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ORIGINAL RESEARCH

Prevalence of Gallstones and Associated Factors Among Children and Adolescents with Sickle Cell Disease in Eastern Uganda

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Background: Sickle cell disease (SCD) predisposes patients to gallstone formation due to chronic hemolysis, which may result in complications such as cholecystitis and pancreatitis. Despite its clinical importance, there is limited data on the prevalence and risk factors of gallstones in children and adolescents with SCD in Uganda. This study aimed to determine the prevalence and associated factors of gallstones in children and adolescents with SCD in Eastern Uganda.

Methods: A hospital-based cross-sectional study was conducted at Mbale Regional Referral Hospital from March to June 2024. We enrolled 208 children and adolescents aged 2–19 years with confirmed SCD. Data on sociodemographic and clinical characteristics were collected using structured questionnaires. Abdominal ultrasound was performed to assess gallstone presence, and blood samples were analyzed for hematological and liver function parameters. Logistic regression was used to identify factors associated with gallstones.

Results: The prevalence of gallstones was 31% (65/208). Adolescents aged 13–19 years were more likely to have gallstones than younger children. Factors significantly associated with gallstones included jaundice adjusted prevalence ratio (aPr = 0.5, 95% CI: 0.4–0.7, $p < 0.001$) and elevated gamma-glutamyl transferase (GGT) levels (aPr = 1.5, 90% CI: 0.9–2.3, $p = 0.051$).

Conclusion: The high prevalence of gallstones in children and adolescents with SCD emphasizes the need for routine screening. Routine ultrasound and liver function tests for children over 10 years may enable early detection and intervention, reducing complications.

Keywords: gallstones, sickle cell disease, children, adolescents, Uganda, liver function

Introduction

Sickle cell disease (SCD) is the most common inherited hemoglobinopathy worldwide, with the highest incidence in Sub-Saharan Africa, the Mediterranean, the Middle East, and the Indian subcontinent.[1] The disease arises from a single-point mutation in the beta-globin gene on chromosome 11, where glutamic acid is replaced by valine at position 6 of the beta-globin chains and this genetic alteration causes red blood cells (RBCs) to distort into a sickle shape under low-oxygen conditions, leading to a loss of RBC flexibility and triggering a cascade of pathophysiological events, including chronic hemolysis and vaso-occlusive crises.[2,3] Specifically, the continuous breakdown of sickled red blood cells leads to an overload of unconjugated bilirubin, which subsequently crystallizes in the gallbladder to form black pigment stones.

The World Health Organization (WHO) has recently recognized SCD as one of the most common and socially significant hemoglobinopathies, emphasizing its public health concern.[1] Globally, the estimated birth prevalence of heterozygous SCD was 4229.72 per 100,000, with Europe having the lowest rate at 803.57 per 100,000 and Africa having the highest rate at 16,121.91 per 100,000 with regional variations in 2018.[4] For the Eastern Mediterranean region,

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more than 100,000 individuals are afflicted with this genetic condition, with roughly 90% of them being Black and 10% being Hispanic.[5] SCD is an important public health problem accounting for about 5% of under-five mortality in Africa.[6] According to a study done in Congo, the factors associated with cholelithiasis (gallstones) included increasing patient age and increased hemolysis.[7] In Uganda, approximately 20,000 newborns in are diagnosed with SCD each year, a number projected to rise significantly by 2050, yet 70–80% of these children do not survive beyond their fifth birthday, contributing to 16.2% of all under-five deaths annually.[6]

Cholelithiasis is a common complication of SCD due to increased hemolysis, which leads to elevated bilirubin levels. A meta-analysis reported a prevalence of gallstones in 25.3% of patients with SCD.[8] Moreover, approximately 75% of patients with SCD who develop gallstones require emergency cholecystectomy, often with poorer outcomes compared to elective procedures.[9] At Mulago National Referral Hospital in Uganda, an unpublished survey by Musasizi found that 22% of children with SCD had gallstones. This study aimed to determine the prevalence and factors associated with gallstones in children and adolescents aged 2–19 years with SCD attending Mbale Regional Referral Hospital (MRRH).

Methods

Study Design and Site

This study was a hospital-based cross-sectional study conducted at MRRH. This study was conducted at the sickle cell clinic and pediatric ward of MRRH, a public teaching hospital in Mbale, Uganda. MRRH serves as a training center for Busitema University and provides healthcare services to the Eastern region. The paediatric section comprises four units: convalescent care (90 beds), acute care, nutrition, and neonatal units, staffed by 12 nurses, five interns, two medical officers, and five paediatricians. The sickle cell clinic, held every Wednesday, attends about 130 patients (70 children, 60 adolescents), with 450 adolescents and 300 children registered.

Eligibility Criteria

Inclusion Criteria

- Children and adolescents aged 2 to 19 years diagnosed with sickle cell disease (SCD) confirmed by hemoglobin electrophoresis.
- Patients attending the sickle cell clinic or admitted to the paediatric ward at MRRH during the study period.
- Those who provided informed consent (for participants aged 18 years and above) or assent (for minors, with parental/guardian consent).
- Patients who were clinically stable and could undergo an abdominal ultrasound (USS) for gallstone detection.

Exclusion Criteria

Patients with a history of cholecystectomy or a critical illness that prevents ultrasound assessment.

Sample Size

The sample size was calculated using the formula by Daniel WW (1999).

$$N = \frac{Z^2 p(1-p)}{e^2}$$

Where N is the required sample size estimate and Z is the critical value for a normal distribution at the 95% confidence level, corresponding to 1.96. P = proportion of gallstones among patients with sickle cell disease, for which a 12.22% was used from the study done from Congo.[7]

q=1-p, therefore,

$$N = \frac{1.96^2 * 0.122(1-0.122)}{0.05^2} = 164$$

Adding 10% of expected non respondents, the sample size considered in this study became 180.

Sample Collection and Methods

Data collection was conducted by trained research assistants using a structured, pre-tested, interviewer-administered questionnaire to capture sociodemographic and clinical data. Eligible participants were consecutively recruited. Before enrollment, each participant and/or their parent/guardian received detailed information about the study, and informed consent and assent were obtained as appropriate. This study was conducted in accordance with the Declaration of Helsinki. A comprehensive clinical assessment was performed by the principal investigator, including a brief medical history and a physical examination to evaluate risk factors for gallstones.

Ultrasound scans (USS) were performed by a certified radiographer at the MRRH radiology department, to confirm the presence or absence of gallstones. Ultrasound has a sensitivity of 85% and a specificity of 100% for the identification of gallstones.[10] To assess hematological factors associated with gallstone formation, blood samples were collected by a laboratory technician for complete blood count (CBC) using 2 mL pediatric EDTA tubes to ensure the proper blood-to-anticoagulant ratio and prevent hyper-dilution and liver function tests (LFTs) (3 mL) and analyzed at the MRRH laboratory.

Data Analysis

Data were analyzed using STATA 14.2 after entry and coding in Microsoft Excel 2016. The dependent variable was gallstone presence, while independent variables included sociodemographic factors (age, sex, BMI), medical history (diabetes, HIV, hypertension, pregnancy status, hydroxyurea use), and hematological (hemoglobin, hematocrit) and biochemical parameters (bilirubin, ALT, AST, ALP, GGT). Descriptive statistics were presented as frequencies and proportions, and associations were tested using Chi-square analysis. Multivariable binomial regression was performed to obtain crude prevalence ratios (cPr) and adjusted prevalence ratios (aPr) with 95% confidence intervals (CI). Variables with $p < 0.2$ in bivariate analysis were included in a multivariable analysis to identify risk factors, ensuring that potential confounding variables were not prematurely excluded from the model due to strict significance testing at the initial stage.

Results

Sociodemographic Characteristics of the Study Participants

A total of 208 patients were recruited for the study, with a median age of 10 years and an interquartile range (IQR) of (5, 13) years. The majority of the patients (58.3%) were aged between 5 and 12 years, followed by those aged 13 to 19 years (36.0%). Gender distribution was relatively balanced, with 55.8% females and 44.2% males. The median BMI of the patients was 15, with an IQR (14, 17) (Table 1).

Table 1 Baseline Characteristics of the Study Participants (N=208)

Variables	Frequencies (N=208)	Percentages (%)/IQR
Age		
2–4	12	5.7
5–12	121	58.3
13–19	75	36.0
Gender		
Male	92	44.2
Female	116	55.8
BMI median (IQR)	15	(14, 17)

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Bivariate Analysis for Social Demographic Characteristics

Among the 208 participants, gallstones were present in 31% (n = 65) and absent in 69% (n = 143). Age was associated with gallstone presence, with children aged 13–19 years having a higher likelihood of gallstones compared to those aged 2–4 years (cPr 2.5; 95% CI 0.6–9.2; p = 0.160). Children aged 5–12 years also showed a slightly increased likelihood (cPr 1.5; 95% CI 0.4–5.6; p = 0.533), though the difference was not statistically significant. Gender was not significantly associated with gallstone presence, as female children had a slightly lower likelihood compared to males (cPr 0.9; 95% CI 0.6–1.4; p = 0.834) (Table 2).

Clinical Characteristic Medications Used and Gallstone Presence

Children with moderate jaundice (14.8%) were significantly more likely to have gallstones compared to those with no jaundice (49.2%) (cPr 1.7; 95% CI 1.1–2.7; p = 0.029). Right upper quadrant (RUQ) pain was reported in 22.7% of children and was associated with a slightly increased likelihood of gallstones, though the association was not statistically significant (cPr 1.1; 95% CI 0.7–1.8; p = 0.662). Anaemia (48.3%) was not significantly linked to gallstone presence compared to those without anaemia (cPr 0.9; 95% CI 0.6–1.4; p = 0.672).

Other symptoms, including fever (12.3%) (cPr 0.9; 95% CI 0.5–1.8; p = 0.997), nausea and vomiting (3.2%) (cPr 0.5; 95% CI 0.1–3.2; p = 0.493), and CNS events (4.3%) (cPr 0.3; 95% CI 0.1–2.2; p = 0.267), were not significantly associated with gallstones. Similarly, headaches (44.5%) (cPr 0.9; 95% CI 0.6–1.4; p = 0.774) showed no significant relationship.

Regarding medications, folic acid use was nearly universal (99.5%), while hydroxyurea use (88.1%) was associated with a slightly higher likelihood of gallstones (cPr 1.6; 95% CI 0.7–3.6; p = 0.207), though this was not statistically significant (Table 3).

Bivariate Analysis of Laboratory Parameters of Participants

Children with normal haemoglobin levels showed a gallstone presence of 35.9% (22/73), while those with below-range levels had a similar proportion of 30.4% (41/135) (cPr 0.9; 95% CI 0.6, 1.5; p = 0.888). For ALT, 8.1% (5/12) of those with above-range levels had gallstones compared to 4.8% (7/196) in those with normal levels (cPr 1.4; 95% CI 0.7, 2.9; p = 0.317). Among children with AST above range, the gallstone presence was 81.0% (51/148) compared to 19.0% (12/60) with normal levels, suggesting a near-significant association (cPr 1.7; 95% CI 0.9, 3.0; p = 0.043). Regarding GGT, 25.8% (16/43) of those above range had gallstones, while 74.2% (119/165) with normal levels did not show significant differences (cPr 1.3; 95% CI 0.8, 2.1; p = 0.218). The occurrence of gallstones was 31.4% (61/194) among participants with haematocrit levels below the normal range, compared to 21.4% (3/14) in those with normal levels (cPr 1.6; 95% CI

Table 2 Bivariate Analysis of Demographic Characteristics and Gallstone Presence

Variables	Total n (%)	Gallstone Presence n=208 (%)		cPr (95% CI)	P value
		Absent	Present		
Age					0.024
2–4	12(5.7)	10(83.3)	2(16.7)	1	
5–12	121(58.3)	90(74.4)	31(25.6)	1.5(0.4, 5.6)	0.533
13–19	75(36.0)	43(57.3)	32(42.7)	2.5(0.6, 9.2)	0.160
Gender					0.834
Male	92(44.2)	63(68.5)	29(31.5)	1	
Female	116(55.8)	81(69.8)	35(30.2)	0.9(0.6, 1.4)	0.834
BMI median (IQR)	15(14, 17)	15(14, 16)	16(14, 18)		

Table 3 Bivariate Analysis of Clinical Factors and Medications Use

Variable	Total	Gallstone Presence		cPr (95% CI)	P
Clinical Presentations		**Absent n (%)**	**Present n (%)**		
RUQ pain	48(23.1)	23(47.9)	25(52.1)	1.1(0.7, 1.8)	0.662
Anaemia	102(49.0)	53(52)	49(48)	0.9(0.6, 1.4)	0.672
Jaundice					0.013
Nil	97(46.6)	56(57.7)	41(42.3)	–	
Tinge	80(38.5)	38(47.5)	42(52.5)	1	
Moderate	31(14.9)	7(22.6)	24(77.4)	1.7(1.1, 2.7)	0.029
Fever	26(12.5)	14(53.8)	12(46.2)	0.9(0.5, 1.8)	0.997
Nausea and vomiting	6(2.9)	4(66.7)	2(33.3)	0.5(0.1, 3.2)	0.493
CNS event	9(4.3)	7(77.8)	2(22,2)	0.3(0.1, 2.2)	0.267
Priapism	7(3.4)	4(57.1)	3(42.9)	0.9(0.3, 3.0)	0.872
Headache	94(45.2)	49(52.1)	45(47.9)	0.9(0.6, 1.4)	0.774
Vomiting	17(8.2)	11(64.7)	6(35.3)	0.8(0.3, 1.8)	0.508
Gingival hyper	16(7.7)	6(37.5)	10(62.5)	1.5(0.8, 2.7)	0.189
Medications received					
Folic acid	208(100)	102(49.0)	106(51)	–	0.504
Hydroxyurea use	185(88.9)	88(47.6)	97(52.4)	1.6(0.7,3.6)	0.207
Sulphadoxine pyrimethamine	206(99.0)	104(50.5)	102(49.5)	1.2(0.2,6.9)	0.795
Erythromycin	1(0.5)	1(100)	0(0.0)	–	0.502
Phenoxy methyl penicillin	17(8.2)	13(76.5)	4(23.5)	–	0.005

0.6–4.4; p = 0.396). Lastly, ALP levels revealed that 28.1% (16/58) of children with normal levels had gallstones versus 70.5% (46/150) in those in the above ranges, which was not significant (cPr 1.1; 95% CI 0.7, 1.7; p = 0.720). (Table 4).

Multivariate Analysis of Factors Associated with the Development of Gallstone Among Children and Adolescents with Sickle Cell Disease

Among the factors that were assessed at multivariate using the generalised linear model to test association with gallstone. Having elevated gamma-glutamyl transferase (GGT) and elevated aspartate aminotransferase (AST) was found to be associated with a higher likelihood of developing gallstones, while jaundice was found to be protective in children. Children who had elevated gamma-glutamyl transferase (GGT) levels have a 1.2 times higher likelihood of developing gallstones compared to those within normal ranges (aPr 1.2; 95% CI 1.1, 1.2; p < 0.001). Similarly, patients with elevated aspartate aminotransferase (AST) levels had a higher gallstone likelihood of having gallstones compared to those in the normal range (aPr 1.5; 95% CI 0.9, 2.3; p = 0.051), while a child who had Jaundice had 0.5 times reduced risk of developing gallstones compared to those who did not have jaundice (aPr 0.5; 95% CI 0.4, 0.7; p < 0.001) (Table 5).

Discussion

This study investigated the prevalence and associated factors of gallstones in children and adolescents with SCD in Eastern Uganda, a region with a high burden of the disease. We found a gallstone prevalence of 31%, a rate that we think

Table 4 Bivariate Analysis of Laboratory Results and Gallstone Presence

Variable	Total	Gallstones Status		cPr (95% CI)	P value
	208	Absent n (%)	Present n (%)		
HB					0.888
Normal (8.0–17.0)	73(35.1)	51(69.9)	22(30.1)	1	
Below range (<8.0)	135(64.9)	94(69.6)	41(30.4)	0.9(0.6, 1.5)	0.888
ALT					
Normal (0–41)	196(94.2)	139(70.9)	57(29.1)	1	0.355
Above range (>41.0)	12(5.8)	7(58.3)	5(41.7)	1.4(0.7, 2.9)	0.317
AST					0.043
Normal (0–40)	60(28.8)	48(80.0)	12(20.0)	1	
Above range (>40)	148(71.2)	97(65.5)	51(34.5)	1.7(0.9, 3.0)	0.057
GGT					0.234
Normal (0–55)	165(79.3)	119(72.1)	46(27.9)	1	
Above range (>55)	43(20.7)	27(62.8)	16(37.2)	1.3(0.8, 2.1)	0.218
Haematocrit					0.595
Normal (26–50)	14(6.7)	11(78.6)	3(21.4)	1	
Below range (<26.0)	194(93.3)	132(68.0)	61(31.4)	1.6(0.6, 4.4)	0.396
ALP					0.503
Normal (40–129)	58(27.9)	42(72.4)	16(27.6)	1	
Above range (>129)	150(72.1)	104(69.3)	46(30.7)	1.1(0.7, 1.7)	0.720

Abbreviations: GGT, gamma-glutamyl transferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; ALT, alkaline transferase.

Table 5 Factors Associated with Gallstones Among Children and Adolescents

Variables	cPr (95% CI)	P value	aPr (95% CI)	P value
Age				
<5	1		1	
5–12	1.5(0.4, 5.6)	0.533	1.3(0.4, 3.7)	0.686
13–19	2.5(0.7, 9.2)	0.160	2.6(0.9, 7.3)	0.078
CNS event				
No	1		1	
Yes	0.3(0.1, 2.2)	0.267	0.3(0.04, 2.1)	0.223
Jaundice				
No	1		1	
Yes	0.6(0.4, 0.9)	0.023	0.5(0.4, 0.7)	<0.001

(*Continued*)

Table 5 (Continued).

Variables	cPr (95% CI)	P value	aPr (95% CI)	P value
Hydroxyurea				
No	1		1	
Yes	1.6(0.7, 3.6)	0.243	1.4(0.6, 3.2)	0.380
AST				
Normal range	1		1	
Above range (>40)	1.7(0.9, 3.0)	0.057	1.5(0.9, 2.3)	0.051
GGT				
Normal range	1		1	
Above range (>55)	1.3(0.8, 2.1)	0.218	1.2(1.1, 1.2)	<0.001
Haematocrits				
Normal range	1		1	
Below range (<26.0)	1.5(0.5, 4.1)	0.469	1.7(0.5, 5.8)	0.384
ALP				
Normal range	1		1	
Above range (>129)	1.1(0.6, 1.7)	0.720	1.4(0.9, 2.2)	0.094
Direct (Bilirubin)				
Normal	1		1	
Above Range	1.4(0.9, 2.2)	0.091	1.2(0.8, 1.8)	0.285

Abbreviations: cPr, crude prevalence ratio; aPr, Adjusted prevalence ratio; GGT-gamma-glutamyl transferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase.

can be explained by the high prevalence of SCD in eastern Uganda according research done in Sironko district.[11] However, our finding aligns with studies in similar SCD populations, such as those conducted in the Democratic Republic of Congo (28–50%)[7] and Eastern Uganda (Sironko District).[11] However, it is higher than the 22% reported at Mulago National Referral Hospital in Uganda[12] and 11.1% reported in Sudan.[12] These variations may be due to differences in diagnostic methods, with ultrasonography potentially increasing detection rates, and sample demographics, as cohorts with older children tend to show higher prevalence rates.

Among the factors associated, we found elevated gamma-glutamyl transferase (GGT) and aspartate aminotransferase (AST) levels, both of which are markers of hepatic dysfunction often linked to gallstone formation. Elevated GGT was particularly significantly associated with the development of gallstones in children. Our findings indicate that the relationship between elevated GGT and gallstone formation may stem from the predisposition of children with certain genetic or metabolic conditions such as sickle cell anaemia or obesity to develop gallstones in addition to haemolysis or altered lipid metabolism, both of which can raise GGT levels and signal hepatobiliary stress, a known factor in gallstone development.[13] Our finding is consistent with other studies which have highlighted GGT as a reliable marker for gallstone risk in patients with sickle cell disease.[14,15] The observed association between elevated GGT and gallstones likely reflects secondary biliary stasis or subclinical cholestasis caused by the stones themselves, rather than acting as a primary cause for stone formation. However, in SCD, chronic hemolysis drives both pigment stone formation and

sickle cell hepatopathy, creating a direct pathophysiological link where altered liver enzymes and gallstones occur concurrently as complications of the same underlying hemolytic process.

Additionally, we also revealed that elevated AST and alkaline phosphatase (ALP) levels were slightly associated with gallstone development in our population though not statistically significant. We think the association we are observing here, could be possible due to the presence of hepatobiliary stress and bile duct obstruction in these children. AST rises with liver cell damage, while ALP increases with biliary tract disturbances. Gallstones can intermittently obstruct bile flow, causing mild, chronic elevation in these enzymes as liver and biliary cells respond to the stress and inflammation associated with impaired bile drainage.[16,17]

Surprisingly, our study found an inverse association between jaundice and gallstone formation, which contrasts with findings in adult populations. This may be due to differences in bilirubin metabolism in pediatric SCD patients, warranting further investigation.[18] This discrepancy may reflect the unique pathophysiology in paediatric SCD patients, who may experience bilirubin processing abnormalities without manifesting in gallstone formation. Similar studies in paediatric SCD populations have suggested that factors like recurrent haemolysis and ongoing liver stress may explain this association, but further investigation is needed to clarify this inverse relationship.[19] Other demographic factors, such as age and gender, did not show significant associations with gallstone development in this study.

This study is the first to specifically investigate gallstones in children and adolescents with SCD in Eastern Uganda, providing valuable insights into an under-researched population with significant clinical implications. Our findings highlight the potential benefit of routine annual ultrasound screenings for patients over 10 years old, which could facilitate early detection, timely intervention, and prevention of severe complications. Identifying key risk factors also enhances clinicians' ability to recognize at-risk patients and optimize monitoring and treatment strategies.

However, ultrasound imaging limitations in differentiating pigment, cholesterol, and mixed stones may have impacted diagnostic accuracy. Additionally, as a cross-sectional study, our findings represent a single time point, limiting causal inferences. Future case–control studies comparing individuals with and without gallstones would provide stronger evidence on risk factors and improve diagnostic precision, ultimately guiding more effective management strategies for this high-risk population.

Conclusion

In conclusion, this study identified a high prevalence of gallstones among pediatric patients with SCD in Eastern Uganda as compared to other studies in Uganda. Elevated GGT and AST levels emerged as significant factors associated with gallstone development, emphasizing the role of hepatic dysfunction in this cohort. Our findings highlight the importance of routine monitoring of liver enzymes in SCD management to detect gallstone risk early. We recommend routine ultrasound screening for children with SCD starting at age 10 to facilitate early detection and management of gallstones.

Abbreviations

ALP, Alkaline Phosphatase; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; GGT, Gamma-Glutamyl Transferase; LFT, Liver Function Test; MRRH, Mbale Regional Referral Hospital; SCD, Sickle Cell Disease; TB, Tuberculosis; USS, Ultrasound Scan; WHO, World Health Organization.

Data Sharing Statement

The datasets used and/or analyses during the current study are available from the corresponding author upon reasonable request from first author OOA.

Ethical Consideration and Consent to Participate

The study was approved by the Busitema University Faculty of Health Sciences Research Ethics Committee (BUFHS-REC) under approval number BUFHS-2023-135. All participants aged 18 and above provided informed consent. Children aged 8–17 provided assent alongside parental/guardian consent. Consent was obtained from the parent/guardian for those younger than 8 years. This study was conducted in accordance with the Declaration of Helsinki.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agreed to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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