



Acute Glomerulonephritis with Hepatitis A- A Rare Complication

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Abstract

Hepatitis A is a necrotising disease of the liver, caused by hepatitis A virus. It can manifest clinically as mild illness to fulminant hepatitis. Though considered to be a self limiting illness it can sometimes lead to considerable morbidity and mortality. It commonly manifest's with hepatic symptoms like jaundice, fever, pain in right hypochondriac region but at times can have extrahepatic manifestations like, pleural effusion, coagulopathy, pancreatitis, arthritis, acute glomerulonephritis. Of these Acute glomerulonephritis (AGN) is a rare complication. We hereby present a case of hepatitis A associated with Acute glomerulonephritis. Patient presented with haematuria and hypertension, later developed ascites, pleural effusion and icterus. The patient was positive for Hepatitis A IgM, he was treated conservatively and improved dramatically.

Keywords: *Hepatitis A, Hematuria, Acute glomerulonephritis.*

Introduction

Hepatitis A is a self limiting disease, most commonly seen in developing countries, children between 5 to 14 years are most commonly affected. Although it is self limiting with recovery within 2 months, it can sometimes cause considerable morbidity⁽¹⁾. It usually manifests as fever, vomiting, hypochondriac pain with or without jaundice, however sometimes may have extrahepatic manifestations. Acute glomerulonephritis is one of the rare extrahepatic manifestation of hepatitis A occurring during the course of illness and mostly resolves spontaneously with the resolution of the disease⁽²⁾. Here we present a case of hepatitis A with Acute Glomerulonephritis.

Case Report

A four year male child was admitted in our hospital with complaints of fever since 2 weeks, vomiting, anorexia and pain in the right hypochondriac region since 2 days. H/O passing red coloured urine 3 hrs prior to admission. No h/o oliguria, burning micturition, trauma, any drug or food consumption causing red coloured urine, no history of any previous renal disease. No family history of any bleeding or renal disorders. On examination child was sick looking, febrile, with a temperature of 38.5 degree Celsius, with HR- 118 beats/min, RR-28/min, peripheral pulses well felt, weight-12kgs, height- 102cm, BP- 110/78 mmHg. Pallor present with no icterus and pyoderma. Abdomen was distended and non tender. Liver palpable 4cm below the right

subcostal margin in the mid clavicular line with the span of 9cm, with rounded borders and soft in consistency, non tender and spleen not palpable. Other systems were normal. Laboratory studies revealed Microcytic Hypochromic anaemia. Liver function test found to be abnormal with raised bilirubin of 2.6 and SGPT/SGOT-587/692. Urinalysis showing plenty of RBC's and mild proteinuria, however urine culture was sterile. Patient was worked up for Acute glomerulonephritis i.e. Renal Function Test, complement levels (C3), ASO titers all were negative. Coagulation profile (PT-APTT) was normal. After 2 days haematuria subsided and child started developing icterus. There was an increase in serum bilirubin from 2.6 to 7.1 and liver enzymes increased SGPT/SGOT- 587/692 to 756/812, anti HAV was sent which was positive. Other Viral markers including hepatitis B, C and E were negative by ELISA. USG abdomen and chest showing ascites and pleural effusion. The child was managed conservatively. Later total bilirubin started dropping and the fever started subsiding.

Discussion

Hepatitis A is a necrotizing inflammatory disease of the liver caused by hepatitis A virus of picornaviridae family. It is transmitted by feco-oral route, accounting for 1.4 million cases worldwide⁽³⁾. Majority of the cases being reported from developing countries due to poor sanitation, overcrowding and poor vaccine coverage. It clinically manifests as mild hepatitis with symptoms like fever, vomiting, abdominal pain and jaundice or may run a more devastating course with fulminant hepatitis, seen in less than 1% of the patients⁽³⁾. Extrahepatic manifestations are unusual. The exact incidence is not known as there are very limited studies. Their incidence ranges from 6-8% of all the cases of hepatitis A⁽⁴⁾. These include arthralgia, cutaneous vasculitis, ascites, pancreatitis, cryoglobulinemia, aplastic anaemia, acute glomerulonephritis, thrombocytopenia, agranulocytosis and pleural

effusion. Among these Acute glomerulonephritis is rare.

Glomerulonephritis is well documented as a complication in hepatitis B, C, Epstein-Barr virus but with hepatitis A is rare^(5,6). The exact mechanism is unknown, but several different mechanisms can occur sequentially or simultaneously. It could be deposition of immune complex in the glomerular capsule or direct cytopathogenic effect of virus on glomerular cells⁽⁷⁾. AGN can present during the course of illness or after the illness. Our patient presented with hematuria and hypertension which were suggestive of acute glomerulonephritis, however other causes of acute glomerulonephritis e.g. post streptococcal glomerulonephritis were ruled out as ASO titers were negative and C3 levels were normal. There was no other cause for acute glomerulonephritis therefore it was considered as a complication of hepatitis A.

Conclusion

Co-existence of Acute glomerulonephritis with hepatitis A infection is a rare complication. Hence the clinician must be aware, to avoid the diagnostic dilemma.

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