ORIGINAL RESEARCH

Assessment of clinical profile of children with Hepatitis A infection

¹Dr. Diksha Sharma, ²Dr. Prachi Jain

¹Assistant Professor, Department of Paediatrics, F H Medical College, Tundla, Firozabad, Uttar Pradesh, India ²Assistant Professor, Department of Paediatrics, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India

Corresponding Author

Dr. Prachi Jain

Assistant Professor, Department of Paediatrics, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh,

India

Received: 12 November, 2021

Acceptance: 16 December, 2021

ABSTRACT

Background: Acute Hepatitis A is a viral infection that affects the liver and is caused by the hepatitis A virus (HAV). The present study was conducted to assess the clinical profile of children with Hepatitis A infection. **Materials & Methods:** 56 patients under the age of 15 years attended the department of paediatrics with acute hepatitis of both genderswere subjected to blood tests like complete blood count, liver function tests, prothrombin time and serum ammonia in cases with altered sensorium. Cases were managed conservatively with symptomatic treatment and dietary advice. **Results:** Age group 0-5 years had 4 males and 6 females, 5-10 years had 7 males and 9 females and 10-15 years had 19 males and 11 female child patients. Clinical features were fever seen in 47, jaundice in 56, abdominal pain in 37, vomiting in 15, dark colored urine in 39, altered sensorium in 21, lethargy in 13 and diarrhoea in 25 patients. The difference was significant (P< 0.05). The normal serum albumin level was 5.8 g/dl, prothrombin time was 13.2 seconds, TLC was 11214.5 cu/mm, total bilirubin was 65.7 U/l and ammonia was 96.2 mcg/dL. **Conclusion:** Although hepatitis A is a self-limiting illness, there are a number of risk factors that can make it worse, including poor eating habits, drug use, co-infection, and underlying liver disease. Therefore, every instance needs to be monitored until full recovery.

Key words: Children, hepatitis A, liver disease

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution -Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

INTRODUCTION

Viral hepatitis continues to be a major health problem in both developing and developed countries.¹ This disorder is caused by the 5 pathogenichepatotropic viruses recognizedto date: hepatitis A(HAV). B(HBV), C(HCV), D(HDV), and E(HEV) viruses. Many other viruses can also cause hepatitis usually as a component of multisystem disease. Acute Hepatitis A is a viral infection that affects the liver and is caused by the hepatitis A virus (HAV).² Hepatitis A is one of the several types of hepatitis viruses, and it is typically transmitted through the fecal-oral route, either by person-to-person contact or by consuming contaminated food or water. The virus primarily targets the liver, leading to inflammation and potentially causing various symptoms.³

The symptoms of acute Hepatitis A can vary but often include fever, fatigue, nausea, vomiting, abdominal pain, dark urine, clay-colored stools, and jaundice (yellowing of the skin and eyes).⁴ Some people infected with HAV may not exhibit symptoms, especially children. The virus is usually spread through contaminated food or water, or by close contact with an infected person.⁵ Poor sanitation and hygiene practices contribute to the spread of the virus.Over the last few years, number of cases with atypical presentation of hepatitis A in children has also been increasing.⁶The present study was conducted to assess the clinical profile of children with Hepatitis A infection.

MATERIALS & METHODS

The present study consisted of 56 patients under the age of 15 years attended the department of paediatrics with acute hepatitis of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. The indications for admission were poor general condition, not being able to eat properly due to protracted vomiting, high fever and altered sensorium. Patients were subjected to blood tests like complete blood count, liver function tests, prothrombin time and serum ammonia in cases with altered sensorium. Cases were managed conservatively with symptomatic treatment and dietary advice. Antibiotics were used whenever required. Data thus obtained

were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS Table I Distribution of patients

Age group (years)	Male	Female
0-5	4	6
5-10	7	9
10-15	19	11

Table I shows that age group 0-5 years had 4 males and 6 females, 5-10 years had 7 males and 9 females and 10-15 years had 19 males and 11 female child patients.

Table II Clinical features

Clinical features	Number	P value
Fever	47	0.87
Jaundice	56	
Abdominal pain	37	
vomiting	15	
Dark colored urine	39	
Altered sensorium	21	
Lethargy	13	
Diarrhoea	25	

Table II, graph I show that clinical features were fever seen in 47, jaundice in 56, abdominal pain in 37, vomiting in 15, dark colored urine in 39, altered sensorium in 21, lethargy in 13 and diarrhoea in 25 patients. The difference was significant (P < 0.05).

Graph I Clinical features



Table III Laboratory parameters

Laboratory parameters	Mean	SD
Albumin (g/dl)	5.8	1.1
Prothrombin time	13.2	1.8
TLC	11214.5	358.2
Total bilirubin(mg/dl)	1.3	0.5
Conjugated bilirubin (mg/dl)	1.1	0.6
Alanine Transaminase (ALT) (U/l)	61.2	3.4
Aspartate Transaminase (AST)(U/l)	65.7	5.8
Ammonia(mcg/dL)	96.2	12.7

Table III show that normal serum albumin level was 5.8g/dl, prothrombin time was 13.2 seconds, TLC was 11214.5 cu/mm, total bilirubin was 1.3mg/dl, conjugated bilirubin was 1.1mg/dl, alanine transaminase (ALT) was 61.2U/l, aspartate transaminase (AST) was 65.7U/l and ammonia was 96.2mcg/dL.

DISCUSSION

The most frequent etiological agent for sporadic acute viral hepatitis in India has been found to be acute hepatitis A (HAV), particularly in the pediatric age range.7 However, this virus can affect people of any age. This prevalent virus primarily spreads by the faeco-oral routeand as a result of inadequate sanitation and hygiene is still widespread in many impoverished nations. Acute hepatitis A infection has a wide range of clinical manifestations.⁸ It may occur with a variety of clinical signs or be asymptomatic. Atypical presentations include cholestatic hepatitis, relapsing hepatitis. and extrahepatic manifestations in children.⁹The present study was conducted to assess the clinical profile of children with Hepatitis A infection.

We found that age group 0-5 years had 4 males and 6 females, 5-10 years had 7 males and 9 females and 10-15 years had 19 males and 11 female child patients. Bhargava et al¹⁰ in their study 27 cases were enrolled. The mean age was 6.5 ± 3.9 years out of 27 cases. Fever (92.6%), gastrointestinal complaints (59.3%) and lethargy (37%) were the common presenting symptoms. 12 patients (44.4%) presented with irritability and altered sensorium. Physical findings were hepatomegaly (96.2%) and jaundice (100%) in all children. More than 4-fold increase in level of Aspartate transaminase and Alanine transaminase were noted in almost all cases. Prothrombin time was deranged in 12(44.4%) cases. Serum Ammonia was raised in 8(27%) patients. 8 cases (27%) developed fulminant hepatic failure out of which 2 cases had co-infection with dengue, 1 case had a history of consumption of nimesulide and 1 case had chronic liver disease & 7 cases (25.9%) succumbed to death. Rest 20 (74.1%) recovered completely.

We found that clinical features were fever seen in 47, jaundicein 56, abdominal pain in 37, vomiting in 15, dark colored urine in 39, altered sensorium in 21, lethargy in 13 and diarrhoea in 25 patients. Thapa et al¹¹ in their study the pattern of viral markers in acute sporadic hepatitis in 329 children and those in 334 healthy school children from North West India were studied. Hepatitis A was found to be the commonest infection in sporadic cases (78 per cent). Of these, 86 per cent were under 10 years and 50 per cent less than 5 years of age. Hepatitis B was positive in 8 per cent, non-A non-B in 13 per cent, A as well as B in 1 per cent, and none had Delta virus infection. Viral markers in healthy school children showed anti-HAV IgG positivity in 96 and 85 per cent in those belonging to low and high socio-economic groups, respectively, indicating past infection. HBsAg was positive in 1 per cent of cases. Viral hepatitis is an important public health problem in children and warrants active immunization.

We observed that normal serum albumin level was 5.8 g/dl, prothrombin time was 13.2 seconds, TLC was 11214.5 cu/mm, total bilirubin was 1.3 mg/dl,

conjugated bilirubin was 1.1 mg/dl, alanine transaminase (ALT) was 61.2 U/l, aspartate transaminase (AST) was 65.7 U/l and ammonia was 96.2 mcg/dL. Sharma et al¹² studied hepatitis A in children which were divided in 3 age groups; 1-5, 6-10 and 11-18 years. Clinical features, laboratory parameters, ultrasound findings were compared in three age groups.Out of 88 cases 48 were boys and 40 girls. In the present study hepatitis A was the most common (85.2%) etiology. Jaundice (90.9%) is the most common symptoms followed by dark colored urine (86.3%), loss of appetite (83.3%) and fever (68.2%) reported in this study. Icterus (90%) is the common sign reported followed most hv hepatomegaly (86%). Out of 88 cases 4 cases died. Out of 4 deaths, 2 cases of hepatitis A and 2 cases positive for Hepatitis B. The cause of death was hepatic encephalopathy in both the cases.

The limitation of the study is the small sample size.

CONCLUSION

Authors found that although hepatitis A is a selflimiting illness, there are a number of risk factors that can make it worse, including poor eating habits, drug use, co-infection, and underlying liver disease. Therefore, every instance needs to be monitored until full recovery.

REFERENCES

- Das AK. Changing patterns of aetiology of acute sporadic viral hepatitis in India: newer insights from north-east India. Int J Cur Res Rev. 2014;6(19):32-9.
- 2. Girish N, Sunil B, Devaranavadagi RA. A clinical study of viral hepatitis in children: a prospective hospital-based study. Int J Contemp Pediatr. 2018;5:563-8.
- 3. Sudhamshu KC, Sharma D, Poudyal N, Basnet BK. Acute viral hepatitis in pediatric age groups. J Nepal Med Assoc.2014;52(193):687-91.
- Pannuti CS, Mendonça JS, Pereira ML, Carvalho MJ, Amato Neto.Sporadic acute viral hepatitis A, B and non-A non-B, A prospective study of 150 consecutive cases in São Paulo, Brazil. Trop Geogr Med. 1985;37(2):136-8.
- Nandi B, Hadimani P, Arunachalam R, Ganjoo RK. Spectrumof acute viral hepatitis in Southern India. Med J Armed Forces India. 2009;65(1):7-9.
- 6. Parekh Z, Modi R, Banker D. Clinical study of hepatitis in children with special reference to viral markers. NHL J Med Sci. 2013;2(1):23-7.
- Behera AK, Jit BP, Purohit P,Nahak SR, Chhatar S, Marndi C, et al. Clinical profile of viral hepatitis in a tertiary health care centre of eastern India. Int J Med Res Rev.2016;4(7):1276-80.
- SalahuddinM, Syed SA, Manzoor H, Mahenaz A, Farhana T. Recent spectrum of acute viral hepatitis in children: an experience in a tertiary centre of Bangladesh. Adv Res Gastroentero Hepatol.2017;6(3):555686.
- 9. Bernuau JR, Durand F. Herbal medicines in acute viral hepatitis: a ticket for more trouble. Eur J GastroenterolHepatol. 2008;20(3):161-3.

- 10. Bhargava A, Panchal S, Gavhane J. Hepatitis A: A clinical spectrum of the disease in children admitted in a tertiary care hospital. Journal of the Pediatrics Association of India. 2019 Jul 1;8(3):1-2.
- 11. Thapa BR, Singh K, Singh V, Broor S, Singh V, Nain CK. Pattern of hepatitis A and hepatitis B virus markers in cases of acute sporadic hepatitis and in

healthy school children from North West India. J Trop Paediatr. 1995;41:328-9.

12. Sharma CM, Gupta S, Aggarwal B, Chaudhary P. Acute viral hepatitis in children: a prospective hospital based study. International Journal of Contemporary Pediatrics. 2020 Jul 22;7(8):1681.