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Original Research Article

Clinicopathological spectrum and diagnostic contribution of liver biopsy in children: a prospective observational study from a tertiary care hospital

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ABSTRACT

Background: Liver biopsy remains an important reference standard for histopathological assessment of selected pediatric liver diseases despite advances in non-invasive investigations. It provides important histopathological diagnosis, assesses disease severity, and guides management. This study evaluated the clinicopathological spectrum and diagnostic contribution of liver biopsy in children.
Methods: This prospective observational study included 25 children aged 1 month to 12 years who underwent percutaneous liver biopsy for suspected liver disease at a tertiary care teaching hospital in Gujarat, India, between September 2006 and September 2008. Demographic, clinical, laboratory, ultrasonographic, and histopathological findings were analysed using descriptive statistics. Diagnostic contribution was defined as confirmation of the suspected diagnosis, establishment of a previously unconfirmed diagnosis, or exclusion of intrinsic hepatic disease.
Results: Twenty-five children underwent liver biopsy, with a male-to-female ratio of 1.5:1. Jaundice (80%), fever (76%), pallor (64%), and hepatomegaly (100%) were the most common clinical features. Hyperbilirubinemia (80%), elevated alanine aminotransferase (72%), hypoalbuminemia (44%), and prolonged prothrombin time (12%) were the predominant laboratory abnormalities. On histopathology chronic hepatitis and its variants were present in 11 (44%) children, including cirrhosis in 2 (8%) children, followed by acute inflammatory liver disease (20%), neonatal hepatitis (8%), metabolic liver disease (8%), hemolytic disorders (8%), portal triaditis (4%), and normal liver histology (8%). Liver biopsy established or confirmed the diagnosis and provided clinically important additional information in 92% of children, including assessment of inflammatory activity and fibrosis, differentiated overlapping disorders, and excluded intrinsic hepatic disease in selected cases.
Conclusions: Liver biopsy remains an important diagnostic tool in pediatric liver disease. When interpreted alongside clinical, laboratory, and imaging findings, it contributes to diagnostic clarification and disease staging, particularly in chronic liver disease, neonatal cholestasis, and suspected metabolic liver disorders. The findings should be interpreted in the historical context of pediatric hepatology during 2006-2008.

Keywords: Liver biopsy, Child, Pediatric liver disease, Histopathology

INTRODUCTION

Pediatric liver diseases comprise a heterogeneous group of infectious, inflammatory, metabolic, cholestatic, and chronic disorders that often present with overlapping clinical and biochemical features. During 2006-2008, diagnosis relied largely on clinical evaluation, biochemical investigations, ultrasonography, serology, and, in selected cases, liver biopsy.

During the study period, liver biopsy provided direct assessment of hepatic architecture, inflammation,

fibrosis, cholestasis, and storage disorders. It was particularly useful when the clinical, laboratory, or radiological findings were inconclusive and could establish or confirm a diagnosis, assess disease severity, or exclude intrinsic hepatic pathology.[1-3]

Data on clinicopathological spectrum of pediatric liver disease and the diagnostic contribution of liver biopsy from Indian tertiary-care centers during this period were limited. Therefore, this prospective observational study was undertaken to evaluate the demographic, clinical, laboratory, ultrasonographic, and histopathological profile of children undergoing liver biopsy at a tertiary-care teaching hospital in Gujarat between September 2006 and September 2008, and to assess its diagnostic utility through clinicopathological correlation.

METHODS

This prospective observational study was conducted in the Department of Pediatrics at a tertiary care teaching and referral hospital in Gujarat, India, over a 2-year period from September 2006 to September 2008. The study included all consecutive children who underwent percutaneous liver biopsy for evaluation of liver disease during the study period.

Children aged 1 month to 12 years who underwent liver biopsy for evaluation of suspected liver disease were eligible for inclusion. Indications for liver biopsy included persistent abnormalities of liver function tests, unexplained jaundice, unexplained hepatomegaly, neonatal cholestasis, suspected chronic liver disease, and systemic disorders with hepatic involvement where histopathological confirmation was considered necessary. Children with uncorrectable coagulopathy, severe thrombocytopenia, massive ascites, hemodynamic instability, suspected vascular liver lesions, inadequate biopsy specimens or whose parents/guardians declined consent for procedure were excluded. A total of 25 children were included in the study with adequate biopsy specimens.

Clinical details were recorded in predesigned data collection form. Demographic characteristics, presenting complaints, duration of illness, anthropometric measurements, clinical examination findings, laboratory investigations, ultrasonographic findings, indications for liver biopsy, histopathological diagnosis, and procedure-related complications were recorded. Laboratory investigations included complete blood count, liver function tests (serum bilirubin, alanine aminotransferase, alkaline phosphatase, serum total protein, albumin and globulin), coagulation profile, and other relevant investigations such as viral serology and serum ceruloplasmin whenever clinically indicated. All children underwent abdominal ultrasonography before liver biopsy. Liver size, echotexture, presence of focal lesions, biliary abnormalities, splenomegaly, ascites, portal vein abnormalities, and other associated findings were documented.

Percutaneous liver biopsy was performed under aseptic precautions using a Vim–Silverman biopsy needle after administration of local anesthesia. The procedure was performed by experienced clinicians after correcting coagulation parameters. Children were monitored for immediate and delayed post-procedure complications, including pain, bleeding, hypotension, and other adverse events. Written informed consent was obtained from the parents or legal guardians before liver biopsy. Patient confidentiality was maintained throughout the study by anonymizing all collected data.

Liver biopsy specimens were fixed in 10% buffered formalin, processed using standard histopathological techniques, and examined by an experienced histopathologist. Specimens containing 11 or more complete portal tracts were considered adequate for histopathological evaluation. Routine sections were stained with hematoxylin and eosin (H and E) to assess hepatic architecture, portal and lobular inflammatory activity, hepatocellular injury, cholestasis, fibrosis, and other morphological abnormalities. Scheuer and Lefkowitch silver staining was performed for evaluation of connective tissue and fibrosis. Perls' Prussian blue staining was used to assess iron deposition and other pigments, including bile and lipofuscin. Periodic acid-Schiff (PAS) staining following diastase digestion (D-PAS) was performed to identify PAS-positive material. Copper staining was performed in children with suspected Wilson disease. Based on the histopathological findings, cases were categorized into major diagnostic groups, including chronic hepatitis and its variants, acute inflammatory liver disease, neonatal hepatitis, metabolic liver disease, hemolytic disorders, portal triaditis, and normal liver histology. In children with chronic hepatitis, the degree of inflammatory activity and fibrosis was assessed wherever applicable. The final histopathological diagnosis was correlated with the clinical presentation, laboratory investigations, and ultrasonographic findings to assess the diagnostic contribution of liver biopsy.

Statistical analysis

Data were entered into a password-protected workbook in Microsoft excel 2007 and internally validated for consistency. Statistical analysis was performed using excel 2007 (version 12.0). Data were summarized descriptively. Continuous variables were expressed as median (IQR), and categorical variables as frequency and percentage. Clinicopathological findings were compared descriptively; no inferential statistical tests were performed because of the small sample size.

RESULTS

Twenty-five children underwent liver biopsy during the study period. There was a male predominance, with 15

(60.0%) boys and 10 (40.0%) girls (male-to-female ratio 1.5:1). The age of the patients ranged from 1 month to 12 years with median age of 36 months (IQR: 24-55.2 months. Jaundice was the most common presenting symptom, observed in 20 (80.0%) children, followed by fever in 19 (76.0%) and pallor in 16 (64.0%). Splenomegaly was present in 12 (48.0%) children, while abdominal distension was noted in 9 (36.0%). Vomiting and peripheral edema were each present in 6 (24.0%) children, and bleeding manifestations occurred in 5 (20.0%). Pruritus and skin rashes were uncommon, each occurring in one child (4.0%). All children had hepatomegaly documented, with liver enlargement ranging approximately 2-7 cm below the right costal margin. A firm liver was the predominant finding (76%), while 6 (24.0%) had a soft liver. A firm liver was most frequently associated with chronic liver diseases, including chronic hepatitis with varying grades of inflammatory activity, fibrosis, and cirrhosis, whereas children with a soft liver predominantly had acute hepatitis or neonatal hepatitis (Table 1).

Laboratory evaluation demonstrated a broad spectrum of biochemical abnormalities. The median total bilirubin was 5.3 (IQR 1.9-7.7) mg/dL, with hyperbilirubinemia present in 20 (80.0%) children. The median serum ALT was 115 (IQR 36-279) IU/L, and 18 (72.0%) patients had elevated ALT levels, indicating hepatocellular injury. The median alkaline phosphatase (ALP) level was 360 (IQR 264-888) U/L; elevated values were observed in 12 (48.0%) children based on age-specific pediatric reference ranges, particularly among those with cholestatic or chronic liver disorders. The median serum albumin concentration was 3.5 (3.2-3.6) g/dL, and hypoalbuminemia was present in 11 of 25 (44%) children in whom albumin values available. Serum ceruloplasmin was measured in 11 children with suspected metabolic liver disease, with a median value of 30.6 (22.6-37.1) mg/dL; One child (9.1%) had a reduced ceruloplasmin level; this finding was interpreted in conjunction with clinical and histopathological findings (Table 2).

Although hepatomegaly was present clinically in all children, ultrasonography documented hepatomegaly in 12 (48%) children and splenomegaly in 8 (32%) children, including 4 (16%) with hepatosplenomegaly and 4 (16%) with isolated splenomegaly. Ascites was observed in 5 (20.0%) patients, pleural effusion in 4 (16.0%), and altered hepatic echotexture in 3 (12.0%), including fatty liver changes in 1 (4.0%). Five (20.0%) children had no significant abnormality on ultrasonography. Splenomegaly and ascites on ultrasonography were commonly observed in children with advanced chronic liver disease and correlated well with histological evidence of fibrosis or cirrhosis.

Liver biopsy provided clinically important diagnostic information in 23/25 (92%) children. Histopathological examination revealed a heterogeneous spectrum of liver diseases. Chronic hepatitis and its variants constituted the largest diagnostic group (11/25, 44%), including chronic hepatitis of varying grades, chronic hepatitis with portal fibrosis, chronic hepatitis with cirrhosis, chronic active hepatitis with bridging necrosis, and chronic hepatitis with cholangitis. This group also includes 2 (8.0%) children with cirrhosis. Acute inflammatory liver diseases were identified in 5 (20.0%) children, comprising acute hepatitis, acute viral hepatitis, and fulminant hepatic necrosis. Neonatal hepatitis and giant cell hepatitis accounted for 2 (8.0%) cases, while metabolic liver diseases, including glycogen storage disease and other storage disorders, were diagnosed in 2 (8.0%) children. Liver biopsy was reported as histologically normal in 2 (8.0%) children, thereby excluding intrinsic hepatic pathology. No immediate/ delayed procedure-related complications, including bleeding, hypotension, bile leak, or pneumothorax, were observed (Table 3).

Histopathology confirmed the clinically suspected diagnosis in 19 (76%) children, established a previously unconfirmed diagnosis in 2 (8%), and excluded intrinsic hepatic disease in 2 (8%). Overall, liver biopsy provided clinically relevant diagnostic information in 23 (92%) children. In 19 children (76%), histopathological examination confirmed the clinically suspected diagnosis while simultaneously characterizing disease severity by grading inflammatory activity and staging fibrosis where applicable. In two children (8%), biopsy established a previously unconfirmed diagnosis of metabolic liver disease, thereby providing diagnostic clarification relevant to disease-specific management. Conversely, two children (8%) had normal liver histology, allowing exclusion of intrinsic hepatic pathology and preventing unnecessary treatment (Table 4).

Table 1: Demographic and clinical characteristics of children undergoing liver biopsy, (n=25).

Variables	N (%)
Age groups (in years)	
<1	4 (16.0)
1–<3	4 (16.0)
3–<5	11 (44.0)
5–<10	5 (20.0)
≥10	1 (4.0)
Sex	
Male	15 (60.0)
Female	10 (40.0)

Continued.

Variables	N (%)
Presenting clinical features	
Jaundice	20 (80.0)
Fever	19 (76.0)
Pallor	16 (64.0)
Hepatomegaly	25 (100.0)
Splenomegaly	12 (48.0)
Abdominal distension	9 (36.0)
Vomiting	6 (24.0)
Edema	6 (24.0)
Bleeding manifestations	5 (20.0)
Itching	1 (4.0)
Rashes	1 (4.0)
Consistency of liver	
Firm	19 (76.0)
Soft	6 (24.0)

Table 2: Laboratory parameters, frequency of laboratory abnormalities and ultrasonographic findings in children undergoing liver biopsy, (n=25).

Laboratory parameters	Median (IQR)
Total bilirubin (mg/dl)	5.3 (1.9-7.7)
Direct bilirubin (mg/dl)	3.3 (1.5-4.3)
Indirect bilirubin (mg/dl)	1.4 (0.9-2.9)
Alanine aminotransferase (ALT, U/l)	115 (36-279)
Alkaline phosphatase (ALP, U/l)	360 (264-888)
Total protein (g/dl)	6.6 (6.4-7.0)
Serum albumin (g/dl)	3.5 (3.2-3.6)
Serum globulin (g/dl)	3.0 (3.0-3.3)
Serum ceruloplasmin (mg/dl)**	30.6 (22.6-37.1)
Laboratory abnormality, N (%)	
Hyperbilirubinemia (Total bilirubin >1.2 mg/dl)	20 (80.0)
Elevated ALT (>40 U/L)	18 (72.0)
Hypoalbuminemia (Serum albumin < 3.5 g/dl)	11 (44.0)
Hyperglobulinemia (Serum globulin >3.5 g/dl)	4 (16.0)
Prolonged prothrombin time*	3 (12.0)
Low serum ceruloplasmin† (<20 mg/dl)	1 (4.0)
Ultrasonographic finding, N (%)	
Hepatomegaly	12 (48.0)
Splenomegaly	8 (32.0)
Ascites/ free intraperitoneal fluid	5 (20.0)
Pleural effusion	4 (16.0)
Altered hepatic echotexture e. g. hepatitis/fatty liver /cirrhosis changes	3 (12.0)

**Reference limit for ALP is based on age-specific pediatric reference ranges. Prolonged PT was defined according to the institutional laboratory reference used during the study period. †Serum ceruloplasmin was measured only in children with suspected metabolic liver disease. **Patients could have more than one ultrasonographic finding; therefore, percentages do not total 100%. †Other findings included medullary sponge kidney (n=1), ectopic kidney (n=1), cholecystitis (n=1).

Table 3: Histopathological diagnosis in children undergoing liver biopsy, (n=25).

Histopathological diagnosis	N (%)
Chronic hepatitis and variants*	11 (44.0)
Acute inflammatory liver diseases†	5 (20.0)
Neonatal hepatitis‡	2 (8.0)
Metabolic liver disease§	2 (8.0)
Hemolytic disorders	2 (8.0)
Portal triaditis	1 (4.0)
Normal liver histology	2 (8.0)

*Includes chronic hepatitis with varying grades of activity, chronic hepatitis with fibrosis, chronic active hepatitis, chronic hepatitis with cholangitis, and chronic hepatitis with cirrhosis. †Includes acute viral hepatitis, acute hepatitis with fatty change, acute fulminant hepatic necrosis, and nonspecific acute hepatitis. ‡Includes neonatal hepatitis and neonatal giant cell hepatitis. §Includes glycogen storage disease and metabolic storage disease.

Table 4: Clinicopathological correlation and diagnostic utility of liver biopsy, (n=25).

Clinical suspicion	Histological diagnosis	Diagnostic contribution of liver biopsy	N (%)
Acute hepatitis	Acute viral hepatitis/ acute hepatitis/ fulminant hepatitis	Confirmed diagnosis and assessed severity	5 (20.0)
Chronic liver disease	Chronic hepatitis with or without fibrosis/cirrhosis	Confirmed diagnosis, assessed inflammatory activity and staged fibrosis; documented architectural distortion.	11 (44.0)
Neonatal cholestasis	Neonatal hepatitis/ giant cell hepatitis	Differentiated etiologies of neonatal cholestasis	2 (8.0)
Suspected metabolic liver disease	Glycogen storage disease/ metabolic storage disease	Established the diagnosis	2 (8.0)
Suspected hemolytic liver disease	Hemolytic process	Confirmed hepatic involvement	2 (8.0)
Suspected hepatic inflammatory pathology	Portal triaditis	Confirmed hepatic inflammation	1 (4)
Indeterminate liver disease	Normal liver histology	Excluded intrinsic hepatic pathology	2 (8.0)

*The 2 cases of cirrhosis are included within the 11 cases of chronic hepatitis and its variants.

DISCUSSION

The present prospective observational study evaluated the clinicopathological spectrum and diagnostic contribution of liver biopsy in 25 children undergoing evaluation for suspected liver disease at a tertiary-care teaching hospital in Gujarat between September 2006 and September 2008. The cohort demonstrated a heterogeneous spectrum of pediatric liver disease, with chronic hepatitis and its variants being the most frequent histopathological category (44%), followed by acute inflammatory liver disease (20%), neonatal hepatitis (8%), metabolic liver disease (8%), hemolytic disorders (8%), portal triaditis (4%), and normal liver histology (8%). Liver biopsy provided clinically relevant diagnostic information in 23 (92%) children through confirmation of the suspected diagnosis, establishment of a previously unconfirmed diagnosis, characterization of disease severity, or exclusion of intrinsic hepatic pathology.

There was a modest male predominance (60%), with children ranging from 1 month to 12 years. Earlier Indian biopsy series have similarly demonstrated a heterogeneous spectrum of pediatric liver disease. Ramakrishna et al reported neonatal hepatitis syndrome, storage disorders, and cirrhosis among the frequent histological diagnoses in 128 Indian children undergoing liver biopsy.[4] North Indian studies of chronic liver disease have also reported jaundice, fever, hepatomegaly, and splenomegaly as common presentations.[5,6] These findings provide useful historical comparison with the present cohort.

Jaundice, fever, pallor, and hepatomegaly were the predominant clinical manifestations. Hepatomegaly was present in all children and clinical splenomegaly in 48%. A firm liver was more frequently associated with chronic liver disease, fibrosis, cirrhosis, and metabolic disorders, whereas a soft liver was more commonly observed in acute and neonatal liver disease. However, clinical examination alone cannot reliably establish the underlying hepatic etiology.

The laboratory profile reflected the heterogeneous nature of the cohort. Hyperbilirubinemia was present in 80%, elevated ALT in 72%, and hypoalbuminemia in 44%, while prolonged PT, defined as a patient-control difference of ≥10 seconds, was present in 12%. These findings indicate hepatocellular injury and impaired synthetic function in some children but are not sufficiently specific to establish etiology. Liver biochemistry therefore required interpretation alongside clinical findings, imaging, and histopathological assessment in selected cases.[7-13]

Ultrasonography demonstrated hepatomegaly in 48% and splenomegaly in 32%, with ascites, pleural effusion, and altered hepatic echotexture occurring less frequently. The difference between clinically detected and ultrasonographically documented hepatomegaly emphasizes that physical examination and imaging provide complementary information. Ultrasonography is useful for identifying hepatosplenomegaly, ascites, biliary abnormalities, and features of portal hypertension but has limitations in defining precise histological etiology or inflammatory activity.[12-14]

Histopathology demonstrated chronic hepatitis and its variants as the largest diagnostic category (44%), including two children with cirrhosis. Acute inflammatory liver diseases accounted for 20%, while neonatal hepatitis and metabolic liver disease each accounted for 8%. This heterogeneous spectrum is consistent with previous Indian biopsy series, although the relative frequency of individual diagnoses varies between studies.[4-7] Routine H and E examination together with special stains permitted assessment of hepatic

architecture, inflammatory activity, fibrosis, cholestasis, iron and other pigments, PAS-positive material, and copper in children with suspected Wilson disease.

An important contribution of biopsy was assessment of disease severity and stage. In children with chronic hepatitis, histopathology allowed characterization of inflammatory activity and fibrosis, including identification of cirrhosis. Liver biopsy provides direct assessment of hepatic architecture and permits evaluation of inflammatory activity, fibrosis, cholestasis, and disease-specific morphological changes. The ESPGHAN Hepatology Committee recognizes liver biopsy as an important diagnostic and prognostic tool, while emphasizing that its indications have evolved with advances in non-invasive investigations.[8]

The diagnostic contribution was notable: histopathology confirmed the clinically suspected diagnosis in 19 (76%) children, established a previously unconfirmed diagnosis in two (8%), and excluded intrinsic hepatic disease in two (8%), providing clinically relevant diagnostic information in 23 (92%) children. This supports selective use of liver biopsy when clinical, biochemical, and imaging findings are insufficient to establish a diagnosis or when histological staging is required.[8,9] The two children with metabolic liver disease illustrate the potential value of histopathology when the diagnosis cannot be established readily by routine investigations.

Neonatal hepatitis and giant cell hepatitis accounted for 2 (8%) cases. In neonatal cholestasis, liver biopsy may provide useful information when the diagnosis remains uncertain after clinical, biochemical, and imaging evaluation. The joint NASPGHAN/ESPGHAN guideline recognizes liver biopsy as a useful investigation in selected diagnostic situations.[10]

No procedure-related complications were observed in this cohort. This should not be interpreted as evidence that liver biopsy is without risk; appropriate patient selection, correction of coagulation abnormalities, adequate tissue sampling, and post-procedure monitoring remain important.[8] Contemporary Indian experience has also demonstrated the role of transjugular biopsy in selected children.[11]

The role of liver biopsy has evolved substantially since this cohort was recruited. During 2006-2008, access to molecular diagnostics and non-invasive fibrosis assessment was more limited. Currently, elastography and other non-invasive techniques can provide useful information regarding hepatic fibrosis and may reduce the need for biopsy in selected children.[14,15] However, these techniques do not provide the complete range of information obtained from tissue examination, including assessment of architecture, inflammatory activity, fibrosis, cholestasis, pigment deposition, storage material, and disease-specific morphological changes. Recent literature therefore continues to support a selective role for pediatric liver biopsy.[15-17]

The strengths of this study include its prospective design, consecutive recruitment, predefined data collection, systematic clinicopathological correlation, adequate biopsy specimens, and use of routine and special histochemical stains. However, the study has limitations, including its small sample size, single-center setting, and heterogeneous cohort, which precluded meaningful inferential statistical analysis. Long-term outcomes and the effect of biopsy findings on subsequent management were not systematically evaluated. Importantly, the study was conducted during 2006-2008; therefore, its diagnostic spectrum and investigative context differ from contemporary pediatric hepatology.

Overall, liver biopsy provided substantial clinicopathological information in this selected group of children, including confirmation or clarification of diagnosis, assessment of inflammatory activity and fibrosis, identification of metabolic disorders, recognition of cirrhosis, evaluation of neonatal liver disease, and exclusion of intrinsic hepatic pathology. These findings should be interpreted in their historical context, while contemporary practice supports a more selective and individualized approach to liver biopsy with increasing availability of non-invasive and molecular diagnostics.

CONCLUSION

When integrated with clinical, biochemical, and radiological findings, liver biopsy provides valuable diagnostic and staging information and remains an important component in the evaluation of selected children with suspected liver disease, despite advances in non-invasive diagnostic techniques.

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