ORIGINAL RESEARCH PAPER

Volume-8 | Issue-4 | April-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CLINICAL SPECTRUM AND OUTCOME OF PATIENTS WITH ACUTE VIRAL HEPATITIS A: A PROSPECTIVE STUDY.

General Surgery

Dr Naveed Anjum Qureshi Medical Officer, District Hospital Poonch.

Dr Viney Sambyal* Lecturer Medicine, Post Graduate Department of Medicine, GMC Jammu. *Corresponding Author

ABSTRACT

Background: Hepatitis is the Liver inflammation which can be described as either a clinical illness or histological findings associated with the disease. Hepatitis can be due to Viral, Drug Induced, Ischemic, Infections (such as Malaria, Enteric Fever), Alcoholic Hepatitis, Autoimmune, and Idiopathic. Acute phase may result in conditions ranging from subclinical disease, self limiting asymptomatic disease to acute fulminant hepatic failure. In this study we evaluate the epidemiology, clinical features and outcomes of Acute Viral Hepatitis due to Hepatitis A virus.

Materials and methods: The study is designed to evaluate all cases presenting with the complaints of Fever, abdominal pain, nausea vomiting, anorexia, rash, altered sensorium, hepatosplenomegaly. Blood samples were taken at the time of presentation and sent to Microbiology Department and LFTs followed at an interval of 2 weeks.

Results: A Total of 76 cases were recorded between June 1st 2018 to 28th February 2019 of which 50(65.8%) were males and rest were females(34.2%). 8 cases (10.38%) were reported after close contact with the confirmed cases while two deaths (2.5%) were noted in the pediatric age group due to acute fulminant hepatic failure in early course of the disease. Hepatis A virus infection was confirmed in 45 patients and variable rise in serum bilirubin (range 1.5 to 32mg/dl) and liver enzymes (SGOT- 350 to 1700 and SGPT- 370 TO 1900) were noted.

Conclusion: Acute viral hepatitis in epidemic form may occur due to feco-oral transmission of hepatitis A virus which may result in a subclinical infection to acute fulminant hepatitis. Patients usually present with nonspecific symptoms which can be attributed to any viral illness; however there is high elevation of liver enzymes in acute viral hepatitis due to hepatitis A infection.

KEYWORDS

INTRODUCTION:

Hepatitis is the Liver inflammation which can be described as either a clinical illness or histological findings associated with the disease. Hepatitis can be due to Viral, Drug Induced, Ischemic, Infections (such as Malaria and Enteric Fever), Alcoholic Hepatitis, Autoimmune or Idiopathic. Acute phase may result in conditions ranging from subclinical, self limiting asymptomatic disease to acute fulminant hepatic failure. In this study we evaluate the epidemiology, clinical features and outcomes of Acute Viral Hepatitis due to Hepatitis A virus (HAV). HAV is transmitted through feco-oral route and crosses through the GIT and enters liver where it replicates in hepatocytes, released in the blood stream and is simultaneously present in bile and shed in feaces. IgM response is detected usually within 6 months, IgG responses persist for life, providing protection against reinfection. Pre and post immunization with pooled human serum immunoglobin (ISG) is 90% effective in preventing hepatitis A (1). Epidemiology of the disease is best defined by the measurement of anti-HAV antibodies (2). Seroprevalence studies reveal that 90% to 100% of the population acquires anti-HAV antibody and becomes immune by aldolesence (3). HAV is a major vaccine preventable, public health problem in developing world with compromised sanitary conditions and is also known as short incubation serum hepatitis as it posses potential for epidemic spread (4).

MATERIALS AND METHODS:

The study is designed to evaluate all cases presenting with the complaints of Fever, abdominal pain, nausea ,vomiting, anorexia, rash, altered sensorium, hepatosplenomegaly. All patients presenting to OPD for a period of 9 months from 1st June 2018 to 28th February 2019 clinically suspected for acute viral hepatitis were included for the study. Questionnaire specifying patients name, age, sex, and address, duration of illness, signs and symptoms of hepatitis were collected and informed consent was taken from patient/guardian. Peripheral blood samples (5ml) were taken which were centrifuged at 3000 rpm for 3 min to obtain serum and divided into 2 parts for microbiology and biochemistry one each. The serum samples were analyzed for LFT and anti-HAV IgM antibodies using ELISA kits and were reported as positive or negative as per the optical density (OD) value.

RESULTS:

A Total of 76 cases were recorded between 1st June 2018 to 28th February 2019 of which 50(65.8%) were males and 26(34.2%) were women with a gender ratio of 1.9:1 (Table 2). The higher seropositivity was noted in paediatric age group (Table 1). The patients above 45 years were observed least effected by HAV with only 1 seropositive case. 8 cases (10.38%) were reported after close contact with the confirmed cases while two deaths (2.5%) were noted in the pediatric age group due to acute fulminant hepatic failure in early course of the disease. Hepatis A virus infection was confirmed in 76 patients and variable rise in serum bilirubin (range 1.5 to 32mg/dl) and liver enzymes (SGOT- 350 to 1700 U/L and SGPT- 370 to 1900 U/L) was noted. Youngest patient was 4 years old and oldest 45 years. Highest number of cases were among age group 11-20 years (42.1%). Also there was clustering of cases in some villages whose common source was traced to a common water supply and contamination with sevage was found.

AGE DISTRIBUTION (TABLE 1)

AGE	NUMBER OF CASES
0-10 YEARS	22(28.9%)
11-20 YEARS	32(42.1%)
21-30 YEARS	16(21.1%)
31-40 YEARS	5(6.6%)
41-50 YEARS	1(1.3%)

TABLE 2 SEX DISTRIBUTIONS

SEX	NUMBER OF CASES
MALE	50(65.8%)
FEMALE	26(34.2%)
TOTAL	**76(100%)**

TABLE 3 SYMPTOMOLOGY OF PATIENTS

SYMPTOM	NO OF CASES
ANOREXIA	76 (100%)
JAUNDICE	76 (100%)
NAUSEA , VOMITING	74 (97.36%)
PAIN ABDOMEN	45 (59.21%)
HEPATOSPLENOMEGALY	12 (15.78%)
ALTERED SENSORIUM	05 (6.57%)
RASH	05 (6.57%)

DISCUSSION:

The spectrum of AVH is distributed worldwide with approximately more than 1.4 million new cases reported annually. The HAV rarely cause fulminant hepatitis and is seldom fatal with a fatality rate of 0 to 2 percent (6-7). The seroprevalence of HAV varies with time and region (8-9). There are very few published data available from India for acute HAV hepatitis and some of them suggest changing epidemiological

outline (10-11). A seropositivity rate of 30.63% and 44.8% for HAV were reported from Brazil and Bagdad respectively (13-14). Joon et al reported 19.31% seropositivity for HAV from Mangalore while Sarthi et al reported 37.25% seropositivity for HAV in patients presenting with AVH from Davengere. Both studies are from different cities of same state, Karnataka (India) with a notable difference in prevalence rate of HAV(15,16). The difference in seroprevalence findings of present study and that of other studies may relate to differences in HAV epidemiology in different geographical location, population group and social habits. Poonch district being a far flung area of Jammu and Kashmir State with poor public health services. The water supply lines were found placed in the sewage drains which resulted in contamination of drinking water due to negative pressure created in water supply lines.

CONCLUSION

The result of present study reveals the epidemic of HAV in Poonch region and still higher among young children. The study at regular interval needs to be performed for to evaluate and to observe changes in pattern of HAV prevalence in this reason. The results of present study can help in framing public health policies like sanitation program and formulation of vaccination strategies for appropriate use and controlling hepatitis A efficiently.

REFERENCES

1. Stepleton JT. Host Immune Response to Hepatitis A Virus. Journal of Infectious Disease.1995;171:S9-S14.
2. Koff RS. Hepatitis A. Lancet.1998; 351(9116):1643–9.
3. Melnick J. History and Epidemiology of HAV. Journal of Infectious Disease. 1995; 171:S2-S8.
4. Acharya SK, Madan K, Dattagupta S, Panda SK. Viral Hepatitis in India. The National Medical Journal of India.2006;19:203-17.
5. Wasley A, Fiore A, Bell BP. Hepatitis A in the era of Vaccination. Epidemiologic Reviews.2006;28:101-11.
6. Lemon SM. Type A viral hepatitis: New developments in an old disease. The New England Journal of Medicine.1985; 313: 1059-1067.
7. U.S. 2016 Surveillance Data for Viral Hepatitis. Statistics & Surveillance. Division of Viral Hepatitis. CDC. Cdc.gov. https://www.cdc.gov/ hepatitis/statistics/2016surveillance/ commentary. htm. Published 2018. Accessed October 23, 2018.
8. Syed R, Mohammed AH, Sindiri PK, Nathani AA, Rao VVR, Satti VP et al. Seroepidemiology of Hepatitis A virus in Hydrabad, South India. Journal of Medical and Allied Sciences.2012; 2:58-61.
9. Arankalle V, Mitra M, Bhave S, Ghosh A, Balasubramanian S, Chatterjee S, et al. Laxmi Rathore et.al. The Prevalence of Hepatitis A Causing Acute Viral Hepatitis in Western Rajasthan International Journal of Research & Review (www.ijrrjournal.com) 19 Vol.6; Issue: 3; March 2019 Changing epidemiology of Hepatitis A virus in Indian children. Vaccine Development and Therapy.2014;4:7-13
10. Tewari R, Makeeja V, Dudeja M. Prevalence of Hepatitis A in southern part of Delhi, India. International Journal of Medical Science and Public Health. 2016; 5:2067-2070.
11. Lakshmi TM, Vaithilingam A, Franklin A, Reddy EP. The prevalence of serological markers of viruses causing acute hepatitis in South Indian population. International Journal of Biological and Medical Research. 2011;2(4):925–8.
12. Jain P, Prakash S, Gupta S, Singh K P, Shrivastava S, Singh D D, Singh J, Jain A. Prevalence of hepatitis A virus, hepatitis B virus, hepatitis C virus, hepatitis D virus and hepatitis E virus as causes of acute viral hepatitis in North India: A hospital based study. Indian Journal of Medical Microbiology. 2013;31:261-5
13. Villar LM, Paula VS, Gaspar AM. Seasonal variation of Hepatitis A virus infection in the city of rio de Janerio. Brazil. Revista do Instituto de Medicina Tropical de Sao Paulo. 2002; 44:289-92.
14. Naaimi AS, Turky AM, Khaleel HA, Jalil RW, Mekhlef OA, Kareem SA et al. Predicting acute viral hepatitis serum markers (A&E) in patients with suspected acute viral hepatitis attending primary health care centers in Bagdad: a one year cross sectional study. Global Journal of Health Sciences.2012;4:172-83.
15. Joon A, Rao P, Shenoy SM, Baliga S. Prevalence of Hepatitis A virus (HAV) and Hepatitis E virus (HEV) in the patients presenting with acute viral hepatitis. Indian Journal of Medical Microbiology.2015; 33:102-105.
16. Sarthi M, Kumar KGR, Jayasimha VL, Kumar CSV, Patil SS, Taj KRS, Basavarajappa KG. Prevalence of Hepatitis A virus as a cause of acute viral hepatitis in central Karnataka, India. International Journal of Contemporary Pediatrics. 2017; 4:87-89.