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Management of a Case of Dengue Shock Syndrome with Liver Failure: A Case Report

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Abstract:

This case report details the management and treatment of a 38-year-old male cook from Dhaka, diagnosed with dengue fever, which progressed to Dengue Shock Syndrome (DSS) accompanied by acute liver failure. Despite initial stabilization with fluid resuscitation, patient's condition worsened, prompting a fresh whole blood transfusion and comprehensive care. This report underscores the necessity of prompt recognition and effective management of severe dengue cases to improve patient outcomes.

Keywords: Dengue fever, Dengue shock syndrome, acute liver failure, Fresh whole blood transfusion.

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Introduction:

The dengue outbreak in Bangladesh has taken a worrisome turn, with a worrying increase in cases and fatalities this year. As of 18 July 2023, the country has already recorded 127 fatalities (female - 73; male - 54) from the mosquito-borne disease, a staggering five times higher than the previous year (2022).[1] Many patients infected with dengue virus remain asymptomatic while symptomatic infection ranges from undifferentiated fever, dengue fever (DF), dengue haemorrhagic fever (DHF) which includes dengue shock syndrome (DSS) and expanded dengue syndrome.[2] In the absence of specific antiviral therapy, meticulous fluid administration is the mainstay of management of DHF and judicious fluid administration during the "critical phase" is vital in reducing mortality.[3] Herein, we present a case of a previously healthy adult who developed acute liver failure in DSS.

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Case Report:

A 38-year-old male cook from Dhaka, presented to department of medicine, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet with a one-day history of continued fever, headache, malaise, and diffuse abdominal pain. He had no previous significant medical history. He was initially diagnosed with dengue fever in critical phase, based on symptoms and positive dengue NS1 antigen. However, his condition deteriorated by the 3rd day of treatment, leading to the diagnosis of DSS evident by tachycardia, cold periphery, restlessness and reduced urine output with accompanying acute liver failure.

During examination, on 2nd day of fever, conscious and alert, but was very lethargic and moderately dehydrated. His pulse rate was 108 beats per minute and blood pressure 110/80mmHg, temperature was 103°F. The heart sounds were normal. His respiratory rate was 20 breaths/min, and his lungs were clear, SpO2 in room air was 98%. His abdomen was soft, with no free fluid; moderate epigastric and right hypochondrial tenderness were present. He was neurologically normal. His tourniquet test was positive and capillary refilling time was normal. His electrocardiogram (ECG) should sinus tachycardia. His dengue NS1 antigen was positive and we reached the diagnosis of dengue fever with warning signs (abdominal pain and lethargy) and although according to patient's statement it was only 2nd day of his fever but patients clinical condition (tachycardia, restlessness) and lab reports (platelet 1,00,000/cmm) prompted us to make the clinical assumption that his critical phase had begun. His total (Oral + I/V) fluid requirement was calculated according to

maintenance+5% deficit formula to be 4800ml in 48 hours and I/V normal saline was started. The sample of urine and blood were sent to investigate haematology, biochemistry, liver function, urine analysis, arterial blood gas and patient was shifted to ICU for better monitoring. Initial lab investigations revealed haemoglobin (Hb%) 14.4 gm/dl, haematocrit (HCT) 43 %, platelet 100K/uL and TC of WBC 6K/uL. S. creatinine was 1.12 mg/dl, S. ALT 106 U/L, S. Albumin 3.4 gm/dl, S. Electrolytes, urine R/E and ABG were normal. [Table I] Chest radiography showed left sided mild pleural effusion.

Although I/V fluid was maintained [Table II], patient's oral intake was not adequate and on the 4th day of fever his condition deteriorated. Patient was confused, restless, severely dehydrated, tachypneic (RR 28 breaths/min), pulse rate 120 bpm, BP was 110/70 mm of hg, urine was high colored. Blood reports showed that the patient's total WBC count had decreased to 4,500 cells/cmm and platelet count had decreased to 60,000 cells/cmm, HCT raised to 51.8%. [Table I] He was diagnosed with dengue shock syndrome and managed accordingly. Initially, the patient's condition improved after giving I/V normal saline 500ml in 1 hour, but when his intravenous fluids were gradually tapered down [Table II], his condition deteriorated.

On 5th day of fever, his platelet count decreased to 15,000 cells/cmm, HCT suddenly fell to 48% and he was very restless, aggressive and developed hematuria and purpuric spots all over his body. He was diagnosed with acute liver failure with grade III hepatic encephalopathy evident by deranged liver biochemistry and altered level of sensorium; HBsAg (ELISA) and anti-HAV and ameuria level ant-HEV were done to rule out acute viral hepatitis and revealed to be negative. His renal function was normal. His ABG showed metabolic acidosis and serum lactate was increased. Ultrasonography of whole abdomen was done which showed bilateral mild pleural effusion and mild intraperitoneal collection; CT scan of brain was normal.

Alongside fluid resuscitation and supportive care, a fresh whole blood transfusion helped to stabilize the patient. [Table II] Blood transfusion was indicated by a fall in hematocrit and active bleeding evident by haematuria. I/V ceftriaxone was started after sending blood for C/S (which yielded no growth) and short course I/V methylprednisolone was added to treat septicemia which was evident by fever with altered mental status, tachypnea (qSOFA score 2)[4]. Gradually, his platelet count and HCT improved, and signs of organ failure diminished. [Table I] By the 7th day of fever, the patient's condition showed significant improvement, and he was shifted to regular ward. Following 3 days of observation, patient was discharged after fulfilling discharge criteria.

Table-I

Represent the laboratory investigational value monitored periodically.

Lab parameter	Investigated value								Normal value
	Day-2	Day-3	Day-4(i)	Day-4(ii)	Day-5	Day-6	Day-7	Day-10	
RBC (million/cmm)	5.05	5.29	6.2	6.06	5.85	5.54	4.28	4.37	4.04-6.13 million/cmm
Haemoglobin (g/dl)	14.4	15.2	18.1	17.3	16.8	16.6	16.2	14.8	12.2-16.5 g/dl
HCT (%)	43	44.9	51.8	50.1	48	45.7	46.3	41.3	37.7-57.7 %
WBC (per cmm)	6,000	4,500	5,400	6,000	8,900	10,400	17,000	14,700	4,000-11,000 /cmm
Platelet (thousand/cmm)	100	60	50	20	15	70	120	150	150-400 thousand/cmm
S. AST (U/L)					2282				Upto 40 U/L
S. ALT (U/L)	106				875			277	Upto 60 U/L
S. Bilirubin (mg/dl)					3.1				0.2-1.3 mg/dl
S. Creatinine (mg/dl)	1.12		1.05		0.77	0.73			0.8-1.5 mg/dl
S. Lactate (mmol/L)					2.5				0.7-2.1 mmol/L
B. Urea (mg/dl)			31.7						19.26-42.8 mg/dl
S. Calcium (mg/dl)			7.5						8.4-10.2 mg/dl
S. Albumin (gm/dl)	3.4		3.1						3.5-5 gm/dl
Prothrombin time (sec)					18	17			11-14 sec

*day of fever

Table-II

Periodic fluid replacement

Day*	Required fluid	Total oral intake	Total I/V fluid infusion	Blood/blood products transfusion
Day 2-3	4800ml/48hrs	1000ml	3000ml	0
Day 4	3020ml/24hrs	0	3020ml	0
Day 5	4800ml/24hrs	0	4300ml	500ml
Day 6	2000ml/24hrs	500ml	1500ml	0

*day of fever

Discussion:

The critical phase of dengue typically begins around the time of defervescence but it might begin as early as the third day after fever onset in patients who are still febrile.[1] In this case the critical phase began as early as 2nd day of fever according to patient's statement. It might be a case where patient missed the early signs of febrile illness, but his clinical symptoms prompted us to take the initial steps in his management, which in the end helped to save his life.

The fluid requirement during the critical stage of DHF is highly variable and majority of patients require fluid in excess of the currently recommended M + 5% deficit to maintain the clinical parameters.[3] Restricting the amount of fluid in fear of fluid overload may lead to impaired organ perfusion and tissue hypoxia.[3]

This case presentation also highlights the importance of close monitoring of patients with DSS, especially during the critical phase of the illness. The patient's condition deteriorated on the 4th day of fever, despite initial improvement. This could have been due to a number of factors, such as inadequate fluid resuscitation (as patient did not take required oral fluid), infection, or progression of the disease.

Corticosteroids are not generally recommended in the treatment of dengue according to our national guideline.[2] But due to lack of adequate evidence to support this non-recommendation, the interest in corticosteroids for treating dengue has continued.[5] Beneficial effects of Corticosteroids was shown by Premaratna R. et al., by administering a single dose of IV methylprednisolone 1 g (equal to 5000 mg of hydrocortisone) to adult patients in severe DSS.[6] Nonetheless, this was not a randomized trial, although the results were striking. Notably, this study recruited the most ill dengue patients, and administration of corticosteroids was a rescue measure; as in our case.

Liver is a frequently involved organ in dengue infection.[7] Even though liver involvement is mild in most of the cases; there are acute liver failure cases associated with high morbidity and mortality due to complications such as encephalopathy, severe bleeding, renal failure and metabolic acidosis. In adults with dengue associated acute liver failure, the outcome was good with spontaneous survival from standard medical therapy alone.[8] Our patient had only grade III hepatic encephalopathy without any other complications and improved with only supportive therapy.

Conclusion:

Early recognition of warning signs and usual manifestation, prompt management, and timely supportive care are vital in mitigating the progression of dengue fever to severe forms. Dengue remains a significant public health concern, emphasizing the need for continued research and enhanced strategies for prevention, diagnosis, and management.

Conflict of interest: There is no conflict of interest relevant to this paper to disclose.

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