



## Case Report: Coinfection with Dengue Hemorrhagic Fever Complicated with Infective Viruses

Nilsson Peter\*

Professor of Medicine, Department of Medicine, Faculty of Medical Sciences, University of Sri Jayewardenepura, Nugegoda, Sri Lanka

### Abstract

Dengue fever caused by dengue virus is a common tropical infection transmitted by the mosquitos *Aedes aegypti* and *Aedes albopictus*. Four strains of the genus flavivirus are responsible for the epidemics of varying severity. Hepatitis A caused by hepatitis A virus is spread by faecal-oral route. The culprit virus is a hepatovirus. Coinfection with dengue virus and hepatitis A virus is rare and is a diagnostic as well as management challenge to the medical professional. We report a patient who presented to us with dengue virus and hepatitis A virus coinfection.

**Keywords:** Dengue; Dengue Hemorrhagic Fever; Hepatitis

### Introduction

Dengue fever is the world's most important viral hemorrhagic fever disease; it is the most geographically wide-spread of the arthropod-borne viruses, especially in the Americas, the Pacific islands and on continental Asia. Dengue virus infection can present a diverse clinical spectrum, ranging from asymptomatic illness to dengue shock syndrome, as well as unusual manifestations, such as hepatitis, encephalitis, myocarditis, Reye's syndrome, hemolytic uremic syndrome and thrombocytopenic purpura [1].

Liver injury due to dengue infection is not uncommon and has been described since 1970. However, in the Americas, this clinical presentation is poorly documented. Painful hepatomegaly, the main clinical symptom observed, is seen in up to 30% of patients. It is most commonly associated with dengue hemorrhagic fever (DHF), and its magnitude has no relationship with the severity of the disease [2]. On the other hand, an increase in aminotransferases can be seen in up to 90% of persons with dengue infection, with levels of aspartate aminotransferase (AST) higher than those of alanine aminotransferase (ALT).

### Case Report

A 34-year-old previously healthy male presented with high grade fever associated with constitutional symptoms of five days' duration. He was complaining of right hypochondrium pain and tea-coloured urine for two days associated with yellowish discolouration of the eyes. He had severe loss of appetite to food and water, but his bowel habits had been normal throughout the illness [3]. On admission, he was afebrile with normal vital signs but was deeply icteric. His abdominal examination revealed a tender, mild hepatomegaly while his cardiovascular, respiratory, and nervous system examination was normal.

His full blood count revealed a white cell count of  $3 \times 10^3 \mu\text{L}$  with a platelet count of  $116 \times 10^3 \mu\text{L}$ . His aspartate aminotransferase and alanine aminotransferase were 5162 U/L and 3964 U/L, respectively [4]. Initial total bilirubin was found to be 61.7  $\mu\text{mol/L}$  with an increased direct fraction. His prothrombin time/international normalized ratio and serum protein were normal throughout the illness. His dengue IgM antibody done by chromatographic immunoassay was positive on the 6th day of the illness. Hepatitis A IgM antibody done by the ELISA technique was positive on day 6 of the illness, while hepatitis A IgG antibody was negative. Other investigations done during the hospital

stay [5].

During the course of the illness, the patient was closely monitored for features of development of liver failure while continuing with the precritical monitoring of the dengue fever. The patient's hospital stay was uneventful, and he did not develop features of acute liver failure or features of plasma leakage as in DHF. He was discharged on day 10 of the illness [6, 7]. On follow-up, his AST, ALT, and bilirubin have become normal.

### Discussion

DF is caused by dengue virus and transmitted by vector *Aedes* mosquito. Hepatitis A infection is caused by hepatitis A virus and transmitted via faecal-oral route. Although both infections are common in the population occurring as isolated infections, coinfection is rare [8].

Coinfection of dengue virus with other infections has been documented in the past. DF per se is associated with hepatic involvement which ranges from minor alterations in the aminotransferase levels to acute hepatitis. DHF is associated with a greater incidence of hepatitis and fulminant hepatitis than simple dengue fever. The pathogenesis of liver involvement in DF is still poorly understood [9]. Direct viral invasion of the liver cells or products of host immune response acting on liver cells is thought to contribute to the liver cell injury. The elevation of transaminases is mild to moderate in most cases of DF, and the level of AST is greater than that of ALT. The levels decrease to normal levels usually within six weeks of resolution of infection. However, jaundice is an uncommon finding in DF [10].

Liver biopsies performed on hepatitis A patients have revealed hepatocellular necrosis with ballooning, eosinophilic degeneration, and infiltration of mononuclear cells, accounting for the liver injury

\*Corresponding author: Nilsson Peter, Professor of Medicine, Department of Medicine, Faculty of Medical Sciences, University of Sri Jayewardenepura, Nugegoda, Sri Lanka, E-mail: [peternilsson@edu.org](mailto:peternilsson@edu.org)

**Received:** 03-Jan-2023, Manuscript No: jcidp-23-85972, **Editor assigned:** 05-Jan-2023, PreQC No: jcidp-23-85972 (PQ), **Reviewed:** 18-Jan-2023, QC No: jcidp-23-85972, **Revised:** 25-Jan-2023, Manuscript No: jcidp-23-85972 (R) **Published:** 30-Jan-2023, DOI: 10.4172/2476-213X.1000172

**Citation:** Peter N (2023) Case Report: Coinfection with Dengue Hemorrhagic Fever Complicated with Infective Viruses. J Clin Infect Dis Pract, 8: 172.

**Copyright:** © 2023 Peter N. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

due to direct viral invasion and cellular immune response. Serum AST/ALT levels both rise rapidly during the prodromal period, reach peak levels, and then decrease by approximately 75% per week [11]. Serum bilirubin concentrations reach peak levels later and decline less rapidly than serum aminotransferases. Complete clinical recovery with restoration of normal serum bilirubin and aminotransferase values is usually achieved by 6 months.

Differentiating between the two infections and determining the possibility of coinfection are important in the management of an acutely ill patient with hepatitis [12,13]. A patient presenting with haemoconcentration, thrombocytopenia, and plasma leakage in the presence of features of hepatitis should alert the clinician about the possibility of DF, while on the contrary, elevated bilirubin levels and deranged coagulation profile should lead towards the possibility of viral hepatitis as they are usually unchanged in DF.

### Conflict of Interest

The authors declare that they have no conflicts of interest.

### References

1. Kobo O, Nikola S, Geffen Y, Paul M (2017) The pyogenic potential of the different *Streptococcus anginosus* group bacterial species: retrospective cohort study. *Epidemiol Infect* 145:3065-3069.
2. Noguchi S, Yatera K, Kawanami T, Yamasaki K, Naito K, et al. (2015) The clinical features of respiratory infections caused by the *Streptococcus anginosus* group. *BMC Pulm Med* 26:115:133.
3. Yamasaki K, Kawanami T, Yatera K, Fukuda K, Noguchi S, et al. (2013) Significance of anaerobes and oral bacteria in community-acquired pneumonia. *PLoS One* 8:e63103.
4. Junckerstorff RK, Robinson JO, Murray RJ (2014) Invasive *Streptococcus anginosus* group infection-does the species predict the outcome? *Int J Infect Dis* 18:38-40.
5. Okada F, Ono A, Ando Y, Nakayama T, Ishii H, et al. (2013) High-resolution CT findings in *Streptococcus milleri* pulmonary infection. *Clin Radiol* 68:e331-337.
6. Gogineni VK, Modrykamien A (2011) Lung abscesses in 2 patients with Lancefield group F streptococci (*Streptococcus milleri* group). *Respir Care* 56:1966-1969.
7. Kobashi Y, Mouri K, Yagi S, Obase Y, Oka M (2008) Clinical analysis of cases of empyema due to *Streptococcus milleri* group. *Jpn J Infect Dis* 61:484-486.
8. Shinzato T, Saito A (1994) A mechanism of pathogenicity of "Streptococcus milleri group" in pulmonary infection: synergy with an anaerobe. *J Med Microbiol* 40:118-123.
9. Zhang Z, Xiao B, Liang Z (2020) Successful treatment of pyopneumothorax secondary to *Streptococcus constellatus* infection with linezolid: a case report and review of the literature. *J Med Case Rep.* 14:180.
10. Che Rahim MJ, Mohammad N, Wan Ghazali WS (2016) Pyopneumothorax secondary to *Streptococcus milleri* infection. *BMJ Case Rep* bcr2016217537.
11. Xia J, Xia L, Zhou H, Lin X, Xu F (2021) Empyema caused by *Streptococcus constellatus*: a case report and literature review. *BMC Infect Dis* 21:1267.
12. Lee YJ, Lee J, Kwon BS, Kim Y (2021) An empyema caused by *Streptococcus constellatus* in an older immunocompetent patient: Case report. *Medicine* 100:e27893.
13. George B, Tanveer N, Boyars M (2021) *Streptococcus Constellatus* Empyema Presenting With Undulant Fever Pattern- A Case Report and Literature Review. *Int J Respir Pulm Med* 8:160.