Case Report

Indian Journal of Emergency Medicine
Volume 3 Number 2, July - December 2017
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.3217.35

G6PD Deficiency as a Precipitant of Haemolysis in Hepatitis E Patients

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Received on 24.05.2017,
Accepted on 13.06.2017

Abstract

Hepatitis E is one of the common forms of Acute viral hepatitis in epidemic proportions in India. It has been seen to cause severe Haemolysis when associated with G6PD deficiency which is rarely seen in the northern India. This case report is of a 35 year old male with Hepatits E who presented to the Emergency Room pale and icteric and on evaluation was found to have G6PD deficiency as the cause of severe haemolysis. Therefore, in patients with acute viral hepatitis and severe anaemia with unconjugated hyperbilirubinemia, it becomes a necessity to rule out G6PD deficiency as a cause of the intravascular haemolysis.

Keywords: G6PD Deficiency; Hepatitis E; Viral Hepatitis; Intravascular Haemolysis; Unconjugated Hyperbilirubinemia; Anaemia.

Introduction

Hepatitis E, is one of the most common forms of acute viral hepatitis in India [1], it is potentially fatal in pregnant females and is a concerning cause of epidemic proportions of viral hepatitis in India. In patients with G6PD deficiency, it has been known to cause complications such as severe anaemia, haemolysis, hepatic, renal impairment or even death [2,3]. Since G6PD deficiency is of very low occurrence in the Indian population, reported between 2.2-14% in northern India [4].

We present the case report of a 35 year old male who presented to the ED with Hepatitis E with icterus, anaemia and was later on evaluation discovered to have G6PD deficiency.

Case Report

A 35 year old male had presented in the ED with a history of fever since past 10 days associated with nausea, vomiting and diarrhoea. Patient also complained of yellowish discolouration of eyes and dark coloured urine since past 2-3 days along with excessive drowsiness. No history of constipation, loose stools, malena, hematemesis, trauma.

On primary survey; his Airway was patent; Breathing, the respiratory rate was 16/min with a saturation of 85% on room air which improved to 89% despite supplementing with high flow oxygen; Circulation, heart rate was 98/min with a blood pressure reading of 130/70 mmHg, Peripheral pulses felt equally and a capillary refill time of less than 3 seconds. The patient was drowsy but responding to verbal commands, moving all four limbs with a GRBS of 220mg/dl.

On secondary survey; conjunctival pallor, icterus was seen, oral mucosa was dry, there were no distended neck veins, chest had equal air entry bilaterally with no adventitious sounds, heart sounds S1S2 heard with no murmurs and a normal JVP; Abdomen was soft, non-tender with mild hepatomegaly, no splenomegaly, shifting dullness present and bowel sounds heard. Central nervous system examination, the patient was drowsy but arousable, moving all four limbs, no sensory or motor deficit, Deep tendon reflexes were normal in all four limbs, the plantar reflexes were flexors bilaterally and flapping tremors were absent. Extremities showed no rashes, deformities or oedema.

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He was a known case of Diabetes Mellitus, Bipolar Mood Disorder and Hypertension for which he was on oral hypoglycaemics, Lithium and Amlodipine.

Among the point of contact tests done in the Emergency, his ECG and Chest X ray were within normal limits. Arterial blood Gas was within normal limit with no hypoxaemia seen and S. Lactate was 1.4mmol/L.

His Lab Investigations revealed as follows:

Haemogram – Haemoglobin was 6.3 g/dl, TLC of 6,400/mm3, Platelets 200,000/mm3;

Renal profile – S.Urea 24 mg/dL, S.Creatinine 0.7mg/dL, S. Sodium 122.5mEq/L, S. Potassium 4.4mEq/L S. Chloride 94mEq/L

Liver function tests – S.Albumin 3.4g/dL, S. Globulin 2g/dL, Total bilirubin 50.3mg/dL, unconjugated bilirubin 19.7 mg/dL, Alkaline phosphatase 422 U/L, SGOT 310 IU/L and SGPT 640 IU/L

Coagulation profile - PT 12.6 S, INR 1.11, APTT 24.6

Abdominal sonography was suggestive of Hepatomegaly, a thickened oedematous Gall Bladder with minimal ascites.

He was admitted with a working diagnosis of Viral Hepatitis with Hepatic Encephalopathy (Grade 1). Investigation results revealed Serum Ammonia 233 mcg/dl and Serum LDH 2244 U/L was seen.

Hepatitis A, Hepatitis B and Hepatitis C were tested negative. Hepatitis E virus was positive. Reticulocyte counts were elevated and G6PD enzyme was found to be 4.1 (low).

No evidence of Malaria, Typhoid, Dengue on investigation.

Coomb's test (Direct/Indirect) was Negative.

Patient was transfused 2 units of PRBCs. Patient was managed conservatively, avoiding all oxidant, hepatotoxic and nephrotoxic drugs, while maintaining an adequate urine output following which, on the fourth day, his lab parameters had improved with haemogram showing Hb of 10 g/dl.

After five days of hospital stay he was discharged in a stable condition with normal vital parameters, diagnosed as Acute Hepatitis E with Haemolytic anaemia due to G6PD deficiency.

Discussion

Viral Hepatitis has been known to cause mild haemolysis which rarely becomes evident clinically [5]. Severe haemolysis has been known in patients with G6PD deficiency on exposure to certain drugs [5,7,9]. But as in our case, viral hepatitis has been known to cause haemolysis in the absence of any such drugs. The patient described above in this case, had a fall in Haemoglobin, reticulocytosis, unconjugated hyperbilirubinemia along with low levels of G6PD which suggested severe intravascular haemolysis due to G6PD deficiency. The presence of severe hyperbilirubinemia in patients with viral hepatitis and G6PD deficiency has been reported previously [8-10]. The mechanism is believed to be through decreased levels of glutathione in RBCs as a result of accumulation of oxidants due to hepatic dysfunction, thus causing haemolysis in presence of G6PD deficiency [6].

Prognosis in these patient is associated with the degree of hepatic injury. Severe haemolysis could lead to increase in free haematin and bilirubin, thus leading to obstruction of renal tubules and acute renal impairment. Renal failure in these patients might be non-oliguric. Hence, renal function monitoring should be done with blood tests and urine osmolality and sodium.

Tests for G6PD deficiency might be negative during or after a haemolytic episode because the old red cells deficient in G6PD have undergone haemolysis and the newer red blood cells with higher content of G6DP might lead to false normal levels.

Hence, a repeat test needs to be done 8 to 10 weeks after the disease resolves. All G6PD-deficient individuals should be vaccinated against Hepatitis A and B.

Conclusion

In patients presenting with acute viral hepatitis and an unexplained severe anaemia with unconjugated hyperbilirubinemia, the possibility of intravascular haemolysis should be considered and evaluated with due consideration to rule out G6PD deficiency.

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