ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 04 | April - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CLINICOPATHOLOGICAL EVALUATION OF SEVERE THIN CHILDREN AGED 5–12 YEARS UNDERGOING UPPER GASTROINTESTINAL ENDOSCOPY: A PROSPECTIVE OBSERVATIONAL STUDY FROM WESTERN INDIA

Paediatrics

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ABSTRACT

Background: Severe thinness (BMI < –3 SD) in school aged children is commonly attributed to nutritional deprivation, but underlying gastrointestinal pathology may also contribute.**Methods:** This prospective observational study evaluated 30 children (15 boys, 15 girls) aged 5–12 years with severe thinness. All participants underwent clinical assessment, laboratory evaluation, upper gastrointestinal endoscopy and duodenal biopsy.**Results:** Pallor, hepatomegaly and dermatological changes were frequent clinical findings. Endoscopy revealed normal mucosa in 16 children, scalloping in 6, gastritis in 3, duodenal nodularity in 3 and peptic ulcer in 1. Histopathology demonstrated normal mucosa in 16 children, mild duodenitis in 9, moderate duodenitis in 2, focal villous atrophy in 1, partial villous atrophy in 1 and giardiasis in 1. **Conclusion:** Severe thinness in school aged children may be associated with many gastrointestinal pathological conditions, some of which may contribute to malnutrition. A structured clinicopathological approach helps identify gastrointestinal conditions including duodenal inflammation and celiac disease.

KEYWORDS

Severe thinness; upper GI endoscopy; duodenal biopsy; celiac diseas

INTRODUCTION

Severe thinness is defined, in children 5-12 years of age, as BMI less than –3 standard deviations according to WHO growth standards. Although inadequate nutritional intake is frequently implicated, persistent growth failure may also reflect underlying gastrointestinal disease. Celiac disease, inflammatory mucosal disorders and parasitic infections may impair nutrient absorption and contribute to growth failure.

Despite the high burden of pediatric under nutrition in India, limited data is available describing the clinicopathological spectrum of severe thin children undergoing gastrointestinal evaluation.

AIM

The present study was undertaken to evaluate the clinical profile, laboratory parameters, endoscopic findings and duodenal histopathology of severe thin children presenting to a tertiary care center in Western India.

MATERIALS AND METHODS

This prospective observational study was conducted on 30 children aged 5–12 years with BMI < –3 SD in the Pediatric Nutrition OPD of a tertiary care centre in Western India. Children with a known chronic illness and those on a gluten-free diet were excluded. All enrolled children underwent detailed clinical evaluation, hematological testing, liver enzyme estimation, celiac serology, upper gastrointestinal endoscopy and gastric and/ or duodenal biopsies. Data were analyzed using the SPSS V 15.0 package.

The study protocol was approved by the Institutional Ethics Committee and written informed consent was obtained from parents or guardians, along with a written informed assent from children above 7 years of age.

RESULTS

Age

A total of 30 children (15 boys and 15 girls), aged 5 - 12 years (mean age 7.4 years), with severe thinness, were included in the study. All participants underwent detailed clinical, laboratory, endoscopic and histopathological evaluation.

Birth weight

80% had a birth weight of more than 2.5 kg and the remaining 20% had a low birth weight, suggesting that malnutrition doesn't always start in the neonatal period and social, dietary and organic factors need to be addressed to determine the cause of severe thinness.

Clinical and laboratory findings

Clinical examination revealed pallor in 16 (53.3%) children, of which 12 had mild pallor, 3 had moderate and 1 had severe pallor. Of the 14 children who had no clinical pallor, 9 showed some degree of anemia on laboratory evaluation, suggesting that mild degree of anemia may not be always evident clinically. The distribution of anemia is given in Table 2. Skin changes suggestive of micronutrient deficiencies were seen in 10 (33.3%) children, including dry scaly skin, ichthyosis, knuckle hyper pigmentation, dermatitis and itchy skin rash. 10 children were found to have oral mucosal abnormalities - 5 had angular cheilitis, 4 had aphthous ulcers and 1 had a magenta red tongue. A notable observation in this cohort was the high prevalence (43.3%) of hepatomegaly (13 children), and an even greater prevalence (70%) of elevated transaminases than more than 2 times the upper limit of normal (SGOT in 21 and SGPT in 19 children).

Table 1: Clinical features

Clinical sign	Number	Percentage
Pallor	16	53.3%
Hepatomegaly	13	43.3%
Skin changes	10	33.3%
Oral lesions	10	33.3%
Edema	7	23.3%
Cervical lymphadenopathy	4	13.3%

Table 2: Severity of Anemia

Severity (Hb in g/dL)	Number	Percentage
No anemia (>11 g/dl)	5	16.7%
Mild (10–11 g/dL)	7	23.3%
Moderate (7–10 g/dL)	11	36.7%
Severe (5–7 g/dL)	5	16.7%
Very severe (<5 g/dL)	2	6.7%

Table 3: Elevated Transaminases

Transaminases (>2× ULN)	Number	Percentage
SGOT	21	70%
SGPT	19	63.3%

Upper GI Endoscopy

Upper GI endoscopy was normal in 16 out of the 30 patients, with 14 children demonstrating the following gross endoscopic abnormalities - scalloping of duodenal folds in 6 (20% of cases), duodenal nodularity in 4 (13.3%), inflammatory changes in the gastric mucosa in 3 and peptic ulcer in 1 child.

Table 4: Gross Upper GI Endoscopic Findings

Finding	Number	Percentage
Normal	16	53.3%
Duodenal scalloping	6	20.0%

Duodenal nodularity	4	13.3%
Gastritis	3	10.0%
Peptic ulcer	1	3.3%

Duodenal biopsy

Inflammatory duodenal changes on histopathological examination were observed in a considerable proportion of children in the present study, with mild and moderate duodenitis being the most frequent abnormal finding, seen in 9 (30%) and 2 (6.7%) patients respectively. Celiac disease represents an important but often under-recognized cause of growth failure in children. In the present study, two children, who also had positive anti-tissue transglutaminase antibodies (anti tTG-IgA), demonstrated focal and partial villous atrophy in one each, consistent with Marsh grade 3A and 3b changes respectively on histopathology, confirming the diagnosis of celiac disease.

Table 5: Duodenal biopsy findings

Finding	Number	Percentage
Normal mucosa	16	53.3%
Mild duodenitis	9	30.0%
Moderate duodenitis	2	6.7%
Focal villous atrophy	1	3.3%
Partial villous atrophy	1	3.3%
Giardiasis	1	3.3%

DISCUSSION

The present study highlights that severe thinness in school aged children may represent the clinical presentation of, or co-exist with, an underlying gastrointestinal pathology rather than being a purely nutritional disorder. In our clinical experience at a tertiary pediatric gastroenterology referral center, children presenting with severe thinness often demonstrate heterogeneous gastrointestinal pathology beyond simple nutritional deficiency.

Hepatic involvement is well recognized in severe malnutrition, particularly in protein-energy malnutrition. Fatty infiltration of the liver and metabolic disturbances may result in hepatomegaly in affected children. Alihonou et al. evaluated malnourished children using clinical and radiological assessment and reported that hepatic steatosis and hepatomegaly were frequently observed, highlighting fatty infiltration of the liver as a common hepatic manifestation of severe malnutrition.[1] Hypertransaminasemia has been described in association with malnutrition as well as gluten-sensitive enteropathy. Karajibani et al. reported significantly elevated serum transaminase levels in malnourished hospitalized children compared with well-nourished controls, suggesting that nutritional deficiency may contribute to hepatocellular injury and biochemical evidence of hepatic dysfunction.[2] Vajro et al. reported that approximately one-third of children with newly diagnosed celiac disease demonstrate elevated transaminase levels, which often normalize following initiation of a gluten-free diet.[3] Our findings of hepatomegaly and elevated transaminases in many from this cohort of severe thin children, of which two were diagnosed as celiac disease, align with these observations. These observations may represent hepatic involvement secondary to metabolic disturbances, fatty infiltration, micronutrient deficiencies, and increased intestinal permeability or immune-mediated mechanisms.

The duodenal abnormalities like scalloping and nodularities observed in our study are known to be associated with malabsorptive disorders, duodenal inflammation and parasitic infestations. Gastric findings suggestive of dyspepsia and peptic ulcer have also been reported by other authors, apart from similar duodenal findings, in association with pediatric malnutrition. Arslan et al. reported that children undergoing endoscopic evaluation for persistent growth retardation frequently demonstrate abnormal findings, with pangastritis and duodenitis being among the most common endoscopic abnormalities.[4]

The histopathological findings of duodenitis and villous atrophy in our cohort suggest that mucosal inflammatory processes may contribute to impaired nutrient absorption in a subset of children presenting with severe thinness. Another theory cited to explain such findings is the concept of environmental enteropathy or environmental enteric dysfunction due to chronic feco-oral pathogen exposure. Keusch et al highlighted that malnutrition may be associated with inflammatory and structural changes in the intestinal mucosa, contributing to impaired nutrient absorption.[5] Although the number of cases was small, the diagnosis of celiac disease in 2 of the 30 (6.66%) severe thin children in our study underscores the importance of screening for celiac disease in children presenting with persistent severe thinness and unexplained growth failure. Similar observations have been reported in pediatric studies evaluating children with malnutrition and growth failure. Barbezat et al., in their biopsy-based study of children with protein-calorie malnutrition, demonstrated structural abnormalities of the small intestinal mucosa including varying degrees of villous atrophy and mucosal damage.[6] Poddar and Yachha have reported that celiac disease in Indian children frequently presents with features such as failure to thrive, anemia and undernutrition, highlighting that gluten-sensitive enteropathy should be considered in children presenting with unexplained growth failure.[7]

The endoscopic and histopathological findings of the present study are consistent with these observations, further supporting the role of targeted gastrointestinal evaluation in children presenting with severe thinness and unexplained growth failure.

CONCLUSION

Severe thinness in school-aged children represents a multifactorial clinical entity rather than a purely nutritional disorder. Our study highlights the presence of inflammatory duodenal pathology, including celiac disease, elevated transaminases and occasional parasitic infection in this population. A structured clinicopathological evaluation, including endoscopy and duodenal biopsy in selected cases, may facilitate early identification of treatable gastrointestinal causes of growth failure and guide targeted management.

LIMITATIONS

The primary limitations of this study include the relatively small sample size and the single-center design. However, the study provides valuable preliminary insights into the endoscopic and histopathological spectrum of severe thin children undergoing gastrointestinal evaluation.

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