Print ISSN: 2321-6379
Online ISSN: 2321-595X
DOI: 10.17354/ijss/2016/625

Original Article

Febrile Jaundice in a Tertiary Care Center: A Prospective Study

K Vasanthan[1], S Manikantan[2], K Vengadakrishnan[3], M Rajkumar[3], N Senthil[3]

[1]Associate Professor, Department of General Medicine, Sri Ramachandra Medical College, Porur, Chennai, Tamil Nadu, India, [2]Resident, Department of General Medicine, Sri Ramachandra Medical College, Porur, Chennai, Tamil Nadu, India, [3]Professor, Department of General Medicine, Sri Ramachandra Medical College, Porur, Chennai, Tamil Nadu, India

Abstract

Background: Acute febrile jaundice is a common entity in clinical practice. A variety of causes have been observed including noninfectious causes.

Aim and Objectives: The aim of this study was to identify the causes of fever with jaundice and to analyze their clinical and laboratory profiles.

Methods: The study included 86 patients (70 males and 16 females) admitted in Sri Ramachandra Medical College Hospital from the year 2007 to 2009. All routine laboratory parameters, chest X-ray, electrocardiogram, and two-dimensional echo were carried out for all patients. Serology for specific bacterial pathogen and virus was performed along with cultures. Computed tomography scan was done when required.

Results: Viral hepatitis was noted in 35 patients (16 patients each with Hepatitis A and B and three patients had Hepatitis E). Nine patients had clinical malaria, seven had leptospirosis, and 32 patients had no definite etiology. 47 patients were found to be alcoholic, and four had history of blood transfusion. 50 patients had clinical hepatomegaly and 62 had splenomegaly. Seven patients had leukocytosis, 47 had thrombocytopenia, 21 had elevated blood urea nitrogen, 7 had elevated creatinine, 19 had elevated alkaline phosphatase, 83 had elevated serum glutamic pyruvic transaminase, and 50 had proteinuria.

Conclusion: Febrile jaundice is a distinct clinical entity. Elevated liver enzymes, thrombocytopenia along with proteinuria, were frequently observed, and leukocytosis is uncommon.

Key words: Hepatitis, Jaundice, Liver enzymes, Proteinuria, Thrombocytopenia

INTRODUCTION

Fever with Jaundice is one of the common presentations among patients. Some of the common causes include malaria, leptospirosis, and viral hepatitis. Various noninfectious conditions including connective tissue disorders, drugs, and malignancies are all known to contribute to febrile jaundice. Coinfection with two organisms worsens the outcome.

Access this article online

www.ijss-sn.com

Month of Submission : 10-2016
Month of Peer Review : 11-2016
Month of Acceptance : 11-2016
Month of Publishing : 12-2016

The approaches to a febrile patient with jaundice/deranged liver functions start with a careful history and physical examination. This is aimed at ruling out other features such as pain and other systemic manifestations which often give clues to alternative diagnosis. In general, patients present with a short febrile illness, and the jaundice appears or is noted by the relatives or the clinician. Patients may be completely unaware of jaundice. If the patients present with ongoing fever with jaundice, one must rule out alternative causes. In endemic countries, tropical infections are important, but one must never forget that fever with jaundice, vomiting, and abdominal pain could be a clinical manifestation of acute onset cholangitis. Jaundice can mask anemia in patients with hemolysis due to infections. Coagulation parameters are usually preserved in patients with tropical jaundice.[1-3]

Corresponding Author: Dr. K Vengadakrishnan, Sri Ramachandra Medical College, Porur, Chennai, Tamil Nadu, India.
E-mail: drkvk1975@gmail.com

While clinically approaching such patients, one must rule out surgical causes of jaundice. Multiorgan dysfunction is rare in acute viral hepatitis unless it is complicated by fulminant hepatic failure. The presence of a normal-sized liver or hepatomegaly along with splenomegaly would indicate a tropical infection.[4] When in doubt, hepatic enzymes and serum bilirubin can help. The clinical picture mimicking acute hepatic failure with preserved coagulation parameters points toward a diagnosis of tropical infections.

The management strategy in patients having tropical jaundice is aimed at treating the underlying causes and supporting the liver till recovery occurs.[5] Since the febrile patient may be vomiting with minimal intake the management starts by assessing the hydration status and hydrate using isotonic fluids. Although malarial infection is the most common cause of tropical hepatopathy, empiric use of antimalarial is not indicated.[5-8]

Antibiotic therapy is indicated for enteric fever, leptospirosis, and scrub typhus, which are the most common causes of tropical infections. Patients should be observed for the development of multiorgan dysfunction, with careful observation on urine output, blood pressure, seizures, encephalopathy, and bleeding. Blood and blood products should be liberally transfused if there is high risk of bleeding. One must use local guidelines for transfusing platelets in patients with thrombocytopenia at high risk of bleeding. Dengue is an important infection which affects liver which can at times be fatal. Prophylactic platelet transfusion in dengue is not indicated even if the platelet count is <10,000.[8-11] Enteric fever can rarely produce moderate to severe hepatitis.[12,13]

This study was conducted to identify the various clinical spectrum of febrile jaundice.

MATERIALS AND METHODS

This study was conducted in Sri Ramachandra University between August 2007 and August 2009. This was a prospective study done on 86 patients who presented with fever and jaundice. All patients who met the inclusion criteria (patients >18 years of age presenting with fever and jaundice or serum bilirubin >2 mg/dl at presentation) were subjected to laboratory and imaging studies.

A questionnaire for detailed history was taken from all patients, and a thorough physical examination was done. Abdomen examination was done to demonstrate hepatomegaly, splenomegaly, free fluid, or cirrhosis of liver. Complete blood count, blood urea, sugar, serum creatinine, liver function tests including coagulation profile, hepatitis B surface antigen (HBsAg), anti-hepatitis C virus (HCV), and urine analysis were done for all patients. Based on their clinical and laboratory profiles, certain special studies were done the collected data were analyzed using Statistical Package for Social Sciences (SPSS) for Windows software. Data were expressed as the mean ± standard deviation. $P < 0.05$ was considered statistically significant.

RESULTS

In this study, we analyzed a group of 86 patients with acute febrile jaundice. Of these 86 patients, 81.4% were males and 18.6% were females. Alcohol consumption was seen in 55.8% patients, sexual promiscuity in 19.8%, blood transfusion history in 4.7%, and respiratory symptoms in 3.5%. History of drug intake was seen in 3.5%. Clinically, liver was palpable in 60.5% and spleen in 73.3% patients. Urine protein was present in 57.65% patients, blood urea nitrogen (BUN) was abnormal in 24.4%, and creatinine was abnormal in 7%. Malarial parasite was positive for 10.5% patients using quantitative buffy coat method. HBsAg was positive for 20.9% patients, anti-hepatitis A virus in 18.6%, and anti-hepatitis E virus in 7%. IgM leptospirosis was positive in 8.1%. In ultrasonography, abdomen hepatomegaly was present in 44.2% patients, splenomegaly in 11.6%, and hepatosplenomegaly in 20.9%. Chest X-ray was abnormal in 5.8% patients. 2.3% patients had dengue and hepatitis C. The platelets were low (<150,000) in 54.85% patients and normal in 45.35%. About 37.2% patients had mild icterus fever, and myalgia predominated rather than jaundice. Almost 37.2% of people were treated for both malaria and leptospirosis and all of them became symptomatically better. One patient presented with respiratory symptoms and suspected to have atypical pneumonia.

DISCUSSION

In our study, of 86 patients, 8 (11.4%) patients of males were malaria positive and 1 (6.3%) of females was malaria positive. *Plasmodium falciparum* was positive for 5 patients and *Plasmodium vivax* was positive for 4 patients.

In a study conducted by Shah *et al.,*[14] 19 malarial hepatopathy in falciparum malaria who had jaundice was studied. Hepatomegaly was present in 30.6% patients. Splenomegaly was seen in 70.9% patients. About 51.6% had bilirubin more than 3 mg. Nearly 45% had alanine transaminase (ALT) levels more than 65. ALT levels were increased in 67.6% patients, of which 11.4% had ALT >3 times normal. Bilirubin was elevated in 81% patients.

About 23% had bilirubin >3 mg/dl. Thrombocytopenia was present in 91.6% patients.

Leptospirosis was positive in 7 patients (6 males and 1 female). Three patients were in age group in this study ranging from 40 to 50 years, 2 patients in 30-40 years, and 2 patients in 18-30 years. Clinically, splenomegaly was present in all 7 patients and hepatomegaly in 6 patients. All patients had normal total white blood cell count. Thrombocytopenia was present in all 7 patients; thrombocytopenia was also present in the study by Berman *et al.*[15]

None had change in mental status. In the study conducted by Edwards *et al.*,[16] altered sensorium was the predominent manifestation. Altered mental status was the strongest predictor of death in Salvador study group.[17]

BUN and creatinine were elevated in 3 patients; all patients had mild proteinuria similar to a study conducted by Lin *et al.*[18]

No patients expired in our study, and in other study conducted by Salvador *et al.*, case fatalities were 15% and 24.1%.

No patients had skin manifestation where skin manifestation was present in 2% in a study conducted by Edwards *et al.*

Liver enzymes, i.e., aspartate transaminase (AST)/alanine transaminase (ALT), was elevated in all patients, but none had elevation above 3-fold from normal, i.e., 8.3% among patients who had elevation >65 units. All patients had a elevated bilirubin with a direct bilirubin elevated >30% of total bilirubin. In hepatitis group, hepatitis A was diagnosed in 16 (18.6%) patients; hepatitis B was diagnosed in 17 (19.8%) patients; and hepatitis E was diagnosed in 3 (3.5%) patients. The maximum number of patients in hepatitis A group was between 18 and 30. The maximum number of patients in hepatitis B group was between 30 and 40. Promiscuity history was present in 13 patients of hepatitis B (76.5%). Three patients in hepatitis B group had history of prior blood transfusion.

Clinically, hepatomegaly was present in 11 (21.2%) patients in hepatitis A group, and 4 patients in hepatitis B group (7.7%). Two patients of hepatitis E had hepatomegaly. Clinically, splenomegaly was present in 1 patient of hepatitis A group and 3 patients of hepatitis B group.

BUN was abnormal in 5 patients of hepatitis group. Creatinine was abnormal in 4 patients. AST and ALT enzymes were elevated >3-fold in all patients of viral hepatitis group. Direct bilirubin was elevated above 30% of total bilirubin in all patients of viral hepatitis group. No patients were positive for HCV.

CONCLUSION

Febrile jaundice is an increasingly common entity in clinical practice. Early evaluation and empirical treatment has a definite role in final outcome.

REFERENCES

1. Bhalla A, Suri V, Singh V. Malarial hepatopathy. J Postgrad Med 2006;52:315-20.
2. Singh V, Bhalla A, Sharma N, Mahi SK, Lal A, Singh P. Pathophysiology of jaundice in amoebic liver abscess. Am J Trop Med Hyg 2008;78:556-9.
3. Mosler P. Management of acute cholangitis. Gastroenterol Hepatol (N Y) 2011;7:121-3.
4. Anand AC, Garg HK. Approach to clinical syndrome of jaundice and encephalopathy in tropics. J Clin Exp Hepatol 2015;5 Suppl 1:S116-30.
5. Theodossi A. The value of symptoms and signs in the assessment of jaundiced patients. Clin Gastroenterol 1985;14:545-57.
6. Anand AC, Ramji C, Narula AS, Singh W. Malarial hepatitis: A heterogeneous syndrome? Natl Med J India 1992;5:59-62.
7. Murthy GL, Sahay RK, Sreenivas DV, Sundaram C, Shantaram V. Hepatitis in falciparum malaria. Trop Gastroenterol 1998;19:152-4.
8. Halstead SB. Dengue. Curr Opin Infect Dis 2002;15:471-6.
9. Seneviratne SL, Malavige GN, de Silva HJ. Pathogenesis of liver involvement during dengue viral infections. Trans R Soc Trop Med Hyg 2006;100:608-14.
10. Chhina RS, Goyal O, Chhina DK, Goyal P, Kumar R, Puri S. Liver function tests in patients with dengue viral infection. Dengue Bull 2008;32:110-7.
11. Itha S, Kashyap R, Krishnani N, Saraswat VA, Choudhuri G, Aggarwal R. Profile of liver involvement in dengue virus infection. Trop Gastroenterol 2005;18:127-30.
12. Ramachandran S, Godfrey JJ, Perera MV. Typhoid hepatitis. JAMA 1974;230:236-40.
13. Khosla SN. Typhoid hepatitis. Postgrad Med J 1990;66:923-5.
14. Shah S, Ali L, Sattar RA, Aziz T, Ansari T, Ara J. Malarial hepatopathy in falciparum malaria. J Coll Physicians Surg Pak 2009;19:367-70.
15. Berman SJ, Tsai CC, Holmes K, Fresh JW, Watten RH. Sporadic anicteric leptospirosis in South Vietnam. A study in 150 patients. Ann Intern Med 1973;79:167-73.
16. Edwards CN, Nicholson GD, Hassell TA, Everard CO, Callender J. Thrombocytopenia in leptospirosis: The absence of evidence for disseminated intravascular coagulation. Am J Trop Med Hyg 1986;35:352-4.
17. Gouveia EL, Metcalfe J, Carvalho AL, Aires TS, Bisneto JC, Queirroz A, *et al*. Leptospirosis-associated severe pulmonary hemorrhagic syndrome, Salvador, Brazil. Emerg Infect Dis 2008;14:505-8.
18. Lin PC, Chi CY, Ho MW, Chen CM, Ho CM, Wang JH. Demographic and clinical features of leptospirosis: Three-year experience in central Taiwan. J Microbiol Immunol Infect 2008;41:145-50.

How to cite this article: Vasanthan K, Manikantan S, Vengadakrishnan K, Rajkumar M, Senthil N. Febrile Jaundice in a Tertiary Care Center: A Prospective Study. Int J Sci Stud 2016;4(9):108-110.

Source of Support: Nil, **Conflict of Interest:** None declared.