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Case Report

Severe Cholestasis in Hepatitis C Infection in IV Drug Users- Add on Drug Effect on Liver?

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Abstract

A 25 year old Caucasian male with history of intravenous drug abuse presented with yellowing of eyes and skin and dark brown urine for 1 week duration. Marked scleral icterus, yellowish discoloration of skin and mild epigastric tenderness was found on physical examination. Laboratory testing revealed markedly elevated Bilirubin 13.7 mg/dl and transaminases (ALT of 2057 IU/L and AST of 2016IU/L) without abnormality in synthetic liver function. Radiological studies were unremarkable and non-conclusive. Hepatitis serology was negative for acute Hepatitis A and E infection, immune for Hepatitis B infection and positive Hepatitis C Antibody screen. Hepatitis C RNA PCR was positive with a viral load of 83,458,000 IU/mL (7.921 log10 IU/mL). Ultra-sonogram of liver was unremarkable. Such level of severe hyperbilurubinemia atypical for hep C infection led us to rule out other coexisting causes of cholestasis and explore further about the possibility of the add on effect of drugs itself.

Keywords: Hepatitis C; Drug; Liver

Introduction

According to CDC, about 3.5 million Americans are currently living with hepatitis C (HCV) and roughly half are unaware of their infection. As per new CDC studies released in 2016 revealed that Hepatitis C kills more Americans than any other infectious disease. The greatest hepatitis C burden falls on baby boomers – those born from 1945 to 1965. However, a new wave of hepatitis C infections among people who inject drugs was striking findings from recent the surveillance data from CDC. It accounts for 20% of acute hepatitis cases, of which only one third are symptomatic. Variable transaminitis is seen with ALT and AST usually > 10x the upper limit. Bilirubin elevations may be seen as well, but is usually <10 mg/dl as per literature review. Herein, we describe a case of Acute HCV infection with severe hyperbilirubinemia as primary presentation of this infectious process.

Case Presentation

A 25 year old Caucasian man presented to ER for after noticing yellowish discoloration of eye, skin and dark brown urine for 1 week duration. He had no significant past medical or family history. He also complained of worsening anorexia and nausea without vomiting with mild abdominal pain located mostly in the epigastrium. He was alert, oriented to time, place and person and no symptoms of encephalopathy. No history of recent blood transfusion or hepatotoxic medication use and in monogamous relationship with one partner. Other history was unremarkable except for active intravenous heroin use. He reported injecting about 20 bags per day on an average since 3 years. He claimed that he reuses needles but does not share needles with others.

On presentation, he was hemodynamically stable, bradycardic (HR 42) and afebrile. Physical examination was remarkable for

marked scleral icterus, yellowish discoloration of skin especially the palms, mild epigastric tenderness with no guarding or rigidity. No organomegaly was appreciated. There were no peripheral stigmata of liver disease.

His lab testing revealed marked hyperbilirubinemia with total bilirubin of 13.7 mg/dl(Direct Bilirubin 11.4 mg/dl) and also transaminitis with ALT of 2057 IU/L and AST of 2016IU/L and minimally elevate ALP. Prothrombin time/international normalized ratio were 16.5/1.4, Ceruloplasmin 12.5 mg/dl, Acetaminophen levels <10 and drug screen was positive only for opiates. HIV 1/2 enzyme immunoassay was non-reactive. Hepatitis A, E, B and C serology was tested which reported negative for Hepatitis A and E, immune for Hepatitis B and anti HCV antibody titer was positive. Following positive serology, HCV RNA quantification showed viral load of 83,458,000 IU/mL (7.921 log10 IU/mL). Due to severe hyperbilirubinemia, gastroenterology was consulted and recommended additional investigations to rule out other causes. Abdominal ultrasound showed mild hepatomegaly, contracted gallbladder without stones, common bile duct of 4mm, and a normal sized spleen. Autoimmune serology was negative for anti-nucleolar antibodies, anti-mitochondrial M2 antibodies, anti-smooth muscle antibodies and anti- liver kidney microsomal 1 antibody. Patient improved symptomatically with resolution of abdominal pain and vomiting. Jaundice improved with downward trend of serum Bilirubin levels. He remained hemodynamically stable with no evident signs of mental deterioration. He was discharged with recommendations of outpatient follow up for Hepatitis C management. Unfortunately, patient failed to comply with follow up visits.

Discussion

Hepatitis C virus (HCV) accounts for approximately 20% of cases of acute and 70% of chronic hepatitis making it one of the major causes of chronic liver disease worldwide [1]. Although, incidence of Acute Hepatitis C is decreasing, cases still occur due to Intravenous drug use (IVDU), Health care associated procedures, unprotected sexual exposure and various unknown causes, of which, IVDU accounts for about 60% of cases [1]. High incidence of HCV infection amongst IVDU in the United States was attributed to sharing of unsterilized equipment in 2 cross-sectional studies [2,3] and 2 prospective studies [4,5].

Up to 70% of acute Hepatitis C cases are asymptomatic due to which majority of acute HCV infection are unrecognized. Symptoms, when evident, resemble other forms of acute hepatitis which are malaise, jaundice of variable severity, abdominal pain and nausea/vomiting [6]. Hepatitis C RNA is detectible in the serum typically within 7-21 days of exposure (average 12 days), followed by anti-HCV antibodies within 20-150 days (average 50 days) [7]. The average time period from exposure to symptom onset is 4–12 weeks.

Bilirubin elevations vary significantly during the acute phase of the disease. Symptomatic acute Hepatitis C has a mild and protracted course with clinically evident Bilirubin elevations seen in <25% of patients [8]. As per Loomba R., *et al*, 10 out of 25 patients who met criteria of acute Hepatitis C were found to be clinically icteric (40%) with average peak bilirubin levels of 2.5 [9]. Another study by Kathereen C., *et al*, resulted that 17 out of 28 patients (60%) bilirubin was elevated with a mean of 4.4 mg/dl (+/- 4.6 mg/dl). It is relatively unusual to present with Bilirubin levels >10mg/dl as seen in this case [10].

As seen in our patient, he presented predominantly for jaundice with some prodrome of symptoms including nausea, mild abdominal pain. His labs were remarkable for only for hyperbilirubinemia and transaminitits but no derangement of the synthetic liver function. The only risk factor identified in the case was IV heroin use. However, there are no robust data or studies that identified heroin as a causative agent of drug induced liver injury (DILI) leading to acute hepatitis. A very few articles look the effect of IV heroin use and impact of direct liver injury. One article in particular by Mary., *et al*. [11] published that the degree of increase in serum bilirubin and transaminase levels are more higher in IV drug abuser as compared to non-addicted persons and also mentioned that high level and acute load of drug augment the tendency to cholestasis in acute episodes of liver disease. They attributed to the direct toxic effect of the drug preparations upon the liver as cause of higher degree of biomarker elevation.

Learning points

- Identify Acute Hepatitis C as a differential diagnosis of acute liver injury in patients with Intravenous drug use specially when presented with severe hyperbilirubinemia
- Recognize atypical laboratory and clinical findings in Hepatitis C infection.

Conflict of Interest

No conflict of interest involved.

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