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RESEARCH ARTICLE
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ETIOLOGICAL PROFILE OF CHRONIC LIVER DISEASE IN CHILDREN: EXPERIENCE FROM A TERTIARY CARE HOSPITAL

Dr Parvaiz Rafieq sohil[1], Dr Shaista Ahmad[2], Dr Jafar Ahmad Tantray[3],* Dr Sourabh Gupta[4], Dr Muzafar jan [5]

[1]Senior Resident, Department of pediatrics , Government Medical College Srinagar, India
[2]Senior Resident, Department of pediatrics , Government Medical College Srinagar, India
[3]Senior Resident, Department of pediatrics , Government Medical College Srinagar, India
[4]Postgraduate Scholar, Department of pediatrics , Government Medical College Srinagar, India
[5]Professor & Head, Department of pediatrics , Government Medical College Srinagar, India

Corresponding author: *Dr Sourabh Gupta
Postgraduate Scholar, Department of pediatrics, Government Medical College Srinagar, India
Email id: sourasachit@gmail.com

Abstract

Background: Chronic liver disease (CLD) in children comprises a diverse group of disorders with varied etiologies, clinical presentations, and outcomes. Early identification of the underlying cause is essential for timely interventions and improving prognosis, particularly in resource-limited settings where advanced diagnostics and liver transplantation facilities may be delayed or inaccessible.

Aim: To assess the clinical profile, etiological spectrum, and outcomes of pediatric patients admitted with chronic liver disease in a tertiary care hospital in Kashmir.

Methods: This was a prospective observational study conducted over two years in the Department of Pediatrics at G.B. Pant Hospital, Government Medical College Srinagar. A total of 44 children aged 1 to 18 years with confirmed or suspected chronic liver disease were enrolled. Detailed history, clinical examination, laboratory investigations, imaging, liver biopsy (where indicated), and genetic analysis were performed. Data were compiled and analyzed using Epi Info software.

Results: Among the 44 children, 59% were males and 41% females, with the majority (70.4%) aged 1–5 years. Abdominal distension (93%) and jaundice (81.8%) were the most common presenting symptoms, while hepatomegaly (77%) and jaundice (79.5%) were the most prevalent signs. Biochemical abnormalities included elevated SGPT/SGOT, hyperbilirubinemia, and prolonged prothrombin time in most patients. The most common etiologies identified were post-Kasai biliary atresia (18.1%), glycogen storage disorders (13.6%), Wilson's disease (9%), PFIC (9%), and autoimmune hepatitis (6.8%). Genetic abnormalities such as hereditary fructose intolerance, Niemann-Pick disease, and Alagille syndrome were also identified. Liver biopsy was performed in 31% of patients. Four patients (9%) had cryptogenic CLD. Many children presented late, with complications including ascites and variceal bleeding; 18% had previously failed Kasai surgery and were referred for liver transplantation.

Conclusion: Chronic liver disease in children presents with a wide range of clinical and biochemical findings. Biliary atresia remains the leading cause in this setting, followed by metabolic and genetic disorders. Delayed referral and limited access to liver transplantation remain major

challenges. Early diagnosis, comprehensive evaluation, and timely surgical or transplant interventions are critical to improving outcomes in pediatric CLD.

Keywords: Chronic liver disease, children, biliary atresia, glycogen storage disease, Wilson's disease, PFIC, autoimmune hepatitis, pediatric hepatology.

Introduction

Chronic liver disease (CLD) in children represents a significant clinical challenge characterized by progressive destruction and regeneration of the liver parenchyma, culminating in fibrosis and, ultimately, cirrhosis. It is associated with impaired synthetic function of the liver, such as diminished production of serum proteins and clotting factors, disrupted glycemic control and ammonia metabolism, abnormal bile secretion, and cholestasis [1]. CLD is typically diagnosed when hepatic symptoms persist for more than six months, although this criterion may not apply to infants, where inborn errors of metabolism may lead to CLD in a shorter time frame [2].

Most acute liver conditions resolve within 12 weeks; therefore, any persistence beyond this period should prompt evaluation for possible chronicity. The etiological spectrum of CLD includes infectious, metabolic, genetic, vascular, autoimmune, and structural disorders that ultimately result in progressive liver damage and complications such as portal hypertension and hepatic encephalopathy [3,4]. In pediatric populations, CLD remains a growing health concern, contributing to considerable morbidity and mortality globally [5]. Although the exact prevalence is unclear, it is estimated that approximately 15,000 pediatric hospital admissions occur annually due to CLD in the United States [6].

Children with CLD may present with nonspecific and overlapping clinical signs, including jaundice, pruritus, abdominal distension, pale stools, and dark-colored urine [7]. Non-alcoholic fatty liver disease (NAFLD) has recently emerged as a common etiology in children, with a pooled prevalence of 7.65% in the general pediatric population and up to 34% among obese children [8]. Notably, 5–15% of pediatric CLD cases remain idiopathic despite extensive evaluation [9].

Etiological patterns of CLD vary geographically. Indian childhood cirrhosis, once a leading cause of pediatric liver disease in the Indian subcontinent, has become rare due to preventive public health interventions [10]. Parasitic infections like hydatid disease and schistosomiasis still contribute to the burden of CLD in endemic regions. In older children, common causes include chronic hepatitis B and C, Wilson's disease, autoimmune hepatitis, cystic fibrosis, and primary sclerosing cholangitis [11]. Indian studies indicate that metabolic disorders (especially Wilson's disease) constitute the most common cause (\~25%), followed by viral hepatitis (8–15%) and autoimmune diseases (2–4%). Nearly 40% of cases remain cryptogenic [12,13].

In contrast, Western data suggest autoimmune hepatitis as the leading etiology, with infectious causes being relatively rare. Nonetheless, idiopathic forms still account for a substantial proportion [14,15]. The diagnosis and evaluation of CLD have significantly advanced with the availability of sensitive serological assays, imaging modalities, and improved histopathological techniques. Despite being invasive, liver biopsy remains the gold standard for diagnostic confirmation, etiological classification, and assessment of disease severity [16].

Non-invasive techniques like transient elastography and biomarkers are being increasingly used to evaluate liver fibrosis in children. Delayed diagnosis of biliary atresia often leads to chronic liver damage in infancy. Similarly, Oriental cholangiohepatitis, common in parts of Asia, may lead to secondary biliary cirrhosis in children [17].

The limited diagnostic infrastructure in many developing regions restricts the identification of metabolic etiologies, which are therefore underrepresented in epidemiological studies. CLD should be suspected in children with a history of prolonged conjugated hyperbilirubinemia, family history of liver or autoimmune disease, recurrence of hepatitis symptoms, or persistent features of acute hepatitis beyond three months. Physical signs can include poor growth, muscle wasting, spider angiomata, hepatosplenomegaly, ascites, asterixis, and altered mental status.

Every case warrants a comprehensive clinical, biochemical, serological, and histopathological workup to establish a definitive diagnosis [18]. Liver biopsy not only aids in establishing the histological diagnosis but also in identifying metabolic disorders through enzyme studies and storage material analysis (e.g., copper and iron quantification). It also helps monitor therapeutic response and identify hepatotoxicity related to treatment [19].

Materials and Methods

This hospital-based observational study was conducted over a period of two years in the Department of Paediatrics at GB Pant Hospital, Government Medical College, Srinagar. Ethical clearance was obtained from the Institutional Ethical Committee prior to initiation of the study.

Inclusion Criteria

All children and adolescents aged between 1 and 18 years presenting with clinical features suggestive of chronic liver disease (CLD) were included in the study.

Exclusion Criteria

Patients were excluded if they:
1. Were less than 1 year or more than 18 years of age.
2. Had acute liver illnesses or focal hepatic lesions.

All patients enrolled underwent a detailed history and clinical examination. The following baseline and diagnostic investigations were performed:

Laboratory Investigations
* Complete blood count (CBC)
* Liver function tests (LFTs)
* Kidney function tests (KFTs)
* Random blood sugar (RBS)
* Routine urine examination (RUE)
* Arterial blood gas analysis (ABG)

In addition to baseline investigations, specific tests were conducted based on clinical indications:
* Coagulation profile: Prothrombin time (PT), International Normalized Ratio (INR), and activated partial thromboplastin time (APTT) using clotting method
* Enzyme assays to evaluate for metabolic disorders such as galactosemia, alpha-1 antitrypsin deficiency, and glycogen storage disorders
* Serum ceruloplasmin levels measured via immunoturbidimetric assay
* 24-hour urinary copper excretion analyzed by Inductively Coupled Plasma Mass Spectrometry (ICP-MS)
* TORCH profile assessed using enzyme-linked immunosorbent assay (ELISA)

For evaluation of autoimmune hepatitis, the following markers were tested:
* Antinuclear antibody (ANA), anti-smooth muscle antibody (ASMA), and anti-liver kidney microsomal (Anti-LKM) antibody using HEP-2 cell line immunofluorescence
* Serum total immunoglobulin G (IgG)

Radiological Investigations
* Chest X-ray
* Abdominal ultrasonography (USG)
* Hepatic Doppler ultrasound to assess hepatic venous flow
* Computed tomography (CT) angiography where indicated
* Magnetic resonance cholangiopancreatography (MRCP) to evaluate biliary tract obstruction

* Echocardiography (ECHO) when clinically indicated

Other Relevant Investigations
* Liver biopsy (performed where indicated and after obtaining informed parental consent)
* Upper gastrointestinal (UGI) endoscopy
* Genetic studies including clinical exome sequencing, in selected cases

Statistical Analysis
Data were compiled using Microsoft Excel and analyzed using Epi Info software. Categorical variables were expressed as frequencies and percentages, while continuous variables were summarized using means and standard deviations.

Results
The study included a total of 44 children diagnosed with chronic liver disease (CLD) based on the predefined inclusion criteria. A demographic overview including gender, age group, and consanguinity status is provided below [Table 1].

Table 1: Demographic Characteristics of the Study Population

Characteristic	Category	No. of Patients	Percentage
Gender	Male	26	59.0
	Female	18	40.9
Age Group (years)	1 – 5	31	70.4
	6 – 10	5	11.3
	11 – 18	8	18.1
Consanguinity	Present	16	36.3
	Absent	28	63.6

The most common symptom reported at presentation was abdominal distension, seen in 93% of patients. This was followed by jaundice (81.8%), itching (50%), and other symptoms such as anorexia, delayed milestones, lethargy, and fever [Table 2].

Table 2: Symptoms at Presentation

Symptoms	No. of Patients	Percentage
Abdominal distension	41	93.0
Jaundice	36	81.8
Itching	22	50.0
Anorexia	21	47.7
Delayed milestones	14	31.8
Clay colored stools	14	31.8
Lethargy	13	29.5
	11	25.0

The most frequently observed clinical signs were jaundice (79.5%) and hepatomegaly (77.2%). Muscle wasting, ascites, splenomegaly, and anemia were also observed in a substantial proportion of patients [Table 3].

Table 3: Signs at Presentation

Signs	No. of Patients	Percentage
Jaundice	35	79.5
Hepatomegaly	34	77.2
Muscle wasting	17	38.6

Anemia	16	36.3
Ascites	16	36.3
Splenomegaly	12	27.2
Edema	10	22.7
Short stature	4	9.0
Clubbing	3	6.8
Caput medusae	2	4.5
Palmar erythema	2	4.5

Biochemical analysis revealed that 52.2% of patients had SGOT levels between 100-400 IU/L, and 43.1% had levels <100 IU/L. SGPT levels showed a similar pattern. Serum bilirubin was elevated in all cases, and prothrombin time was prolonged in 72.7% of patients [Table 4].

Table 4: Key Biochemical Parameters

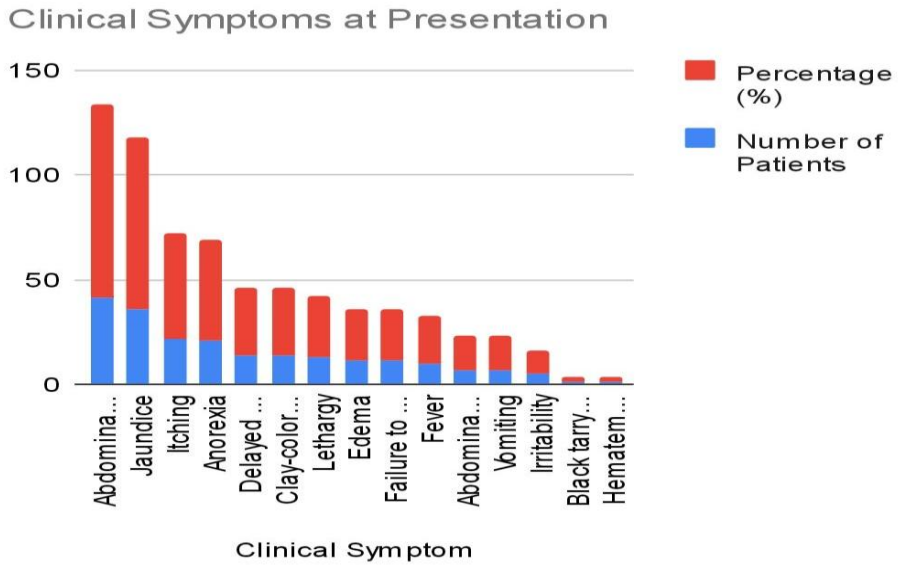
Parameter	**Values**	**No. of Patients**	Percentage
SGOT	<100	19	43.1
	100-400	23	52.2
	>400	2	4.5
SGPT	<100	23	52.2
	100-400	19	43.1
	>400	2	4.5
Serum Albumin	<2.5	9	20.4
	2.5-3.5	18	40.9
	>3.5	17	38.6
Prothrombin Time	Normal	12	27.2
	Elevated	32	72.7
Serum Bilirubin	1-2	11	25.0
	2-4	10	22.7
	4-6	3	6.8
	>6	20	45.4

Etiological evaluation was performed using clinical, biochemical, histopathological, imaging, and genetic studies. The most common etiologies identified included post-Kasai for biliary atresia (18.1%), Wilson's disease (9%), autoimmune hepatitis (6.8%), Alagille syndrome (6.8%), and several forms of glycogen storage disease [Table 5].

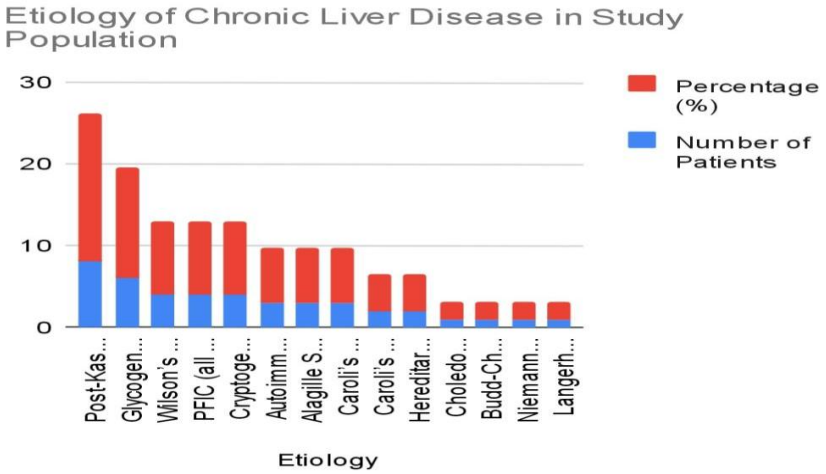
Table 5: Etiological Profile of Chronic Liver Disease

Etiology	**No. of Patients**	**Percentage**
Post-Kasai for biliary atresia (BA)	8	18.1
Wilson's disease	4	9.0
Autoimmune hepatitis	3	6.8
Alagille syndrome	3	6.8
Glycogen storage disease (types 1b, 3, 6)	5	11.3
Progressive familial intrahepatic cholestasis (PFIC 1-4)	4	9.0
Hereditary fructose intolerance	2	4.5
Caroli's disease and syndrome	5	11.3
Niemann-Pick disease	1	2.2
Choledochal cyst with PBC	1	2.2
Chronic Budd-Chiari syndrome	1	2.2
Langerhans cell histiocytosis (LCH)	1	2.2
Cryptogenic	4	9.0

Bar graph: Clinical symptoms at presentation.



Bar graph : Etiology of chronic liver disease in study population.



Discussion

This prospective observational study was conducted over two years at the Department of Pediatrics, G.B. Pant Hospital, Government Medical College Srinagar, involving 44 pediatric patients with chronic liver disease (CLD). A male predominance was noted, with 59% males and 41% females, yielding a male-to-female ratio of approximately 1.4:1. These findings align with other studies reporting male dominance, including Behera et al. [20], Hanif et al. [21], Akinbami et al. [22], Abou-Telab et al. [23], and Maheswari et al. [24]. In contrast, Dar et al. [25] reported a female predominance.

The most common symptom In this study was abdominal distension (93%), followed by jaundice (81.8%). Similar patterns were observed in Behera et al. [20], Seerat et al. [26], Dar et al. [25], and Shah et al. [27], where jaundice ranged from 70% to 84%. Hanif et al. [21] and Seerat et al. [26] also identified abdominal distension as a primary symptom in 80% and 100% of patients, respectively.

Hepatomegaly was the most frequently observed clinical sign (77%), consistent with Jena et al. \[28] (74%), Shah et al. [27] (71%), Behera et al. [20] (70%), Hanif et al. [21] (64%), and Dhole et al. [29] (63%). However, Dar et al. [25] reported a lower prevalence (22%). Splenomegaly (27.2%), ascites (36.3%), and anemia (36.3%) were commonly observed, reflecting the findings of Behera et al. [20] and Maheswari et al. [24].

Anemia was present in 37% of patients, lower than the findings of Hanif et al. \[21] and Mehnaz et al. \[30], who reported anemia in 95% and 75% of cases, respectively. Anorexia (47.7%), irritability (11.3%), and failure to gain weight (25%) were comparable to those in Maheswari et al. [24] and Shah et al. [27]. Additional signs like short stature, abdominal pain, fever, and acholic stools were less common but reported similarly by Lodhi et al. [31].

Biochemical investigations revealed hyperbilirubinemia in 90% of patients, consistent with Hanif et al. [21]. SGOT and SGPT were moderately elevated in most cases. Serum alkaline phosphatase was raised in 70% of patients, and prothrombin time was prolonged in 72.7%, indicative of hepatic dysfunction as described by Dhole et al. [29] and Maheswari et al. [24].

Upper gastrointestinal endoscopy was performed in 35 patients; 80% had esophageal varices. This is comparable to Choudhary et al. [32], who reported varices in 50% of cases. Liver biopsies were performed in 14 patients (31%), revealing biliary atresia with fibrosis, cirrhosis, and autoimmune features, findings in accordance with earlier studies [25, 29, 33].

The most frequent etiology In our cohort was biliary atresia post-Kasai procedure (18.1%), which matches Lodhi et al. \[31], Choudhary et al. [32], and Muthuphei et al. [34] (17.9%–20.8%). Glycogen storage disorders (13.6%) were also significant contributors, aligning with Lodhi et al. [31], Shah et al. [27], and Seerat et al. [26], though Abou-Telab et al. [23] reported a higher incidence (26.5%).

Progressive familial intrahepatic cholestasis (PFIC) was diagnosed in 9%, similar to Lodhi et al. [31] (13%) and Tahir et al. [35] (23.7%). Autoimmune hepatitis was found in 6.8% of cases, comparable to Dar et al. [25], Jena et al. [28], Yachha et al. [36], and Rafeey et al. [37]. Higher rates were reported by Hanif et al. [21] and Seerat et al. [26]; lower rates by Mehnaz et al. [30] and Murtaza et al. [38].

Wilson's disease was seen in 9% of our patients, aligning with Maheswari et al. [24], Seerat et al. [26], and Lodhi et al. [31]. However, markedly higher incidences were reported by Abou-Telab et al. [23], Choudhary et al. [32], Behera et al. [20], and Shehata et al. [39].

Rare causes included Alagille syndrome (6.8%), Budd-Chiari syndrome (2.2%), Niemann-Pick disease (2.2%), choledochal cyst (2.2%), Caroli's disease (6.8%), Caroli's syndrome (4.5%), and LCH (2.2%). Cryptogenic CLD was found in 9%. These distributions match other regional and international studies on pediatric CLD [20, 24, 27, 31].

Limitation

This study was single-centered and limited by the unavailability of advanced diagnostic tools for metabolic and genetic disorders, which could result in underdiagnosis.

Conclusion

The most frequent cause of CLD in this cohort was biliary atresia, often post-Kasai procedure, highlighting delays in diagnosis and surgical intervention. Other significant etiologies included progressive familial intrahepatic cholestasis (PFIC), glycogen storage disorders (GSD), Wilson's disease, autoimmune hepatitis, and rare genetic syndromes such as Alagille syndrome and Niemann-Pick disease. A proportion of patients remained cryptogenic despite extensive evaluation.

The study underlines the importance of a structured diagnostic approach incorporating clinical, biochemical, imaging, histopathological, and genetic work-up to ascertain the underlying cause of CLD. Timely referral and access to liver transplantation remain critical, particularly in progressive conditions such as biliary atresia and PFIC. Furthermore, the findings call for improved early screening for metabolic and genetic liver diseases and enhanced awareness among primary care providers to facilitate early diagnosis and management.

In conclusion, pediatric chronic liver disease presents with a broad spectrum of symptoms and etiologies. Comprehensive diagnostic strategies and multidisciplinary care are essential for optimizing outcomes. Future multicenter studies with larger sample sizes and genetic panels may provide a deeper understanding of the evolving patterns of CLD in children in resource-constrained settings.

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