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# Dengue Shock Syndrome with Unusual Cardiac Manifestation in a Child: A Case Report

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**ABSTRACT**

**Introduction:** Dengue fever is a viral infectious disease transmitted through arthropods (arboviruses) that is most common throughout the world with the greatest burden occurring in tropical and subtropical regions. The clinical manifestations of dengue infection range from asymptomatic to severe dengue. Neutofil to lymphocyte ratio (NLR) was reported can predict the severity of myocarditis and the severity of dengue infection in children. Cardiac involvement in dengue patients has been reported in some cases and may be characterized by abnormal heart rhythms.

**Case Presentation:** We describe the case of an 11-year-old patient with dengue shock syndrome and cardiac manifestation. The patient was given fluid therapy according to WHO guidelines for dengue shock syndrome and supportive treatment. In the convalescence phase, ECG examinations were carried out and the results showed sinus bradycardia and premature atrial complex. NLR was also found to increase in the early phase and decrease with the convalescence phase. The patient had no clinical manifestation of myocarditis, dyspnea or chest pain so there were no additional treatments for this condition. After four days of treatment, the patient was discharged from the hospital with an improved condition.

**Conclusion:** Cardiac involvement can occur in dengue shock syndrome which can be characterized by abnormal cardiac rhythm on the ECG. NLR is a laboratory parameter that can be used as a predictor of the severity of dengue and myocarditis. Early diagnosis, supportive management and close monitoring with clinical and a laboratory parameter can reduce the mortality of dengue shock syndrome with cardiac manifestation in pediatric patients.

**KEYWORDS:** Dengue shock syndrome, Pediatric, Cardiac manifestation, NLR

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## INTRODUCTION

Dengue fever is an infectious disease caused by dengue virus (DENV) infection which consists of four subtypes (DENV 1, 2, 3, and 4). The DENV is transmitted through the Aedes genus mosquito which is widespread in subtropical and tropical regions of the world. The incidence of dengue infections has continued to increase in the last two decades and remains a global health problem. In 2023, the highest number of dengue infections was reported with more than 6.5 million cases and caused more than 7.300 dengue related-deaths in more than 80 countries. Southeast Asia and tropical regions of America are reported to have the highest incidence of dengue. Indonesia is included in the 30 countries with the highest dengue endemic rate in the world with dengue cases still one of the leading causes of hospitalization and death in children.[1,2] Dengue infection has been reported to become the most common cause of acute fever in Indonesia, approximately 31.9% of 1.464 cases. Exposure to DENV in children is also reported to occur from an early age which was more than 40% in children aged 5 and more than 90% of children at the age of 12 years.[3]

In 2009, the World Health Organization (WHO) classified dengue infection according to the severity of the disease as dengue without warning signs, dengue with warning signs, and severe dengue including dengue shock syndrome (DSS).[4] The pathophysiology of dengue hemorrhagic fever (DHF) is

related to activation of endothelial cells which can mediate plasma leakage. This condition caused by activation of infected monocytes and T cells, the complement system, production of mediators, monokines and cytokines. Cross-reactive T cells and antibodies play a critical role in secondary dengue infections which are associated with severe dengue.[5] In critical condition, dengue patients with severe plasma leakage lead to decreased intravascular volume, decreased blood flow to the heart and decreased left ventricle wall movements in the diastolic period that can affect various organs of the body.[6] WHO in 2011 introduced the term expanded dengue syndrome (EDS) to cover unusual clinical manifestations of dengue infection involving organs such as the liver, kidneys, bone marrow, brain and heart.[7]

Dengue patients can sometimes experience unusual cardiac manifestations as a result of viral invasion and direct cardiac toxicity such as dysrhythmias, pericarditis, myocarditis and heart block.[7,8] The incidence and severity varies between studies among 16.7% to 71%, characterized by clinical symptoms of heart failure, increased cardiac enzymes, abnormal electrocardiograms, and echocardiogram changes. Electrocardiography (ECG) can be used as an initial examination to assess cardiac involvement in dengue infection, especially in DSS. ECG abnormalities in severe dengue have been reported in 44-75% of patients. Various ECG abnormalities in DHF have been reported previously, such as ST segment abnormalities, sinus bradycardia, sinoatrial (SA) block, atrioventricular (AV) block, transient AV block, transient ventricular arrhythmias, premature atrial contractions (PAC), and premature ventricular contractions (PVC). [9,10]

The neutrophil to lymphocyte ratio (NLR) is a systemic inflammatory marker. Increased the value of NLR may indicate a severe immune response, potentially leading to worse outcomes in dengue patients.[11] NLR also has been reported in several studies as a predictor of cardiovascular events and other disease such as asthma, rheumatic fever and cancer. NLR as the ratio between the number of neutrophils and lymphocytes in peripheral blood reflects immune system, innate immunity (neutrophils), and adaptive immunity (lymphocytes).[12] Chen et al. (2018) have reported that high NLR was associated with myocardial damage in acute myocardial patients.[13] A study by Aryani et al. (2022) reported that high NLR (cutoff point 3.4) was significantly associated with the severity of carditis (p= 0.001) in children with rheumatic fever. [14] In this case report, we describe children with dengue shock syndrome with cardiac involvement in the pediatric intensive care unit at Sanjiwani Hospital to add limited knowledge regarding the management of these patients.

## CASE PRESENTATION

An 11-year-old boy was referred to the emergency room in Sanjiwani Hospital, with complaints of fever four days before admission accompanied by stomach pain, nausea, vomiting, diarrhea and slight shortness of breath with cold sweat. The patient was treated at the primary health clinic one day before being referred to the hospital due to worsening conditions and haemoconcentration. A history of previous illnesses such as diabetes mellitus, kidney problems, heart disease or genetic diseases was denied. The history of preexisting heart disease in the family was denied. This time was the first time patients were infected with dengue.

On general examination found that the patient was compos mentis, weak, with vital signs, pulse rate of 103x/minute weak palpable, blood pressure was 101/72 mmHg, temperature 36.0 $^0$C, respiratory rate 20x/minute. The patient's weight was 50.8 kg, height 1.52 m, and body mass index 21.98 kg/m$^2$. On physical examination, epigastric and right hypochondriac tenderness were found, both extremities were cold with the capillary refill time more than 2 seconds. A serial complete blood count (CBC) was done in the primary clinic and hospital according to the patient's clinical status with the results presented in **Table 1.** There was a decline of platelet count with an increase in hematocrit. NLR was calculated and increased in the early onset of dengue infection. The patient was diagnosed with dengue shock syndrome (Febris Day 4). In the emergency room for the initial treatments, the patient was given oxygenation with 3-4 lpm nasal cannula, then given a loading of crystalloid fluids ringer lactated (RL) 10 ml/kg, which was 500ml/hour for one hour, then RL 7ml/kg/hours, which was 350ml/hour for one hour, then RL 5ml/kg/hour wich was 250ml/hour for four hours. The patient was also given an injection of 500 mg of paracetamol, 50 mg of ranitidine and 4 mg of ondansetron. Monitoring of resuscitation response, repeated shocks, urine output and fluid balance were done.

Cyto's CBC was done three hours post-resuscitation and revealed that the hematocrit 50.9%, platelets 62 x10^3/uL, hemoglobin 16.7 g/dL and white blood cells 5.12 x10^3/uL **(Table 1).** Fluid therapy with RL 5ml/kg/hour (250ml/hour) was given for six in total, a second infusion line was installed with each infusion rate of 125ml/hour (125 ml on line 1 and 125 ml on line 2). A dower catheter with urobag and abdominal circumference was installed for monitoring urine production, fluid balance and abdominal distention during treatments in the pediatric intensive care unit. Complete blood count examination eight hours post fluid resuscitation revealed that hematocrit 48.6 %, platelet 52 x10^3/uL, hemoglobin 15.9 g/dL and white blood cell 6.93x10^3/uL. Maintenance fluid was continued with RL at 3ml/kgbb/hours which was 150 ml/hour (75ml on line 1 and 75ml on line 2). On the second day of treatment (20 hours after resuscitation) the patient's condition was stable with a complete blood count showing decreased hematocrit (48.6%), platelets 67 x10^3/uL, hemoglobin 12.0 g/dL and white blood cells 5.55 x10^3/uL **(Table 1),** therefore RL was reduced to 100ml/hour in one infusion line.

On the fourth day of admission (7$^{th}$ day of fever) patient was in convalencent phase and patient no longer complained of

fever, abdominal pain, nausea or vomiting. The patients didn't complain of shortness of breath either chest pain or chest discomfort. On physical examination of vital signs, blood pressure was 115/80 mmHg, heart rate 58x/minute, respiration rate 20x/minute, temperature 36.7 $^0$C and oxygen saturation 98% on room air. On general physical examination no remarked abnormality was found, the results of the chest examination showed symmetrical movements, no retractions, vesicular breath sounds, no rhonchi, and no wheezing. On cardiac examination, single S1 and S2 sounds were regular and there were no murmurs or regurgitation. On abdominal examination, there was no epigastric and right hypochondriac tenderness, no distension, normal bowel sounds, and no hepatosplenomegaly. CBC revealed decreased hematocrit at 36.1%, platelets 115 x10^3/uL, hemoglobin 11.5 g/dL, white blood cells 4.47 x10^3/uL **(Table 1).** An ECG examination was carried out on the patient and the results were sinus bradycardia and premature atrial complex **(Figure 1).** The patients were observed and had no complaints, there were no additional treatments for abnormal cardiac rhythm findings in these patients. The patient was allowed discharged from the hospital and planned control five days later at Paediatric Policylinic. During the control at the Pediatric Polyclinic, the patient had no complaints and was able to carry out light activities. On physical examination was within normal limits.

**Table 1. Haematological examination result**

| NAME OF TEST AND INDICATOR | RESULTS | | | | | | | | | | | UNIT | REFERENCE VALUE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| COMPLETE BLOOD COUNT | 23/07/24 | 24/07/24 | | 25/06/24 | | | | 26/06/24 | | 27/07/24 | 28/06/24 | | |
| | 19.45 | 06.41 | 22.23 | 02.14 | 06.02 | 11.26 | 20.02 | 06.19 | 18.09 | 06.32 | 06.34 | | |
| Erytroctes (RBC) | 4.75 | 4.96 | 5.71 | 6.12 | 5.83 | 6.00 | 5.43 | 4.87 | 4.60 | 4.26 | 4.30 | 10^6/uL | 3.50-5.50 |
| Leukocyte (WBC) | 3.5 | 3.8 | 4.7 | 5.12 | 6.93 | 8.80 | 10.30 | 8.01 | 5.55 | 5.12 | 4.47 | 10^3/uL | 4.00-12.00 |
| Neu# | 2.5 | 2.3 | 2.8 | 2.76 | 4.15 | 5.21 | 5.28 | 4.08 | 2.53 | 2.34 | 2.31 | 10^3/uL | 1.80-8.00 |
| MCV | 80.5 | 80.2 | 79.9 | 82.3 | 81.9 | 81.3 | 81.2 | 82.0 | 82.7 | 84.0 | 84.0 | fL | 80.0-100.0 |
| Platelet count | 128 | 101 | 69 | 62 | 52 | 64 | 64 | 64 | 67 | 69 | 115 | 10^3/uL | 150-450 |
| MPV | 10.2 | 10.6 | 10.1 | 8.8 | 9.5 | 10.6 | 10.5 | 11.1 | 11.5 | 11.3 | 9.5 | fL | 6.5-12.0 |
| Lym# | 0.7 | 1.2 | 1.3 | 1.66 | 1.68 | 1.96 | 2.59 | 2.17 | 1.89 | 1.87 | 1.65 | 10^3/uL | 1.20-5.80 |
| Hematocrit | 38.3 | 39.8 | 45.6 | 50.9 | 48.6 | 48.8 | 44.1 | 39.9 | 38.0 | 35.8 | 36.1 | % | 35.0-49.0 |
| MCH | 27.3 | 27.2 | 27.9 | 27.3 | 27.3 | 26.6 | 26.8 | 26.6 | 26.1 | 26.2 | 26.7 | pg | 27.0-34.0 |
| Lym% | 18.7 | 31.6 | 26.6 | 32.4 | 24.2 | 22.2 | 25.1 | 27.0 | 34.0 | 36.5 | 36.9 | % | 20.0-65.0 |
| Hemoglobin (HGB) | 13.0 | 13.5 | 15.6 | 16.7 | 15.9 | 15.9 | 14.6 | 12.9 | 12.0 | 11.2 | 11.5 | g/dL | 11.0-16.0 |
| Neu% | 71.6 | 60.9 | 60.2 | 53.8 | 59.8 | 59.3 | 51.3 | 50.9 | 45.5 | 45.6 | 51.6 | % | 25.0-70.0 |
| PDW | 10.6 | 10.9 | 14.7 | 16.0 | 16.4 | 16.9 | 17.1 | 17.0 | 16.9 | 17.1 | 16.3 | fL | 9.0-17.0 |
| MCHC | 33.9 | 33.9 | 34.2 | 32.8 | 32.7 | 32.7 | 33.1 | 32.4 | 31.6 | 31.2 | 31.8 | g/dL | 32.0-36.0 |
| RDW-SD | 39.8 | 39.8 | 38.9 | 38.1 | 38.0 | 48.4 | 47.9 | 47.0 | 47.5 | 46.8 | 37.8 | fL | 35.0-56.0 |
| NLR | 3.82 | 1.92 | 2.26 | 1.66 | 2.47 | 2.67 | 2.04 | 1.88 | 1.33 | 1.24 | 1.39 | | |

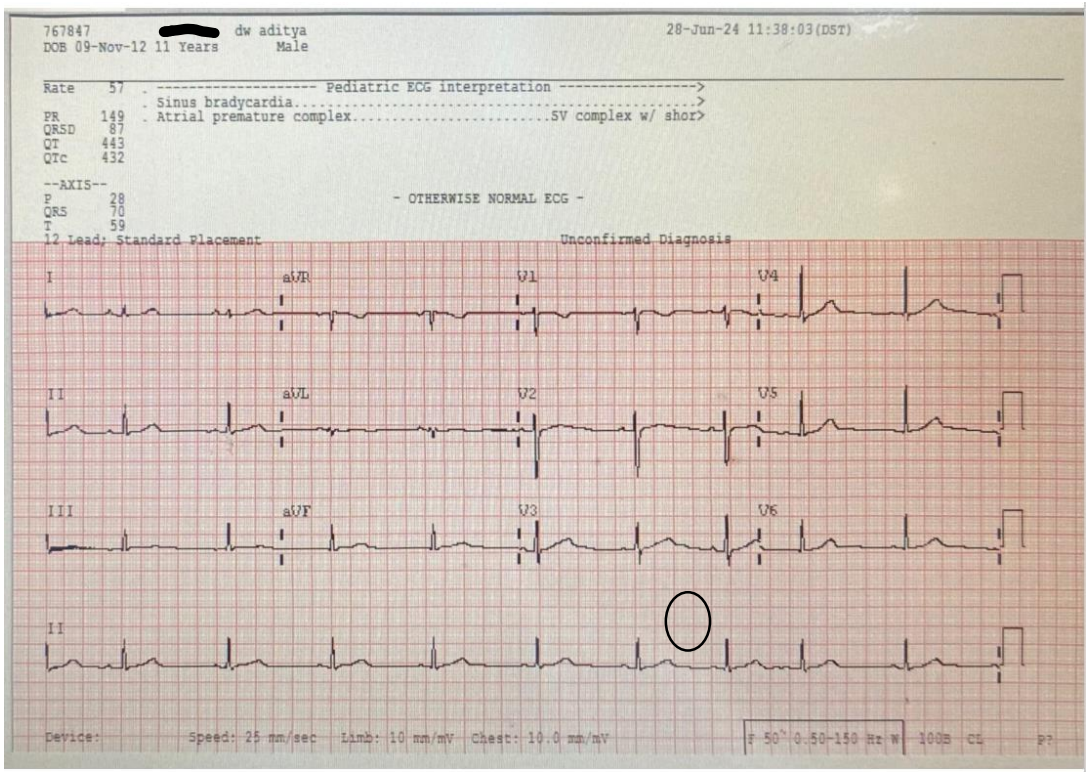


**Figure 1. ECG results of the patients. The result showed sinus rhythm, rate 57 bpm, normoaxis, normal P wave (< 0,12 seconds), normal PR interval, QRS complex <0,12", normal ST and T wave. Conclusion: sinus bradycardia and premature atrial complex.**

## DISCUSSIONS

Dengue fever (DF) is an acute infectious disease characterized by fever with headache or retro-orbital pain, nausea and vomiting, abdominal pain, myalgia, skin rash, loss of appetite, bleeding manifestations, leukopenia, and thrombocytopenia. Dengue hemorrhagic fever (DHF) is a dengue infection characterized by increased permeability of blood vessels which causes intravascular plasma leakage into the interstitial compartment leading to hemoconcentration, low blood pressure and shock.[15,16] Dengue shock syndrome (DSS) in severe dengue is caused by extravasation of intravascular fluids to third spaces (peritoneal spaces, pleural cavity and tissue plains) leading to hypovolemia and tissue hypoperfusion.[17] The mortality rate from untreated severe dengue can be 13% or higher but with early diagnosis and appropriate management can be reduced to <1%.[18]

We report a case of dengue shock syndrome pediatric patient with cardiac involvement characterized by rhythm abnormalities on the ECG. The diagnosis is based on the clinical manifestation of dengue infection with symptoms of fever, abdominal pain, nausea and vomiting. On physical examination, we found increased heart rate palpable weak and thready, tenderness on the epigastric and hypochondriac region with both extremities were cold and the capillary refill time > 2 seconds. On CBC there was haemoconcentration with decreased platelets and leukopenia. The clinical findings in these patients indicated that the patient was experiencing early stages of hypovolemic shock in DSS. In this stage, tachycardia with weak palpable pulses was the early sign that we found, resulted from compensatory mechanisms to maintain normal systolic blood pressure. Furthermore, cold extremities and delayed capillary refill time caused by peripheral vasoconstriction with decreased skin perfusion. Further shock is characterized by restlessness, increased diastolic pressure, narrowing of the pulse pressure ≤20 mmHg, hypotension, delayed capillary refill (>3 seconds) and cold extremities. If not managed properly, the condition of the patient can worsen to uncompensated shock which is characterized by blood pressure and/or pulse becoming invisible and leading to death due to progressive multi-organ damage organ in hypoperfusion conditions.[4,7,19]

Dengue fever has various manifestations in children, including cardiac involvement. The clinical manifestations of dengue myocarditis range from asymptomatic to symptoms of heart failure (chest pain, dyspnea), pulmonary edema, and cardiogenic shock.[5,20] Myocarditis in dengue can be identified by clinical symptoms of heart failure, increased cardiac enzymes (eg troponin T, creatine phosphokinase-myocardial band [CPK-MB]), abnormal ECG and echocardiogram.[21] Research by Buntubatu et al (2019) in Yogyakarta, reported that myocarditis was diagnosed in 53% of DF, 75% of DHF, and 96% of DSS pediatric patients. Myocarditis was significantly more prevalent in DSS than in DF patients (p=0.003).[22] ECG abnormalities in dengue infection patients also significanlty correlated with the severity of dengue infection (P=0.022). Abhinaya et al. (2021) reported that cardiac involvement in children with dengue occurred in 60 (46.2%) patients and occurred more frequently in children

with severe dengue (72.7%), followed by dengue with warning signs (53.8%) and dengue fever without warning sign (28.6%).[21] Other study in India reported that of 39 pediatric patients with dengue, cardiac manifestations were seen in 22 (56%) patients, which were seen in 5 (71%) of 7 patients with severe dengue fever, 7 (53%) of 13 patients with dengue fever with warning signs and 10 (52%) of 19 with dengue fever without warning signs.[23]

ECG abnormalities are often benign and may be the only sign of cardiac involvement with normal cardiac enzyme and echocardiography finding.[22] In this study, we found sinus bradycardia and atrial premature complex (PAC) in convalesent phase of dengue. Sinus bradycardia rhythm and PAC in children with dengue infection have been reported. The study in India reported ECG abnormalities that was found in dengue patients were sinus bradycardia (17%), T wave inversion in V5 and V6 (70%), ST elevation or depression (8%), pathological Q waves and narrow QRS complex each in 1 patient.[21] Menwal et al (2020) reported that the most common ECG results in dengue fever were sinus tachycardia (45%) followed by sinus bradycardia (10%), ST elevation and high T waves and right axis deviation were also observed in 8% of subjects. [24] The results of research by Nerella el at. (2020) in children with dengue reported that abnormal ECG were found in 78 cases (52%) out of a total of 150 in terms of rate.[10] Yantie et al. (2016) reported 10 cases of dengue infection with ECG abnormalities in normal heart structures in the covalence phase, which were sinus arrhythmia and sinus bradycardia each found in 3 patients, first-degree AV block in 2 patients, sinus tachycardia in 1 patient and 1 patient found AV second degree Mobitz type I. No structural abnormalities were found in all patients.[25]

The mechanism of cardiac dysfunction or myocarditis in dengue infection is thought to direct viral invasion of myocytes and an immune response due to the release of inflammatory mediators such as TNF-alpha, cytokines, interleukins and oxygen free radicals during viral infection.[23] DENV is phagocytosed by macrophages with T cell activation, thereby inducing a cascade of vasoactive mediators and proinflammatory cytokines which can cause increased vascular permeability resulting in plasma leakage. This condition can cause cardiac dysfunction due to decreased intravascular volume and preload as well as changes in coronary microcirculation. Increases in resting diastolic calcium ion levels and myocardial autonomic tone induced by dengue may be associated with arrhythmias and decreased left ventricular function. Myocardial damage can lead to poor ventricular function and myocarditis as well as signal conduction abnormalities that can manifest in ECG abnormalities. This abnormality will resolve with the resolution of the infection thus explaining transient ECG changes, as well as Echo findings.[5,21,22,23]

In these patients, sinus bradycardia and PAC were obtained on ECG examination in the covalent phase (fever day 7). In most cases of dengue fever, ECG abnormalities are temporary and tend to occur during the recovery phase. Other studies show that ECG abnormalities can occur at any phase of the course of dengue fever.[5] The mechanism of sinus bradyarrhythmia with stable hemodynamics in dengue fever sufferers is still not known with certainty. However, it is stated that in the recovery phase, systemic blood vessel leakage stops and the extravasated third space fluid begins to be reabsorbed. Hemodilution causes a decrease in hematocrit, a rapid increase in the number of white blood cells and is followed by an increase in platelets. However, bradyarrhythmias can be dangerous if occur in the critical phase when hypovolemia is an obvious concern, as the inability to mount an appropriate response to heart rate to maintain potential cardiac output will increase hemodynamic instability and may affect the outcome of the disease.[21]

Besides clinical and laboratory parameters (trombosit, hematocrit, haemoglobin and leukocyte), NLR has been studied as an early predictive marker for severe dengue.[26] Leukocyte components, neutrophils and lymphocytes make up 80% of leukocytes and play an active role in the body's response to inflammation and infection, so the immune response can be inferred from NLR value. In an immune response to inflammation or systemic stress, there is an increase in neutrophil production and encourages lymphocyte apoptosis, resulting in neutrophilia and lymphopenia causing an increase in NLR.[27,28] An increase in NLR has also been reported to predict mortality in pediatric intensive care units.[29] High initial NLR at hospital admission was associated with adverse outcomes for adult patients with dengue infection.[27] NLR has also been proposed as a surrogate marker of endothelial dysfunction and inflammation in different populations.[28]

The NLR value has been studied as a prognostic predictor in cardiovascular diseases, infections, inflammatory diseases and cancers.[30,31] Several studies have reported normal reference values for the NLR in various populations. In adults, normal NLR is between 1-2, values higher than 3.0 and below 0.7 are pathological.[32] Normal values of NLR in the pediatric population based on age and gender were reported by Moosmann et al. (2022). At the age of 3-18 years, the 50th percentile NLR continued to increase from 0.99 to 1.76 in both boys and girls. In pediatric patients with asthma, the NLR was reported to be higher compared to healthy children with an average NLR of 2.07 compared to 1.77 in healthy controls. The cut-off value of NLR to predict systemic involvement was 2.73.[33] Another study reported that higher NLR was associated with a more severe clinical course in children with acute exacerbation of asthma and a higher rate of hospitalization.[34] Aryani et al. (2022) reported that with a strong positive correlation between the NLR value and the severity of carditis ($r = 0.66$, $p = 0.001$) using a cutoff of 3.4, NLR can be used to predict severe carditis with a sensitivity of 81.8% and a specificity of 91.3%.[14]

In this case, an increase in the NLR value was found at the beginning of the illness, then the NLR value decreased as the

patient's condition improved (febrile H-6) as in **Table 1**. At the beginning of the disease phase, the NLR value in this patient was 3.82 then decreased to 1.88 on day 6 of fever. There are no definite guidelines for normal NLR values in children based on age. Nevertheless, based on Moosmann et al. (2022), the average normal NLR value for boys aged 10-11 years is 1.625. In this patient, an increase in NLR was found, thus indicating the severity of the disease which is the patient experienced DSS and indicating cardiac involvement, myocarditis, apart from being characterized by abnormal ECG (sinus bradycardia and PAC). A decrease in the NLR value in patients was also found as the patient's clinic improved and laboratory parameters such as leukocytes, hematocrit and platelets began to normalize. This is consistent with the course of dengue infection. In the early days of dengue infection, there will be an increase in the percentage of neutrophils so that the NLR ratio will be higher but as the disease progresses from the acute febrile phase to the critical phase there will be an increase in lymphocytes due to reactive lymphocytosis and there will be a reversal of the NLR ratio in the early days 6 to day 9.[30,35]

Management of this patient was carried out according to WHO guidelines. Initial management of the patient was given oxygenation with a 3-4 lpm nasal cannula, then given a loading of crystalloid fluids Ringer lactated (RL) 10 ml/kg in one hour. If clinical monitoring shows an improvement in the condition of the resuscitation phase, fluid administration can be given at a dose of 7–5–3 ml/kg/hour. Reduction of intravenous fluid administration is carried out gradually after the patient's hemodynamic status stabilizes and when the plasma leak rate decreases towards the end of the critical phase as indicated by adequate urine output and a decrease in hematocrit. Periodic complete blood tests are also carried out to assess hematocrit levels. At three hours post-resuscitation, the results of a complete blood count revealed that the hematocrit was 50.9%, platelets 62 x10^3/uL, hemoglobin 16.7 g/dL and white blood cells 5.12 x10^3/uL, so fluid therapy with RL 5cc /kg/hour (250cc/hour) was continued for six hours in total. On the second day of treatment (20 hours after resuscitation) the patient's condition was stable with a complete blood count showing hematocrit 48.6%, platelets 67 x10^3/uL, hemoglobin 12.0 g/dL and white blood cells 5.55 x10^3/uL maintenance fluid was reduced to 2cc/kgBB/hour. We do close monitoring during the critical phase to assess resuscitation response, warning signs, recurrent shock, urine production and fluid balance. The patient showed a good response to fluid resuscitation and there was no excess fluid such as hepatomegaly, edema, rhonchi in the lungs, pleural effusion or ascites.

Early detection of critical periods or plasma leakage and appropriate fluid replacement are very important in the treatment of dengue patients. In the management of DSS patients, intravenous fluid is administered with a sufficient volume to maintain effective circulation during periods of plasma leakage but avoid overload fluid therapy.[36] Crystalloids such as normal saline (0.9%) and Ringer's lactate (RL) are the fluid therapy of choice in DSS, followed by colloids for patients with refractory shock. Ringer's lactate is a better choice in patients who have hyperchloremia, hyperchloremic acidosis, or hypernatremia while normal saline may be chosen for initial resuscitation in patients with hyponatremia and normal chloride levels (95–105 mmol/L). Additional colloids are required in less than half of DSS cases while blood products are given only occasionally. A study reported on 61 cases of dengue fever that developed more severe during hospitalization and were recovered with adequate fluid therapy, mainly using a standard first-line treatment regimen with crystalloids. [15,37,38,39]

In this patient, there were no symptoms of shortness of breath or chest pain so there were no additional treatment was given for cardiac manifestations. However, the patient was monitored until discharged from the hospital and an evaluation was carried out a few days later. The same findings were also reported with no special symptoms found in cases of dengue infection with ECG abnormalities so special treatment was not given. Additionally, no evidence of structural cardiac abnormalities was found. Clinical improvement and improvement in ECG evaluation results after several days indicate that myocardial involvement in dengue infection is benign without long-term complications.[17,21,22,25] In the covalent phase, the cardiac manifestations of dengue fever are benign, temporary, and can resolve on their own. Children with abnormal ECG findings in dengue infection were monitored until discharge from the hospital.

## CONCLUSION

Cardiac involvement can be found in paediatric patients with DSS and should be detected early and managed carefully. Clinical manifestation of cardiac involvement in dengue patients can be characterized by abnormal cardiac rhythm on the ECG which can be a precautionary sign. High NLR can be a predictor of the severity of dengue infection and myocarditis in pediatric patients. Fluid management and monitoring hemodynamic status are very important in the management of DSS. Close monitoring is needed in patients who experience heart rhythm abnormalities. Further research is needed on the correlation of NLR with dengue myocarditis in children and to determine the relationship between severity and NLR values.

## RESEARCH ETHICS

Before obtaining patient data through medical records, the authors have obtained patient consent to be reported in this case report.

## CONFLICT OF INTEREST

There are no conflict of interest.

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**AUTHORS' CONTRIBUTION**

All authors contributed to the data writing process up to publication.

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