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RESEARCH ARTICLE

UNUSUAL PRESENTATION OF HEPATITIS A VIRAL INFECTION

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ABSTRACT

Hepatitis A virus is a nonenveloped 27-nm, heat-, acid-, and ether-resistant RNA virus in the Hepatovirus genus of the picornavirus family. Its virion contains four capsid polypeptides, designated VP1 to VP4, which are cleaved posttranslationally from the polyprotein product of a 7500-nucleotide genome. Hepatitis A has an incubation period of 4 weeks. Its replication is limited to the liver, but the virus is present in the liver, bile, stools, and blood during the late incubation period and acute preicteric phase of illness. Despite persistence of virus in the liver, viral shedding in feces, viremia, and infectivity diminish rapidly once jaundice becomes apparent. The diagnosis of hepatitis A is made during acute illness by demonstrating anti-HAV of the IgM class. After acute illness, anti-HAV of the IgG class remains detectable indefinitely, and patients with serum anti-HAV are immune to reinfection. Neutralizing antibody activity parallels the appearance of anti-HAV, and the IgG anti-HAV present in immune globulin accounts for the protection it affords against HAV infection. Patients develop abrupt onset of prodromal symptoms of malaise, joint pain (11%), right upper quadrant pain, and evanescent rash (14%) even weeks before developing jaundice (40% to 70%) in acute infection. Atypical manifestations of prolonged cholestasis, relapsing hepatitis, and extra hepatic involvement, which are rare unlike in hepatitis B or hepatitis C, may be present. Extra hepatic manifestations may include acute kidney injury, urticarial and maculopapular rash, polymyositis, arthralgias, and suppurative parotitis that can be seen in both prodrome and acute infection. Polymyositis can result in rhabdomyolysis, as seen in our patient with elevated creatinine phosphokinase and myoglobinuria. Many cases of acute kidney injury associated with hepatitis A infection have been described in endemic areas. We present an unusual complication of Hepatitis A infection presenting in a 14 year old young boy with encephalitis and refractory seizures.

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INTRODUCTION

14 year old male, became symptomatic in the last week of December 2014 with intermittent fever and chills. Initially he did not seek any medical attention. On 06 Jan 2015, he had an episode of transient loss of consciousness while at his home. Profile suggestive of a generalized tonic clonic seizure. He was admitted at a hospital where he was found to have jaundice (serum bilirubin- 5.8 mg/dL, AST-2206 IU/L and ALT-3259IU/L. IgM anti HAV done at that centre was positive. MRI (Brain, non contrast) showed Lt Sylvian fissure cortical lesion. He received inj. Midazolam and inj. Dexamethasone. He continued to be febrile and was shifted to this hospital, on 07 Jan 2015. No h/s/o pain abdomen, vomiting, hematemesis, maelena, drug abuse. There was no past history of jaundice, chronic medical illness or prolonged drug intake. No past history of blood transfusion. On initial evaluation the pt was Icteric, normotensive, febrile (Temp: 101F).

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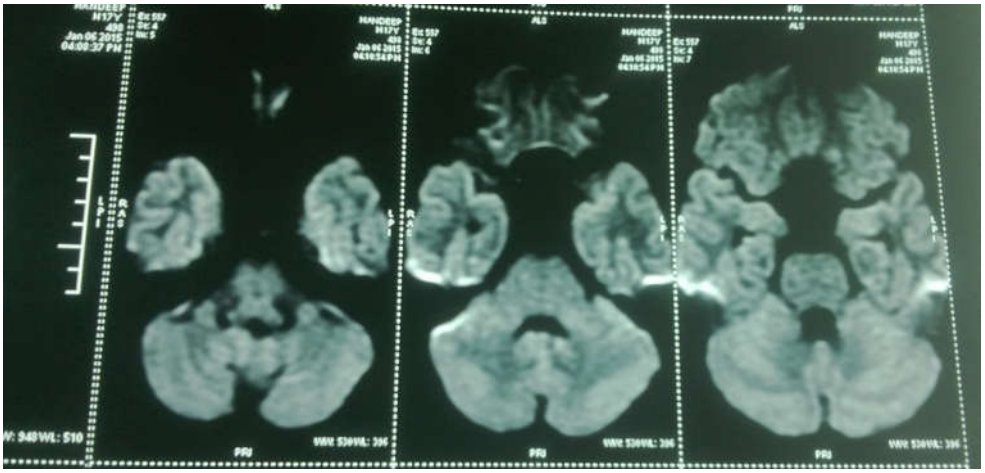
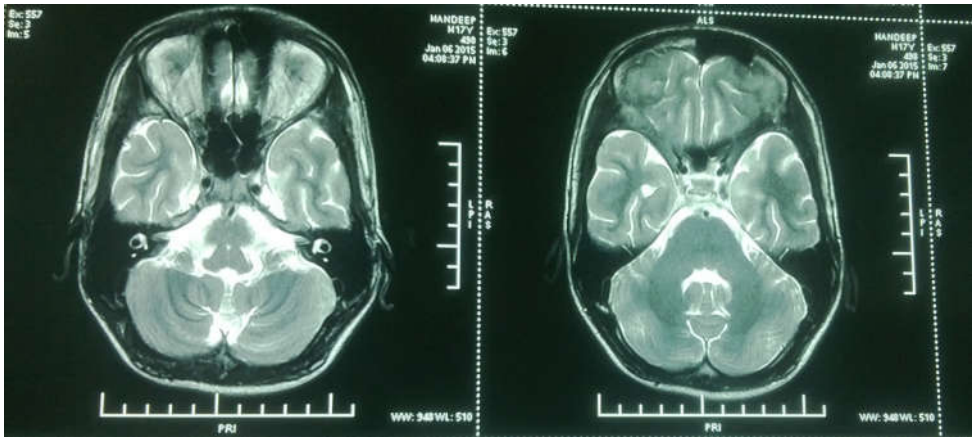
There was no pedal edema, clubbing or lymphadenopathy. No stigmata of chronic liver disease. Neurologically he was drowsy but arousable. Pupils were b/l equally reacting to light. Fundus-normal. No cranial nerve deficit. Bulk tone was normal all muscle groups. DTR -3+ b/l. Plantar Flexor b/l. No signs of meningeal irritation. However astrexis were present. Liver was just palpable below rt costal margin. No splenomegaly. A slit lamp examination performed at day 2 of admission ruled out KF rings. A provisional diagnosis of fulminant hepatic failure was considered. However in view of persisting fever with jaundice and features of encephalitis a differential diagnosis of cerebral malaria, Enteric fever, Ricketsial infection and viral encephalitis was also considered.

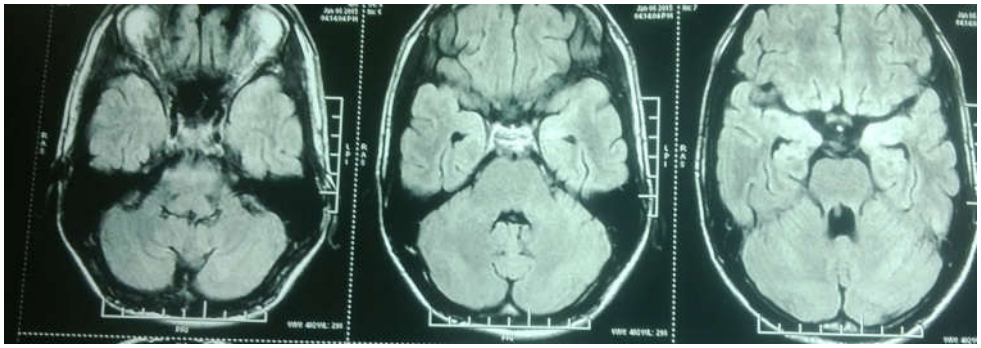
Summary of investigations as per table attached

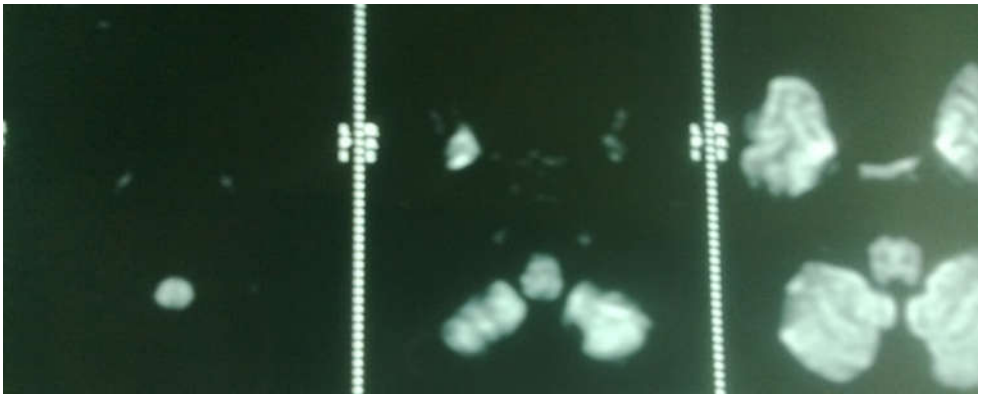
CSF analysis showed features of viral encephalitis (WBC-14/mm3, Lymphocyte predominant. RBC-240/mm3, proteins-45 mg/dL, globulins- not increased, CSF glucose- 90 mg/dL, matching blood glucose-129mg/dL, culture sterile, No organism seen on gram stain, AFB or India ink preparation).

Date	Hb	TLC	Platelets	INR	Serum Albumin	Serum Bilirubin	AST/ ALT	Alk Po4	Urea/ Creatinine	Na+/ K+
07 Jan	15.9	11, 200	1,14,000	1.43	3.4	7.5	1800/1080	150	18/0.7	137/3.7
08 Jan	13.9	5,800	1,17,000	1.21	3.5	6.5	825/530	128	18/0.9	129/3.5
10 Jan	13.8	6,800	1,25,000	1.2	3.0	8.5	450/325	115	20/0.9	138/2.9
12 Jan	13.0	8,100	1,54,000	1.2		7.9	360/309		18/0.9	134/3.4
14 Jan	13.4	8,700	1,51,000	1.2	2.9	7.0	324/289	101	16/0.9	130/3.3
15 Jan	14.6	5,500	1,05,000	1.14		6.5	121/175		15/0.7	136/3.5









Initial management included iv hydration(NS)., empirical antibiotics(Inj Ceftriaxone, antimalarial- Inj Artesunate + Cap Doxycycline, Syp Lactulose, Inj Vit K besides supportive management. His general condition deteriorated by day 03 of hospital stay when he developed status epilepticus (recurrent GTCS without regaining consciousness in between). He was given inj Lorazepam and started on antiepileptic drugs – Inj Sodium Dilantin. He continued to be febrile with max temp of 103F. In view of recurrent seizures and altered sensorium he was intubated and placed on mechanical ventilator support as well. By day 04 he continued to be febrile. He now started having Rt focal seizures despite being on benzodizapines (Inj midazolam) and inj sodium Dilantin. Although his hematological andbiochemical parameters started improving, his clinical condition continued to deterioratewith continuous fever and persistingRt focal seizure. At this point inj Levetiracetam was also added to the treatment regimen. Close monitoring of his nutritional status and electrolytes was being done. He was also planned for a repeat neuroimaging which however could not be done because of his worsening clinical condition. A repeat CSF done on 13 Jan 2015 showed increased WBC- 240/mm3, Lymphocytes, RBC-490/mm3, proteins- 60mg/dL, Globulins increased, CSF glucose-90 mg/dL, matching serum glucose- 129mg/dL, culture sterile, No organism seen on gram stain, AFB or India ink preparation).
His coagulation parameters remained normal throughout his hospital stay. In view of persistent fever, recurrent seizures and CSF picture in the setting of normal coagulation parameters, the diagnosis was revised to – possible viral encephalitis secondary to ?viral hepatitis A infection. Despite of best efforts his condition continued to deteriorate and he succumbed to his underlying illness on 16 Jan 2016.

PBS- Normocytic normochromic cells. Platelets adequate on smear. No atypical cells. No left shift. No MP seen.
IgM Anti HAV(CMIA)- 10.27 units, Reference range< .80
IgM Anti HEV- .19, Reference range <.90
Hbs Ag, Anti HCV- negative, HIV antibody- not detected
ICT for MP - Negative
USG(Abdomen)-Liver size- 14.5 cm in max. span. Echo texture appears minimally hypo echoic. No focal lesion seen. No intrahepatic biliary dilatation seen. CBD & portal vein- not dilated. Gall bladder- contracted, wall thickening noted.Pancreas, Spleen, Kidneys normal. Minimal ascites+.

DISCUSSION

Hepatitis A virus is a non enveloped 27-nm, heat-, acid-, and ether-resistant RNA virus in the *Hepatovirus* genus of the picornavirus family. Its virion contains four capsid polypeptides, designated VP1 to VP4, which are cleaved posttranslationally from the polyprotein product of a 7500-nucleotide genome. Hepatitis A has an incubation period of 4 weeks. Its replication is limited to the liver, but the virus is present in the liver, bile, stools, and blood during the late incubation period and acute preicteric phase of illness. Despite persistence of virus in the liver, viral shedding in feces, viremia, and infectivity diminish rapidly once jaundice becomes apparent. the diagnosis of hepatitis A is made during acute illness by demonstrating anti-HAV of the IgM class. After acute illness, anti-HAV of the IgG class remains detectable indefinitely, and patients with serum anti-HAV are immune to reinfection. Neutralizing antibody activity parallels the appearance of anti-HAV, and the IgG anti-HAV present in immune globulin accounts for the protection it affords against HAV infection (Harrison's principles of internal medicine). Patients develop abrupt onset of prodromal symptoms of malaise, joint pain (11%), right upper quadrant pain, and evanescent rash (14%) even weeks before developing jaundice (40% to 70%) in acute infection (Alarcon and Townes, 1973; Lednar *et al.,* 1985; Schiff, 1992). Atypical manifestations of prolonged cholestasis, relapsing hepatitis, and extra hepatic involvement, which are rare unlike in hepatitis B or hepatitis C, may be present. Extra hepatic manifestations may include acute kidney injury, urticarial and maculopapular rash, polymyositis, arthralgias, and suppurative parotitis that can be seen in both prodrome and acute infection (Routenberg *et al.,* 1979; Franczak and Matysiak, 1988). Polymyositis can result in rhabdomyolysis, as seen in our patient with elevated creatinine phosphokinase and myoglobinuria (Ann *et al.,* 2009). Many cases of acute kidney injury associated with hepatitis A infection have been described in endemic areas (Kim *et al.,* 2006). Acute hepatitis can be associated with evanescent rash in up to 14% of the cases, but it is usually a transient phenomenon. Spontaneous resolution occurs once the infection has cleared (Schiff, 1992). Hepatitis A has been known to even mimic Adult Onset Still's Disease, characterized by fever, arthralgia, rash, and leukocytosis (Yamaguchi *et al.,* 1992).

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