Salmonella Hepatitis -A Case Report

S. K. Chowdhury

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Abstract

Salmonella hepatitis is a rare complication of Typhoid fever in tropics. Typhoid fever is an infectious disease associated with high morbidity and mortality. The classical pattern of presentation is continuous fever with a ?step ladder? rise, headache, abdominal pain, diarrhea, constipation, and relative bradycardia. In a rare instance, Typhoid fever presented with the feature of acute hepatitis like hepatomegaly and deranged liver functions with hepatic encephalopathy. After taking proper informed consent, we have reported a young patient who presented with low-grade fever, diarrhea, and jaundice. Subsequently he was found to have acute salmonella hepatitis secondary to typhoid fever. Recognition of Salmonella hepatitis is of clinical importance as it can mimic acute viral hepatitis. Early institution of specific therapeutic intervention and supportive care improve the prognosis in these patients.

Index terms— typhoid fever, salmonella hepatitis, typhoid hepatitis.

1 Introduction

yphoid fever, a type of enteric fever caused by gram-negative bacteria Salmonella typhi. There is various type of bacteria, but S.typhi can only live in human. The usual manifestations are fever, headache, anorexia, abdominal pain, diarrhea, constipation, skin rash, relative bradycardia. It is a common infectious disease of tropics, associated with high morbidity and mortality. Each year, typhoid and paratyphoid fever, respectively, cause an estimated 26 million and 5 million illnesses globally. [1] It spreads through the consumption of contaminated food and water. Populations that have lack access to potable water, and adequate sanitation and hygiene are most affected. Incidence is highest in southern Asia and sub-Saharan Africa [2]. Various authors have highlighted the protean manifestations of this common tropical infection. Systemic complications ranging from intestinal perforation, hepatitis to neurologic manifestations have been well documented [3]. It usually starts as an acute systemic disease without localization and is clinically indistinguishable from other infections, including malaria, bacterial, and viral infections. After being ingested of the contaminated food or water organism invade the intestinal mucosal cell and secret sif A inside host cell. Which manipulate the immune function of the host cell and help multiplication of organism and release toxin and manifest inflammatory feature. Hepatic involvement could be considered important, as it may be associated with a higher relapse rate. [4] However, cholestasis secondary to typhoid fever also reported in a few instances. Typhoid fever usually diagnosed by the presence of an organism in blood and stool and urine. High-risk groups and complicated cases need an antibiotic to treat. Typhoid fever with a clinical picture of acute hepatitis is a rare complication. We report a young patient who presented with low-grade fever, diarrhea, and jaundice and had acute hepatitis secondary to typhoid fever.

2 II.

3 Case Report

Our patient, 30 years old serving soldier of a paramilitary force admitted in a tertiary level hospital with a history of low-grade fever and diarrhea of seven days duration on 5 September 2019. He was reasonably well 7 days back. There were no histories of cough, chest pain, dyspnea, abdominal pain, vomiting, dysuria, skin rashes, pruritus, dark color urine, clay color stool, or any hemorrhagic manifestations. He was not known to have any significant diseases like hypertension, diabetes mellitus, ischemic heart disease, or bronchial asthma. There was no history

of surgical intervention or tooth extraction and hospital admission. He denied drug abuse, blood transfusion, tattooing, unprotected sexual activity, and intravenous drug intake and alcohol consumption. He did not take any herbal and indigenous medicine. His family history of jaundice, congenital hyperbilirubinemia, and hemolytic anemia was negative. On examination, he was ill-looking, mildly dehydrated, and febrile. There was no edema, cyanosis, clubbing, koilonychia, leuconychia. His vitals were within the normal range. His relevant systemic examination was unremarkable. His routine blood picture shows ESR was 25 mm/h and the CRP was elevated 90 mg/l. ICT for malaria and thick and thin film for malaria parasites were negative. RFT and coagulation profile were normal. LFT shows a mild elevation of transaminase (AST-83U/L, ALT-114 U/L, ALP-196U/L) and S bilirubin (1.6gm/dl). Relevant investigations (Blood C/S, urine C/S, Hepatic viral markers and febrile antigen, NS1 antigen for dengue and serology for leptospirosis) were sent to rule out Viral hepatitis, Enteric fever, Leptospirosis, Dengue fever. The patient was treated empirically with IV fluid and antipyretic. Two days later, he has developed a yellow color of the sclera and vague right upper abdominal discomfort. His liver enzymes showed worsening (AST-792 U/l, ALT-2893U/L), and LDH level T was 378 U/L. His jaundice was apparent clinically, with a total bilirubin level of 6.14m mg/dl. The serum ALT: Lactate dehydrogenase (LDH) ratio was < 9. There was no evidence of viral hepatitis, Leptospirosis, and Dengue from the investigation report. The serological test for enteric fever was strongly positive in 1:320 dilution for both "O" and "H" antigens. His blood culture yielded growth of Salmonella typhi after 72 hours incubations, with antibiotic sensitivity to Ciprofloxacin (MIC <0.5 mg/L) and Ceftriaxone (MIC <0.25 mg/L). The strain was resistant to Vancomycin. Injection Ceftriaxone 2 gm twice daily was started after getting blood culture reports. With the above treatment, the patient showed rapid clinical improvement along with a gradual decline in liver transaminases levels. He got released from hospital after twenty days with follow up advice after one month. The patient report to the outpatient department after one month with liver function test which shows normal transaminase level.

4 III.

5 Discussion

Typhoid fever is a disease of high prevalence in Southeast Asia and Subsaharan Africa. Any febrile patient in this region demands evaluation for Typhoid and Malaria. As this disease manifest without any localization feature, other possibilities like a viral infection, acute enterocolitis, malaria, dengue, flu, other febrile illness needs exclusion before starting treatment. The confirmatory laboratory diagnostic tool is blood culture, other test like urine, feces, duodenal content culture may use but those can be positive in carrier state also. Now a day various diagnostic tools like ELISA test to measure anti lipopolysaccharides antibody, Rapid diagnostic test to detect Ig M & IgG antibody, real-time PCR assay, TUBEX, and TPTEST are used. [5]The factors predisposing to varying degrees of hepatic injury in typhoid fever are not known. Possibly, there is an interplay of the micro-organism factors and immunity, which causes liver injury. [6] Histopathological study and immune histochemistry of the liver biopsy reveals typhoid nodules, cloudy swelling, ballooning degeneration, moderate fatty change, and mononuclear cell infiltrate in few focal areas and intact bacilli in the liver parenchyma. [7] Approximately 21 to 60% patient shows a mild increase of transaminase level without hepatomegaly. [8] severe hepatic involvement in typhoid fever is a rare complication. Hepatic involvement clinically presents with vague discomfort in the right upper abdomen, enlarged liver, hepatic encephalopathy without asterixis. Only 4 to 5% patient shows severe hepatic derangement simulating acute viral hepatitis. [9] Early recognition of this clinical condition and institution of specific therapy is particularly important in tropical countries where malaria and viral hepatitis are quite common and clinical features are indistinguishable. [10] As in our setting typhoid fever and viral hepatitis are common ailment and Salmonella hepatitis is a rare incident so, we like to highlight the case to reduce causality in the future. The timely institution of antimicrobial therapy has reduced typhoid case-fatality rates from 15%-20% to less than 1%. [11]Other clues that raise the possibility of Salmonella hepatitis include fever, relative bradycardia, jaundice at the peak of fever, and left shift of WBCs. The serum ALT/LDH ratio (ALT: LDS <9) is the best discriminator between both entities. In some cases rise alkaline phosphatase and 5adenosine nucleotidase, hypofibrinogenemia, low prothrombin index and low platelets observed. [12] IV.

6 Conclusions

Salmonella hepatitis responds well to specific antibiotic therapy and jaundice resolves with clinical improvement within a few days. The clinical course can be severe, with a mortality rate as high as 20%, particularly with delayed treatment or in patients with other complications of salmonella infection. So as Typhoid fever is a common infection in a certain area of the globe, the recognition of salmonella hepatitis and other complication is of clinical importance. [1]

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