GCS, low SpO_2 Low pH, low HCO_3, high lactate, high corrected AG
Comorbid Risk Factors	Diabetes, hypertension, cardiovascular disease
Mortality by AKI Cause	Prerenal: 68 deaths Renal: 33 deaths Postrenal: 3 deaths
Corrected AG & Outcomes	Higher AG → more mortality, less RRT use, shorter stays, lower recovery

Conclusion

This study found that a high initial anion gap is a strong predictor of mortality in critically ill AKI patients. Mortality was associated with low pH, low bicarbonate, high lactate, low blood pressure, low GCS, and low oxygen saturation. Comorbidities like diabetes, hypertension, and cardiovascular disease further increased risk, while CKD and liver disease had less impact. Prerenal AKI was linked to higher mortality than renal or postrenal causes.

A higher corrected anion gap was associated with reduced likelihood of receiving RRT, likely due to patient instability. It also correlated with shorter hospital stays and unresolved AKI, suggesting it reflects more severe illness and poor outcomes.