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WHEN OBESITY MASKS SHOCK: A CASE OF COMPENSATED DENGUE SHOCK SYNDROME IN AN EARLY ADOLESCENT

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Abstract

Dengue fever is a major public health problem, and its incidence has markedly increased over the past two decades. Severe dengue, particularly when accompanied by shock (dengue shock syndrome [DSS]), is a life-threatening complication. Obesity may mask early signs of hypovolemia, leading to delayed recognition of shock. This study aims to report a case of compensated DSS in an early adolescent with obesity, highlighting the diagnostic challenges. We report the case of a 13-year-old obese male (BMI 38.9 kg/m²) with compensated DSS. Clinical and laboratory data were collected during his hospitalization at Wangaya Regional General Hospital, Denpasar. The patient presented with fever, abdominal pain, and petechiae. Despite initially stable vital signs, he developed oliguria and narrowed pulse pressure within 18–24 hours of admission. Laboratory findings showed progressive thrombocytopenia and hemoconcentration, indicating compensated shock. The patient responded well to rehydration and intensive monitoring in the PICU. Obesity can mask early clinical manifestations of DSS, delaying recognition. Strict monitoring of subtle signs, such as urine output and pulse pressure, is crucial for timely intervention and improved outcomes in obese pediatric patients with dengue.
Keywords: dengue; dengue shock syndrome; obesity; pediatrics

INTRODUCTION

The global incidence of dengue has markedly increased over the past two decades. In the WHO Southeast Asia region, 10 out of 11 Member States are known to be endemic for dengue virus. In particular, India, Indonesia, Myanmar, Sri Lanka and Thailand rank among the world's 30 most highly endemic countries. Indonesia give 0.72% for the number of CFR (World Health Organization, 2023).

Severe dengue is a life-threatening manifestation of dengue virus infection and may progress to dengue shock syndrome (DSS), which is characterized by plasma leakage, hemoconcentration, and circulatory failure (Guzman et al., 2021; Sim et al., 2020). The critical phase of dengue typically occurs between days 3 and 7 after the onset of fever, during which defervescence may be accompanied by rapid clinical deterioration, thrombocytopenia, and shock (Lima et al., 2020; Narvaez et al., 2021; Kementerian Kesehatan RI, 2021).

While the pathophysiology of dengue is well-described, host factors that modify disease presentation and progression are increasingly recognized. One such factor is nutritional status. Zulkipli et al. (2018), in a systematic review and meta-analysis, established a significant association between obesity and increased dengue severity in pediatric patients. Similarly, a prospective study by Ahmed and Islam (2023) found obesity to be a significant risk factor for severe dengue in children, with an odds ratio of 1.38 (95% CI: 1.10--1.73), and reported higher mortality among this subgroup. These studies highlight the prognostic importance of obesity in dengue infection.

Pathophysiology of DHF/DSS involves various inflammatory cytokines as as IL-6, IL-8, IL-10, TNF-α,MIF, VEGF, IFN-αβand IFN-γ). These cytokines affect vascular permeability and trigger plasma leakage. a research has shown that TNF-α synergize with dengue virus to modify adhesion molecule on endothelial cell and increases vascular permeability. Cytokine IL-8 was initially known to increase vascular permeability and cause thrombocytopenia in patients with dengue infection (Pramudito et al., 2020).

Managemenet of dengue fever can devied into 3 groups, there was Group A for dengue without warning sign, Group B for dengue with warning sign or certain condition, and Group C for severe dengue (Chart. 1)(Kementerian Kesehatan RI, 2021). This report describes a case of compensated DSS in an early adolescent with obesity, highlighting the diagnostic challenges and emphasizing the importance of strict monitoring and timely management to improve clinical outcomes.

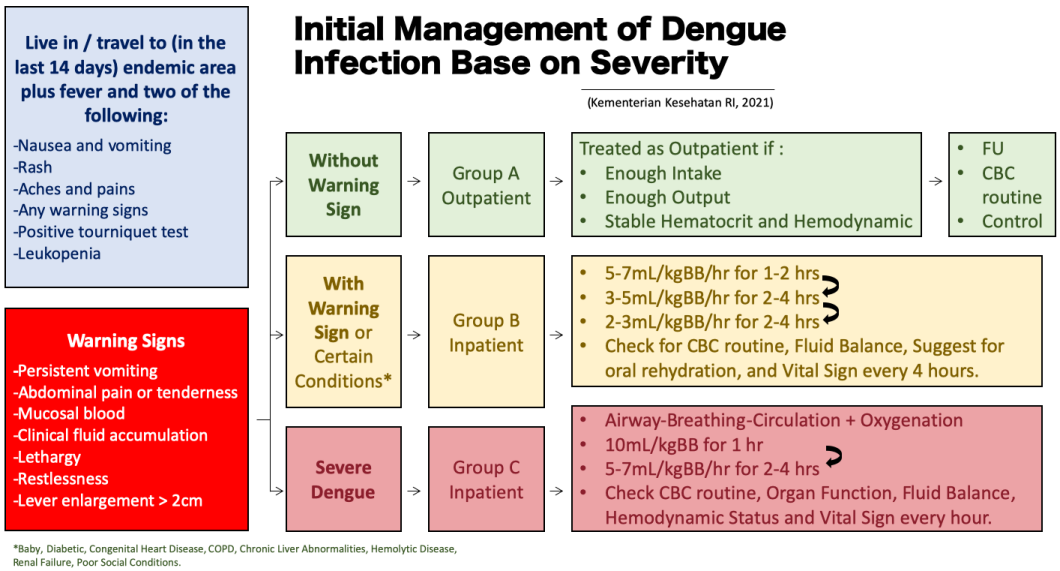

Figure 1. Initial Management of Dengue Infection Base on Severity

CASE PRESENTATION

A 13-year-old Balinese boy was admitted to Wangaya Regional General Hospital, Denpasar, with a chief complaint of fever since four days. The patient had a body weight of 106 kg and a height of 165 cm, resulting in a body mass index (BMI) of 38.9 kg/m². Based on the Centers for Disease Control and Prevention (CDC) growth chart, this value is consistent with obesity.

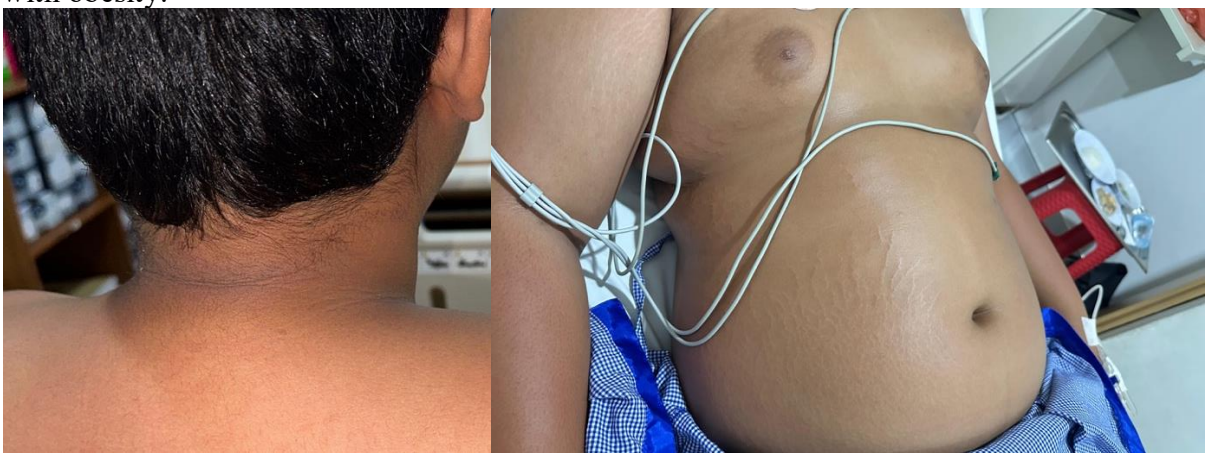

Figure 2. Acanthosis Nigricans, Short Neck, Stretch Mark and Fat Fold on Belly in 13-year-old Balinese boy with (BMI) of 38.9 kg/m2 that suffered DSS.

The fever began on July 12, 2025, and was initially managed at a primary care clinic. Although the fever temporarily subsided, it recurred on July 15, 2025, accompanied by nausea and abdominal discomfort. When arrived at the emergency room, the patient appeared pale and lethargic. He also complained of a sensation of mucus in the throat, nausea, and abdominal

pain. There was a past medical history of hypertension diagnosed in 2023. However, anti-hypertensive treatment had been discontinued, and the patient had not undergone any medical follow-up.

On physical examination at admission, his vital signs were within normal limits. But, there is notable findings included acanthosis nigricans on the posterior neck, abdominal striae, epigastric tenderness, and hepatomegaly palpable 2 cm below the costal margin. Initial laboratory investigations revealed thrombocytopenia (Table 1). Based on clinical and laboratory findings, the patient was diagnosed with dengue fever with warning signs and admitted for inpatient management.

Laboratory examination results were obtained:

Table 1. Laboratory Findings

Name of Test and Indikators	Result					Unit	Reference Value
Complete Blood Count	15/7/25 11:38	15/7/25 18:03	16/7/25 08:14	17/7/25 07:58	19/7/25 10:08		
WBC	4.40	3.02	7.22	8.46	5.40	10^3/uL	4.0-10.0
Eritrosit		6.30	7.46	5.97	5.07	10^3/uL	4.50-6.20
HB	14.5	14.7	17.0	13.8	11.8	g/dL	13.0-18.0
HCT	45.9	45.6	51.6	42.0	36.4	%	40.0-54.0
MCV	72.3	72.4	69.2	70.4	71.8	fL	81.0-96.0
MCH	22.8	23.3	22.8	23.1	23.3	Pg	27.0-36.0
MCHC	31.6	32.2	32.9	32.9	32.4	g/dL	31.0-37.0
PLT	132	107	59	45	83	10^3/uL	150-400

The therapy provided includes rehydration using ringer lactate (RL) fluid infusion 250 cc/hour (5cc/kgBB/hour, IBW = 50kg) for 2 hours. Anti-emetics in the form of Intravenous Ondansentron Injection at 4 mg IV every 12 hours, Ranitidine injection as gastro-protector at 50 mg every 8 hours if needed, and Paracetamol as antipyretic at 500 mg given orally every 6 hours if needed. A repeat complete blood count performed six hours later demonstrated leukopenia and progressive thrombocytopenia (Table 1).

Within 18–24 hours of hospitalization, the patient developed clinical signs of shock, including narrowed pulse pressure (110/90 mmHg), generalized petechiae, and oliguria, with a urine output of 0.34 mL/kg/hour (Table 2). Laboratory evaluation at that time showed an increased hematocrit level and worsening thrombocytopenia, indicating ongoing plasma leakage. The patient was subsequently transferred to the pediatric intensive care unit (PICU) for close monitoring and further management.

Table 2. Urine Production and Fluid Balance

Time	15/7/25	16/7/25	17/7/25	18/7/25	19/7/25	20/7/25	21/7/25
24.00–06.00	–	0.34	–	–	1.7	1.1	1.9
06.00–12.00	–	–	1.6	1.5	2.5	2.0	1.6
12.00–18.00	0.6	1.5	–	1.6	1.6	2.0	–
18.00–24.00	0.47	–	–	1.6	1.4	1.6	–
Time	**15/7/25**	**16/7/25**	**17/7/25**	**18/7/25**	**19/7/25**	**20/7/25**	**21/7/25**
24.00–06.00	–	+1003	–	–	+124	-342	-124
06.00–12.00	–	–	+32	+54	+42	-242	+58
12.00–18.00	+618	-1032	–	+79	+58	-242	–
18.00–24.00	+1002	–	–	+58	+158	-42	–

In the PICU, the patient received supplemental oxygen via nasal cannula and continued fluid therapy according to severe dengue management protocols. Following treatment, hemodynamic parameters improved, with blood pressure stabilizing at 129/67 mmHg and urine

output increasing to 1.6 mL/kg/hour. Serial laboratory examinations showed gradual improvement, with persistent but recovering thrombocytopenia (Table 2). After two days of PICU, the patient's clinical condition improved, and he was transferred back to the ward. Llast complete blood count showed improvement result (Table 1). After observation 24 hour at ward, The Patient was discharged in a stable condition.

RESULTS AND DISCUSSION

Dengue is an acute systemic viral infection caused by the dengue virus subtypes DEN-1 to DEN-4, with *Aedes aegypti* and *Aedes albopictus* as the principal vectors (Zulkipli et al., 2018). Clinically, dengue fever classified into dengue without warning signs, dengue with warning signs, and severe dengue. warning sign include persisten vomiting, abdominal pain or tenderness, mucosal blood, clinical fluid accumulation, lethargy or restlessness, lever enlargement > 2cm. Severe dengue is condition of dengue infection with or without warning sign that experiencing symptoms like severe plasma leakage, severe haemorrhage or severe organ impairment (Kementerian Kesehatan RI, 2021).

Southeast Asia accounts for the highest proportion of severe dengue cases worldwide. Previous reports indicate that approximately 15% of dengue cases in this region progress to severe disease, with plasma leakage being the most frequent manifestation, followed by severe bleeding and organ dysfunction (Kementerian Kesehatan RI, 2021). Children are affected particularly those under 15 years of age, who exhibit a higher incidence of plasma leakage and shock during dengue infection (Ferdous et al., 2024). In line with these observations, the present case involved a 13-year-old obese boy who developed plasma leakage and shock on day 4 of illness, corresponding to the critical phase of dengue.

The pathophysiology of DSS involve various chemokines and cytokines are produced such as TNFα, IFNα, IFNγ, IL-6, IL-8, and IL-10. These cytokines play a significant role in vasculopathy, coagulopathy, and bleeding tendencies (World Health Organization of Timor Leste, 2022). They affect vascular permeability and trigger plasma leakage. Research has shown that TNF-α synergize with dengue virus to modify adhesion molecule on endothelial cell and increases vascular permeability. These cytokines would form 'cytokine storms' that will increase vascular permeability and cause vascular leakage (Ferdous et al., 2024).

Hypotension is a late manifestation of dengue shock syndrome. During the early or compensated phase, substantial intravascular volume depletion may already be present while systemic blood pressure remains preserved through increased sympathetic activity and systemic vascular resistance. This condition is often referred to as *masked shock*, in which classical signs of hypovolemic shock, particularly hypotension, are absent despite ongoing plasma leakage. In obese individuals, a higher baseline circulating blood volume and increased cardiac output may further delay the onset of hypotension, thereby masking early clinical indicators of shock such as intravascular volume depletion. As a result, the progression to severe dengue may appear clinically silent until compensatory mechanisms fail, leading to sudden hemodynamic decompensation and overt shock (Guzman and Martinez, 2024).

Children were found to be more susceptible to shock than adults because of children have less well-developed mechanism in minimizing heart decompensation against increasing vascular permeability, thus making children more vulnerable to hemodynamic instability (Ferdous et al., 2024).

Obesity has increasingly been recognized as a risk factor for severe dengue. Adipose tissue functions as an active endocrine organ that produces pro-inflammatory cytokines, including IL-6. In obese individuals, an increased number of adipocytes leads to persistently elevated IL-6 levels, contributing to chronic low-grade systemic inflammation (Midah et al., 2021) (Medellu et al., 2023). In addition, obesity is associated with increased production of other inflammatory mediators such as IL-1β and TNF-α, which promote oxidative stress and

endothelial dysfunction, thereby exacerbating vascular leakage during dengue infection (Medellu et al., 2023). Persistent elevation of IL-6 has also been linked to chronic inflammatory states that may worsen disease severity (Susantiningsih and Mustofa, 2018).

In the present case, obesity may have contributed to both diagnostic and clinical challenges. Physical findings related to obesity, such as acanthosis nigricans, abdominal striae, and increased fat folds, complicated clinical assessment for signs of fluid accumulation, including ascites. Despite initially stable vital signs, the patient developed oliguria and narrowed pulse pressure within 24 hours of admission, indicating a compensated shock state. Prompt recognition of these subtle signs led to timely escalation of care and transfer to the pediatric intensive care unit.

Several studies have demonstrated an association between obesity and increased severity of dengue infection. Chiu et al. reported that obese patients were more likely to develop severe dengue manifestations, including shock (Chiu et al., 2023). Similarly, Ahmed and Islam found that obesity was a significant risk factor for severe dengue in pediatric patients, with an odds ratio of 1.38 (95% CI: 1.10–1.73), and reported higher mortality among obese children with severe dengue (Ahmed and Islam, 2023). These findings support the role of obesity as a prognostic factor in dengue infection.

This case highlights the importance of heightened clinical vigilance in obese pediatric patients with dengue infection. Early signs of hypovolemia may be subtle or masked, necessitating strict monitoring of urine output, pulse pressure, hematocrit trends, and platelet counts. Timely recognition of compensated shock and appropriate management according to established guidelines are essential to prevent rapid deterioration and improve clinical outcomes.

CONCLUSION

This case report concludes that obesity can significantly mask the early clinical manifestations of Dengue Shock Syndrome (DSS) in children. The physiological reserve of a higher baseline blood volume in obese patients can delay the onset of hypotension, a traditional sign of shock, while insidious plasma leakage and end-organ hypoperfusion, as indicated by rising hematocrit, narrowing pulse pressure, and oliguria, are already underway. This "masked shock" phenomenon creates a diagnostic challenge, increasing the risk of delayed recognition and escalation of care. For future research, multi-center prospective cohort studies are recommended to quantify the risk and characterize the clinical presentation of compensated shock in obese versus normal-weight children with dengue. Such studies could help develop and validate a specific risk-stratification scoring system incorporating anthropometric data (e.g., BMI) with early warning indicators like urine output and pulse pressure trends. This would provide clinicians with a practical, evidence-based tool for the early identification of high-risk patients, ultimately guiding more aggressive monitoring and timely intervention to reduce morbidity and mortality in this vulnerable population.

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