Original Article

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Paediatrics Section

Serum Cortisol and Lipid Profile as Predictors of Dengue Severity in Children: A Prospective Observational Study

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ABSTRACT

Introduction: Early prediction of severe dengue in children continues to be difficult, even with World Health Organisation (WHO) warning signs. Alterations in stress hormones and lipid metabolism have been associated with dengue severity; however, prospective paediatric data are limited. Serum cortisol and lipid profile are routinely available laboratory parameters and may provide additional information regarding disease severity.

Aim: To evaluate the significance of serum cortisol and lipid profile in forecasting the advancement to severe dengue in children.

Materials and Methods: This prospective observational study was conducted in the Department of Paediatrics, SRM Medical College Hospital and Research Centre, Kattankulathur, Chennai, Tamil Nadu, India, over two years (July 2024-July 2026). A total of 90 dengue NS1 or IgM ELISA-positive children aged between 0-14 years with warning signs and presenting between days 3 and 7 of illness were enrolled after informed consent. Serum cortisol and fasting lipid profile were measured at enrolment, and the day of illness at sampling was recorded. Children were classified according to subsequent progression to severe dengue. Categorical variables were compared using the Chi-square test and continuous variables using the independent-samples t-test; p-value <0.05 was considered statistically significant.

Results: In the present study, 15 out of 90 (16.67%) children progressed to severe dengue. Boys constituted 57/90 (63.33%) of the cohort, and the largest age group was 1-5 years (32/90; 35.56%). Fluid accumulation, mucosal bleeding, lethargy, and significant liver enlargement were significantly associated with progression to severe dengue. Severe dengue was also associated with higher total leucocyte count and serum urea, lower serum sodium and bicarbonate, and prolonged prothrombin time. Mean serum cortisol was higher in children who progressed to severe dengue than in those who did not (18.74±6.14 vs 15.46±4.48 µg/dL; p-value =0.017). Mean triglyceride levels were also higher (220.07±141.71 vs 162.97±62.69 mg/dL; p-value =0.014), whereas mean total cholesterol levels were lower (99.67±23.70 vs 122.01±26.71 mg/dL; p-value =0.003). Very Low-Density Lipoprotein (VLDL), Low-Density Lipoprotein Cholesterol (LDL-C), and High-Density Lipoprotein (HDL) levels did not differ significantly between groups.

Conclusion: High levels of cortisol and triglycerides with low levels of total cholesterol at the time of admission are strongly linked to the development of severe dengue.

Keywords: Hypocholesterolemia, Lipid metabolism, Plasma leakage, Stress response, Triglycerides

INTRODUCTION

Dengue Virus (DENV) is a positive-sense RNA virus of the family *Flaviviridae* transmitted primarily by *Aedes aegypti* and *Aedes albopictus* [1,2]. Dengue remains an important arboviral disease in tropical and subtropical regions, including South-East Asia [1,3]. In 2019, nearly 5.2 million dengue cases were reported worldwide [1]. Early identification of children at risk is important because timely supportive care and appropriate fluid management can reduce mortality [4]. Dengue continues to contribute substantially to morbidity and mortality to India [5].

Dengue has a broad clinical spectrum, ranging from asymptomatic or mild febrile illness to severe disease with plasma leakage, haemorrhage, shock and multiorgan dysfunction [1,3]. WHO classifies dengue as dengue without warning signs, dengue with warning signs, or severe dengue [3]. Although warning signs facilitate early recognition of patients at risk, no single clinical parameter reliably predicts progression to severe disease [3]. Reliable biomarkers that complement clinical assessment may therefore be useful.

Dengue pathogenesis involves viral replication, host immune responses, endothelial dysfunction and cytokine-mediated vascular permeability [2,6]. Acute infection activates the Hypothalamic-Pituitary-Adrenal (HPA) axis, increasing cortisol secretion as part of the stress response [7,8]. Bongsebandhu-phubhakdi C et al., reported higher cortisol concentrations in severe dengue and suggested potential value as an adjunctive biomarker [7]. Previous studies have evaluated cortisol changes during dengue haemorrhagic fever and the relationship between cortisol, ACTH and disease severity [9,10]. However, prospective paediatric evidence remains limited.

Dengue infection is also associated with alterations in lipid metabolism. Changes in cholesterol and lipoprotein metabolism may accompany disease progression, with lower total cholesterol and LDL-C levels reported in more severe disease [11,12]. A systematic review and meta-analysis found an association between lower cholesterol/LDL-C concentrations and severe dengue [12]. Increased triglyceride concentrations have also been described and may reflect altered lipolysis and fatty-acid metabolism during acute inflammation [11,13].

Most available evidence on cortisol and lipid abnormalities in dengue is derived from adults or mixed-age populations [7,9-13]. Paediatric patients may differ in their physiological and immune responses to infection. Therefore, this prospective study evaluated the association of serum cortisol and fasting lipid profile with progression to severe dengue in children, together with clinical warning signs and routine laboratory parameters.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Paediatrics, SRM Medical College Hospital and Research Centre, Kattankulathur, Chennai, Tamil Nadu, India, over two years (July 2024-July 2026). Study was commenced after approval from the Institutional Ethics Committee (SRMIEC–ST0724-1829).

Inclusion criteria: Ninety admitted children aged from birth to 14 years with dengue NS1 or IgM ELISA positivity and WHO warning signs were included [3]. Eligible children were enrolled between days 3 and 7 of fever, after valid written consent from parents or guardians.

Exclusion criteria: Children with negative dengue tests, co-infections, illness beyond day 7, obesity, immunodeficiency, use of immunosuppressive therapy, or refusal of consent were excluded from the study.

Days 3-7 were selected because this interval encompasses the late febrile and early critical phases, during which clinical deterioration and plasma leakage may emerge. The day of illness at enrolment and sample collection was recorded. Because cortisol and lipid values may vary across this interval, sampling timing was considered a potential source of confounding and is acknowledged as a limitation of the study.

Sample size: Based on the previous cross-sectional observational study used for sample-size estimation, the required sample size was calculated using $n=(Z\alpha/2)^2pq/L^2$. The values used were $Z\alpha/2=2.58$, p=65% (prevalence proportion used in the original sample-size calculation), q=35%, and L=13% (20% relative precision of p), giving a calculated sample size of 89.6. The final sample size was rounded up to 90 [13].

Study Procedure

A predesigned study proforma was used to record demographic details, day of fever at admission, past history of dengue, heart rate, respiratory rate, blood pressure, dengue NS1/IgM ELISA status, clinical warning signs, and laboratory findings. Warning signs assessed included fluid accumulation, persistent vomiting, mucosal bleeding, lethargy/restlessness, liver enlargement >2 cm, and rising haematocrit with a concurrent decrease in platelet count. Severe dengue was assessed using WHO-based clinical criteria, including respiratory distress due to plasma leakage, signs of shock, severe bleeding, and severe organ involvement, including marked transaminase elevation [3,6].

Routine haematological and biochemical investigations included haematocrit/PCV, platelet count, total leucocyte count, renal function tests, serum electrolytes, liver transaminases, coagulation profile, serum cortisol, and fasting lipid profile. An early-morning serum sample was collected after admission during the eligible day 3-7 window, together with routine samples, for cortisol and fasting lipid profile estimation.

The laboratory parameters, units, methods of estimation and institutional reference intervals used in the study are presented in [Table/Fig-1]. Reference ranges/cut-off values were based on the age-appropriate institutional laboratory reference intervals used at SRM Medical College Hospital and Research Centre.

Parameters	Unit	Method of estimation	Reference framework
PCV/ haematocrit	%	Automated haematology analysis	34-40% (institutional laboratory reference interval); no study-specific diagnostic cut-off
Platelet count	/µL	Automated haematology analysis	150,000-410,000 /µL (institutional laboratory reference interval); no study-specific diagnostic cut-off
Total leucocyte count	/µL	Automated haematology analysis	5,000-15,000 /µL (institutional laboratory reference interval); no study-specific diagnostic cut-off
Urea	mg/dL	Urease-GLDH	15-45 mg/dL (institutional laboratory reference interval); no study-specific diagnostic cut-off
Creatinine	mg/dL	Jaffe's kinetic	≤0.5 mg/dL (institutional laboratory reference interval); no study-specific diagnostic cut-off
Sodium, Potassium, Chloride, Bicarbonate	mEq/L	ISE-Indirect	Sodium: 135-145; Potassium: 3.5-5.0; Chloride: 95-105; Bicarbonate: 21-31 mEq/L (institutional laboratory reference intervals); no study-specific diagnostic cut-off
AST, ALT	U/L	UV Kinetic/IFCC	AST: <31 U/L; ALT: <45 U/L (institutional laboratory reference intervals); no study-specific diagnostic cut-off
PT, APTT, INR	Seconds/ratio	Automated coagulation analysis	11.9-15.1 seconds/ 25.4-33.8 Seconds/ 0.9-1.1 (institutional laboratory reference intervals); no study-specific diagnostic cut-off
Serum cortisol	µg/dL	Institutional laboratory immunoassay platform (CMIA)	8 AM cortisol: 3.7–19.4 µg/dL (institutional laboratory report); no study-specific diagnostic cut-off
Total cholesterol, triglycerides, VLDL, LDL, HDL	mg/dL	Fasting lipid profile by institutional automated laboratory methods (automated enzymatic colorimetric method on spectrophotometric chemistry analyser)	Total cholesterol: <200; triglycerides: 40-150; VLDL-C: <40; LDL-C: 80-130; HDL-C: >40 mg/dL (institutional laboratory reference intervals); no study-specific diagnostic cut-off

[Table/Fig-1]: Laboratory parameters, units, methods of estimation and institutional reference intervals used in the study.
PCV: Packed cell volume; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; PT: Prothrombin time; APTT: Activated partial thromboplastin time; INR: International normalised ratio; CMIA: Chemiluminescent microparticle immunoassay; VLDL-C: Very-low-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; HDL-C: High-density lipoprotein cholesterol

STATISTICAL ANALYSIS

Continuous variables were summarised as mean±standard deviation (SD), with median and range where appropriate. Categorical variables were summarised as frequency and percentage. The Chi-square test was used for categorical associations, and the independent-samples t-test was used to compare mean values between children with and without progression to severe dengue. A p-value <0.05 was considered statistically significant. Statistical analyses were performed using IBM Statistical Package for the Social Sciences (SPSS), version 25.0. No ROC/AUC analysis or predictive cut-off values were calculated because the present analysis evaluated associations rather than diagnostic or predictive accuracy.

RESULTS

[Table/Fig-2] shows that the cohort had a male predominance, with the 1-5-year age group being the largest. Significant liver enlargement was the most frequent warning sign, followed by persistent vomiting. Fluid accumulation, mucosal bleeding, respiratory distress and shock were observed in smaller proportions of children.

Parameters	n (%)
Age (years)	
<1	6 (6.67)
1-5	32 (35.56)
6-10	29 (32.22)
11-15	23 (25.56)
Sex	
Female	33 (36.67)
Male	57 (63.33)
Past H/O dengue	
Absent	88 (97.78)
Present	2 (2.22)
Fluid accumulation	
Present	15 (16.67)
Absent	75 (83.33)
Persistent vomiting	
Present	48 (53.33)
Absent	42 (46.67)
Mucosal bleed	
Present	8 (8.89)
Absent	82 (91.11)
Lethargy	
Present	29 (32.22)
Absent	61 (67.78)
Significant liver enlargement	
Present	68 (75.56)
Absent	22 (24.44)
Respiratory distress	
Absent	79 (87.78)
Present	11 (12.22)
Signs of shock	
Absent	84 (93.33)
Present	6 (6.67)
AST/ALT >1000 IU/L	
Absent	86 (95.56)
Present	4 (4.44)

[Table/Fig-2]: Descriptive analysis of demographic and clinical parameters.

[Table/Fig-3] demonstrates the baseline haematological and biochemical profile of the study population. Cortisol and triglyceride concentrations showed considerable variability, while the lipid parameters were reported with their corresponding units. These baseline values were subsequently compared between children with and without progression to severe dengue.

Parameters	Units	Mean±SD
PCV	%	36.9±4.44
Platelet	/µL	149956.67±118783.59
Total count	/µL	5405.16±4585.62
Urea	mg/dL	22.43±8.34
Creatinine	mg/dL	0.47±0.14
NA	mEq/L	133.3±3.34
K	mEq/L	3.96±0.56
Cl	mEq/L	103.23±3.81
HCO_3	mEq/L	21.59±2.5
AST	U/L	93.08±62.09
ALT	U/L	42.73±32.22
PT	seconds	14.11±3.12
APTT	seconds	34.73±5.17
INR	ratio	1.06±0.3
S. Cortisol	µg/dL	16.01±4.91
S. Triglycerides	mg/dL	172.49±82.97
S. VLDL	mg/dL	42.32±16.81
S. LDL	mg/dL	72.76±16.7
S. HDL	mg/dL	35.16±15.88
S. Cholesterol	mg/dL	118.29±27.41

[Table/Fig-3]: Descriptive analysis of haematological and biochemical parameters in study population (N=90).

[Table/Fig-4] shows statistically significant differences in total leucocyte count, serum urea, sodium, bicarbonate and prothrombin time between children with and without progression to severe dengue. Children who progressed had higher total leucocyte count, urea and prothrombin time, with lower sodium and bicarbonate. Other baseline haematological and biochemical parameters did not differ significantly between the groups.

Parameters	Progression to Severe Dengue (Mean±SD)		p-value
	Yes (N=15)	No (N=75)	
PCV	36.07±5.93	37.07±4.11	0.429
Platelet	161760±204250.45	147596±95094.98	0.676
Total count	8720±9166.66	4742.19±2592.37	0.002*
Urea	26.8±10.71	21.55±7.57	0.025*
Creatinine	0.43±0.11	0.48±0.14	0.229
Sodium	131.13±3.44	133.73±3.16	0.005*
Potassium	4.14±0.81	3.92±0.49	0.168
Chloride	103.33±4.34	103.21±3.73	0.912
Bicarbonate	20.33±3.72	21.84±2.13	0.033*
SGOT	89±48.35	93.91±64.77	0.782
SGPT	35.6±19.83	44.18±34.11	0.350
Prothrombin time	15.57±6.98	13.81±1.37	0.047*
APTT	36.57±9.93	34.35±3.53	0.130
INR	1.16±0.57	1.04±0.2	0.154

[Table/Fig-4]: Comparison of mean baseline haematological and biochemical parameters between progression to severe dengue (N=90).
* indicates statistical significance; p<0.05 was considered statistically significant.

[Table/Fig-5] shows significant associations of fluid accumulation, mucosal bleeding, lethargy and significant liver enlargement with progression to severe dengue. Persistent vomiting did not show a statistically significant association. [Table/Fig-6] shows that children who progressed to severe dengue had higher mean serum cortisol and triglyceride levels and lower mean total cholesterol. These differences were statistically significant. VLDL, LDL and HDL did not differ significantly between the two groups.

DISCUSSION

The present study assessed serum cortisol and lipid profile as preliminary biomarkers of severity in paediatric dengue. Most of the children in the present study were aged between 1 and 5 years and boys were more numerous than girls (63.3%). This data was in accordance with the study conducted by Gupta N et al., [5]. Most of the patients presented between days 4 and 5 of illness when plasma leakage usually begins [3,6].

Signs statistically significant for an individual worsening with severe dengue were fluid accumulation, mucosal bleeding, lethargy and hepatomegaly >2 cm. Plasma leakage is a key component of the development of severe dengue, resulting from endothelial dysfunction and cytokine-mediated increased vascular permeability [2,6]. Thus, the present study pathophysiologically substantiates the association of fluid accumulation with severe dengue. Mucosal bleeding is a sign of coagulopathy and capillary fragility is common in severe disease

Clinical parameters	Progression to severe dengue (n=15)		Progression to severe dengue (n=75)		Chi-square test χ^2	p-value
	Yes	No	Yes	No		
Fluid accumulation	11 (73.33%)	4 (26.67%)	4 (5.33%)	71 (94.67%)	41.61	<0.005*
Persistent vomiting	8 (53.33%)	7 (46.67%)	40 (53.33%)	35 (46.67%)	0.00	0.999
Mucosal bleed	5 (33.33%)	10 (66.67%)	3 (4.00%)	72 (96.00%)	13.280	0.003*
Lethargy	13 (86.67%)	2 (13.33%)	16 (21.33%)	59 (78.67%)	24.431	<0.001*
Significant liver enlargement	15 (100.00%)	0	53 (70.67%)	22 (29.33%)	5.824	0.018*

[Table/Fig-5]: Association of clinical warning signs with progression to severe dengue.
*Statistically significant; p<0.05. Exact p-values are presented based on the reported Chi-square statistics.

Parameter	Progression to severe dengue (mean±SD)		p-value
	Yes (N=15)	No (N=75)	
S. Cortisol	18.74±6.14	15.46±4.48	0.017*
S. Triglycerides	220.07±141.71	162.97±62.69	0.014*
S. VLDL	39±13.29	42.99±17.43	0.405
S. LDL	66.8±18.26	73.95±16.24	0.131
S. HDL	36.53±19.62	34.88±15.16	0.715
S. Cholesterol	99.67±23.7	122.01±26.71	0.003*

[Table/Fig-6]: Comparison of mean parameters between progression to severe dengue (N=90).
* indicates statistical significance. A p-value < 0.05 was considered statistically significant

[6]. Lethargy and restlessness may indicate systemic inflammation or early circulatory compromise [6]. Hepatomegaly or enlarged liver is likely due to direct injury to the liver from the virus and immune-mediated inflammation of the liver [6]. Persistent vomiting and a rise in haematocrit with thrombocytopenia did not have independent statistical significance, supporting previous reports that individual warning signs may not be reliable predictors of severity [3].

The significant baseline laboratory tests associated with severe dengue were total leucocyte count, serum urea, sodium, bicarbonate and prothrombin time. An increased leucocyte count may indicate an exaggerated inflammatory response or a superinfection [2]. High blood urea may be a sign of renal involvement or increased catabolic stress [6]. Hyponatraemia is frequently seen in severe dengue. It may be caused by capillary leakage, excess antidiuretic hormone or renal dysfunction [6]. Low bicarbonate levels mean metabolic acidosis due to low blood flow to tissues. Long prothrombin time means that coagulation pathway is involved [6]. These findings were in accordance with the known mechanisms of severe dengue pathophysiology [2,6].

One of the key findings of this study was that higher levels of cortisol in the blood were associated with the progression of severe dengue. The HPA axis is activated during an acute infection. This causes the body to produce more cortisol as a stress response [7,8]. Previous studies have noted increased cortisol levels during the febrile phase of dengue [9,10]. Recent prospective study found that cortisol levels were significantly higher in cases of severe dengue than in non severe cases. Thus, cortisol levels could be an early marker of the disease [7]. The present study findings support these reports and suggest that serum cortisol might be a useful biomarker for severity of dengue in children.

The DENV changes the host's lipid pathways to make it easier for the virus to replicate. This alters the synthesis of cholesterol and the catabolism of lipoproteins [11]. In present study, children with severe dengue had much lower levels of total cholesterol and much higher levels of triglycerides. Previous studies have shown that total cholesterol, LDL-C, and HDL-C concentrations decrease as dengue severity increases [11,12]. A systematic review and meta-analysis has demonstrated that there is a strong association between having low cholesterol levels and having severe dengue [12]. Hypocholesterolaemia in severe dengue is due to the liver being unable to produce adequate cholesterol, ingesting too much cholesterol, and cytokines changing the body's cholesterol metabolism [11]. Increased triglyceride levels may represent increased lipolysis and altered fatty acid metabolism during systemic inflammation [11]. Similar results for triglycerides were reported by Van Gorp ECM et al., who showed that patients with dengue infection had significantly increased triglyceride levels together with decreased cholesterol levels indicating marked changes in lipid metabolism during the acute phase of infection [13].

In the present study, there were no statistically significant differences in the levels of VLDL, LDL and HDL. However, the strong correlation between total cholesterol and triglycerides supports the clinical utility of checking lipid profiles on admission. Most of the data on changes in cortisol and lipids in dengue come from adults [7,11]. Paediatric-specific evidence remains limited. Reliable early biomarkers are particularly important in children, whose physiological and immune responses differ from those of adults.

To summarise, the present study data suggest that severe dengue is associated with not only clinical deterioration but also major endocrine and metabolic dysregulation. The combination of serum cortisol and lipid profile tests with clinical warning signs may be helpful in better determining which children are at high-risk for dengue.

Limitation(s)

This study had a relatively small sample size, with only 15 children progressing to severe dengue, limiting statistical precision and adjustment for potential confounders. The single-centre observational design limits generalisability and does not establish causality. Serum sampling occurred between days 3 and 7 of illness; although the day of illness was recorded, variation in sampling time may have influenced cortisol and lipid values. The study also did not perform ROC/AUC analysis or derive diagnostic cut-off values. Therefore, the findings should be interpreted as associations that require validation in larger multicentre prospective studies with standardised sampling and predictive-accuracy analyses.

CONCLUSION(S)

The present study showed that higher levels of cortisol in the blood were strongly linked to severe dengue, which indicates the body's stress response was activated during a serious illness. Lower total cholesterol and higher triglyceride levels were also strongly linked to severity, which supports the idea that changes in lipid metabolism play a role in the development of dengue. Fluid build-up, bleeding from the mucous membranes, tiredness/restlessness, and hepatomegaly were all strong signs of severe dengue. Severe disease was strongly associated with high total leucocyte count, high serum urea, low sodium, low bicarbonate and prolonged prothrombin time. The results suggest that serum cortisol and lipid profile measured early, in addition to routine tests and clinical warning signs can be used to identify high-risk kids earlier. These tests are simple and widely available and so could be useful biomarkers, particularly in resource-limited settings, to help identify children at risk of severe disease based on the above laboratory findings. Large prospective studies in children are necessary to confirm these results and to establish definitive cut-off values for clinical use.

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