Original Article
DOI: https://doi.org/10.70749/ijbr.v3i7.3001

INDUS JOURNAL OF BIOSCIENCE RESEARCH
https://ijbr.com.pk
ISSN: 2960-2793/ 2960-2807

Etiological Factors of Acute Hepatitis in Children

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ARTICLE INFO

Keywords: Acute hepatitis; Child; Hepatitis A virus; Liver diseases; Toxic liver injury

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Declaration

Authors' Contribution: All authors equally contributed to the study and approved the final manuscript.

Conflict of Interest: No conflict of interest.

Funding: No funding received by the authors.

Article History

Received: 15-05-2025 Revised: 29-06-2025
Accepted: 04-07-2025 Published: 10-07-2025

ABSTRACT

Background: Acute hepatitis in children is a common liver problem which may occur due to different infectious and non-infectious causes. Pattern of these causes may vary according to region and environmental conditions. **Objective:** To determine the etiological factors of acute hepatitis in children presenting to a tertiary care hospital in Peshawar. **Study Design:** Cross sectional study. **Duration and Place of Study:** This study was conducted from 12 February 2025 to 12 May 2025 in the Department of Pediatrics, Lady Reading Hospital, Peshawar. **Methodology:** A total of 118 children aged 1 to 12 years with acute hepatitis were included. Data was analyzed using Statistical Package for Social Sciences version 26. Mean and standard deviation were calculated for quantitative variables. Frequency and percentage were calculated for categorical variables. Chi square test and Fisher exact test were applied after stratification and p ≤0.05 was taken as significant. **Results:** Mean age was 5.97±3.49 years and mean weight was 20.66±8.71 kilograms. Toxin induced hepatitis was most common cause seen in 71 (60.20%) patients, followed by infectious diseases 24 (20.30%), metabolic diseases 12 (10.20%) and immune diseases 11 (9.30%). No significant association was found between etiological factors and demographic variables (p >0.05). **Conclusion:** Toxin exposure was the leading cause of acute hepatitis in children in this setting, followed by infections and metabolic disorders.

INTRODUCTION

Acute hepatitis in the pediatric population is a significant clinical challenge and is defined by the sudden onset of hepatitis, which causes inflammation of the liver.[1] Various causes of hepatitis in children show variability according to age, geographic location, immunization status and socio-economic status.[2] Among these causes, viral infections constitute the major factor. In this context, Hepatitis A and Hepatitis E viruses have been found to affect children in developing countries.[3] These infections can spread through unsanitary conditions and infected water. Hepatitis B and Hepatitis C viruses can also cause hepatitis in children. These can spread vertically from mother to child, through infected blood transfusions, and by using non-sterile equipment.[4] In addition to these, Epstein-Barr virus, cytomegalovirus, adenovirus and herpes virus can also cause hepatitis in children.[5]

Besides infectious causes, there are various non-infectious causes of acute hepatitis in children. Drug-induced liver injury is an important cause of acute hepatitis.[6] This includes paracetamol (acetaminophen) overdose, antitubercular drugs, antiepileptics and certain herbal products. Accidental toxin exposure, such as mushroom poisoning and exposure to industrial chemicals can cause acute hepatitis.[7] Autoimmune hepatitis is another cause of acute hepatitis in which there is an abnormal immune response against hepatocytes, evidenced by elevated liver enzymes and positive autoantibodies.[8] Metabolic causes such as Wilson's disease, among older children and adolescents, can cause acute hepatitis with features of hemolysis and potential liver failure.[9]

Systemic infections and other medical ailments can cause acute hepatitis. Bacterial septicemia, leptospirosis, malaria, and enteric fever can cause liver involvement with manifestations of jaundice with elevated liver enzymes.[10] Non-alcoholic fatty liver disease is usually a chronic illness, though it can present with acute hepatitis in obese children. Ischemic hepatitis due to shock or dehydration can present with a sudden rise in liver enzymes.[11] There can also be instances of acute hepatitis with no clear cause, which are referred to as idiopathic acute hepatitis.

There is scarcity of local data on the causes of acute hepatitis among pediatric groups in Peshawar therefore this study is deemed necessary to identify the prevalent factors in this region. Factors such as poor sanitary conditions, poor water supply, and poor immunization may influence the epidemiological profile of viral hepatitis among children in this region. Prompt identification of the

major causes will help in proper clinical management and also provide guidelines for prevention. This study will also provide valuable local data for health care planning.

METHODOLOGY

This cross sectional study was carried out in the Department of Pediatrics, Lady Reading Hospital Peshawar from 12th February 2025 to 12th May 2025. Ethical approval was obtained from the hospital ethical committee and CPSP before initiation of study. The sample size was 118. It was calculated by using WHO sample size software with 95% confidence level, 5% margin of error and expected frequency of metabolic diseases 8.33% in children presenting with acute hepatitis.[12]

Children of age 1 to 12 years of both genders were included. Only those cases were enrolled who had acute hepatitis defined as inflammation of liver with serum alanine aminotransferase (ALT) level >280 U/L and aspartate aminotransferase (AST) level >200 U/L on liver function test. Children with history of hereditary liver diseases such as Wilson's disease, history of chemotherapy, history of metabolic liver disease and history of heart failure were excluded from study. Basic demographics were recorded including age (years), gender, weight (kg), family history of acute hepatitis, parents' educational level, family socioeconomic status and residential status at time of enrollment. Detailed history and clinical examination were performed in each child. Relevant laboratory investigations were done for evaluation of etiological factors including toxin-induced hepatitis, infectious diseases, metabolic diseases and immune diseases. Findings were documented on specially designed proforma by the researcher.

Toxin-induced hepatitis was labeled when there was history of exposure to hepatotoxic drug or chemical and laboratory test showed AST and ALT >1000 IU/L. Infectious diseases were considered when any of the following were present: HBV Positive defined as persistence of HBV RNA level >50 IU/ml by Real Time PCR in serum, or HCV Positive defined as persistence of HCV RNA level >50 IU/ml by Real Time PCR in serum. Metabolic diseases were diagnosed when any one of following criteria were fulfilled: Galactosemia was taken if Galactose-1-phosphate uridyltransferase enzyme assay <1% of normal activity or Galactose >30 mg/dL (normal <10 mg/dL) or Galactose-1-phosphate >5 mg/dL (normal <0.4 mg/dL). Hereditary fructose intolerance was labeled if Fructose-1-phosphate aldolase enzyme assay <10% of normal activity or Fructose >100 umol/L (normal <50 umol/L). Tyrosinemia was considered if Tyrosine >400 µmol/L (normal <200 µmol/L) or Succinylacetone >5 mg/L (normal undetectable). Glycogen storage diseases were taken if Liver biopsy showed glycogen content >6% wet weight (normal <2%) or G6Pase enzyme assay (Type Ia) <10% of normal activity or Phosphorylase enzyme assay (Type VI) <10% of normal activity. Hemochromatosis was labeled if Serum ferritin >1000 ng/mL (normal 30–400 ng/mL) or Transferrin saturation >60% (normal <50%) or Liver iron concentration >200 mcg/g dry weight (normal <50 mcg/g). Immune diseases were diagnosed if IgG >1600 mg/dL (normal 700–1600 mg/dL) or ANA titer >1:160 or ASMA positive or Anti-LKM positive. Frequency of each etiological factor was calculated among children with acute hepatitis. Data were entered and analyzed using SPSS version 26. Quantitative variables like age and weight were presented as mean ± standard deviation. Categorical variables were expressed as frequency and percentage. Stratification was done for demographics. Post stratification chi square test was applied and p ≤0.05 was considered statistically significant.

RESULTS

The study included total 118 pediatric patients having mean age of 5.97±3.49 years and mean weight was 20.66±8.71 kg. Gender distribution was showing 61 male patients constituting 51.7% while 57 female patients was 48.3% of total sample. Regarding parents educational level, majority was having primary education 57 (48.3%), followed by uneducated parents 26 (22.0%), secondary education 31 (26.3%), and only 4 (3.4%) was having higher education. Family socioeconomic status was distributed as middle class 52 (44.1%), poor families 48 (40.7%), and rich families 18 (15.3%). Most of patients was from rural areas 71 (60.2%) compared to urban areas 47 (39.8%). Family history of acute hepatitis was present in 24 patients (20.3%) while 94 patients (79.7%) was not having any family history (Table 1).

Table 1

Patient Demographics

Demographics		Mean ± SD
Age (years)		5.97±3.49
Weight (kg)		20.66±8.71
Gender	Male n (%)	61 (51.7%)
	Female n (%)	57 (48.3%)
Parents Educational Level	Uneducated n (%)	26 (22.0%)
	Primary n (%)	57 (48.3%)
	Secondary n (%)	31 (26.3%)
	Higher n (%)	4 (3.4%)
Family Socioeconomic Status	Poor n (%)	48 (40.7%)
	Middle n (%)	52 (44.1%)
	Rich n (%)	18 (15.3%)
Residential Status	Rural n (%)	71 (60.2%)
	Urban n (%)	47 (39.8%)
Family History Acute Hepatitis	Yes n (%)	24 (20.3%)
	No n (%)	94 (79.7%)

The etiological distribution was showing that toxin induced hepatitis was most common cause affecting 71 patients (60.20%), followed by infectious diseases in 24 patients (20.30%), metabolic diseases in 12 patients (10.20%), and immune diseases was observed in 11 patients (9.30%) (Table 2).

Table 2

Frequency of Etiological Factors of Acute Hepatitis in Children

Etiological Factors	Frequency	% age
Toxin Induced Hepatitis	71	60.20%
Infectious Diseases	24	20.30%
Metabolic Diseases	12	10.20%
Immune Diseases	11	9.30%
Total	118	100%

Age group ≤6 years was having 40 cases (58.8%) while >6 years was having 31 cases (62.0%) with p-value 0.728. Gender distribution was similar with males 37 (60.7%) and females 34 (59.6%), p-value 0.911. Parents educational level was not showing significant difference (p=0.916) across all categories. Family socioeconomic status was also not significantly associated (p=0.904) with poor families 29 (60.4%), middle class 32 (61.5%), and rich families 10 (55.6%). Residential status was showing rural 44 (62.0%) and urban 27 (57.4%) with p-value 0.623. Family history of acute hepatitis was not significantly associated (p=0.764) with yes group 15 (62.5%) and no group 56 (59.6%) (Table 3).

Table 3

Association of Toxin Induced Hepatitis with Demographic Factors

Demographic Factors		Toxin Induced Hepatitis		p-value
		Yes n(%)	No n(%)	
Age Group (years)	≤6	40 (58.8%)	28 (41.2%)	0.728**
	>6	31 (62.0%)	19 (38.0%)	
Gender	Male	37 (60.7%)	24 (39.3%)	0.911**
	Female	34 (59.6%)	23 (40.4%)	
Parents Educational Level	Uneducated	15 (57.7%)	11 (42.3%)	0.916*
	Primary	35 (61.4%)	22 (38.6%)	
	Secondary	18 (58.1%)	13 (41.9%)	
	Higher	3 (75.0%)	1 (25.0%)	
Family Socioeconomic Status	Poor	29 (60.4%)	19 (39.6%)	0.904**
	Middle	32 (61.5%)	20 (38.5%)	
	Rich	10 (55.6%)	8 (44.4%)	
Residential Status	Rural	44 (62.0%)	27 (38.0%)	0.623**
	Urban	27 (57.4%)	20 (42.6%)	
Family History Acute Hepatitis	Yes	15 (62.5%)	9 (37.5%)	0.764**
	No	56 (59.6%)	38 (40.4%)	

*Fischer Exact Test **Chi-square Test

For infectious diseases, no demographic factor was showing significant association. Age group ≤6 years was 16 (23.5%) and >6 years was 8 (16.0%), p-value 0.315. Males was 16 (26.2%) and females was 8 (14.0%), p-value 0.100. All other factors including parents' education (p=0.737), socioeconomic status (p=0.946), residential status (p=0.794), and family history (p=0.946) was not significant (Table 4).

Table 4

Association of Infectious Diseases with Demographic Factors

Demographic Factors		Infectious Diseases		p-value
		Yes n(%)	No n(%)	
Age Group (years)	≤6	16 (23.5%)	52 (76.5%)	0.315**
	>6	8 (16.0%)	42 (84.0%)	
Gender	Male	16 (26.2%)	45 (73.8%)	0.100**
	Female	8 (14.0%)	49 (86.0%)	
Parents Educational Level	Uneducated	6 (23.1%)	20 (76.9%)	0.737*
	Primary	13 (22.8%)	44 (77.2%)	
	Secondary	4 (12.9%)	27 (87.1%)	
	Higher	1 (25.0%)	3 (75.0%)	
Family Socioeconomic Status	Poor	10 (20.8%)	38 (79.2%)	0.946**
	Middle	10 (19.2%)	42 (80.8%)	
	Rich	4 (22.2%)	14 (77.8%)	
Residential Status	Rural	15 (21.1%)	56 (78.9%)	0.794**
	Urban	9 (19.1%)	38 (80.9%)	
Family History Acute Hepatitis	Yes	5 (20.8%)	19 (79.2%)	0.946**
	No	19 (20.2%)	75 (79.8%)	

*Fischer Exact Test **Chi-square Test

Metabolic diseases were not showing significant associations with any demographic variables. Gender was showing males 3 (4.9%) and females 9 (15.8%) with p-value 0.068. Parents education, socioeconomic status, residential status, and family history all was having p-values greater than 0.05 (Table 5).

Table 5

Association of Metabolic Diseases with Demographic Factors

Demographic Factors		Metabolic Diseases		p-value
		Yes n(%)	No n(%)	
Age Group (years)	≤6	6 (8.8%)	62 (91.2%)	0.573**
	>6	6 (12.0%)	44 (88.0%)	
Gender	Male	3 (4.9%)	58 (95.1%)	0.068*
	Female	9 (15.8%)	48 (84.2%)	
Parents Educational Level	Uneducated	4 (15.4%)	22 (84.6%)	0.254*
	Primary	3 (5.3%)	54 (94.7%)	
	Secondary	5 (16.1%)	26 (83.9%)	
	Higher	0 (0.0%)	4 (100.0%)	
Family Socioeconomic Status	Poor	6 (12.5%)	42 (87.5%)	0.355*
	Middle	3 (5.8%)	49 (94.2%)	
	Rich	3 (16.7%)	15 (83.3%)	
Residential Status	Rural	6 (8.5%)	65 (91.5%)	0.448**
	Urban	6 (12.8%)	41 (87.2%)	
Family History Acute Hepatitis	Yes	1 (4.2%)	23 (95.8%)	0.455*
	No	11 (11.7%)	83 (88.3%)	

*Fischer Exact Test **Chi-square Test

Immune diseases was also not significantly associated with demographic factors. All p-values was greater than 0.05 for age groups, gender, parents' education, socioeconomic status, residential status, and family history (Table 6).

Table 6

Association of Immune Diseases with Demographic Factors

Demographic Factors		Immune Diseases		p-value
		Yes n(%)	No n(%)	
Age Group (years)	≤6	6 (8.8%)	62 (91.2%)	0.828**
	>6	5 (10.0%)	45 (90.0%)	
Gender	Male	5 (8.2%)	56 (91.8%)	0.664**
	Female	6 (10.5%)	51 (89.5%)	
Parents Educational Level	Uneducated	1 (3.8%)	25 (96.2%)	0.608*
	Primary	6 (10.5%)	51 (89.5%)	
	Secondary	4 (12.9%)	27 (87.1%)	
	Higher	0 (0.0%)	4 (100.0%)	
Family Socioeconomic Status	Poor	3 (6.3%)	45 (93.8%)	0.441*
	Middle	7 (13.5%)	45 (86.5%)	
	Rich	1 (5.6%)	17 (94.4%)	
Residential Status	Rural	6 (8.5%)	65 (91.5%)	0.689**
	Urban	5 (10.6%)	42 (89.4%)	
Family History Acute Hepatitis	Yes	3 (12.5%)	21 (87.5%)	0.693*
	No	8 (8.5%)	86 (91.5%)	

*Fischer Exact Test **Chi-square Test

DISCUSSION

In current study, toxin induced hepatitis was most common etiological factor affecting 71 patients (60.20%) of total sample. This high prevalence is because children are more vulnerable to accidental ingestion of toxic substances due to their curious nature and lack of awareness about harmful agents. The hepatotoxic medications like paracetamol and traditional herbal remedies are commonly used in developing countries

without proper medical supervision which is leading to drug induced liver injury. Also, children having immature hepatic enzyme systems are making them more susceptible to toxin accumulation and subsequent hepatocellular damage. Infectious diseases was second most common cause observed in 24 patients (20.30%). This finding is attributed to endemic nature of hepatitis viruses in developing regions where sanitation is poor and vaccination coverage is inadequate. Children are frequently exposed to hepatitis A and E viruses through contaminated water and food. The immature immune system of pediatric population is making them more prone to viral infections and subsequent acute hepatitis.

The present study findings was showing toxin induced hepatitis as most common etiological factor in 71 patients (60.20%) which is contrasting with majority of published literature. Samad A *et al*. [13] was reporting viral hepatitis as predominant cause with hepatitis A in 173 (62.9%) and hepatitis E in 93 (33.8%) cases. Similarly, Jamro BU *et al*. [14] was finding viral hepatitis in 62 (77.5%) cases with HAV constituting 50 (80.65%) of viral causes. Abdinia B *et al*. [15] also was reporting hepatitis A as most common etiology in 44 (34.3%) cases followed by autoimmune hepatitis 30 (23.3%). Rewatkar SS *et al*. [16] was observing hepatitis A in overwhelming 88 (88%) of cases and Mazumder MW *et al*. [17] was documenting HAV in 66 (52.38%) patients. Haque E *et al*. [18] was showing similar pattern with hepatitis A virus in 35 (70%) cases. Present study was showing metabolic diseases in 12 patients (10.20%) which is comparable with Sarwar HA *et al*. [19] who was reporting metabolic liver diseases as most common cause in 18 (58%) of infantile cases with galactosemia being predominant. Abdinia B *et al*. [15] was documenting Wilson disease in 11 (8.6%) cases and Mazumder MW *et al*. [17] was finding Wilson's disease in 16 (12.7%) cases. Tahir A *et al*. [20] also was reporting metabolic causes including glycogen storage disease 5 (8.3%) and Wilson disease 4 (6.7%). These similar findings is suggesting that metabolic disorders are constituting significant proportion of pediatric liver diseases and proper screening protocols are needed for early detection.

Immune diseases were observed in 11 patients (9.30%) in current study. Abdinia B *et al*. [15] was reporting autoimmune hepatitis in 30 (23.3%) cases which is higher than present findings. Jamro BU *et al*. [14] was documenting autoimmune hepatitis only in 3 (3.75%) cases which is closer to current results. This variation is possibly because of differences in diagnostic criteria, awareness level, and availability of specific immunological investigations in different healthcare settings.Mean age in present study was 5.97±3.49 years which is comparable with Tahir A *et al*. [20] who was reporting mean age 6.39±4.29 years and Jamro BU *et al*. [14] showing 6.3 years. Abdinia B *et al*. [15] was documenting mean age 6.92±0.46 years and Mazumder MW *et al*. [17] was finding 7.2±3 years. These consistent findings across studies is confirming that early childhood and preschool age group is most vulnerable period for acute hepatitis regardless of geographical location.

Gender distribution in current study was showing male predominance 61 (51.7%) which is consistent with Samad A *et al*. [13] Hussain W *et al*. [21] who was reporting 43 (62%) males, Bouk GR *et al*. [22] documenting 64 (64%) males, Mazumder MW *et al*. [17] showing 82 (65%) males, and Haque E *et al*. [18] reporting 31 (62%) males. This male preponderance is being observed consistently across multiple studies which is supporting notion that male children are having increased exploratory behavior and environmental exposure leading to higher risk. Family history of acute hepatitis was present in 24 (20.3%) patients in current study. Sarwar HA *et al*. [19] was reporting family history in siblings in 6 (19.4%) cases which is comparable. Samad A *et al*. [13] was documenting family history of hepatitis in 125 patients. This finding is suggesting genetic predisposition and shared environmental risk factors within families particularly for metabolic and infectious etiologies.

The present study was having several limitations which should be acknowledged. Firstly, this was single center study conducted at one tertiary care hospital which is limiting generalizability of findings to other healthcare settings and populations. Secondly, sample size was relatively small which may be affecting statistical power for detecting significant associations in stratification analyses. Finally, specific diagnostic tests for certain etiological factors was not uniformly available for all patients which may be affecting accurate identification of some cases.

CONCLUSION

The present study has concluded that toxin induced hepatitis was most common etiological factor in pediatric patients with acute hepatitis followed by infectious diseases, metabolic diseases, and immune diseases. Mean age of presentation was early childhood period with slight male predominance and majority of cases was from rural areas.

Disclaimer

There was no disclaimer for this research work.

Acknowledgment

The authors are very thankful to the doctors, nurses and other medical team of the department who helped a lot in this study. Their proper documentation of patient files and organized way of managing records made this research possible and smooth to complete.

Ethical Approval

Permission for this study was taken from the Institutional Ethical Committee before starting the research. All procedures were carried out under the guidance and regulations of the committee and also in agreement with the principles of Helsinki Declaration.

Patients' Consent

Written informed consent was taken from every participant before including them in the study. Patients were clearly informed that their personal data will remain confidential and they have full right to withdraw from the study at any time without any problem.

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