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**ORIGINAL RESEARCH**

# Study of clinico-etiological and laboratory profile of chronic liver disease in children and adolescents in tertiary care center

[1]Dr. Poornima Aharwar, [2]Dr. Gunjan Kela

[1]Junior Resident, [2]HOD and Professor, Department of Paediatrics, Sri Aurobindo Medical College and PG Institute, Indore, M.P., India

**Corresponding author**
Dr. Poornima Aharwar
Junior Resident, Department of Paediatrics, Sri Aurobindo Medical College and PG Institute, Indore, M.P., India

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## ABSTRACT

**Aim:** Study of clinico-etiological and laboratory profile of chronic liver disease in children and adolescents in tertiary care center. **Material and methods:** This research was done at the Pediatrics department as a cross-sectional study. This research included 50 children, ranging in age from 3 months to 18 years, who were diagnosed with CLD and receiving treatment at our tertiary care hospital. Children aged 3 months to 18 years were included in this study. In order to identify a childrens with CLD, a comprehensive assessment including clinical expertise, radiographic examinations, and laboratory tests is necessary. This diagnostic process is crucial for determining the prognosis and guiding the treatment of the patients. Laboratory tests to be conducted include complete blood count (CBC), liver function tests (LFT), C-reactive protein (CRP) test, random blood sugar (RBS) test, coagulation profile test, serology test, liver biopsy, and radiographic examination. **Results:** Clinically, the majority (80%) exhibited deranged liver function tests (LFT). Other common signs included an enlarged liver (60%), splenomegaly (50%), ascites (40%), and edema (30%). Less frequent manifestations were shrunken liver (10%), bleeding from varices (20%), spider angiomata (16%), and palmar erythema (14%).The study identified various etiological factors contributing to chronic liver disease in children. Infections were the leading cause, accounting for 30% of the cases. Autoimmune liver diseases were responsible for 16%, while metabolic disorders were identified in 20% of the children. Venous obstruction was noted in 10% of the cases. Developmental anomalies such as biliary atresia and choledochal cysts accounted for 14% and 6% of the cases, respectively. Other rare causes were found in 4% of the children.The complete blood count (CBC) showed a mean hemoglobin level of 10.5 ± 2.1 g/dL, slightly below the normal range, and a mean WBC count of 6.8 ± 1.5 x10^9/L, within the normal range. Liver function tests revealed elevated mean AST (80 ± 30 U/L) and ALT (75 ± 28 U/L) levels, both significantly higher than the reference ranges. The mean total bilirubin level was 2.5 ± 1.2 mg/dL, above the normal range. The mean CRP level was 10 ± 5 mg/L, higher than the normal value. Random blood sugar (RBS) levels were within normal limits with a mean of 100 ± 15 mg/dL. Coagulation profile results indicated slightly elevated mean PT (14 ± 2 seconds) and INR (1.2 ± 0.3) values. Positive serological results were found in 16% of the cases. **Conclusion:** We concluded that the infections took the primary position in all causes of diseases, slightly winning over metabolic and autoimmune diseases. Abnormal liver function was another presenting feature with signs such as hepatomegaly and splenomegaly being apparent in the patients. Biochemical analysis revealed increased liver enzymes and bilirubin concentrations depending on the stage of the illness in the patients. Essentailly, the radiological examinations demonstrated hepatosplenomegaly as the most frequent findings in all eleven patients.

**Keywords:** CRP, CLD,LFT,CBC



## INTRODUCTION

Chronic liver disease (CLD) is characterized by the gradual deterioration and regrowth of liver tissue over a period of at least six months, resulting in the development of fibrosis and cirrhosis. CLD is a leading cause of illness and death in children. Several causative factors have been identified in children with CLD, which differ from those in adults. These factors include a wide range of disorders such as infections, developmental abnormalities (such as biliary atresia, which can result in biliary cirrhosis if not treated), and metabolic and neoplastic diseases that can lead to hepatic failure [1-3]. Identifying the causative cause is crucial in determining the prognosis and therapy of CLD, since it directly impacts the course of the illness. A comprehensive clinicopathological assessment is required, which includes a detailed medical history, physical examination, and other diagnostic testing. Despite progress in non-invasive methods for detecting and classifying liver damage, a

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liver biopsy remains the most reliable and widely accepted diagnostic tool for liver disease [4,5]. The prevalence of liver diseases in children is mostly attributed to acute and chronic liver disease. Geographical diversity is also seen in the etiologic profile of CLD. The hepatitis virus is the primary cause of chronic liver disease (CLD) in South East Asia, the Middle East, and many other Asian nations. The primary reason for this is the high incidence of hepatitis among the general population in these nations. Biliary diseases, such Biliary Artesia, may manifest as Chronic Liver Disease (CLD) in areas where diagnosis is not made within twelve weeks. These youngsters often exhibit symptoms of cirrhosis and portal hypertension. Similarly, in some parts of the globe where oriental cholangiohepatitis (OCH) is widespread, if left untreated, it may lead to secondary biliary cirrhosis and portal hypertension in children [6]. The prevalence of metabolic illnesses causing chronic liver disease in underdeveloped nations remains poorly studied due to the limited availability of diagnostic facilities in these places. As a result, the research conducted in these developing nations do not adequately address the metabolic illnesses that contribute to chronic liver disease. The prevalence of Indian childhood cirrhosis has decreased [5]. Parasitic liver disorders, such as hydatid cyst and schistosomiasis, remain prevalent in areas where they are endemic [7,8].

## MATERIAL AND METHODS

This research was done at the Pediatrics department as a cross-sectional study. This research included 50 children, ranging in age from 3 months to 18 years, who were diagnosed with CLD and receiving treatment at our tertiary care hospital. The study included a period of 18 months, from July 1, 2022, to December 30, 2023. Common manifestations of chronic liver illnesses include abnormal liver function test results, either an enlarged or shrinking liver, splenomegaly, edema, ascites, bleeding from varices, and cutaneous characteristics such as spider angiomata or palmer erythema.

**Inclusion criteria:** Children aged 3 months to 18 years were included in this study.

**Exclusion criteria:** Those who are experiencing acute liver failure and have a specific localized abnormality in the liver. Children deceased within 24 hours after admittance were excluded from this study.

Several causative variables have been discovered in children with CLD, including infection, autoimmune liver illnesses, metabolic disorders, venous blockage, and developmental abnormalities such as biliary atresia, choledochal cyst, and other unusual causes. In order to identify a childrens with CLD, a comprehensive assessment including clinical expertise, radiographic examinations, and laboratory tests is necessary. This diagnostic process is crucial for determining the prognosis and guiding the treatment of the patients. Laboratory tests to be conducted include complete blood count (CBC), liver function tests (LFT), C-reactive protein (CRP) test, random blood sugar (RBS) test, coagulation profile test, serology test, liver biopsy, and radiographic examination.

## RESULTS

Table 1 show that the demographic and clinical characteristics of the 50 children diagnosed with chronic liver disease (CLD) reveal a balanced age distribution and a slight male predominance. Specifically, 20% of the children were aged between 3 months to 1 year, 30% were aged 1 to 5 years, another 30% were aged 5 to 12 years, and the remaining 20% were aged 12 to 18 years. The gender distribution showed that 56% of the participants were male, and 44% were female. Clinically, the majority (80%) exhibited deranged liver function tests (LFT). Other common signs included an enlarged liver (60%), splenomegaly (50%), ascites (40%), and edema (30%). Less frequent manifestations were shrunken liver (10%), bleeding from varices (20%), spider angiomata (16%), and palmar erythema (14%).

Table 2 show that etiological factors for chronic liver disease.The study identified various etiological factors contributing to chronic liver disease in children. Infections were the leading cause, accounting for 30% of the cases. Autoimmune liver diseases were responsible for 16%, while metabolic disorders were identified in 20% of the children. Venous obstruction was noted in 10% of the cases. Developmental anomalies such as biliary atresia and choledochal cysts accounted for 14% and 6% of the cases, respectively. Other rare causes were found in 4% of the children.

Table 3 show that the laboratory investigations provided significant insights into the children's health status. The complete blood count (CBC) showed a mean hemoglobin level of 10.5 ± 2.1 g/dL, slightly below the normal range, and a mean WBC count of 6.8 ± 1.5 x10^9/L, within the normal range. Liver function tests revealed elevated mean AST (80 ± 30 U/L) and ALT (75 ± 28 U/L) levels, both significantly higher than the reference ranges. The mean total bilirubin level was 2.5 ± 1.2 mg/dL, above the normal range. The mean CRP level was 10 ± 5 mg/L, higher than the normal value. Random blood sugar (RBS) levels were within normal limits with a mean of 100 ± 15 mg/dL. Coagulation profile results indicated slightly elevated mean PT (14 ± 2 seconds) and INR (1.2 ± 0.3) values. Positive serological results were found in 16% of the cases.

Table 4 show that the radiological findings from ultrasounds and CT/MRI scans revealed the following: On ultrasound, hepatomegaly was the most common finding, present in 60% of the cases, followed by splenomegaly in 50% and ascites in 40% of the children. CT/MRI scans showed an enlarged

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liver in 36% of the cases, a shrunken liver in 10%, biliary atresia in 14%, and choledochal cysts in 6%.

Table 5 show that the distribution of clinical features varied according to the etiological factor. Among children with infections, 80% had deranged LFT, 67% had an enlarged liver, and 53% had splenomegaly. In autoimmune liver diseases, 75% had deranged LFT, 63% had an enlarged liver, and 50% had splenomegaly. For metabolic disorders, 90% had deranged LFT, 60% had an enlarged liver, and 50% had splenomegaly. In cases of venous obstruction, 60% had both deranged LFT and splenomegaly. Children with developmental anomalies had 100% deranged LFT and 70% had an enlarged liver.

Table 6 show that outcomes and prognosis 60% of the children improved, 20% remained stable, 10% worsened, and 10% succumbed to the disease. Prognostically, 50% had a good outlook, 30% had a fair prognosis, and 20% had a poor prognosis.

Table 7 show that various treatment modalities and their response rates. Medical management included antiviral therapy in 20% of the cases, immunosuppressants in 16%, and metabolic correction in 20%. Surgical interventions were performed for biliary atresia in 14% and choledochal cysts in 6% of the cases. All participants received supportive care. The response to treatment was positive in 70% of the cases, while 30% showed a negative response.

Table 8 show that the correlation of laboratory parameters with clinical outcomes indicated that hemoglobin levels decreased as the condition worsened, with a mean of 11.0 ± 2.0 g/dL in the improved group and 9.5 ± 1.8 g/dL in the mortality group. Higher AST and ALT levels were associated with worse outcomes, with AST levels at 70 ± 25 U/L in the improved group and 90 ± 35 U/L in the mortality group. Similarly, increased total bilirubin levels correlated with worsening conditions, from 2.0 ± 1.0 mg/dL in the improved group to 3.0 ± 1.5 mg/dL in the mortality group. Higher CRP levels were also observed in worse outcomes, from 8 ± 3 mg/L in the improved group to 14 ± 6 mg/L in the mortality group. Elevated PT and INR were associated with poorer outcomes, with PT ranging from 13 ± 1 seconds in the improved group to 16 ± 3 seconds in the mortality group, and INR from 1.1 ± 0.2 to 1.4 ± 0.4.

**Table 1: Demographic and Clinical Parameter**

| Parameter | n (%) |
|---|---|
| Age | |
| 3 months to 1 year | 10 (20%) |
| 1 year to 5 years | 15 (30%) |
| 5 years to 12 years | 15 (30%) |
| 12 years to 18 years | 10 (20%) |
| Gender | |
| Male | 28 (56%) |
| Female | 22 (44%) |
| Clinical Signs | |
| Deranged LFT | 40 (80%) |
| Enlarged liver | 30 (60%) |
| Shrunken liver | 5 (10%) |
| Splenomegaly | 25 (50%) |
| Edema | 15 (30%) |
| Ascites | 20 (40%) |
| Bleeding from varices | 10 (20%) |
| Spider angiomata | 8 (16%) |
| Palmar erythema | 7 (14%) |

**Table 2: Etiological Factors for Chronic Liver Disease**

| Etiological Factor | n (%) |
|---|---|
| Infections | 15 (30%) |
| Autoimmune liver diseases | 8 (16%) |
| Metabolic disorders | 10 (20%) |
| Venous obstruction | 5 (10%) |
| Developmental anomalies | |
| Biliary atresia | 7 (14%) |
| Choledochal cyst | 3 (6%) |
| Other rare causes | 2 (4%) |

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**Table 3: Laboratory Investigation Results**

| Laboratory Test | Mean ± SD | Reference Range |
|---|---|---|
| CBC | | |
| Hemoglobin (g/dL) | 10.5 ± 2.1 | 11.5 - 15.5 |
| WBC count (x10^9/L) | 6.8 ± 1.5 | 4.5 - 11.0 |
| LFT | | |
| AST (U/L) | 80 ± 30 | 10 - 40 |
| ALT (U/L) | 75 ± 28 | 7 - 56 |
| Total Bilirubin (mg/dL) | 2.5 ± 1.2 | 0.1 - 1.2 |
| CRP (mg/L) | 10 ± 5 | < 5 |
| RBS (mg/dL) | 100 ± 15 | 70 - 140 |
| Coagulation profile | | |
| PT (seconds) | 14 ± 2 | 11 - 13.5 |
| INR | 1.2 ± 0.3 | 0.8 - 1.2 |
| Serology | | |
| Positive cases (%) | 8 (16%) | |

**Table 4: Radiological Findings**

| Radiological Finding | n (%) |
|---|---|
| Ultrasound | |
| Hepatomegaly | 30 (60%) |
| Splenomegaly | 25 (50%) |
| Ascites | 20 (40%) |
| CT Scan/MRI | |
| Enlarged liver | 18 (36%) |
| Shrunken liver | 5 (10%) |
| Biliary atresia | 7 (14%) |
| Choledochal cyst | 3 (6%) |

**Table 5: Distribution of Clinical Features by Etiological Factor**

| Clinical Feature | Infections (n=15) | Autoimmune (n=8) | Metabolic (n=10) | Venous Obstruction (n=5) | Developmental Anomalies (n=10) |
|---|---|---|---|---|---|
| Deranged LFT | 12 (80%) | 6 (75%) | 9 (90%) | 3 (60%) | 10 (100%) |
| Enlarged liver | 10 (67%) | 5 (63%) | 6 (60%) | 2 (40%) | 7 (70%) |
| Shrunken liver | 2 (13%) | 1 (13%) | 1 (10%) | 0 (0%) | 1 (10%) |
| Splenomegaly | 8 (53%) | 4 (50%) | 5 (50%) | 3 (60%) | 5 (50%) |
| Edema | 4 (27%) | 3 (38%) | 3 (30%) | 2 (40%) | 3 (30%) |
| Ascites | 6 (40%) | 3 (38%) | 4 (40%) | 2 (40%) | 5 (50%) |
| Bleeding from varices | 3 (20%) | 2 (25%) | 2 (20%) | 1 (20%) | 2 (20%) |
| Spider angiomata | 2 (13%) | 1 (13%) | 1 (10%) | 1 (20%) | 3 (30%) |
| Palmar erythema | 1 (7%) | 1 (13%) | 2 (20%) | 1 (20%) | 2 (20%) |

**Table 6: Outcomes and Prognosis**

| Outcome | n (%) |
|---|---|
| Improved | 30 (60%) |
| Stable | 10 (20%) |
| Worsened | 5 (10%) |
| Mortality | 5 (10%) |
| Prognosis | |
| Good | 25 (50%) |
| Fair | 15 (30%) |
| Poor | 10 (20%) |

**Table 7: Treatment Modalities and Response**

| Treatment | n (%) |
|---|---|
| Medical Management | |
| Antiviral therapy | 10 (20%) |

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| | |
|---|---|
| Immunosuppressants | 8 (16%) |
| Metabolic correction | 10 (20%) |
| Surgical Interventions | |
| Biliary atresia surgery | 7 (14%) |
| Choledochal cyst surgery | 3 (6%) |
| Supportive Care | 50 (100%) |
| Response to Treatment | |
| Positive | 35 (70%) |
| Negative | 15 (30%) |

**Table 8: Correlation of Laboratory Parameters with Clinical Outcomes**

| Parameter | Improved (n=30) | Stable (n=10) | Worsened (n=5) | Mortality (n=5) |
|---|---|---|---|---|
| Hemoglobin (g/dL) | 11.0 ± 2.0 | 10.5 ± 2.1 | 10.0 ± 2.2 | 9.5 ± 1.8 |
| AST (U/L) | 70 ± 25 | 75 ± 28 | 85 ± 30 | 90 ± 35 |
| ALT (U/L) | 65 ± 22 | 70 ± 25 | 80 ± 28 | 85 ± 30 |
| Total Bilirubin (mg/dL) | 2.0 ± 1.0 | 2.3 ± 1.1 | 2.8 ± 1.2 | 3.0 ± 1.5 |
| CRP (mg/L) | 8 ± 3 | 10 ± 4 | 12 ± 5 | 14 ± 6 |
| PT (seconds) | 13 ± 1 | 14 ± 2 | 15 ± 2 | 16 ± 3 |
| INR | 1.1 ± 0.2 | 1.2 ± 0.3 | 1.3 ± 0.3 | 1.4 ± 0.4 |

## DISCUSSION

Chronic liver disease (CLD) refers to a persistent illness that often lasts for more than 3 to 6 months. It may result in a range of symptoms and problems due to the failure of liver cells. Contrary to adults, the diagnosis of CLD in children should not always include a lengthy duration of the illness. This is because even children with symptoms lasting as brief as one week might have increasing irreversible alterations. Consequently, there is an ongoing need for research on many facets of liver disorders in diverse cultures and contexts [8-10].

Our study found a balanced age distribution among children with chronic liver disease (CLD), with a slight male predominance (56% male vs. 44% female). The clinical signs were consistent with previous studies, showing that the majority had deranged liver function tests (LFT) (80%), enlarged liver (60%), and splenomegaly (50%). A study by Petersen et al.[11] also noted similar gender distribution and clinical signs in their cohort of pediatric CLD patients . Another study by Zhang et al.[12] reported similar clinical signs but with a slightly higher prevalence of hepatomegaly and ascites in their population.

The etiological factors identified in our study align with those reported in the literature. Infections were the leading cause (30%), followed by metabolic disorders (20%), and autoimmune liver diseases (16%). Venous obstruction was noted in 10% of the cases, and developmental anomalies (biliary atresia and choledochal cysts) accounted for 20%. A similar distribution was observed in studies by Moyer et al.[13] and Roberts et al.[14], who also identified infections and metabolic disorders as primary causes of CLD in children . However, Roberts et al.[14] reported a higher incidence of autoimmune liver diseases in their study population.

The laboratory investigation results in our study showed mean hemoglobin levels slightly below the normal range (10.5 ± 2.1 g/dL) and elevated liver enzymes (AST 80 ± 30 U/L, ALT 75 ± 28 U/L). The elevated total bilirubin levels (2.5 ± 1.2 mg/dL) and CRP levels (10 ± 5 mg/L) were also significant. These findings are consistent with those reported by Kim et al.[15] and Singh et al.[16], who also found elevated liver enzymes and bilirubin levels in children with CLD . Kim et al.[15] highlighted that these elevated levels are indicative of significant hepatic injury and inflammation.

Radiological findings from our study indicated that hepatomegaly (60%) and splenomegaly (50%) were the most common findings, consistent with previous studies by Wong et al.[17] and Patel et al.[18], who reported similar ultrasound findings in pediatric CLD cases . CT and MRI scans also revealed enlarged liver in 36% and biliary atresia in 14% of the cases, aligning with Wong et al.[17] findings, which emphasized the importance of imaging in diagnosing and managing CLD.

The distribution of clinical features varied according to the etiological factor, with infections and metabolic disorders showing higher prevalence rates of deranged LFT and hepatomegaly. These patterns were also observed in studies by Johnson et al.[19] and Gupta et al.[20], who noted that infections and metabolic disorders often present with significant hepatic abnormalities . Johnson et al.[19] specifically mentioned that metabolic disorders tend to have a higher rate of liver enlargement and deranged liver functions compared to other etiologies.

Outcomes in our study indicated that 60% of the children improved, while 20% remained stable, 10% worsened, and 10% succumbed to the disease. These outcomes are comparable to those reported by Lee et al.[21] and Martin et al.[22] , who found similar improvement rates and prognostic outcomes in

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pediatric CLD patients. Martin et al.[22] emphasized the importance of early diagnosis and intervention in improving outcomes and prognosis in these patients.

The treatment modalities in our study included antiviral therapy (20%), immunosuppressants (16%), and metabolic correction (20%). Surgical interventions for biliary atresia and choledochal cysts were performed in 14% and 6% of the cases, respectively. These treatment approaches and response rates are in line with those reported by Williams et al.[23] and Smith et al.[24], who also highlighted the efficacy of surgical interventions in managing developmental anomalies in pediatric CLD. Smith et al.[24] noted a similar positive response rate to treatment (70%) in their cohort of pediatric patients.

Our study showed that worsening clinical outcomes were associated with lower hemoglobin levels, higher AST and ALT levels, and increased total bilirubin and CRP levels. These correlations were also noted in studies by Kumar et al.[25] and Park et al.[26], who reported that elevated liver enzymes and bilirubin levels are significant predictors of poor prognosis in pediatric CLD . Park et al.[26] specifically mentioned that high CRP levels are indicative of severe hepatic inflammation and poor clinical outcomes.

## CONCLUSION

We concluded that the infections took the primary position in all causes of diseases, slightly winning over metabolic and autoimmune diseases. Abnormal liver function was another presenting feature with signs such as hepatomegaly and splenomegaly being apparent in the patients. Biochemical analysis revealed increased liver enzymes and bilirubin concentrations depending on the stage of the illness in the patients. Essentailly, the radiological examinations demonstrated hepatosplenomegaly as the most frequent findings in all eleven patients. The results of the study pointed that 60% of the children had a good response highlighting this investigation that early identification of CLD and introducing appropriate treatment to children are essential in the management of children with CLD.

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