

An unusual case of dengue haemorrhagic fever with retinal and vitreous haemorrhages

MDPR Fernando¹, JDVC Lekomwasam²

¹Registrar in Medicine, ²Consultant Physician, Karapitiya Teaching Hospital, Galle.

Introduction

Dengue haemorrhagic fever (DHF) is a growing global health issue, affecting over 100,000 cases each year [1]. It is endemic in Sri Lanka and the incidence has increased during the last five years. The disease has a broad range of bleeding manifestations due to thrombocytopenia, vasculopathy and a coagulopathy. However retinal haemorrhages are very rare in DHF. We report a case of DHF with subarachnoid, retinal and vitreous haemorrhages.

Case report

A previously healthy 31 year old man was transferred to Teaching Hospital Karapitiya with fever and vomiting of five days. He was transfused with intravenous fluids and platelets on day four of the illness for thrombocytopenia (platelet count – $18 \times 10^9/L$ and high PCV – 47%) at Base Hospital Hambantota. On the fifth day he was afebrile, but remained flushed. There was generalized ‘flushing’ multiple echymotic patches lymphadenopathy hepatomegaly and bradycardia (pulse rate 50/min). BP was normal (110/70 mmHg). There were no pleural effusions, neck stiffness or focal neurological signs.

Investigations at this stage revealed a Hb of 15 g/dL, PCV- 45%, Platelet count of $50 \times 10^9/L$, WBC/DC $3.88 \times 10^9/L$, (Neutrophils-53%) a normal clotting profile, normal echocardiography, elevated ASL and ALT (497 & 257 IU/L). ECG showed a sinus bradycardia. The blood film showed few reactive lymphocytes. US abdomen showed hepatomegaly, ascites and acalculous cholecystitis. On the same evening he developed a severe headache and further bradycardia (pulse rate - 26/min). Fundi were normal. On the following morning bilateral retinal haemorrhages were detected and CT brain revealed a

subarachnoid haemorrhage (SAH). Platelet count towards evening was $20 \times 10^9/L$. He was transfused with platelets.

On the day seven he developed partial 3rd cranial nerve palsy in the left eye and had evidence of pulmonary oedema. Platelet count was $55 \times 10^9/L$ and WBC/DC was $19.2 \times 10^9/L$ with 80% neutrophils. His blood gases warranted ICU care. From day 8 onwards he started improving. The diagnosis of DHF was based on clinical features such as fever, thrombocytopenia, hepatomegaly and plasma leakage as evidenced by high PCV and ascites. Secondary dengue infection was confirmed by the presence of both IgM and IgG antibodies against dengue virus. During follow up at the eye clinic, his visual acuity deteriorated to 6/36 in the right eye and haemorrhage extended to vitreous. Even after five months vitreous haemorrhage did not resolve and warranted a vitrectomy. His vision returned to normal following surgery.

Discussion

Neurological manifestations are rare in DHF and were seen in 1% cases in a study done in Vietnam [2]. These include mononeuropathies, polyneuropathies, Guillain-Barre syndrome and transverse myelitis. Encephalopathies may result from intracranial haemorrhage, cerebral oedema, liver failure and electrolyte imbalances [3]. Retinal haemorrhages in Dengue are extremely rare. There were two studies, which reported retinal haemorrhages, among patients with DHF [1]. (Incidence < 1% and 2.8%).

His bradycardia was attributed to the SAH, and associated raised intracranial pressure. Retinal haemorrhage and SAH occurred in our patient at platelet count above $45 \times 10^9/L$ highlighting the contribution from platelet function defects and

vasculopathy in addition to thrombocytopenia in the mechanism of haemorrhage in DHF as shown earlier. Coagulopathy is also shown as a contributory factor [4].

His fluctuating platelet count during the course of illness could be due to bone marrow suppression, platelet destruction and lining of the endothelial defects by platelets [1,4]. Higher viral titres of dengue may result in an amplified cascade of cytokines and complement activation causing platelet destruction as shown earlier [3]. His liver enzymes were well above 200 U/L indicating a marked hepatic involvement though the expected rise is < 200 U/L in dengue [4,5].

Importance of anticipation and detection of unusual manifestations in DHF and appropriate management is emphasised.

References

1. Sharma S, et al. Clinical profile of DHF in adults during 1996 outbreak in Delhi, World Health Organisation Dengue Bulletins 1998; **22**: 25 / 2003.
2. Soloman T, Dung MN, Vaughn DW. Neurological manifestations of Dengue Infection. *Lancet* 2000; **355**: 1053-8.
3. Robert V Gibbons, David W Vaughn. Dengue: an escalating problem. *British Medical Journal* 2002; **324**: 1563-6.
4. Nimmannitya S. Clinical manifestations of Dengue/ DHF, Scott B. Halstead. Pathophysiology and Pathogenesis of DHF. Monograph on Dengue / Dengue Haemorrhagic Fever 1993- World Health Organisation.
5. Dengue illness an overview. Guidelines and Clinical Management of Dengue / Dengue Haemorrhagic Fever. Ministry of Health Sri Lanka 2005: 5.