

ORIGINAL ARTICLE

Nutrition

Liver function tests among children with moderate acute malnutrition: A secondary analysis of a randomized trial from Burkina Faso

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Abstract

Objectives: Hepatic steatosis has been described in children with severe acute malnutrition, but the degree of liver damage in children with moderate acute malnutrition (MAM) is unknown. This study aimed to investigate the development of malnutrition-associated liver damage by describing liver function tests among children with MAM.

Methods: This study was a randomized 2 × 2 × 3 factorial trial, Treatfood, conducted in Burkina Faso. Children (6–23 months) with MAM received either a lipid-based nutrient supplement or a corn-soy blend, containing either dehulled soy or soy isolate and different quantities of dried skim milk. Malaria rapid diagnostic tests and liver function tests were performed at inclusion and after 3 months of supplementation and compared to Danish reference intervals. Associations were analyzed with linear regressions or tobit regressions, and odds ratios were calculated with logistic regressions, all age- and sex-adjusted.

Results: In total, 1405 children had one or more liver function tests measured at baseline. The median age was 11.4 months [interquartile range: 8.2–16.1]. Few children had elevated concentrations of liver function tests at baseline. Concentrations of total bilirubin were 19% (95% confidence interval: 2–40) greater among children with length-for-age Z-scores <−3 compared with children with Z-scores ≥−2. The concentrations of total bilirubin and alkaline phosphatase were negatively associated with age. Minor changes in the serum concentrations of liver function tests were observed after intervention.

Conclusions: Liver function tests were largely within the normal range, whereby no signs of manifest liver damage were found. Children with stunted growth had higher bilirubin, which might indicate subclinical liver damage.

Trial Registration: ISRCTN number: ISRCTN42569496

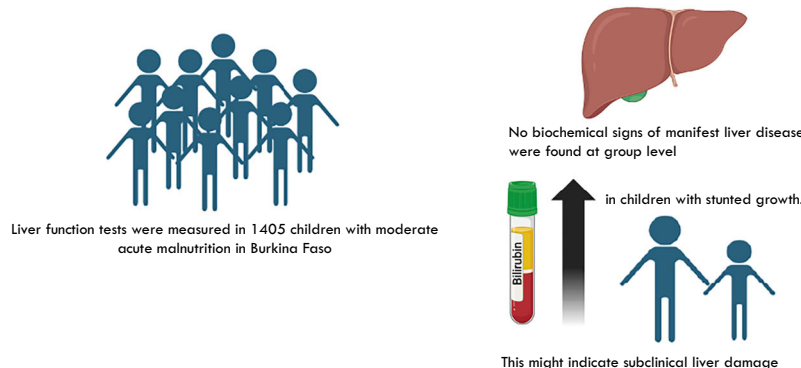
Christina L. Winther and Thora W. Helt shared first-authorship.

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Are liver function tests elevated among children with moderate acute malnutrition?



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KEYWORDS

liver parameters, middle- and low-income countries, pediatric, wasting

1 | INTRODUCTION

In 2022, the WHO estimated a prevalence of 45 million children under the age of 5 suffering from wasting, of whom 31 million suffered from moderate acute malnutrition (MAM).¹ Malnutrition is a global health burden that poses medical, developmental, social, and economic risks to both individuals and society.² Malnutrition is associated with an increased risk of infections, and it is estimated to be linked to nearly 50% of deaths in children under the age of 5. Most of these live in low- and middle-income countries.² Hepatomegaly and hepatic steatosis have been described in children with severe acute malnutrition (SAM).³ Several mechanisms have been suggested as possible explanations for the development of steatosis in children with SAM. These include a reduced ability to break down lipids due to mitochondrial and peroxisomal dysfunction, choline deficiency which indirectly leads to impaired hepatic synthesis and export of very-low-density-lipoproteins, as well as L-carnitine depletion, the amino acid transporting fatty acids across the inner mitochondrial membrane.^{3–5} In addition, high serum concentrations of circulating ferritin, proliferation of ethanol-producing bacteria in the gut microbiota, and exposure to aflatoxins have been described in children with edematous SAM, all of which could lead to toxic damage to the hepatocytes.⁴ Nevertheless, the pathogenesis of malnutrition-associated liver damage is poorly understood, and the degree of liver damage in children with MAM is unknown. Therefore, this study aimed to investigate the early development of malnutrition-associated liver damage by describing parameters of liver function among children with MAM.

What is Known

- Hepatic steatosis has been described in children with edematous severe acute malnutrition.
- The pathogenesis of malnutrition-associated liver damage is poorly understood, and the degree of liver damage in children with moderate acute malnutrition (MAM) is unknown.

What is New

- Serum concentrations of liver function tests were largely within the normal range, whereby no biochemical signs of manifest liver damage were found at the group level.
- Serum concentrations of total bilirubin were higher among children with stunted growth, which might indicate subclinical liver damage.
- Future research studies could include imaging techniques in children with both stunting and MAM to further investigate the development of malnutrition-associated liver damage.

2 | METHODS

2.1 | Ethics statement

The study was approved by the Ethics Committee for Health Research in Burkina Faso (2012-8-059), and consultative approval was obtained from the Danish National Committee on Biomedical Research Ethics (1208204). The trial was registered in the ISRCTN

registry (ISRCTN42569496). Information and consent forms were translated into the local language (Moré) and back-translated to ensure accuracy. Caregivers gave verbal and written consent (signature or fingerprint) before enrollment.

2.2 | Study design and population

This study was part of Treatfood, a randomized $2 \times 2 \times 3$ factorial trial (ISRCTN42569496). It was conducted between September 9, 2013 and August 29, 2014 at five sites in the Province du Passoré, Burkina Faso, to assess the effectiveness of food supplements on fat-free mass.⁶ Children aged 6–23 months were included if they resided near a study site at the time of inclusion and suffered from MAM. Children with SAM, recent or in current need of hospitalization, participation in other nutritional programs, suspected food allergies, or severe disabilities were excluded. MAM was defined as a weight-for-length Z-score (WLZ) ≥ -3 and < -2 from the median of the WHO child growth standards or as a mid-upper arm circumference (MUAC) ≥ 115 and < 125 mm. The children received 500 kcal per day as a lipid-based nutrient supplement or corn-soy blend, containing either dehulled soy or soy isolate, and different quantities of dried skim milk (0%, 20%, or 50% of total protein) for 12 weeks. Nutritional recovery was defined as WLZ > -2 and MUAC > 125 mm. The trial was double-blind regarding the quality of the soy and quantity of milk, although not the matrix (lipid-based nutrient supplement compared with corn-soy blend). Additional information on study characteristics was previously published.⁶

2.3 | Data collection

At inclusion and after 3 months of supplementation, age, sex, and anthropometry data were recorded. In addition, a history of diet and previous illnesses based on a 14-day maternal recall was taken, and a physical examination was carried out. MUAC was measured to the nearest 1 mm at the midpoint between the acromion process and olecranon, using a standard measuring tape. Weight was measured with an electronic scale (Seca model 881 1021659, Seca GmbH & Co. KG) to the nearest 100 g. Length was measured with a wooden height board to the nearest 1 mm. All measurements were performed twice by trained personnel, and the average was recorded.

Trained study nurses collected 2.5 mL of venous blood from the arms at each visit, of which 1 drop was used for malaria rapid diagnostic tests for *Plasmodium falciparum* (SD Bioline Malaria Ag P.f., Standard Diagnostics Inc.). The blood samples were collected with serum vacutainers (Becton, Dickinson and Company

reference #368492) and stored in a cold box at 2–8°C during transportation to the trial laboratory. The samples were centrifuged at 700g for 5 min at room temperature (EBA 20S, Andreas Hettich GmbH & Co. KG) to isolate serum, and serum was stored at –20°C. For the analysis of serum concentrations of alanine transaminase (ALT), albumin, alkaline phosphatase (ALP), amylase, total bilirubin, γ -glutamyl transferase (GGT), and phosphate, serum was sent to the Department of Clinical Biochemistry, University of Copenhagen–Rigshospitalet, Copenhagen, Denmark. These parameters were determined on a Cobas 8000 analyzer system, according to the manufacturer's instructions (Roche Diagnostics GmbH, Mannheim, Germany). The analyses of C-reactive protein (CRP) and α_1 -acid glycoprotein (AGP) were carried out by VitMin Lab, Willstaett, Germany, using a simple sandwich enzyme-linked immunosorbent assay.⁷ Danish laboratory reference intervals were presented along with blood test results, which were compared with these reference values.⁸

2.4 | Statistics

Data were entered twice into EpiData Entry 3.1 (EpiData Association). Statistical analyses were performed using STATA 18 (StataCorp). Children with missing values were excluded from the analyses. Distributions of variables were assessed visually with histograms and probability plots. Variables were described as percentages (n), means (\pm SD) for normally distributed variables, or medians [interquartile range (IQR)] for nonnormally distributed variables. Associations were analyzed by either linear regressions or Tobit regressions, both age- and sex-adjusted, with \log_{10} transformation and backtransformation of the outcomes. Tobit regressions were carried out to take left-censoring into account by using lower limit censored points at 5 U/L for ALT, 2.5 μ mol/L for bilirubin, and 3 U/L for GGT. If data were not censored, differences between visits were examined with paired t tests, and 95% CIs were calculated with bias-corrected bootstrapping (10,000 rep). If data were censored, differences between visits were analyzed with Tobit random-effects models adjusted for site as a fixed effect and ID as a random effect. Odds ratios were calculated with both unadjusted and age- and sex-adjusted logistic regressions.

3 | RESULTS

Of the 1609 children enrolled, 87.2% (1403) had one or more liver function tests at baseline. A participant flow chart can be found in the supplementary materials (Figure S1). Of these, 54% were females, and the

TABLE 1 Baseline characteristics of 1403 children aged 6–23 months with moderate acute malnutrition who had one or more liver parameters measured at baseline.

	<i>n</i>	
Age (months)	1403	11.4 [8.2–16.1]
Female sex	1403	54% (759)
Breastfed currently	1401	95% (1324)
Anthropometry		
Length-for-age (Z-score)	1403	−1.7 (1.1)
$Z \geq -2$		62% (870)
$Z < -2$ and ≥ -3		28% (391)
$Z < -3$		10% (142)
Weight-for-age (Z-score)	1403	−2.5 (0.6)
Weight-for-length (Z-score)	1403	−2.2 (0.5)
Mid-upper arm circumference (mm)	1403	123 [120–124]
Illness		
Ill in the last 2 weeks ^a	1403	76% (1070)
Diarrhea ^a	1403	20% (281)
Cough ^a	1401	29% (407)
Fever ($\geq 37.5^\circ\text{C}$) ^b	1401	17% (244)
Malaria rapid diagnostic test (positive)	1398	40% (564)
Serum concentrations		
C-reactive protein (mg/L)	1402	2.4 [0.8–9.3]
α_1 -acid glycoprotein (g/L)	1402	1.22 [0.88–1.63]

Note: Values are presented as % (*n*), mean (\pm SD), or median [interquartile range].

Abbreviation: SD, standard deviation.

^aBased on maternal recall and physical examination at inclusion by a trained study nurse.

^bBased on physical examination at inclusion by a trained study nurse.

median [IQR] age was 11.4 months [8.2–16.1] (Table 1). The 206 children with no data on liver function tests had a higher mean (\pm SD) hemoglobin (10.2 ± 1.7 g/L vs. 10.0 ± 1.6 g/L, $p = 0.047$) than those with liver function tests measured at baseline. No other differences in baseline characteristics were found between the two groups ($p > 0.05$, data not shown).

3.1 | Liver parameters at baseline

At baseline, serum concentrations of ALT, ALP, amylase, total bilirubin, and GGT were largely within the normal range, with only 0.9%, 0.6%, 12%, 0.3%, and 3% of the children having elevated values, respectively. Moreover, serum phosphate was low in 4% of children while concentrations of serum albumin were elevated

and low in 52% and 13% of the children, respectively (Table 2). When adjusted for age and sex, total bilirubin was 19% (95% CI: 2–40) greater among children with length-for-age Z-score < -3 when compared to children with Z-scores ≥ -2 . In addition, age between 15 and 23 months was associated with 14% (95% CI: 17–11) lower ALP and 14% (95% CI: 22–5) lower total bilirubin compared with age between 6 and 14 months. Compared with nonbreastfed children, breastfed children had 16% (95% CI: 8–25) higher ALP. No associations were found between WLZ, MUAC, weight-for-age Z-scores, or sex and serum concentrations of ALT, ALP, GGT, or total bilirubin (Table S1, unadjusted results in Table S2).

Total serum bilirubin concentrations were greater among children with elevated concentrations of CRP and AGP as well as those with recent illness, fever, or a positive malaria test. Moreover, serum ALP was lower in children with elevated concentrations of CRP and AGP as well as in children with fever or a positive malaria test. Finally, serum GGT was higher among children with elevated concentrations of CRP and AGP (Table S3, unadjusted results in Table S4).

3.2 | Changes in liver parameters between baseline and after intervention

After 3 months of intervention, we observed increases in the concentrations of serum ALT, amylase, ALP, and albumin as well as decreases in the serum concentrations of total bilirubin and GGT. All changes were minor. We found no changes in serum phosphate concentrations (Table 3).

3.3 | Baseline liver parameters as predictors of nutritional recovery

Lower baseline serum phosphate concentrations were associated with lower odds of nutritional recovery with an age- and sex-adjusted odds ratio of 0.31 (95% CI: 0.31–0.84). We found no associations between nutritional outcome and the baseline serum concentrations of ALT, albumin, ALP, amylase, total bilirubin, or GGT (Table 4).

4 | DISCUSSION

Few children had elevated serum concentrations of ALT, ALP, amylase, bilirubin and GGT, thereby no biochemical signs of manifest liver damage were found at group level. These results contrast with those of studies on children with SAM where the serum concentrations of ALT, AST, total bilirubin and serum bile acids were elevated while concentrations of albumin and amylase were low.^{5,9,10} We found serum

TABLE 2 Liver tests in 1403 children aged 6–23 months with moderate acute malnutrition who had one or more liver parameters measured in serum at baseline.

	<i>n</i>	Median [IQR]	% (<i>n</i>) with high values	% (<i>n</i>) with low values	Applied reference intervals	
Alanine transaminase (U/L)	908	6.2 [<5–8.9]	0.9% (8)		0–18 years of age	5–45
Albumin (g/L)	1402	38.2 [34.9–41.2]	52% (733)	13% (185)	0–1 years of age 1–4 years of age	26–34 34–42
Alkaline phosphatase (U/L)	1403	169.9 [139.7–205.4]	0.6% (8)		6–24 months of age 2–6 years of age	120–470 120–290
Amylase (U/L)	1190	5.3 [<3–10.4]	12% (148)		0–1 years of age 1–4 years of age	0–8 0–31
Bilirubin (μmol/L)	1400	<2.5 [<2.5–3.5]	0.3% (4)		0–18 years of age	5–25
γ-glutamyl transferase (U/L)	1385	13 [9–17]	3% (47)		0–18 years of age	10–45
Phosphate (mmol/L)	1397	1.73 [1.56–1.87]	0.4% (6)	4% (52)	2 days to 1 year of age 1–5 years of age	1.36–2.26 1.16–1.81

Abbreviation: IQR, interquartile range.

TABLE 3 Liver tests in 1403 children with moderate acute malnutrition after 3 months of intervention.

Liver test	<i>n</i>	3-month follow-up		Change from baseline to 3-month follow-up	
		Median [IQR]	<i>n</i>	Change (95% CI)	<i>p</i>
Alanine transaminase (U/L)	823	7.1 [<5–9.6]	767	12% (6–19)	<0.001
Albumin (g/L)	1375	39 [36.3–41.3]	1223	0.8 g/L (0.5–1.1)	<0.001
Amylase (U/L)	1349	7.5 [4.1–12.6]	1050	29% (24–33)	<0.001
Alkaline phosphatase (U/L)	1379	180.6 [152.2–214.2]	1227	9.7 U/L (5.0–15.4)	<0.001
Bilirubin (μmol/L)	1378	<2.5 [<2.5–2.8]	1223	–20% (–24 to –16)	<0.001
γ-glutamyl transferase (U/L)	1368	12 [9–16]	1201	–4% (–8 to –1)	0.016
Phosphate (mmol/L)	1375	1.80 [1.67–1.93]	1218	0.2 mmol/L (–0.1 to 0.5)	0.066

Abbreviations: CI, confidence interval; IQR, interquartile range.

TABLE 4 Baseline liver tests as predictors of nutritional recovery during supplementation.^a

Liver test	<i>n</i>	Unadjusted		Age- and sex-adjusted	
		Odds ratio (95% CI)	<i>p</i>	Odds ratio (95% CI)	<i>p</i>
Alanine transaminase (U/L)	893	1.00 (0.99–1.01)	0.96	1.00 (0.99–1.01)	0.98
Albumin (g/L)	1354	0.98 (0.96–1.00)	0.081	0.98 (0.96–1.01)	0.20
Amylase (U/L)	1154	1.00 (0.99–1.01)	0.75	1.00 (0.99–1.01)	0.86
Alkaline phosphatase (U/L)	1355	1.00 (1.00–1.00)	0.35	1.00 (1.00–1.00)	0.68
Bilirubin (μmol/L)	1352	1.03 (0.99–1.08)	0.13	1.04 (0.99–1.08)	0.098
γ-glutamyl transferase (U/L)	1338	1.01 (1.00–1.02)	0.030	1.01 (1.00–1.02)	0.027
Phosphate (mmol/L)	1349	0.48 (0.30–0.76)	0.003	0.51 (0.31–0.84)	0.009

Note: Data are presented as changes in odds of recovery per unit change in predictor value.

Abbreviation: CI, confidence interval.

^aHad mild or no acute malnutrition after 3 months of intervention.

concentrations of total bilirubin to be higher among children with stunted growth, which might indicate subclinical liver damage. These associations were found in serum levels largely within the normal range and would need to be validated in future studies. Future studies could investigate this subgroup of children with both MAM and stunting further through noninvasive measurements of hepatic steatosis and fibrosis with transient elastography rather than measuring biomarkers of liver damage with often low availability in the Global South.

As previously reported, serum concentrations of AGP and CRP were slightly elevated at baseline, which was only partially explained by clinical infections, suggesting subclinical infection.¹¹ Total bilirubin was positively associated with all markers of inflammation. This could be due to increased red blood cell hemolysis secondary to malaria infection or a decreased excretory function of the liver due to underlying liver damage. In addition, we found ALP to be negatively associated with markers of inflammation. Total serum ALP is divided into four isoenzymes, but tissue non-specific ALP from the liver, kidneys and bones makes up the largest fraction of circulating total serum ALP.¹² Low concentrations of ALP can be secondary to malnutrition as well as conditions causing decreased bone formation,¹³ and our findings could thereby indicate suppressed bone formation among acutely malnourished children with concurrent infections.

Overall, the liver parameters remained within the normal range, and changes between the two visits were small. We found serum ALP concentrations to increase during intervention, indicating increased bone formation in response to improved nutritional status.¹⁴ Moreover, the increase in serum albumin concentrations could indicate clearance of previous infections, as albumin is a negative acute-phase protein.^{15,16} A study from Ghana, a neighboring country of Burkina Faso, found higher pediatric reference values for albumin¹⁷ when compared to the Danish reference values. When applying these reference values, we found 30.5% of children in our cohort to have hypoalbuminemia at baseline, but only 21.0% after nutritional intervention. Meanwhile, only 0.2% and 0% of children had hyperalbuminemia at baseline and after intervention, respectively. This indicates that pediatric reference values of serum albumin might differ between Danish and West African populations. Serum concentrations of amylase and ALT increased during the intervention period while total bilirubin and GGT decreased. The increase in ALT is surprising, as a previous study on children with SAM reported that the serum concentrations of ALT decreased after nutritional intervention.¹⁸ Another study on young adolescents with anorexia nervosa reported mild to moderate increases in serum concentrations of ALT during refeeding. Moreover, elevated ALT concentrations during refeeding were associated with a delay in

the start of weight gain when compared with patients with ALT concentrations within the normal range.¹⁹ As the authors suggested, this could be caused by metabolic damage and hepatic vulnerability due to nutritional deficiencies. This is also a possible explanation for the increase in ALT observed in this study. Still, as 34% of ALT samples at baseline and 26% of ALT samples after intervention were subjected to left-censoring, the results of this study need to be validated in future studies. Finally, serum concentrations of phosphate were within the normal range at both visits; thus, no signs of re-feeding syndrome²⁰ were found.

We found lower serum concentrations of phosphate to predict nutritional recovery. Previously, a study reported a positive correlation between serum phosphate concentrations and thymus size in children with SAM.²¹ To our knowledge, no previous studies have investigated the correlation between serum phosphate and anthropometric outcomes in children with malnutrition, and our finding needs to be validated in future studies.

This study is an analysis of secondary outcomes of the original study, which increases the risk of chance findings. To reduce this risk, we formulated a hypothesis, and we engaged researchers who had no previous knowledge of the data set to perform the data analysis as well as write the initial draft. In addition, we applied reference values from the Danish laboratory that analyzed the blood samples to be able to account for methodological considerations and uncertainties. This may have introduced information bias in the case of differing optimal reference values among the pediatric population of Burkina Faso. Unfortunately, we found no reference values for liver function tests in children from Burkina Faso. When comparing the Danish reference values to pediatric reference values established from a cohort from Ghana, we found small variations except for higher normal ranges for both albumin and amylase.¹⁷ Thereby, we find the risk of bias to be small in this regard. We excluded children with severe disabilities and children requiring hospitalization, but we might unknowingly have included children with undiagnosed conditions affecting the liver function tests. This could potentially have confounded our results. Still, we believe this risk to be small, as pediatric liver diseases are rare. Moreover, we found serum concentrations of thyroid-stimulating hormone and immunoglobulin G to be within the normal ranges (data not shown). This indicates that the children were not suffering from either hypothyroidism, hyperthyroidism, or autoimmune hepatitis, all of which could have caused elevated liver function tests.

5 | CONCLUSION

Liver function tests were largely within the normal range, whereby no biochemical signs of manifest liver damage were found. Serum concentrations of total

bilirubin were higher among children with stunted growth, which might indicate subclinical liver damage.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data described in the manuscript will not be made available as the Danish Act on Data Protection does not allow for personal data to be made available to others without prior individual approval from the Danish Data Protection Agency.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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