J Vector Borne Dis 52, September 2015, pp. 265–266

Case Reports

Hepatitis A and dengue coinfection

Ira shah & Amit Dey

Pediatric Liver Clinic, BJ Wadia Hospital for Children, Mumbai, India

Key words Dengue; hepatitis A; serositis

Coinfection of both dengue fever and hepatitis A in an individual is rare and can present as a challenging diagnosis to any medical professional. Dengue, leptospirosis, malaria, and viral hepatitis, have many overlapping clinical symptoms making it difficult to diagnose them together. A series of case reports have been published on various coinfections with dengue in an individual. Coinfection of dengue with leptospira, malaria, hepatitis A, hepatitis E and typhoid fever have been reported in literature[1-2]. We report three cases of children presented with dengue and hepatitis A coinfection.

Case reports

Case 1: A two-yr-old boy presented with fever for eight days along with vomiting and jaundice for one day. There was no abdominal distension or bleeding. On examination, weight was 12 kg and height was 86.5 cm. He had pallor, jaundice and hepatomegaly with ascites and bilateral pleural effusion. Blood pressure (BP) and other systems were normal. Investigations at the time of admission are depicted in Table 1. His dengue IgM and hepatitis A IgM by Elisa were positive. He was treated with maintenance intravenous (i.v.) fluids with half-strength dextrose normal saline and vitamin K for three days. His ascites, pleural effusion and fever responded in next one week. His liver function test (LFT) normalized in next one month.

Case 2: A 10-month-old boy presented with fever and abdominal distension for eight days along with diarrhoea. There was no jaundice or vomiting. He was on breast feed and weaning diet. On examination, weight was eight kg and height was 72 cm. He had pallor, splenohepatomegaly and ascites. Blood pressure was normal. Other systems were normal. Investigations at the time of admission are depicted in Table 1. His dengue IgM and hepatitis A IgM by Elisa were positive. He was treated

Table 1. Investigation details of reported cases

Parameters/Tests	Case 1	Case 2	Case 3
Hemoglobin (g/dl)	9.7	8	6.5
WBC count (cells/mm^3)	7500	11,300	16,500
Platelets (cells/mm^3)	2,41,000	2,30,000	2,82,000
Bilirubin (mg/dl)	3.1	0.8	4.5
SGOT (IU/L)	1460	83	110
SGPT (IU/L)	1920	56	250
Total proteins (g/dl)	5.4	6.5	7.2
Albumin (g/dl)	3	3	3.2
USG abdomen	Ascites and bilateral pleural effusion	Hepatosplenomegaly and ascites	Hepatosplenomegaly and ascites with right pleural effusion
Dengue IgM (AI) (> 1.1)	8.2	1.5	(+)
Hepatitis A IgM	(+)	(+)	(+)
Prothrombin time (sec)	16.1	11.3	11.4
Partial thromboplastin time (sec)	35.4	38.6	30.9
Malaria OptiMAL	(–)	(–)	(–)
Widal	(–)	(–)	(–)

IU/L— International units per litre; (+) = Positive; (–) = Negative.

with oral fluids and vitamin K for one day. His ascites resolved in three days.

Case 3: A two-yr-old boy presented with fever and abdominal distension for two weeks. There was no vomiting. On examination, his weight was nine kg and height was 84 cm. He had jaundice, pallor, ascites, and hepatosplenomegaly. BP and other systems were normal. Investigations at the time of admission are depicted in Table 1. His dengue IgM and hepatitis A IgM by Elisa were positive. He was treated with i.v. fluids and vitamin K for two days; jaundice and ascites decreased in 10 days.

DISCUSSION

Hepatitis A virus (HAV) causes acute hepatitis, associated with significant morbidity and occasional mortality; and sometimes with rare complications such as acalculous cholecystitis, pleural effusion and ascites[3]. The pathology of ascites is thought to be venous or lymphatic obstruction due to liver involvement or reduction of oncotic pressure due to hypoalbuminemia during the course of infection[4].

Dengue infections often present with hepatomegaly, mild-to-moderate increase in transaminase levels with hemoconcentration and third spacing due to plasma leakage; leading to ascites and pleural effusion. Presentation with jaundice can simulate acute hepatitis and that is why, it is very important to differentiate it from hepatitis A. A series of case reports have been reported regarding mortality from dengue associated with liver cell failure[5]. In dengue, liver involvement can occur due to direct effect of the virus or host immune response on liver cells or localized vascular leakage inside the liver capsule and tissue tropism of particular viral serotypes or genotypes[6].

Serum aminotransferase levels are markedly elevated in viral hepatitis (8–10 times of normal) as compared to that in dengue fever in which they are elevated 2–3 times the normal value and the ratio of aspartate aminotransferase/lactate dehydrogenase (AST/LDH) is >four in viral hepatitis[7].

Other differentiating features of dengue fever include hemoconcentration, thrombocytopenia, and third space fluid losses[8]. In the present cases, though the patients had third spacing in form of ascites or pleural effusion, they did not have hemoconcentration or thrombocytopenia. However, since all had fever and serositis at the time of presentation, it was unlikely that hepatitis A alone was the cause of all abnormalities.

An abnormal coagulation profile should alert one to an underlying infection with a hepatotropic virus as the coagulation profile is usually not deranged in dengue[5]. Prolonged fever, highly elevated liver enzymes, deranged prothrombin time along with positive IgM for hepatitis and dengue made us suspicious about the possibility of coexistent viral hepatitis.

The endemic areas of both dengue and viral hepatitis throughout the world are most often superimposed on each other and thus one must rule out both these infections when someone presents with acute hepatitis. Presence of acute hepatitis with fever and ascites in an endemic area for hepatitis A and dengue should make one suspicious of coinfection.

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Correspondence to: Dr Ira Shah, 1/B Saguna, 271/B St Francis Road, Vile Parle (W), Mumbai–400 056, India.
E-mail: irashah@pediatriconcall.com

Received: 6 January 2015 *Accepted in revised form:* 15 April 2015