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THE RISK OF DEVELOPING ACUTE KIDNEY INJURY AMONG SICKLE CELL DISEASE PATIENT DURING CRISIS: A RETROSPECTIVE STUDY IN A SINGLE TERTIARY CENTER AT JEDDAH, SAUDI ARABIA

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ABSTRACT

Introduction

Background: Sickle cell disease (SCD) is a common inherited hemoglobinopathy. Acute kidney injury (AKI) is a serious complication of SCD, often precipitated by crises, dehydration, or nephrotoxic exposure. Data on AKI prevalence in SCD patients in Saudi Arabia remain limited.
Objectives: To determine the prevalence and risk factors of AKI among SCD patients presenting with crises at King Abdulaziz University Hospital, Jeddah.
Design: Retrospective cohort study.
Setting: King Abdulaziz University Hospital, Jeddah, Saudi Arabia.
Patients and Methods: Medical records of patients aged ≥15 years admitted with SCD crises between January 2016 and December 2022 were reviewed. Demographics, comorbidities, medications, crisis type, and laboratory parameters were collected. AKI was defined according to kidney Disease: Improving Global Outcomes (KDIGO) criteria. Statistical analyses included t-test, ANOVA, chi-square, and correlations, with $p<0.05$ considered significant.
Main Outcome Measures: Prevalence of AKI and its association with demographic, clinical, and laboratory variables.
Sample Size: A total of 354 admissions were screened; 183 patients met the inclusion criteria.
Results: The mean age was 30 years, and 51% were female. VOC was the predominant crisis type (91%). AKI occurred in 15% of patients (n=27), most commonly within the first day of admission (67%). Significant predictors of AKI included higher BMI (p=0.001), history of prior AKI (p=0.017), hemoglobin reduction during admission (p=0.044), and creatinine dynamics (p=0.001–0.014). Fever was significantly associated with both crisis triggers ($p<0.001$) and urine culture positivity (p=0.001). No significant association was observed between AKI and use of analgesics, NSAIDs, hydroxyurea, or SCD-modifying therapies. The mean hospital stay was 7 days.
Conclusions: AKI occurred in 15% of SCD patients during crises, with early presentation in most cases. BMI, prior renal events, hemoglobin decline, and creatinine trends were significant predictors, highlighting the need for early renal monitoring and preventive strategies.

KEYWORDS: Acute kidney injury , Adult , Crises , Kidney injury , Sickle cell disease , SCD

INTRODUCTION

Sickle Cell Disease (SCD) is described as a collection of inherited blood conditions in which hemoglobin is produced in an abnormal form, resulting in red blood cells (RBCs) being deformed into crescent or sickle shapes. [1] Circulation is disrupted by these abnormal RBCs through adherence to endothelial walls and the induction of blockages. Hemolysis and recurrent vaso-occlusive crises (VOC) are thereby caused, producing both acute and chronic pain.[2] In our region, various risk factors for SCD have been identified, mainly due to consanguineous marriages that increase the risk of genetic transmission, together with restricted access to genetic counseling and screening, which hinder effective prevention strategies.[3] In a 2024 Saudi Arabian study, it was reported that 2–4% of the population is affected by SCD, with the Eastern Province showing the highest rate at 145 cases per 10,000 people, whereas lower prevalence rates were reported in the Southern, Western, and Central regions.[3,4]
Acute Kidney Injury (AKI) has been recognized as a serious complication in SCD, being characterized by rapid kidney dysfunction that may be triggered by VOC, insufficient hydration, or exposure to nephrotoxic drugs such as inflammatory drugs (NSAIDs). [5] Frequent complication of hospitalizations for SCD by AKI has been documented. It

has been observed in a variety of crisis presentations, including hepatic sequestration, acute chest syndrome, lower respiratory tract infections, and VOC.[6] Numerous studies have been conducted to examine the connection between SCD and renal problems. In 2008, a prospective study was carried out to evaluate renal abnormalities in adolescent and adult Saudi patients with sickle cell anemia (SCA). It was found that 38% of the subjects had low serum creatinine levels and 22.5% had reduced creatinine clearance. Proteinuria was reported as the most common abnormality, being detected in 41% of individuals. Hematuria was identified in 9.5% of cases, while hemoglobinuria was observed in 16%. These findings indicated that proteinuria was the most prevalent renal complication in SCA patients.[7] In a retrospective study conducted at the University of Alabama at Birmingham, AKI was observed in 17% of pediatric SCD patients referred for VOC, with 52% of cases being present at the time of ER admission.[5] Additional evidence was provided by a 2022 Ugandan study, in which VOC was shown to markedly elevate AKI risk in SCD patients. In this study, hospitalized pediatric cases were analyzed, and AKI was found to develop in 48.7% of VOC admissions, 33% upon hospital arrival, and an additional 23.4% during hospital stay.[8] Furthermore, a prospective cross-sectional study carried out in eastern Saudi Arabia revealed that microalbuminuria in adults was an early manifestation of sickle cell nephropathy (SCN) and was a common finding in patients with SCD.[9] Despite previous research on AKI in crisis among SCD patients, to the best of our knowledge no validated study has measured the incidence of AKI among SCD patients during crisis globally. Therefore, this study aims to evaluate the prevalence and risk of developing AKI among SCD patients during sickle cell crisis at King Abdulaziz University Hospital in Jeddah, Saudi Arabia.

METHOD:

Study design and setting

The primary outcome of this retrospective study was to identify the risk of developing AKI in SCA patients with crisis at King Abdulaziz University Hospital, Jeddah, Saudi Arabia, from 1 January 2016 to 30 December 2022. After reviewing all ICD-10- defined SCA with crisis from KAUH, 354 admissions for pain during this time were identified. The inclusion criteria included patients with sickle cell disease, aged more than 15 years, while the exclusion criteria were inability to determine a crisis record or baseline SCr level, or lack of creatinine level recording during the admission.

Sample Size and Sampling Procedure

A total of 354 participants were assessed for eligibility. Among them, 183 participants were enrolled in the final analysis after ensuring accuracy of data and meeting inclusion criteria (detailed in Fig. 1).

Data Collection

Demographic variables recorded for each patient included age, gender, body mass index (BMI), and chronic diseases such as congestive heart failure (CHF), diabetes mellitus (DM), hypertension, thalassemia, and chronic kidney disease (CKD). Regular medications were also documented, including analgesia, folic acid, hydroxyurea, and non-steroidal anti-inflammatory drugs (NSAIDs). The SCD phenotype included sickle cell anemia (SCA) and mixed type (SCA–thalassemia). Clinical variables of the crisis event included evidence of fever, chief complaint, triggers of crisis, and type of sickle cell crisis (VOC, ACS, aplastic crisis, or hemolytic crisis). Sickle cell–modifying therapies were also documented, including hydroxyurea, blood transfusion, combination of hydroxyurea with blood transfusion, or no use of SCA-modifying therapy. In addition, total hospital admission days and evidence of AKI before current admission were recorded. Laboratory data collected consisted of creatinine level at admission, highest creatinine level, hemoglobin level, white blood cell (WBC) count, and urine culture. AKI was determined according to the Kidney Disease Improving Global Outcomes (KDIGO) definition of an increase in serum creatinine (SCr) by ≥0.3 mg/dL or a 50% increase in SCr from baseline.

Statistical Analysis

For univariate analysis, frequencies, means, minimum, maximum, and percentages of each variable were measured using measures of central tendency tests. Furthermore, ANOVA, the Independent Sample t test, and correlation were employed in bivariate analysis with 95% CI. The P value was considered significant if it was less than 0.05 for all tests.

Research Ethics

The study was approved by the Research Ethics Committee of KAUH [Ref. No: 52–24]. Data were collected from hospital records after ethical approval was obtained.

RESULT

Univariate analysis

This study aimed to demonstrate the incidence of developing AKI among SCD patients during crises at King Abdulaziz University Hospital in Jeddah, Saudi Arabia.

Final analysis enrolled 183 patients with a mean age of 30 years; 94 were female (51%) and 89 were male (49%). The diagnosis of participants was either SCD or mixed type, with proportions of 91% and 9%, respectively. Body mass index (BMI) was classified into four groups: <18, 18–24, 25–30, and ≥30, accounting for 37 patients (20%), 103 patients (56%), 32 patients (18%), and 11 patients (6%), respectively. Regarding chief complaints, less than ninety percent of patients presented with pain (n = 162, 89%) (Table 1). Fever during crises was observed in 46 patients (25%), while 137 (75%) did not have fever. Medical illness among participants was also documented: none had hypertension, whereas congestive heart failure was present in seven participants, representing the highest number among comorbidities.

Regarding triggers of crisis, most patients (n = 135, 74%) did not exhibit a clear trigger. The remainder had multiple triggers (Table 2). With respect to type of crisis, more than ninety percent of patients were admitted due to vaso-occlusive crisis (n = 167, 91%), compared to acute chest syndrome (n = 11, 6%), aplastic crisis (n = 1, 0.5%), generalized fatigue (n = 1, 0.5%), and hemolytic crisis (n = 3, 2%). Regular medication use was documented: 96 patients (52%) were taking analgesia, while the remainder were not. Folic acid was taken regularly by 127 patients (70%).

Hydroxyurea was administered regularly to 101 patients (55%). Most patients did not take non-steroidal anti-inflammatory drugs (NSAIDs) regularly; only 45 patients (25%) reported regular use. Sickle cell disease–modifying therapy was also documented, though 28 participants had missing data. Twenty percent of patients (n = 37) did not receive any disease-modifying therapy. Among those who did, 72 patients (39%) received hydroxyurea only, 19 patients (10%) received hydroxyurea with blood transfusion, and 30 patients (16%) received blood transfusion only.

Hospital admission days had a mean of 7 (range 0–102 days).

AKI was found in 27 patients (15%), while 156 patients (85%) did not develop it. Creatinine levels during admission had a mean of 76, with a minimum of 17 and a maximum of 766. The highest creatinine levels were observed on the first day of admission in 123 patients (67%). On the second and third days, the highest levels were observed in 9 and 13 patients, respectively (Table 3).

Regarding AKI before the current admission, 159 patients (86.9%) had no previous AKI, 18 patients (9.8%) had one episode, 5 patients (2.7%) had two episodes, and one patient (0.5%) had four episodes.

Hemoglobin levels had a mean of 8 (minimum 3). White blood cell (WBC) count had a mean of 16 and a maximum of 313. Urine culture was obtained during admission in 73 patients: 66 were negative (36%) and 7 showed positive bacterial growth (4%) (Table 4).

Bivariate Analysis

The Independent Sample t test for gender and AKI development showed no significant difference (P = .486). BMI was significantly associated with both previous AKI and AKI during admission (P = .017 and P = .001, respectively). Fever was significantly associated with crisis triggers and urine culture (P = .000, P = .001). Triggers of crisis were also significantly associated with urine culture (P = .000).

Conversely, triggers of crisis showed no significant association with analgesia, steroids, hydroxyurea, or AKI during admission (P > .05). Similarly, type of crisis was not significantly associated with analgesia (P = .374), steroids (P = .609), hydroxyurea (P = .124), AKI during admission (P = .078), or urine culture (P = .011).

ANOVA was used to test associations between SCD-modifying therapy and total hospital admission days, number of prior AKI episodes, and creatinine at admission; all were insignificant (P = .929, P = .876, P = .394, respectively).

Chi-square analysis between SCD-modifying therapy and AKI during admission showed no significant association (P = .706).

Regarding AKI during admission, significant associations were found with creatinine at admission (P = .001), day of highest creatinine (P = .014), and hemoglobin levels (P = .044), using the Independent Sample t test. In contrast, creatinine at admission was not significantly correlated with WBC or hemoglobin levels.

Discussion

The aim of this study was to determine the prevalence of acute kidney injury (AKI) among sickle cell disease (SCD) patients during crises at King Abdulaziz University Hospital in Jeddah, Saudi Arabia. The final sample included 183 patients, with a mean age of 30 years. Increasing age was found to be associated with a higher risk of developing AKI. Another Saudi Arabian study identified age as an independent risk factor for sickle cell nephropathy (SCN). [10] In this cohort, 91% of patients had SCD, while 9% had a mixed genotype. Most patients had a normal body mass index (BMI) of 18–24, accounting for 56% of the sample. In contrast, Ibemere and Oyedeji reported that 43% of adults aged 20–45 years with SCD were overweight or obese, indicating a higher prevalence of elevated BMI in their population. [11] A significant association was also revealed between BMI and prior AKI episodes, as well as AKI development during admission. Similarly, Farooqui et al. reported that normal BMI was associated with fewer hospital admissions. [12] Pain was the most common presenting symptom, reported by nearly 88% (n = 162) of patients. Fever was noted in 25% (n = 46). A similar local study involving 212 emergency department visits in children with SCD reported fever in 37.7%, pain in 47.2%, and both symptoms in 15% of cases. [13] Regarding the type of crisis, this population (mean

age 30 years) demonstrated a high rate of vaso-occlusive crises (VOC) at 91%, and significantly lower rates of aplastic (0.5%) and hemolytic crises (1.6%). In contrast, a pediatric cohort (mean age 6.6 years) showed VOC in 76.2% and aplastic crisis in 14.3%, reflecting differences in infection exposure, age-related disease manifestation, and clinical care. [3] More than half of the patients (n = 96) reported regular use of analgesics, yet no association was found between AKI development and either the use or dosage of analgesics. This finding is consistent with a 2024 pediatric study that found no significant relationship between ketorolac dosage and AKI. [14] However, Sujatha et al. reported a contrasting result, where both the total duration and dose of ketorolac were significantly associated with AKI. [5] Folic acid was used by 70% of patients, 39% were on hydroxyurea, and only 25% reported consistent NSAID use. In this cohort, hydroxyurea use was not associated with AKI development. However, previous studies have shown hydroxyurea to improve quality of life in 50.7% of patients, reduce severe pain in 47.0%, and decrease hospital admissions in 46.4%. [15] The mean hospital stay was 7 days (range 1–107). No significant association was found between AKI development and length of hospitalization. This contrasts with findings by Sujatha et al., who reported a significant link between AKI and prolonged hospital stay. [5]

In this study, 85% of patients did not experience AKI during the current admission, while 9.8% had one episode, 2.7% had two episodes, and one patient had up to four episodes. Overall, AKI occurred in 15% of patients. This is higher than the 4.3% incidence reported by Asnani et al., who used the Acute Kidney Injury Network (AKIN) criteria and observed AKI in 2.3% of pain crises, 6.9% of moderate acute chest syndrome (ACS), and 13.6% of severe ACS (P = .03).[16] Another study reported AKI in 17% of 197 admissions for VOC and up to 52% in emergency department presentations.[5]

In this cohort, the mean hemoglobin level was 8 g/dL, with values as low as 3 g/dL, and this reduction was significantly associated with the occurrence of AKI (P = .044). Baddam et al. similarly highlighted the contribution of anemia, reporting that every one-unit decrease in hemoglobin from baseline to admission increased the risk of AKI by 49%. Although the mean white blood cell count was elevated, with a mean of nearly 16 × 10^9/L, this elevation was not significantly associated with creatinine levels at admission. This suggests that leukocytosis reflects systemic inflammation during vaso-occlusive crises rather than directly predicting AKI.

Similarly, urine cultures obtained in 73 patients were predominantly negative (36%), with only 4% positive for bacterial growth, indicating that bacterial infection was not a major contributor to AKI episodes in this population. This is consistent with prior reports that non-infectious mechanisms such as hemolysis, hypovolemia, and dehydration may drive renal injury during sickle cell crises. [5]

The mean creatinine level at admission was 76.2 µmol/L. Notably, 67.2% of patients reached their peak creatinine on the first day of admission, while others peaked on day two (n = 9) or day three (n = 13). These findings suggest that renal injury, when it occurs, tends to happen early during hospitalization, highlighting the need for early and close monitoring.

A pediatric study by Baddam et al. found a rise in creatinine from 0.2 to 0.3 mg/dL, a 1.5-fold increase in children with AKI. Though the absolute increase appears small, it was sufficient to meet AKI criteria, emphasizing the importance of recognizing even subtle changes in renal function in SCD patients. [8] the main limitations of this study include its single-center retrospective design, which may limit the generalizability of the findings. Additionally, incomplete documentation of medication dosages and precipitating crisis triggers may have influenced the observed associations.

CONCLUSION

This retrospective study demonstrates that AKI is a common complication among SCD patients during crises, with a prevalence of 15%. Significant predictors of AKI included abnormal BMI, history of prior AKI, hemoglobin decline during admission, and creatinine trajectory, indicating that both baseline health status and acute hematologic changes contribute to renal vulnerability. Fever and crisis triggers showed significant associations with urine culture results, although bacterial infection was not the primary driver of AKI. Most AKI cases manifested on the first day of hospitalization, emphasizing the need for early renal function testing and intervention. Importantly, no significant association was found between AKI and the use of NSAIDs, analgesics, or hydroxyurea, suggesting that patient-related factors may outweigh medication exposure in this context. Preventive strategies such as hydration, vigilant monitoring, and early recognition of renal dysfunction should be prioritized in the care of SCD patients presenting with crises.

DISCLOSURE STATEMENT

Conflict of Interest: None declared.

DATA SHARING STATEMENT

The data that support the findings of this study are available from King Abdulaziz University Hospital, but restrictions apply to the availability of these data, which were used under institutional license for the current study and are not

publicly available. De-identified data may be made available from the corresponding author on reasonable request and with permission of King Abdulaziz University Hospital.

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Author Contributions

Dr. Rana: Supervision, review & editing. **Rzan:** Introduction. **Maha:** Methodology. **Hanin:** Formal analysis, Software. **Rawan:** Discussion. **Hanan:** Result, Discussion.

The authors testify that all designated persons qualify for authorship and have checked the article for plagiarism. All authors meet the following criteria: (1) substantial contributions to the conception, design of the work, the acquisition, analysis, and interpretation of data; (2) drafting the work or revising it critically for important intellectual content; and (3) final approval of the version to be published.

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Table1. Demographic Details

Variable	Frequency (%)
Gender	
Male	**89 (49%)**
Female	**94 (51%)**
Age	
10-18	**21 (11.5%)**
19-35	**109 (59.6%)**
36-45	**46 (25.1%)**
46-65	**6 (3.3%)**
>65	**1 (1=0.5%)**
Diagnosis	
Sickle Cell Anemia (SCA)	**166 (91%)**
Mixed Type	**17 (9%)**
Body Mass Index (BMI)	
<18	**37 (20%)**
18-24	**103 (56%)**
25-29	**32 (18%)**
≥ 30	**11 (6%)**
Fever	
Yes	**46 (25%)**
No	**137 (75%)**
Chronic diseases	
Congestive Heart Failure (CHF)	**7 (4%)**
Diabetes Mellitus (DM)	**5 (3%)**
Hypertension	**6 (3%)**
Thalassemia	**1 (0.5%)**
Chronic Kidney Disease (CKD)	**3 (2%)**

Table2. Crisis Details

Variable	Frequency (%)
Type of Crisis	
VOC	**168 (91.8%)**
Acute Chest Syndrome (ACS)	**11 (6%)**
Hemolytic Crisis	**3 (1.6%)**
Aplastic Crisis	**1 (0.5%)**
Regular Medication	
Analgesia	**96 (53%)**
Folic Acid	**127 (70%)**
Hydroxyurea	**101 (55%)**
NSAIDs	**45 (25%)**
Steroids	**10 (6%)**
Sickle Cell Disease (SCD) Modifying Therapy	
Hydroxyurea only	**72 (39%)**
Blood Transfusion only	**30 (16%)**
Hydroxyurea + Blood Transfusion	**19 (10%)**
No SCD Modifying Therapy	**37 (20%)**
Chief Complaint	
Pain	**162 (88.5%)**
Tachypnea	**3 (1.6%)**
Dizziness	**2 (1.1%)**
Shortness of Breath (SOB)	**7 (3.8%)**
Loss of Consciousness (LOC)	**1 (0.5%)**
Constipation	**1 (0.5%)**
Diarrhea	**1 (0.5%)**
Fatigue	**4 (2.2%)**
Chest Pain	**2 (1.1%)**

Table3. Incidence of crises and creatinine levels

Variable	Frequency (%)
Number of Patients had AKI	
Not Develop AKI	**159 (88.9%)**
One	**18 (9.8%)**
Two	**5 (2.7%)**
Four	**1 (0.5)**
Day of highest Creatinine levels	
Day one	**123 (67.2%)**
Day two	**9 (4.9%)**
Day three	**13 (7.1%)**
Day four	**5 (2.7)**
Day five	**8 (4.4%)**
Day six	**3 (1.6%)**
Day 7-10	**10 (5.4%)**
Day 11-16	**6 (3.2%)**
Day 22	**1 (0.5%)**

Table4. Creatinine and laboratory levels Details

Variable (N)	Mean (S.D)
Creatinine levels at admission (N=183)	**76.2 (103.2)**
Day Number of highest Creatinine levels (N=178)	**2.5 (3.3)**
Total Hospital Admission Days (N=183)	**7 (10.6%)**
Hemoglobin levels (N=182)	**8.1 (3.3)**
White blood cell levels (N=182)	**15.6 (23.1 %)**

Fig1. Participants Details.