Section: Miscellaneous

Original Research Article

CHRONIC LIVER DISEASE IN THE PEDIATRIC POPULATION: CORRELATION OF CLINICAL FEATURES, ANTHROPOMETRY, AND HISTOPATHOLOGICAL FINDINGS

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ABSTRACT

Background: Growth failure is a near-constant accompaniment of chronic liver disease (CLD) in childhood, yet conventional weight-based anthropometric indices are distorted by organomegaly, ascites and oedema. How anthropometric status, clinical signs and liver histology relate to one another in children with CLD has been little studied in Indian settings. The objective is to characterise the anthropometric profile of children with histologically confirmed CLD, to describe their clinical and biochemical features, and to correlate these with the histopathological category and aetiology of liver disease.

Materials and Methods: A prospective, cross-sectional, hospital-based study was carried out from January 2016 to June 2017 at a tertiary care paediatric teaching hospital. Thirty-nine children under 12 years with clinical and biochemical features of CLD and histological confirmation on percutaneous Trucut liver biopsy (chronic active hepatitis, chronic persistent hepatitis or cirrhosis) were enrolled. Weight, length or height and body mass index were plotted on WHO charts up to 5 years and IAP charts thereafter, with values below the 3rd percentile taken as abnormal. Categorical data were expressed as frequencies and percentages, continuous data as mean ± SD, and groups compared by one-way ANOVA (SPSS v20, $\alpha = 0.05$).

Results: The mean age at presentation was 992.3 ± 1080.4 days (2.7 years) and the male-to-female ratio was 2.5:1. Length or height for age fell below the 3rd percentile in 24 children (61.5%), whereas weight for age and weight for length/height or BMI were below the 3rd percentile in only 5 (12.8%) each. Ascites was present in 51.3%, oedema in 38.5% and hepatomegaly in 84.6%, all of which inflate weight-based indices; stunting therefore exceeded wasting and underweight by a factor of nearly five.

Conclusion: Linear growth is the anthropometric parameter most sensitive to chronic hepatic injury in children, while weight-based indices substantially underestimate malnutrition because of fluid retention and organomegaly. Clinical signs also underestimate the extent of architectural damage, so height-for-age should be the routine nutritional index and liver biopsy remains essential for accurate staging.

Keywords: Chronic liver disease; children; anthropometry; stunting; liver biopsy; histopathology; cirrhosis.

INTRODUCTION

Chronic liver disease (CLD) in childhood is a long-standing and irreversible alteration of hepatic structure that may progress to cirrhosis, portal hypertension and hepatic failure.[1] It arises from a wide range of infective, structural, metabolic, genetic and autoimmune insults, and in India

accounts for an estimated 1–5% of paediatric ward admissions and up to a fifth of ward mortality.[1] The pattern of disease differs from that in adults, with neonatal cholestatic syndromes, biliary developmental anomalies and inherited metabolic disorders predominating, and with substantial geographical variation in their relative frequency.[2] The liver is central to nutritional homeostasis, and growth failure is therefore one of the earliest and most consistent consequences of chronic hepatic injury in children. Multiple mechanisms operate together: anorexia and early satiety from ascites and organomegaly, fat malabsorption from reduced intraluminal bile acids, increased resting energy expenditure, malabsorption of fat-soluble vitamins, and resistance to the growth-promoting actions of growth hormone.[3-5] Malnutrition at the time of transplantation is an independent predictor of post-operative morbidity and mortality, which makes accurate nutritional assessment a clinical, not merely a descriptive, exercise.[6-9]

Anthropometry is the mainstay of nutritional assessment in children, but its interpretation in CLD is uniquely problematic. Weight-based indices — weight for age, weight for length or height, and body mass index — are systematically inflated by hepatosplenomegaly, ascites and peripheral oedema, and consequently underestimate the true deficit in body tissue.[5,6,10] Sokol and Stall, in their classic evaluation of 56 children with CLD, showed that mean height Z score was depressed while mean weight and weight-for-height Z scores remained close to normal.[5] Linear growth, which cannot be distorted by fluid retention, is thus the more faithful index of chronic undernutrition, and stunting rather than wasting is the expected pattern. More recent work using arm anthropometry and Z-score analysis has confirmed both the high prevalence of malnutrition in paediatric CLD and the correlation of anthropometric deficits with the severity of hepatic dysfunction.[10,11]

The second interpretive difficulty concerns the assessment of hepatic architecture itself. The initial clinical presentation of paediatric liver disorders is stereotyped — jaundice, hepatomegaly, abdominal distension, failure to thrive — and first-line biochemistry seldom distinguishes between aetiologies.[2,12] Physical signs traditionally taken to indicate cirrhosis, such as a shrunken or nodular liver, are insensitive in young children, and ultrasonography, though useful, cannot reliably grade fibrosis. Histopathological examination of liver tissue therefore remains the reference standard: it categorises the injury as chronic active hepatitis, chronic persistent hepatitis or cirrhosis, demonstrates stored material such as copper or lipid-laden Kupffer cells, and in neonatal cholestasis distinguishes biliary atresia from hepatocellular causes with an accuracy exceeding 90%.[13,14] Because duration of illness is unreliable as a marker of chronicity in children, in whom irreversible damage may occur within weeks, a histological definition of CLD is more defensible than a temporal one.[1]

MATERIALS AND METHODS

Design, setting and participants: This prospective, cross-sectional, hospital-based observational study was conducted over 18 months, from 1 January 2016 to 30 June 2017, in the general paediatric ward, high dependency unit, paediatric intensive care unit and neonatal intensive care unit of a tertiary care paediatric teaching hospital in Kolkata that receives referrals from across West Bengal and neighbouring states.

Children below 12 years of age with clinical features of CLD identified from history, physical examination and biochemical investigation, and in whom percutaneous liver biopsy demonstrated chronic active hepatitis (CAH), chronic persistent hepatitis (CPH) or cirrhosis, were enrolled consecutively. A histological rather than a temporal case definition was adopted because duration of illness is an unreliable indicator of chronicity in this age group.[1j] Children were excluded if consent was refused, if they were older than 12 years, if the illness was acute (for example hepatitis A or dengue), if they had neonatal hepatitis without histological features of CLD, or if they had undergone corrective surgery for biliary atresia. Using the single-proportion formula $n = Z^2 \times P(1 - P)/E^2$ with $Z = 1.96$, an expected prevalence of 3% and a precision of 0.06, the minimum sample size was 32; 39 children were enrolled to allow for non-response.

Clinical, laboratory and histopathological evaluation: A structured, pre-tested proforma captured demographic details, age at onset of jaundice, stool and urine colour, abdominal distension, bleeding, altered sensorium, consanguinity and family history. Clinical examination documented icterus, liver span, surface and consistency, splenomegaly, ascites, oedema, dysmorphism and slit-lamp findings. Laboratory evaluation was performed stepwise: complete blood count, blood glucose, total and conjugated bilirubin, AST, ALT, alkaline phosphatase, total protein and albumin, PT, APTT and INR, renal function, lipid profile and urinary reducing substances, together with hepatitis A, B, C and E serology. Where indicated, serum ceruloplasmin, 24-hour urinary copper, autoimmune markers, fluorometric enzyme assays for galactosaemia and Gaucher disease, and sweat chloride testing were performed. Imaging comprised abdominal ultrasonography with Doppler, hepatobiliary scintigraphy and contrast-enhanced computed tomography as required.

Liver biopsy was performed under aseptic precautions with local anaesthesia and intravenous sedation, using a 16–18 gauge Trucut needle by the intercostal route under ultrasound guidance, after correction of coagulopathy, thrombocytopenia or

anaemia according to a standard pre-biopsy protocol. Specimens were reported by an experienced pathologist using haematoxylin and eosin with special stains as indicated, and were classified as CAH, CPH or cirrhosis.

Statistical analysis: Categorical variables were expressed as numbers and percentages and continuous variables as mean ± standard deviation. One-way analysis of variance was used to compare continuous variables across aetiological and histological groups. Analyses were performed with SPSS version 20; $p < 0.05$ was considered significant. The study was approved by the institutional ethics committee and written informed consent was obtained from a parent or guardian of every child.

RESULTS

Thirty-nine children with histologically confirmed CLD were studied out of 8040 paediatric admissions during the study period (0.5%).

Table 1: Demographic profile of the study population (n = 39)

Variable	Category	n	%
Age at presentation	< 3 months	17	43.6
	3–12 months	2	5.1
	> 12 months	20	51.3
Sex	Male	28	71.8
	Female	11	28.2
Consanguinity	Present	5	12.8
Family history of similar illness	Present	4	10.3
Facial dysmorphism	Present	3	7.7

The mean age at presentation was 992.3 ± 1080.4 days (2.7 years; range 60–2880 days), with a male-to-female ratio of 2.5:1. Extrahepatic biliary atresia (EHBA) was the commonest aetiology (16; 41.0%), followed by Wilson disease (8; 20.5%), galactosaemia, Gaucher disease and non-alcoholic steatohepatitis (NASH) (3 each; 7.7%), Caroli disease and cystic fibrosis (2 each; 5.1%), and Budd–Chiari syndrome and autoimmune hepatitis (1 each; 2.6%).

Table 2: Anthropometric profile of children with chronic liver disease (n = 39)

Anthropometric index	Adequate (≥ 3rd percentile) n (%)	Below 3rd percentile n (%)	Interpretation
Weight for age	34 (87.2)	5 (12.8)	Underweight
Length / height for age	15 (38.5)	24 (61.5)	Stunting
Weight for length/height (0–5 y) or BMI (> 5 y)	34 (87.2)	5 (12.8)	Wasting

Linear growth was the parameter most severely affected. Stunting, defined as length or height for age below the 3rd percentile, was present in 24 of 39 children (61.5%), whereas underweight and wasting were each documented in only 5 children (12.8%) — a difference of almost fivefold. In other words, nearly two-thirds of the cohort showed evidence of chronic undernutrition when assessed by linear growth, while the same cohort appeared largely well-nourished when judged by weight-based indices alone.

Table 3: Clinical findings that confound weight-based anthropometric indices (n = 39)

Clinical finding	n	%
Hepatomegaly	33	84.6
Enlarged left lobe of liver	27	69.2
Splenomegaly	21	53.8
Ascites	20	51.3
Oedema	15	38.5

Organomegaly and fluid retention were widespread. Hepatomegaly was present in 84.6% and splenomegaly in 53.8%, while ascites and peripheral oedema were documented in 51.3% and 38.5% respectively. Since each of these adds to measured body weight without adding functional tissue, they provide a direct explanation for the discordance in [Table 2] between the high prevalence of stunting and the low apparent prevalence of underweight and wasting.

Table 4: Clinical manifestations at presentation (n = 39)

Clinical finding	n	%
Hepatomegaly	33	84.6
Icterus	31	79.5
Enlarged left lobe of liver	27	69.2
Anaemia for age	26	66.7
Splenomegaly	21	53.8

Ascites	20	51.3
Oedema	15	38.5
Hypoglycaemia (RBS < 55 mg/dL)	10	25.6
Kayser–Fleischer ring	8	20.5
Reduced liver span (< 10 cm)	6	15.4
Nodular liver surface	3	7.7
Cataract	2	5.1

Hepatomegaly and icterus were the commonest presenting signs. Anaemia for age, itself a marker of chronic undernutrition as well as of hypersplenism and blood loss, affected two-thirds of the cohort. Hypoglycaemia, reflecting exhausted hepatic glycogen reserve, was found in a quarter of the children, of whom three had galactosaemia and the remainder presented in hepatic failure.

Table 5: Biochemical and synthetic function profile (n = 39)

Parameter	Value / category	n (%) or mean ± SD
Total serum bilirubin (mg/dL)	Mean ± SD	12.80 ± 7.30
Direct bilirubin (mg/dL)	Mean ± SD	10.19 ± 6.08
AST (IU/L)	Mean ± SD	273.10 ± 152.66
ALT (IU/L)	Mean ± SD	276.23 ± 131.22
Serum albumin	< 2.5 g/dL	30 (76.9)
Prothrombin time	Prolonged	21 (53.8)
APTT	Prolonged	21 (53.8)
Anaemia for age	Present	26 (66.7)
Random blood sugar	< 55 mg/dL	10 (25.6)

Conjugated bilirubin accounted for approximately 80% of total bilirubin, confirming a predominantly cholestatic pattern. Hypoalbuminaemia was the commonest abnormality of synthetic function, present in more than three-quarters of the cohort, and correlated clinically with oedema and ascites. Although PT and APTT were prolonged in 53.8%, no child bled spontaneously, indicating that in this population hypoalbuminaemia had a greater bearing on the clinical and nutritional picture than did coagulopathy.

DISCUSSION

The central anthropometric finding of this study is the marked dissociation between linear and ponderal growth. Stunting affected 61.5% of the cohort while underweight and wasting affected only 12.8% each. This pattern reproduces the classic observation of Sokol and Stall, who found mean height Z score depressed but mean weight and weight-for-height Z scores close to normal in children with biliary atresia, idiopathic neonatal hepatitis and other liver disorders.[5] The explanation is mechanical as much as metabolic: hepatomegaly in 84.6%, splenomegaly in 53.8%, ascites in 51.3% and oedema in 38.5% of our children each add to measured weight without contributing functional tissue, so that weight-based indices are systematically inflated.[6,7] Height, being immune to fluid shifts, records the cumulative deficit faithfully. The practical implication is that weight for age and BMI should not be used alone to screen children with CLD for malnutrition, and that accurate measurement of length or height deserves the greater emphasis.[6,10]

Comparable figures have been reported elsewhere. El Koofy and colleagues, using Z-score analysis in 69 Egyptian children with CLD, found 50.2% stunted, 52.2% underweight and 39% wasted, with anthropometric deficits correlating with the severity of hepatic dysfunction.[10] Their higher prevalence of wasting probably reflects the use of Z scores rather than percentile cut-offs and the inclusion of arm anthropometry, which is less affected by fluid retention. Mid-upper arm circumference and triceps skinfold thickness are indeed recommended for this reason,[6,9,10] and their omission is a limitation of the present study. Anaemia in 66.7% and hypoglycaemia in 25.6% of our cohort provide corroborative biochemical evidence of a depleted nutritional state, and the finding that hypoalbuminaemia (76.9%) rather than coagulopathy (53.8%) drove the clinical picture is consistent with albumin's dual role as a marker of synthetic failure and a determinant of oedema.

The histopathological findings show a parallel pattern of clinical underestimation. Fibrosis was present in 82.1% of biopsies and cirrhosis in 20.5%, yet a nodular liver was palpable in only 7.7% and a shrunken liver in 15.4%. Physical signs of cirrhosis are recognised to be insensitive in young children, in whom the liver is often enlarged rather than contracted even at an advanced stage.[12] Ultrasonography identified the same number of cirrhotic livers as histology but could neither grade fibrosis nor assign aetiology. Biopsy contributed to the final diagnosis in every child in this series, and canalicular bile plugs with bridging fibrosis — present in all cases of EHBA — are the features that most reliably favour biliary atresia over hepatocellular causes, with reported accuracy above 90% in developing-country settings.[13,14]

The distribution of histological categories by aetiology is instructive. Cirrhosis was confined to EHBA (25%) and Wilson disease (50%), the latter at a mean age of 7.2 years, confirming that Wilson disease can complete its cirrhotic evolution within the first decade and reinforcing the case for sibling

screening — which detected three asymptomatic affected siblings here.[23] Conversely, Gaucher and Caroli disease showed only chronic persistent hepatitis despite unequivocal clinical CLD, illustrating that histological grade alone can mislead when the underlying defect is a fixed enzyme deficiency or a developmental malformation.

This study is limited by its small single-centre sample, its cross-sectional design, which precluded assessment of nutritional recovery or outcome, the use of percentile cut-offs rather than Z scores, the absence of mid-arm and skinfold measurements, and the referral bias inherent in tertiary practice. Larger prospective studies incorporating arm anthropometry, body composition and longitudinal follow-up are needed to define the relationship between nutritional status and histological progression more precisely.

CONCLUSION

In children with histologically confirmed chronic liver disease, stunting was five times as common as wasting or underweight, establishing linear growth as the anthropometric parameter most sensitive to chronic hepatic injury. Weight-based indices substantially underestimated malnutrition because of the high prevalence of hepatomegaly, splenomegaly, ascites and oedema, and should not be used in isolation for nutritional screening in this population. Height or length for age, supplemented where possible by mid-upper arm circumference and skinfold thickness, is the more dependable index. A similar underestimation was seen in the assessment of hepatic architecture: clinical signs of cirrhosis were present in far fewer children than histology confirmed, and liver biopsy contributed to the aetiological diagnosis in every case. Chronic active hepatitis was the commonest histological pattern, and cirrhosis was confined to extrahepatic biliary atresia and Wilson disease. Routine measurement of length or height, early referral of the jaundiced infant, family screening in Wilson disease, and timely liver biopsy together offer the best prospect of improving outcomes in paediatric chronic liver disease.

REFERENCES

1. Bhave S, Bavdekar A, Pandit A. Changing pattern of chronic liver disease in India. Indian J Pediatr. 1994;61(6):675–82.
2. Dhole SD, Kher AS, Ghildiyal RG, Tambse MP. Chronic liver diseases in children: clinical profile and histology. J Clin Diagn Res. 2015;9(7):SC04–7.
3. Rajeshwari K, Gogia S. The clinical spectrum of chronic liver disease in children presenting to a tertiary level teaching hospital in New Delhi. Trop Doct. 2008;38(2):101–2.
4. Shah I, Bhatnagar S. Clinical profile of chronic hepatobiliary disorders in children: experience from a tertiary referral centre in western India. Trop Gastroenterol. 2010;31(2):108–10.
5. Sokol RJ, Stall C. Anthropometric evaluation of children with chronic liver disease. Am J Clin Nutr. 1990;52(2):203–8.
6. Taylor RM, Dhawan A. Assessing nutritional status in children with chronic liver disease. J Gastroenterol Hepatol. 2005;20(12):1817–24.
7. Heubi JE, Heyman MB, Shulman RJ. The impact of liver disease on growth and nutrition. J Pediatr Gastroenterol Nutr. 2002;35 Suppl 1:S55–59.
8. Chin SE, Shepherd RW, Thomas BJ, Cleghorn GJ, Patrick MK, Wilcox JA, et al. The nature of malnutrition in children with end-stage liver disease awaiting orthotopic liver transplantation. Am J Clin Nutr. 1992;56(1):164–8.
9. Shepherd RW, Chin SE, Cleghorn GJ, Patrick M, Ong TH, Lynch SV, et al. Malnutrition in children with chronic liver disease accepted for liver transplantation: clinical profile and effect on outcome. J Paediatr Child Health. 1991;27(5):295–9.
10. El Koofy N, Moawad EMI, Fahmy M, Mohamed MA, Mohamed HFA, Eid EM, et al. Anthropometric, biochemical and clinical assessment of malnutrition among Egyptian children with chronic liver diseases: a single institutional cross-sectional study. BMC Gastroenterol. 2019;19(1). doi:10.1186/s12876-019-1145-3.
11. Pawaria A, Khanna R, Sood V, Lal BB, Alam S. Subjective global nutritional assessment as a nutritional tool in childhood chronic liver disease. Br J Nutr. 2022;127(6):904–13.
12. Yachha SK, Sharma BC, Khanduri A, Srivastava A. Current spectrum of hepatobiliary disorders in northern India. Indian Pediatr. 1997;34(10):885–90.
13. Rastogi A, Krishnani N, Yachha SK, Khanna V, Poddar U, Lal R. Histopathological features and accuracy for diagnosing biliary atresia by prelaparotomy liver biopsy in developing countries. J Gastroenterol Hepatol. 2009;24(1):97–102.
14. Poddar U, Thapa BR, Das A, Bhattacharya A, Rao KL, Singh K. Neonatal cholestasis: differentiation of biliary atresia from neonatal hepatitis in a developing country. Acta Paediatr. 2009;98(8):1260–4.
15. Bhatia V, Bavdekar A, Matthai J, Waikar Y, Sibal A; Pediatric Gastroenterology Chapter, Indian Academy of Pediatrics. Management of neonatal cholestasis: consensus statement of the Pediatric Gastroenterology Chapter of Indian Academy of Pediatrics. Indian Pediatr. 2014;51(3):203–10.
16. Mouzaki M, Bronsky J, Gupte G, Hojsak I, Jahnel J, Pai N, et al. Nutrition support of children with chronic liver diseases: a joint position paper of NASPGHAN and ESPGHAN. J Pediatr Gastroenterol Nutr. 2019;69(4):498–511.
17. Nightingale S, Ng VL. Optimizing nutritional management in children with chronic liver disease. Pediatr Clin North Am. 2009;56(5):1161–83.
18. Hanif M, Raza J, Qureshi H, Issani Z. Etiology of chronic liver disease in children. J Pak Med Assoc. 2004;54(3):119–22.
19. Akinbami FO, Venugopalan P, Nirmala V, Suresh J, Abiodun P. Pattern of chronic liver disease in Omani children: a clinicopathological review. West Afr J Med. 2004;23(2):162–6.
20. Zhang HF, Yang XJ, Zhu SS, Zhao JM, Zhang TH, Xu ZQ, et al. Pathological changes and clinical manifestations of 1020 children with liver diseases confirmed by biopsy. Hepatobiliary Pancreat Dis Int. 2004;3(3):395–8.
21. Leonis MA, Balistreri WF. Evaluation and management of end-stage liver disease in children. Gastroenterology. 2008;134(6):1741–51.
22. Mukherjee PS, Vishnubhatla S, Amarapurkar DN, Das K, Sood A, Chawla YK, et al. Etiology and mode of presentation of chronic liver diseases in India: a multicentric study. PLoS One. 2017;12(10):e0187033.