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Original Research Article

Prevalence of Dengue in Sickle Cell Disease in Pre-School Children

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Abstract

Introduction: Millions of people are affected with dengue fever every year, which drives up healthcare expenses in many low-income countries. Organ failure and other serious symptoms may result. Another worldwide public health problem is sickle cell anaemia, which is most prevalent in Africa, the Caribbean, and Europe. Dengue epidemics have reportedly occurred in locations with a high frequency of sickle cell disease, compounding the health problems in these areas.

Aims and objectives: This study examines dengue infection in sickle cell disease-afflicted pre-schoolers.

Method: This Retrospective cohort study examined paediatric patients. Young people with sickle cell disease (SCD), dengue infection, and a control group without SCD or dengue were studied. Data on demographics, SCD consequences, medical treatments, and laboratory findings were gathered to analyse the influence of SCD on dengue severity and clinical outcomes, classified as severe or non-severe by the 2009 WHO classification. Using fever or admission symptoms, the research estimated acute illness duration.

Result: Table 1 compares haemoglobin genotype-based dengue episode features in SS, SC, and controls. Table 2 shows that severe dengue cases are older, have longer admission delays, and have particular symptoms. Table 3's multivariate analysis indicates SS genotype's high connection with severe dengue, multiorgan failure, and acute pulmonary problems. Table 4 relates severe dengue to greater white blood cell counts, anaemia, liver enzymes, and reduced lactate dehydrogenase.

Conclusion: This study is valuable but confined to hospitalised dengue patients with sickle cell illness. Small cohorts limit comparisons. Further study is needed since findings contradict predictions.

Keywords: Dengue, chills, headache, severe myalgia, vomiting, nausea, and prostration

Introduction

In poorer nations, dengue fever has become a prevalent cause of fever. Among the most serious viral illnesses that people can contract from arthropods and which has an impact on both mortality and quality of life is dengue. The World Health Organisation states that dengue fever affects between 50 and 100 million people worldwide each year, significantly increasing the cost of healthcare. Dengue fever has thus emerged as one of the most significant issues in public health. Dengue fever can show as anything from a severe fever accompanied by chills, headache, and severe myalgia to vomiting, nausea, and prostration [1]. Dengue fever involves several systems, leading to multiple organ failure and a higher death rate, even in the youngest cases. A higher incidence of viremia, which is linked to organ involvement such as the liver, is observed with an increase in DENV infection. The hepatic symptoms associated with dengue fever can be attributed to either direct viral toxicity or dysregulated immune-mediated liver damage [2]. The dengue virus primarily targets hepatocytes and Kupffer cells, which results in severe liver damage. As dengue fever is a rare manifestation of hepatitis, the doctor should be cautious not to rule out other possible causes of hepatitis in patients who have dengue fever. Therefore, When determining the aetiology of hepatitis, leptospirosis, malaria, and dengue fever should be on the differential list as well as hepatitis A, B, and C [3].

The most common virus transmitted by mosquitoes to humans is dengue. The ribonucleic acid (RNA) viral belonging to the Flaviridae family is the root cause of this endemo-epidemic illness. There are four different dengue serotypes (dengue serotypes 1, 2, 3, and 4). Main vector is Aedes aegypti. With significant geographic spread into new countries as well as more specifically, in subtropical & tropical

regions, the prevalence disease dengue has risen 30-fold during the previous five decades [4]. There are many different clinical manifestations of dengue, typically with uncertain clinical development and outcome. Even though most individuals recover following a self-limiting, mild course of treatment, a small minority may develop a severe illness that is primarily distinguished by plasma leakage in addition to haemorrhage. The following symptoms can occur in conjunction to cause severe dengue: 1-plasma leaks that could result in surprise (dengue shock) and/or water accumulation, regardless of the presence of breathing difficulties; (2) serious bleeding; and (3) significant organ damage. The WHO, or World Health Organisation, established these two clinical phenotypes in 2009 [5].

The synthesis of The mutation known as sickle haemoglobin S (HbS) causes just one amino acid (GluVal) to be substituted at the sixth residue of the -chain, which constitutes the normal haemoglobin (Haemoglobin A) molecule is the hallmark of A class of autosomal-dominate genetic illnesses is sickle cell disease (SCD). The most severe form of SCD is generally regarded as homozygous SS (sickle cell anemia) [6]. Compound heterozygotes, which include HbS in combination with a distinct deletion of a second -globin gene, which causes -globin synthesis to be reduced and causes HbC, D, OArab, or -thalassemia, can also be impacted and exhibit a variety of symptoms. SCD is characterised by chronic hemolysis, vasculopathy, & a prothrombotic state, which are brought on by sticky red blood cells (RBCs) that have an irregular shape and work in conjunction with endothelium or white blood cells (WBCs) [7]. A few conditions that might cause chronic pain include end-organ failure, stroke, lifelong agony, a mediocre level of life, and early mortality serious problems that these processes may cause. The death rate linked with sickle cell disease was significantly lowered by Combined by early comprehensive care for affected newborns, including prophylaxis against Streptococcus pneumoniae infection & parental education for prevention, and universal newborn screening was implemented [8].

A significant issue for worldwide public health is believed to be sickle-cell disease (SCD). Every year, there are about 250,000 births. Africa is the region with the greatest illness burden. However, the USA of North America, the Caribbean, and particularly Europe all see a lot of cases. The gene frequencies for the -globin chain anomalies S and C in Jamaica are 0.055 and 0.019, respectively [9]. Diabetic haemoglobin SC (HbSC) and homozygous haemoglobin SS (HbSS) diabetes have different phenotypic manifestations because of differences in the properties of haemoglobin C & S, such as the far more prominent K+ loss and dehydration of haemoglobin SC red blood cells. It is unknown whether the genotypes exhibit different phenotypic responses to dengue disease. One Sickle Cell Unit (SCU), with over 5,000 patients registered, is the only all-inclusive clinic in Jamaica offering care for sickle cell disease [10].

There have been reports of dengue fever (DF), a viral disease spread by mosquitoes, epidemics in several nations with high rates of sickle cell disease (SCD). Dengue fever, which is common in more than One hundred nations spread over Asia, the Pacific, Africa, the Americas, & the Caribbean are indeed a serious public health problem, much like sickle cell disease. It is estimated that about one-third of the global populace resides in these high-risk regions [11]. DF is a primary cause of death in areas that are tropical or subtropical (Centres to Prevention and Control of Diseases, 2013). The World Health Organisation (WHO) estimates that dengue causes between fifty and one hundred million infections yearly, 500,000 cases of dengue hemorrhagic fever (DHF), and 22,000 fatalities—the majority of which are children—each year. Over the previous 50 years, the prevalence of dengue has climbed 30-fold (World Health Organisation, 2014) [12].

DF is suspected in sickle cell disorder (SCD) patients if the patient has two or more of the following symptoms in addition to a fever: headache, retro-orbital discomfort, myalgia, arthralgia, or rash. Based on their clinical state, patients received therapy as outpatient care or were admitted to the hospital. During outbreaks, individuals suspected to possess DF are often classified as probable cases due to clinical criteria rather than being tested individually, in accordance with Ministry of Health reporting procedures [13].

Method

Research Design

This prospective cohort study included pediatric departments from our hospital in North Maharashtra from May 2022 to April 2023.120 Children with sickle cell disease (SCD) who had never had dengue fever in the past, children with confirmed dengue infection, and a control group without SCD or a dengue history were the research population. The participants provided demographic information including age and gender, as well as SCD complications such as acute splenic or hepatic sequestration, acute chest syndrome, vasoocclusive crises, and more. Data on narcotic pain treatment, transfusions, ICU hospitalizations, and past dengue infections (where known) were also documented. Hemoglobin, hematocrit, platelet, white blood cell, and liver function tests were also recorded for SCD and dengue patients. The study examined how SCD affects dengue severity and clinical outcomes. Dengue severity was divided into severe and non-severe cases per the 2009 WHO classification. The

duration of acute illness was assessed from fever or, in situations without fever, admission symptoms.

Inclusion and Exclusion Criteria

Inclusion

- Children who visited our hospital were included.
- Children without dengue fever who have sickle cell disease (SCD), homozygous SS or compound heterozygotes SC.
- The research included children hospitalized for their first dengue illness in the pediatric departments.
- Control group: Children admitted for confirmed dengue in the same pediatric departments without a history of dengue fever or chronic illnesses like SCD.

Exclusion

- Patient with dengue fever history.
- Children with SCD who did not fulfil homozygous SS or compound heterozygotes SC requirements.
- Clients who are at least 15 years old.
- Non-SCD chronic illness patients.
- People who didn't visit for follow-up
- SCD cases not detected by universal newborn screening.
- Medical records with missing or partial data limit accurate variable evaluation.
- Non-dengue patients having symptoms.

Statistical Analysis

Statistical tests and regression analyses were likely used to compare severe and non-severe dengue cases to determine SCD's impact on dengue complications. To detect severe dengue variables, we compared patients by illness severity. Data were presented as counts and percentages for categorical data and medians with IQR for quantitative data. We quantified the biological data admission-steady-state ratio. For proportions, Fisher's exact or Chi square test was used, and for median differences, Mann-Whitney U. We used a significance criterion of $P < 0.05$ for all two-tailed comparisons. The final model includes single covariate variables showing significant outcome associations. The statistical analysis was done by SPSS 25.

Result

Table 1 compares dengue episode characteristics in SS, SC, and control groups by haemoglobin genotype. At admission, SS patients are younger and have a shorter symptom start to admission delay. More precursors include painful vasoocclusive crises and acute splenic sequestration/splenomegaly. SC patients experience more fever, vomiting, stomach discomfort, and myalgia/arthralgia. Acute anemia, cutaneous problems, severe vasoocclusive crises, and acute pulmonary consequences are more common in SS and SC patients during hospitalization. Hospitalization is longer for SS and SC patients. These categories also had more severe episodes and deaths, especially SC patients. Haemoglobin genotypes affect dengue episode features and outcomes.

Table 1: Comparison of dengue episodes characteristics between hemoglobin genotypes

Variables		SS patients n = 30	SC patients n = 30	Controls n = 30	P-value	p*
Gender	Female	12 (40.00%)	14 (46.66%)	13 (43.33%)	0.8	
	Male	18 (60.00%)	16 (53.33%)	17 (56.66%)		
Age at admission in years – Median (IQR)		8 (5; 14)	9 (6; 15)	1 (0,5; 5)	<0.001	0.001
Delay time between first clinical symptoms and admission in days	<=3	22 (73.33%)	23 (76.66%)	1 (3.33%)	<0.001	
	>3	8 (26.66%)	7 (23.33%)	29 (96.66%)		
Antecedents						
Painful vasoocclusive crises		18 (60.00%)	19 (63.33%)		0.467	
Acute chest syndrome		5 (16.66%)	4 (13.33%)		0.714	
Stroke		3 (10.00%)	0 (0.0)		0.114	
Acute splenic sequestration/Splenomegaly		12 (40.00%)	3 (10.00%)		0.001	
Cholelithiasis		4 (13.33%)	4 (13.33%)		0.962	
Transfusions/Exchange program		12 (40.00%)	8 (26.66%)		0.063	
Infections		8 (26.66%)	9 (30.00%)		0.803	
Clinical symptoms at admission						
Fever		25 (83.33%)	29 (96.66%)	30 (100)	0.3	
TRC		8 (26.66%)	9 (30.00%)	0(0)	0.001	
Vomiting		9 (30.00%)	7 (23.33%)	14 ()	<0.001	
Abdominal pain		6 (20.00%)	9 (30.00%)	4 (13.33%)	0.04	
Splenomegaly		8 (26.66%)	7 (23.33%)	0 (0)	0.02	
Myalgia / Arthralgia		4 (13.33%)	8 (26.66%)	4 (13.33%)	0.1	
Hepatomegaly		5 (16.66%)	3 (10.00%)	6 (20.00%)	0.6	
Neurologic symptoms		4 (13.33%)	2 (6.66%)	1 (3.33%)	0.1	
Bleeding		1(3.33%)	0 (0.0)	0 (0)	0.9	
Hypotension / Shock syndrome		0 (0.0)	2 (6.66%)	0 (0)	0.9	
Complications during hospitalization						
Acute anemia (Hyperhemolysis, bleeding, …)		15 (50.00%)	8 (26.66%)	3 (10.00%)	0.2	
Skin complications		1 (3.33%)	2 (6.66%)	11 (36.66%)	0.001	

Painful vasoocclusive crisis	2 (6.66%)	6 (20.00%)	0 (0)	0.004	
Acute splenic sequestration	0 (0.0)	2 (6.66%)	0 (0)	0.3	
Acute pulmonary complications (pneumopathy, ACS, …)	2 (6.66%)	4 (13.33%)	0 (0)	0.02	
Multiorgan failure	8 (26.66%)	9 (30.00%)	0 (0)	0.001	
Shock syndrome	0 (0.0)	4 (13.33%)	0 (0)	0.8	
Oxygen dependence/Acidosis	1 (3.33%)	2 (6.66%)	0 (0)	0.7	
Treatments during hospitalization					
Hyperhydration	26 (86.66%)	29 (96.66%)	0 (0)	0.8	
Transfusion	14 (46.66%)	5 (16.66%)	0 (0)	0.08	
Transfer in intensive care unit	1 (3.33%)	7 (23.33%)	0 (0)	0.004	0.01
Antibiotics	18 (60.00%)	21 (70.00%)	0 (0)	<0.001	
Analgesia				<0.001	<0.001
No analgesia (antalgics)	0 (0.0)	1 (3.33%)	0 (0)		
Analgesics	28 (93.33%)	14 (46.66%)	30 (100)		
Duration of hospitalization (days) M, IQR	6 (3; 7)	5 (3; 7.5)	3 (3; 5)	<0.001	
Severe episodes	11 (36.66%)	18 (60.00%)	0 (0)	<0.001	
Death	1 (3.33%)	3 (10.00%)	0 (0)	0.02	

Table 2 highlights severity-based dengue episode features. It comprises severe and non-severe dengue parameters. A greater median age at admission (10.5 vs. 5 years) and longer delay between symptom onset and admission (>3 days) in severe cases are notable. Vomiting, stomach discomfort, and delayed capillary refill time characterize severe dengue, while non-severe cases have greater fever. Multiorgan failure, acute pulmonary problems, and transfusions and antibiotics are more common in severe instances. Unfortunately, severe cases had a higher fatality rate (13.33% vs. 0) and lengthier hospital stays. This data shows substantial clinical differences between severe and non-severe dengue patients and may improve early identification and therapy.

Table 2: Comparison of dengue episodes characteristics according to the severity.

Characteristic		**Severe dengue**	**Non-severe dengue**	**P-value**
N		**45**	**45**	
Gender	Female	25 (55.55%)	30 (66.66%)	0.1
	Male	20 (44.44%)	15 (33.33%)	
Age at admission in years – Median (IQR)		10.5 (5; 15)	5 (1; 10)	0.001
Delay time between first clinical symptoms and admission in days	**<=3**	40 (88.88%)	25 (55.55%)	0.005
	>3	5 (11.11)	20 (44.44%)	
Clinical symptoms at admission				
Fever		29 (64.44%)	40 (88.88%)	0.5
Prolonged Capillary refill time		9 (20.00%)	8 (17.77%)	0.007
Vomiting		8 (17.77%)	30 (66.66%)	0.05
Abdominal pain		9 (20.00%)	15 (33.33%)	0.02
Splenomegaly		8 (17.77%)	12 (26.66%)	0.06
Myalgia/arthralgia		6 (13.33%)	8 (17.77%)	0.8
Hepatomegaly		5 (11.11%)	4 (8.88%)	0.4
Neurologic symptoms		4 (8.88%)	4 (8.88%)	0.4
Bleeding		3 (6.66%)	1 (2.22%)	0.4
Hypotension/shock syndrome				
Complications during hospitalization				
Acute anemia (hyperhemolysis, bleeding,…)		11 (24.44%)	18 (40.00%)	0.2
Skin complications		5 (11.11%)	21 (46.66%)	0.2
Painful vasoocclusive crisis		6 (13.33%)	6 (13.33%)	0.1
Acute splenic sequestration		1 (2.22%)	1 (2.22%)	0.5
Acute pulmonary complications* (pneumopathy, ACS, …)		6 (13.33%)	1 (2.22%)	0.007
Multiorgan failure		25 (55.55%)	2 (4.44%)	<0.001
Shock syndrome		6 (13.33%)	0 (0)	
Oxygen dependence/acidosis		4 (8.88%)	1 (2.22%)	0.08
Treatments during hospitalization				
Hyperhydration		41 (91.11%)	25 (55.55%)	0.8
Transfusion		17 (37.77%)	6 (13.33%)	0.002
Transfer in intensive care unit		8 (17.77%)	0 (0)	
Antibiotics		31 (68.88%)	11 (24.44%)	<0.001
Analgesia				<0.001
No analgesia (antalgics)		0 (0.0)	1 (2.22%)	
Mild pain		0 (0)	45 (100)	

Moderatepain	21 (46.66%)	29 (64.44%)	
Severe pain	17 (37.77%)	4 (8.88%)	
Duration of hospitalization (days) M, IQR	6 (4,5; 9)	3 (3; 5)	<0.001
Death	6 (13.33%)	0 (0)	

A multivariate analysis of severe and non-severe dengue (n=45) is shown in Table 3. The analysis covers hemoglobin genotypes, multiorgan failure, and acute pulmonary consequences. Hemoglobin genotypes SS had a substantially higher adjusted odds ratio (AOR) of 5.8 (CI 95%: 2.1-16.4) and p-value of 0.001. Multiple organ failure is substantially linked to severe dengue (AOR: 38, CI 95%: 6.8-213.7, p<0.001), whereas acute pulmonary complications have a significant connection (AOR: 20, CI 95%: 1.7-240, p=0.01). Each variable's sensitivity, specificity, PPV, and NPV percentages help evaluate these parameters' prediction capacities in discriminating severe and non-severe dengue cases. This data suggests that severe dengue is associated with hemoglobin genotypes SS, multiorgan failure, and acute pulmonary consequences.

Table 3: Multivariate analysis

Variables	**Severe dengue (n = 45)**	**Non severe dengue (n = 45)**	**AOR CI 95 %**	**P-value**
Hemoglobin genotypes			5.8 [2.1-16.4]	0.001
SS	14 (31.11%)	20 (44.44%)		
SC	30 (66.66%)	14 (31.11%)		
Normal	1 (2.22%)	11 (24.44%)		
Multiorgan failure				<0.001
Yes	22 (48.88%)	14 (31.11%)	38 [6.8–213.7]	
No	4 (8.88%)	30 (66.66%)		
Acute pulmonary complication				0.01
Yes	8 (17.77%)	20 (44.44%)	20 [1,7–240]	
No	1 (2.22%)	31 (68.88%)		
Sensitivity %	**Specificity %**	**PPV %**	**NPV %**	
64	96	88	85	

The table 4 below compares biological data from 45 instances of severe dengue fever with 45 cases of less severe dengue fever. White blood cell (WBC) counts are much higher in severe cases, with the odds ratio (OR) being 3.2. Platelet levels are comparable. Severe instances are characterized by elevated aspartate aminotransferase (ASAT) and alanine aminotransferase (ALAT) and decreased hemoglobin and hematocrit, suggesting the presence of anemia. Severe instances had noticeably reduced levels of lactate dehydrogenase (LDH). These findings imply that higher WBC counts, anemia, and liver enzyme levels are related with dengue severity and may be useful for clinical evaluation and therapy.

Table 4: Characteristics of biological data according to the severity.

Variables, median (IQR)	**Severe dengue (n = 45**	**Non severe dengue (n = 45)**	**OR (CI 95 %)**	**p**
Platelets (G/L)	248 (127;366)	250 (167;269)	0.84 [0.3–0.5]	0.8
WBC (G/L)	9.2 (5.1;12.3)	7.0 (4.1;10)	3.2 [0.98–10.5]	0.05
Neutrophils (G/L)	4.05 (2.7; 6.3)	4.8 (2.65; 7.25)	1.89 [0.62–5.8]	0.3
Lymphocytes (G/L)	2.56 (1.7; 4.17)	2.19 (1.35; 4.48)	2.19 [0.7–6.7]	0.2
Hémoglobin (g/dL)	9.2 (8.4; 11.2)	9.6 (7.9; 10.2)	0.16 [0.4–0.6]	0.007
Hématocrit (%)	26,3 (24.3; 32)	26 (22.8; 28.9)	0.12 [0.03–0.58]	0.008
ASAT (IU/L)	38 (16; 66)	38 (25; 68)	0.5 [0.2–1.4]	0.2
ALAT (IU/L)	32.5 (22; 44.5)	28 (20; 39)	0.2 [0.1–0.6]	0.005
LDH (IU/L)	416 (286; 761)	569 (327; 757)	0.06 [0.01–0.3]	<0.001

Platelets (G/L) - Platelets per liter of blood; WBC (G/L) - White Blood Cells per liter of blood; Neutrophils (G/L) - Neutrophils per liter of blood; Lymphocytes (G/L) - Lymphocytes per liter of blood; Hémoglobin (g/dL) - Hemoglobin concentration in grams per deciliter of blood; Hématocrit (%) - Hematocrit level in percentage; ASAT (IU/L) - Aspartate Aminotransferase in

International Units per liter of blood; ALAT (IU/L) - Alanine Aminotransferase in International Units per liter of blood; LDH (IU/L) - Lactate Dehydrogenase in International Units per liter of blood; OR Odds Ratio; CI Confidence Interval

Discussion

In the western region of India, sickle cell disease was more prevalent in rural populations. It is among the frequent reasons why children experience morbidity, death, and repeated hospital stays. Few studies have focused on the epidemiology of sickle cell disease within this community, therefore this one was done to evaluate the clinicohematological image of the juvenile sickle cell disease community in a rural tertiary care hospital in western India. As sickle cell disease is diagnosed in paediatric patients, the most common symptoms are vasoocclusive crises and generalised body pain; nevertheless, the haematological picture suggests microcytic hypochromic anaemia. The age of presentation and the degree of anaemia at diagnosis are positively correlated [14].

In Jamaica, dengue disease is extremely common and spreads at exponential rates during epidemics. The reason for this study was the lack of local information and the possibility of large-scale epidemics in a nation where children make up one-third of the population [15]. During the 2012 dengue fever outbreak that swept the island, we assessed the course of dengue in paediatric individuals hospitalised to Mona, Jamaica's University Hospital the West Indies (UHWI). All hospitalised children under the age of 15 who had a physician diagnosis of dengue are included in this retrospective analysis. Short height and a delayed presentation were strongly linked to severe dengue [16].

Children having sickle cell illness need more time in the hospital. The percentage of fatal cases was 3.73%. Utilising effective and safe dengue vaccinations could lessen the impact of children's illness and death linked to dengue [17].

The role of haemoglobin type in the dengue fever intensity is not well understood. The purpose of the research was to ascertain the actual fatality rate and the influence of genotype on individuals diagnosed with dengue fever and the sickle cell illness. In the course of the 2010–2012 study period, 40 individuals with confirmed cases of dengue with sickle cell disease were included in this retrospective analysis [18].

Compared to the overall population's 0.41% case fatality ratio, individuals with either homozygous SS illness or haemoglobin SC disease had a much higher ratio of 12.5%. When comparing the group who had homozygous SS disease to those who had haemoglobin SC disease, the unadjusted chances of death were OR = 4.4. The two factors that were independent of the sickle cell disorder genotypes were the haemoglobin concentration at presenting (OR = 0.57) and the decline in haemoglobin intensity beyond the steady state (OR = 0.59) [19]. When the presentation's haemoglobin concentration was taken into account, the SC genotype's risk of mortality was shown to be higher than that of the SS genotype OR = 13.4. Patients having a relatively moderate genotype (haemoglobin SC) could have a greater chance of developing deadly dengue [20].

There has been evidence in human parvovirus B19 infections that More serious outcomes including acute chest syndrome as well as vaso-occlusive crisis, which cause fast respiratory failure from many pulmonary emboli, are linked to a non-SS genotype. This severe development is the result of widespread sickle cell disease and bone marrow necrosis, which causes fat embolism syndrome. Previous studies have demonstrated that the increased risk for fat embolisation syndrome (FES) and bone marrow necrosis (BMN) in HbSC patients can be explained by their greater baseline haemoglobin level & higher blood viscosity. Tsitsikas et al. (2014) suggested that a "single triggering event"—such as an HPV B19 infection, but not only—caused the acute extensive BMN in large-scale The distinctive discharge of fat & necrotic marrow into the bloodstream of FES. This reaction was then complex by a pathological immunology response that led to endothelial inflammation as well as dysfunction. A comparable mechanism may account for the elevated severity prevalence of dengue infection in SC patients, according to our hypothesis. To test this idea, however, prospective studies are necessary [21].

In actuality, no particular treatment is effective in treating dengue illness. Blood transfusions were utilised more frequently in our study's SS patients, most likely due to their higher frequency of acute anaemia presentations. The severe course and elevated mortality rate among individuals with SC may be explained by the correlation between greater haemoglobin levels and hemoconcentration associated with dengue, or perhaps caused by BMN/fat embolisms [22]. Another theory is that capillary leakage—even subclinical—could hasten the rheologic consequences. The optimal course of treatment is Early, vigorous exchange transfusions would be recommended for all sickle cell patients with severe dengue infection to lower circulating HbS stages, hyperviscosity, and hyperhydration [23].

To use clinical characteristics and the updated WHO recommendations to categorise probable dengue investigations into severe dengue, dengue fever, and dengue with warning signs, with a focus on serology. Dengue fever was detected in 568 youngsters; 4.2% of the population studied was younger than one year old. The majority (42.6%)

were in the 5–10 year age range. The bulk of the kids had fever and flushing. The serious dengue group was more likely to experience gastrointestinal bleeding. Immunoglobulin M (IgM) was positive in 15.8% of cases, antibody G (IgG) in 14.6%, and dengue nonstructural peptide antigen (NS1Ag) in 78% of cases [24]. More problems were seen in children with IgG, however, they were not statistically significant. 1.2% of deaths were recorded. Dengue serology did not benefit the patient, although it did aid in confirming the diagnosis. There are many parallels between the symptomatology of severe dengue and dengue with warning signs. Additional investigation is required to ascertain the kind and intensity of therapy response in newborns and obese adolescents with severe dengue [25].

Conclusion

The study has concluded although no specific therapy is effective in managing dengue infection, but blood transfusion was necessary in SS patients as acute anemia was more frequent in them. Patients with SC genotypes had shown higher death rate because of capillary leakage and rheologic complications. The study also drawn conclusion that the management of these patients should be done aggressively by exchange transfusion to reduce the circulating HbS and hyperviscosity and thereby managing the dehydration. Our study is useful yet limited. Only hospitalized dengue patients with sickle cell illness were examined. Our small cohort prevents us from comparing dengue to other infectious illnesses in SCD patients. However, our work is the first complete dengue-affected pediatric SCD cohort investigation. Strong points include well-documented SCD genotypes and symptoms and dengue case confirmation. Our data contradict conventional thinking, showing that children with the SC genotype, typically associated with milder illness, had a greater rate of severe dengue and fatality than those with the SS genotype. These findings are useful, but prospective research is needed. Further study is needed to understand the complicated relationship between SCD genotypes and dengue severity to improve clinical care and patient outcomes.

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