


## Case Report

# A case report of dengue hemorrhagic fever complicated by anterior chest wall haematoma requiring blood transfusion which later progressed to hepatic encephalopathy.

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## Introduction

Dengue has become the most important vector borne disease in Sri Lanka. It is caused by one of four antigenically distinct RNA viruses in the family Flaviviridae. The four serotypes are DENV-1, DENV-2, DENV-3 and DENV-4 [1]. The primary vector is *Aedes aegypti*. In 2020, 31162 suspected dengue cases were reported to the Epidemiology Unit, while 19339 suspected dengue cases were reported between January and June 2022 [2]. The dengue fever outbreak that occurred in 2017 in Sri Lanka was due to DENV-2 virus [3]. That led to a national level approach to control the dengue virus with new strategies.

Dengue fever ranges from a simple, self-limited, febrile illness to haemorrhagic manifestations and dengue expanded syndrome with multiple organ involvement. The commonest organ involved is the liver which can result in acute liver failure with hepatic encephalopathy [4]. The haemorrhagic manifestations can range from simple petechial lesions and purpura to gastrointestinal bleeding, haematuria and severe central nervous system bleeding. Muscle haematoma formation is a rare complication of dengue haemorrhagic fever and can result in a fatal outcome. Therefore, early recognition is essential for a better patient outcome.

## Case presentation

A 49-year-old, previously known patient with triple vessel disease who had undergone primary percutaneous coronary intervention (PCI) to the left anterior descending, left circumflex and left obturate arteries 10 years previously, presented with fever for one day with a positive NS1 antigen. He complained of generalized body aches with arthralgia

and myalgia. He was initially treated as dengue febrile illness. On admission, he was febrile with a temperature of 103°F. He was not dehydrated, not pale and not icteric. Blood pressure was 110/80mmhg with no postural drop. Pulse rate was 84bpm and CRFT <2 seconds. The respiratory examination was unremarkable, with equal air entry on both lungs. Abdomen was soft. The neurological examination was normal with a GCS 15 out of 15.

Patient's full blood count (FBC) showed WBC 2.69 ( $3.6-11.0 \times 10^9/L$ ), platelets 114 ( $140-400 \times 10^9/L$ ) and hemoglobin 14.2 (13-18g/L). His initial AST level was 47 (8-33 U/L) and ALT was 45 (7-56 U/L). On day five of fever, he entered the critical phase; evidenced by reduced air entry in the right-side lung base. But there was no abdominal tenderness. Ultrasound scan revealed free fluid in the hepato-renal pouch, a peri-cholecystic fluid collection and right sided pleural effusion.

Critical phase management was started. The patient complained of a spontaneous ecchymotic patch on the right-side of the anterior chest wall after 12 hours of critical phase. The patient had severe pain over the anterior chest wall and there was an obvious swelling with haematoma formation. His platelet count was 20 ( $140-400 \times 10^9/L$ ), hemoglobin 14.2 (13-18g/L), hematocrit 42.5 (40-54%) and WBC 3.91 ( $3.6-11.0 \times 10^9/L$ ) at that time. In spite of these results, we continued the management of dengue critical phase; The haematoma gradually increased in size. Expansion of the haematoma was closely monitored by surface marking on the chest wall. The clotting profile came as high APTT with normal PT/INR. As the patient was clinically pale, tachycardic, with a low HCT of 24 after 38 hours of critical phase, he was transfused with one unit of blood, along with 6 packs of platelets. Following platelet transfusion, the platelet count rose to 23 ( $140-400 \times 10^9/L$ ), Hb 8.3 (13-18g/L) and WBC 5.62 ( $3.6-11.0 \times 10^9/L$ ) and APTT 48 (30-40sec). Subsequently, 15ml/kg of FFP transfusion was given after the 45<sup>th</sup> hour of the critical phase. The haemoglobin level was 5.8 (13-18g/L) in the repeated full blood count. Thromboelastogram was performed due to suspected coagulopathy which showed low platelets and fibrinogen. Therefore, another 2 units of blood, 6 units of platelets, 15 units of cryoprecipitate and 10ml/kg of FFP were given at the 56<sup>th</sup> hour after the start of the critical phase.

We noted very deranged liver function test results with elevated PT/INR and APTT at 72 hours of the critical phase on day eight of fever. Therefore, following discussion with the gastroenterology team we arranged an IV N-acetylcysteine infusion. The patient was started on a liver failure regime, along with IV cefotaxime 1g eight hourly. The patient developed rectal bleeding and was drowsy with stage 2 hepatic encephalopathy at the 84<sup>th</sup> hour after the start of the critical phase and was transferred to the ICU for further management of acute liver failure.

As the patient improved clinically, he was transferred back to the ward HDU after two days. Patient was conscious and rational at day 11 of admission and haemodynamically stable. His platelet count and liver function gradually improved. We discharged him on day 13, after restarting his oral aspirin as his platelet count was more than 50000. At one

week of follow up, he was well with improved liver function and normal FBC. His chest wall haematoma was well settled.

**Table 01: Basic investigations and clinical events**

FEVER DAY	WBC COUNT	PLATELET COUNT	HAEMOGLOBIN	HAEMATOCRIT	AST	ALT	PT/INR	APTT	CLINICAL EVENTS
2	2.69	114	14.2	38.9	47	45			
3	1.63	78	13.1	36.2	181	79.7			
4	3.1	47	13.9	36.8	143	105.8			
5	2.64	30	16.2	43.2	336	188			Patient entered the critical phase
6	3.91	20	14.2	42.5	351	205	1.13	32.8	Right side anterior chest wall haematoma
7	2.98	16	13.9	37.5	954	440	1.10	32.2	Haematoma progressed
8	5.67	9	9.4	24.9	7691	2337	1.12	48.6	Hepatic encephalopathy stage 1
	5.67	23	8.3	22			2.02	41.6	Developed melaena
9	5.42	9	5.8	16.4	5960	2063	2.24	39.3	Hepatic encephalopathy stage 2
	7.93	47	7.6	21	4053	1628			ICU admission
10	7.85	56	10.8	31	1234	928	1.4	36.7	Transferred to HDU
11	7.71	62	11	30.4	967	799			Encephalopathy improved. melaena settled.
12	7.86	61	11.5	33.7	561	538			Haemodynamically stable

Normal ranges: WBC (3.6–11.0x10<sup>9</sup>/L), Platelet Count (140-400 x10<sup>9</sup>/L), Haemoglobin (13-18g/L), Haematocrit (41-50%), AST(8-33 U/L), ALT(7-56 U/L), PT/INR (10-20 sec/1-2 sec), APTT (30-40sec)



**Figure 1: Anterior chest wall haematoma**

## Discussion

Dengue fever has a spectrum of clinical manifestations which may range from simple fever to life threatening illness. Muscle haematoma formation is a rare complication. Clinicians should keep a close look out for different manifestations of the illness as it may help in future patient management. Currently there are only a few case reports of muscle haematoma in dengue including psoas haematoma [5,6,7], spontaneous rectus sheath haematoma [7,8] and retroperitoneal haematoma formation [9].

We did not come across any previous case reports of spontaneous bleeding into the anterior chest wall in dengue. Pectoralis major muscle haematoma usually occurs following invasive medical procedures, trauma to chest or in patients using blood thinner medications such as warfarin. The exact mechanism of bleeding in dengue fever is still not well described. The causes for bleeding can include thrombocytopenia, vascular leakage, hepatomegaly, viral load, virus sero-type or abnormal immune responses [10]. Activation of fibrinolytic mechanisms as well as cross reaction of developing antibodies with plasminogen have shown to be involved in the pathogenesis of dengue haemorrhagic fever [5].

It is important to identify muscle haematomas as early as possible as monitoring of the extension of the haematoma may help in assessing the requirement of blood and blood factor for transfusion. Otherwise, the patient may become haemodynamically unstable due to massive bleeding, leading to decompensated shock. Timely intervention with fluid resuscitation, blood product transfusion, monitoring of the PCV and haemoglobin levels may play a pivotal role in preventing such complications. These muscle haematomas can also result in compartment syndrome and nerve compression and the patient may need haematoma evacuation in such cases.

Acute liver failure is also a rare manifestation of dengue that was encountered in the latter part of the course of dengue hemorrhagic fever in our patient. It usually occurs in young adults in the second week of dengue fever [11]. It is diagnosed by evidence of hepatic failure along with derangement in the clotting profile [11]. Although the incidence of acute hepatic failure in DHF is very low (0.31%-1.1%), it is associated with a relatively high mortality rate (20%-68.3%) [4,12]. The pathophysiology could be the direct cytopathic effect of the DENV causing hepatocyte apoptosis, immune-mediated hepatocyte injury, cytokine storm and hepatic hypoperfusion [4]. We used N- acetyl cysteine, along with rest of hepatic failure management regime, in our patient. N-acetylcysteine is produced as a result of acetylation of the L-cysteine amino acid. It acts as a source of reduced glutathione and has the ability to scavenge free radicals in the body. Patients with liver failure in dengue have shown a positive response to N-acetylcysteine which encouraged us to use it in our patient [13]. Ultimately, the early detection of warning signs and prompt treatment is the main strategy to prevent morbidity and mortality in dengue fever.

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