Liver Involvement in Pediatric and Adult Patients with Anorexia Nervosa

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Abstract: *Background:* Feeding and eating disorders, including anorexia nervosa, are among the leading causes of malnutrition in industrialized countries.

Hepatic involvement is an increasingly recognized complication in these patients.
The aim of this study was to investigate the prevalence and severity of liver dysfunction in pediatric and adult patients with anorexia nervosa and to evaluate the effect of nutritional rehabilitation over a 12-month follow-up period.

Methods: A retrospective single-center study was conducted, including pediatric and adult patients. Liver involvement was assessed via biochemical markers and abdominal ultrasonography. These parameters were analyzed in relation to nutritional status and duration of illness. Findings were compared between pediatric and adult patients.–

Results: 100 consecutive patients were enrolled (59 children and 41 adults). Hypertransaminasemia was identified in 30.5% of pediatric and 36.6% of adult patients. Liver steatosis was detected in 61% and 56% of children and adults, respectively. Other main causes of liver diseases were excluded in all cases. Severity of liver impairment significantly correlated with malnutrition status. Following nutritional rehabilitation, 86.7% of patients normalized liver enzyme levels, and improvement grade was significantly correlated with weight gain ($r = 0.61$, $p < 0.001$).

Conclusions: Liver involvement is common in both pediatric and adult patients with feeding and eating disorders, with severity closely linked to nutritional status and improvement following nutritional rehabilitation. It remains important to exclude other causes of liver disease. Overall, our findings support the importance of routine liver function monitoring in patients with eating disorders and emphasize the need for timely multidisciplinary interventions that address both somatic and psychiatric aspects of care.

Keywords: Liver enzymes, Hepatic steatosis, Anorexia nervosa, Malnutrition.

INTRODUCTION

Feeding and eating disorders are among the leading causes of malnutrition in industrialized countries. According to the Diagnostic and Statistical Manual of Mental Disorders, fifth edition text revision (DSM-5-TR™), anorexia nervosa falls under this category [1]. It is defined by a distorted body image, an intense fear of gaining weight, and persistent dietary restriction, leading to severe weight loss [2]. These disorders have a substantial impact on physical and psychological health, particularly during adolescents and early adulthood, with a prevalence of approximately 0.5% in the general population, rising to 1% among adolescents, with a higher incidence in females [3].

Anorexia nervosa can be associated with several medical complications, including cardiovascular abnormalities (*e.g.*, bradycardia, hypotension), gastrointestinal dysfunction (*e.g.*, decreased colonic motility leading to constipation), endocrine and electrolytes disorders, amenorrhea, and liver function tests abnormalities [4, 5]. Among these, hepatic involvement remains a critical concern. Several studies have reported that patients with an inadequate nutritional intake often exhibit signs of liver dysfunction, detectable via elevated liver enzymes or ultrasound findings of liver steatosis [6-8].

In cases of extreme malnutrition, particularly with BMI <13 kg/m² and BMI z-score < -3 for adults and children, respectively, patients may exhibit subtle signs of minimal hepatic encephalopathy, including cognitive slowing, emotional lability, and reduced engagement in treatment [9]. Cognitive impairments, including slowed processing speed and attention deficits, have been documented in patients with eating disorders and

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correlate with the degree of malnutrition [10, 11], potentially complicating treatment adherence and nutritional rehabilitation efforts.

Malnutrition-induced liver disease is frequently observed in cases of profound underweight and may progress to acute liver failure in extreme cases [12]. Although, the mechanism of liver injury in anorexia nervosa remains still unclear, various hypotheses have been proposed, including ischemic hepatitis, hepatic autophagy, and the refeeding syndrome [13, 14]. Elevation of liver transaminases is a common finding, reflecting a hepatocellular damage. During refeeding, hypertransaminasemia may be related to hepatic steatosis caused by excessive glucose accumulation within hepatocytes [15].

Recent studies have investigated the histological and morphological liver features in anorexia nervosa, revealing changes such as fatty degeneration and glycogen accumulation [16]. These may represent an adaptative response to prolonged nutritional deprivation. Contributing factors to hepatic fat accumulation include increased triglyceride synthesis, impaired fatty acid oxidation, and oxidative stress, which may be exacerbated by iron overload [17, 18].

The primary objective of this study was to evaluate the prevalence and severity of liver dysfunction in pediatric and adult patients diagnosed with anorexia nervosa in the context of a university hospital. In addition, we investigated the relationship between liver impairment and both the severity and duration of malnutrition and examined the effects of nutritional rehabilitation over time.

MATERIAL AND METHODS

This single-centre, retrospective study was conducted at the Paediatric Department, Neuropsychiatric and Psychiatric Unit of the University of Naples “Federico II”. A 12-month prospective follow-up was performed on a subgroup of patients to evaluate the impact of nutritional rehabilitation.Patients with DSM-5-TR™ diagnosed feeding and eating disorders attending outpatient or inpatient services were included. Exclusion criteria included chronic liver disease, chronic drug treatments, or concomitant neurological or psychiatric disorders.

Paediatric patients were categorized as: the outpatient group, which included patients with stable or moderately impaired clinical conditions, and the inpatient group, included those requiring hospitalization due to more clinical instability [19].

Hepatic involvement was assessed via liver enzyme panels [alanine aminotransferase (ALT), aspartate aminotransferare (AST), gammaglutamyl-transferase (GGT), alkaline phosphatase (AP), total/direct bilirubin, INR, albumin] and abdominal ultrasonography. Liver steatosis on abdominal ultrasound was categorized as mild (grade 1), moderate (grade 2) or severe (grade 3) based on the ratio between echogenicity of renal cortex and liver tissue [20]. In patients with liver involvement (biochemical and/or ultrasound evidence), potential secondary causes of liver disease (viral hepatitis; autoimmune liver disease; metabolic disorders such as Wilson disease and alpha-1-antitrypsin deficiency; celiace disease; nonalcoholic steatohepatitis; endocrinological disorders; chronic drug exposure) were systematically excluded.Additional measurements included BMI and BMI z-score (for pediatrics), mid-upper arm circumference (MUAC), mid-thigh circumference (MTC), and mid-thigh skinfold thickness (MTSF) were measured using standardized methods.

Malnutritional classification based on BMI for adult:

- Mild: BMI ≥ 17.0 - 18.5 kg/m²
- Moderate: BMI ≥ 16.0 – 16.99 kg/m²
- Severe: BMI ≥ 15.0 – 15.99 kg/m²
- Extremely severe: BMI < 15.0 kg/m²

For children a BMI z-score classification was used:

- < −1 to ≥ −2: mild malnutrition
- < −2 to ≥ −3: moderate malnutrition
- < −3: severe malnutrition

Patients with liver involvement underwent reassessment after 12 months to evaluate treatment response (clinical status, weight, liver function, imaging).

Additionally, information on nutritional therapy, weight changes, and liver enzyme trends was also recorded during follow-up to evaluated treatment efficacy.

ETHICS STATEMENT

The study conformed to the ethical guidelines of the 1975 Declaration of Helsinki. Ethical consent for data collection was waived, given the retrospective nature of the study and the anonymous treatment of data.

STATISTICAL ANALYSIS

The data were managed and analysed via SPSS software v.29. Descriptive data are reported as the

means and standard deviations for quantitative variables, or as absolute numbers and relative frequencies for qualitative variables. Univariate analysis was performed using the unpaired Student's t test or ANOVA. We used the two-tailed Fisher's exact test to assess associations between variables. Correlation between the variables was assessed using Pearson's. A *p-value* <0.05 indicated statistical significance.

RESULTS

A total of 100 consecutive patients with feeding and eating disorders were included in the study, comprising 59 pediatric and 41 adult individuals. The overall cohort was predominantly female (92%). In the pediatric cohort, the mean age at disease onset was **11.2 ± 1.7 years**, and the mean BMI z-score was **−2.3 ± 0.6**. In the adult group, the mean age at diagnosis was **21.0 ± 3.9 years**, with a mean BMI of **15.1 ± 1.2 kg/m²**.

Biochemical Liver Alterations in Pediatric and Adult Patients

In the pediatric cohort, hypertransaminasemia was observed in 19 of 59 patients (32.2%). Among these, 14/19 (74%) had isolated ALT elevations, while 5/19 (26%) showed combined ALT and AST elevation. The mean ALT and AST levels in these patients were **82.4 ± 38.6 IU/L** and **69.3 ± 27.5 IU/L**, respectively. INR (mean: **1.0 ± 0.07**) and GGT (mean: **18.5 ± 6.3 IU/L**) values were within normal limits. No clinical signs suggestive of chronic liver disease (hepatosplenomegaly, spider nevi, abdominal venous reticulum, or scratch lesions) were noted. Three children presented with lower limb edema, attributed to hypoalbuminemia secondary to malnutrition. Other liver tests were reported in Table **1**. No other causes of liver disease were identified. Abdominal ultrasound revealed liver steatosis in 36 of the 59 pediatric patients (61%). Steatosis was graded as mild in 20 cases (55.6%), moderate in 10 (27.8%), and severe in 5 (16.6%). A positive association emerged between steatosis severity and the presence of hypertransaminasemia: elevated transaminases were found in 5 patients with mild, 4 with moderate, and 2 with severe steatosis ($p < 0.01$), supporting an association between hepatic steatosis and biochemical liver injury.

Biochemical Liver Alterations in Adult Patients

Among adults, hypertransaminasemia was observed in 16 of 41 patients (38.1%). The mean ALT level was 93.7 ± 52.1 IU/L (range: 41–218 IU/L), while the mean AST was notably higher, at 104.2 ± 64.7 IU/L. Combined ALT and AST elevation was seen in 75% of cases. Compared to the pediatric cohort, adult patients more frequently exhibited elevated AST levels, suggesting that prolonged disease duration and advanced metabolic adaptation, including muscle catabolism, contribute to this enzyme pattern. Despite these differences, the overall prevalence of liver enzyme elevation did not differ significantly between groups ($p = 0.42$), although AST levels were significantly higher in adults ($p = 0.03$). Other hepatic tests, including INR, bilirubin, and GGT, remained within normal limits in most cases. Only five adult patients presented mildly elevated GGT values (mean: 45.6 IU/L), while no pediatric patients exhibited GGT, bilirubin, or INR abnormalities. None of the patients displayed clinical signs suggestive of chronic liver disease.

Hepatic Steatosis on Abdominal Ultrasound

Abdominal ultrasonography was performed in all patients. Liver steatosis was identified in 36/59 (61%) of pediatric patients and 23/41 (56%) of adults. Among pediatric cases, liver steatosis on abdominal ultrasound was graded as mild in 20 (55.6%), moderate in 10 (27.8%), and severe in 6 (16.6%). In adults, 13 cases (56.5%) were classified as mild, 7 as moderate (30.4%), and 3 as severe (13.1%). A progressive increase in transaminase levels was associated with higher steatosis grades. In patients with severe steatosis, mean ALT reached 108.5 ± 27.6 IU/L, compared to 62.3 ± 18.9 IU/L in those with mild steatosis ($p < 0.01$). This trend was consistent across both age groups.

Table 1: Liver Tests in Paediatric and Adult Patients at Diagnosis

	Pediatrics (n=59)	Adults (n=41)	*p-value*
Hypertransaminasemia (%)	32.2%	38.1%	0.42
ALT (IU/L, mean ± SD)	82.4 ± 38.6	93.7 ± 52.1	*ns*
AST (IU/L, mean ± SD)	69.3 ± 27.5	104.2 ± 64.7	0.03
GGT abnormalities (%)	0	12%	
Hepatic steatosis (%)	61%	56%	*ns*

Relationship between Malnutrition and Liver Dysfunction

A significant association was found between the severity of malnutrition and liver enzyme abnormalities. In the pediatric cohort, hypertransaminasemia occurred in 25% of patients with mild malnutrition (BMI z-score > −1.5), 40% with moderate malnutrition (BMI z-score between −2.5 and −1.5), and 59% with severe malnutrition (BMI z-score ≤ −2.5) (p=0.012). In adults, hypertransaminasemia was observed in 20% of patients with BMI ≥ 17.0 Kg/m², 38.5% of those with BMI between 16.0 and 16.99 Kg/m², and 50% with BMI < 16.0 Kg/m² (p = 0.036) (Table **2**). A strong negative correlation was observed between BMI and ALT levels (r = −0.48, p < 0.001), indicating that lower BMI was associated with greater liver enzyme elevation (Figure **1**).

Among pediatric patients, those requiring hospitalization due to clinical instability (inpatients) had a slightly higher prevalence of hypertransaminasemia compared to outpatients (33% vs 26.3%), although this difference was not statistically significant (p = 0.41). No difference in liver steatosis prevalence was found between inpatient and outpatient subgroups.

Follow-up and response to nutritional rehabilitation

A subgroup of 30 patients (18 pediatric, 12 adults) was followed prospectively for 12 months after initiation of structured nutritional rehabilitation. At baseline, the mean ALT level in this group was 88.4 ± 39.1 IU/L, which declined significantly to 34.2 ± 13.6 IU/L at the 12-month follow-up (p < 0.001). AST values followed a similar trend. Overall, 86.7% (26/30) of patients achieved complete normalization of liver enzymes, while 4 patients had persistent mild elevations with minimal weight gain and remained malnourished (BMI < 16.0 kg/m²) (Table **3**). A significant positive correlation was identified between BMI improvement and reduction in transaminase levels (r = 0.61, p < 0.001), highlighting the reversibility of hepatic impairment with adequate nutritional rehabilitation.

DISCUSSION

Anorexia nervosa is a complex psychiatric disorder with significant somatic complications, including hepatic dysfunction, which is increasingly recognized as both prevalent and clinically meaningful. Although often asymptomatic, liver impairment in malnourished individuals is frequently reflected by elevated transaminase levels, indicative of systemic metabolic stress [21].

Table 2: Association between Malnutrition Severity and Liver Enzyme Elevation

Nutritional Severity (BMI or z-score)	Hypertransaminasemia (%) Pediatrics	Hypertransaminasemia (%) Adults
Mild	25%	20%
Moderate	40%	38.5%
Severe	59%	50%
p-value	0.012	0.036

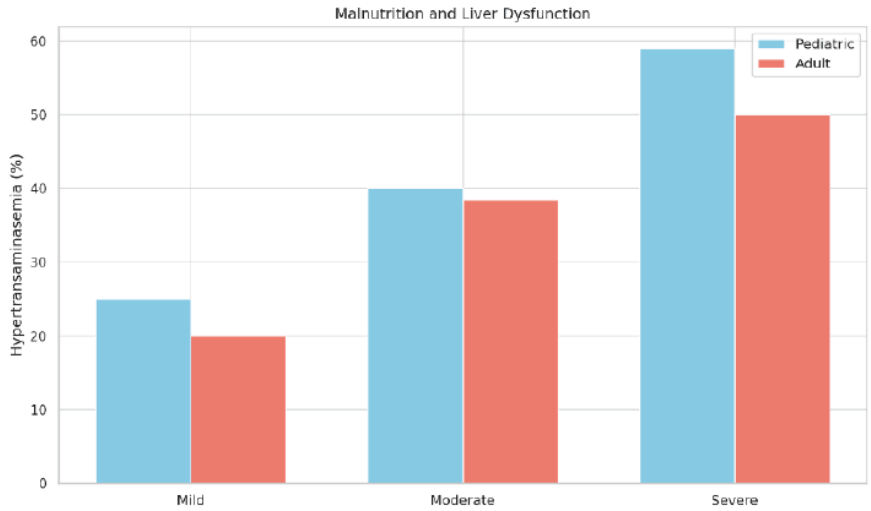

Figure 1: Impact of Malnutrition on Liver Enzyme Abnormalities.

Table 3: Follow-Up after Nutritional Rehabilitation in Children and Adults (12 Months)

	Baseline (n = 30)	12-Month Follow-Up	*p-value*
Mean BMI (Kg/m²)	15.3 ± 1.2	17.2 ± 1.1	< 0.001
ALT (IU/L)	88.4 ± 39.1	34.2 ± 13.6	< 0.001
AST (IU/L)	91.7 ± 42.5	32.6 ± 11.8	< 0.001
Patients with normal ALT levels (%)	-	26 (86.7%)	—
Patients with persistent ALT elevation (%)	-	4 (13.3%)	—
Mean BMI in Patients with Persistent ALT elevation (kg/m²)	14.7 ± 0.9	15.2 ± 1.0	*ns*

Unlike previous studies conducted exclusively on adults or adolescents, our study included adolescents and adults from the same geographic area observed in the same hospital setting.

Our findings confirm that liver enzyme abnormalities are common in both pediatric and adult patients with eating disorders, affecting approximately one-third of individuals in each group. The most prevalent pattern was an isolated elevation in alanine aminotransferase (ALT), which correlated significantly with the degree of malnutrition. Notably, these transaminase elevations were reversible with nutritional rehabilitation, as evidenced by enzyme normalization in patients achieving weight restoration during follow-up.

A strength of this study is the inclusion of a 12-month prospective follow-up, which allowed a more accurate assessment of liver enzymes following nutritional improvement.

The pathophysiology underlying hepatic involvement in anorexia nervosa remains understood and is likely multifactorial. Proposed mechanisms include starvation-induced autophagy, hepatic hypoperfusion, and metabolic adaptations to prolonged caloric deficiency [22]. These factors contribute to the characteristic hepatocellular pattern of enzyme elevation. Moreover, the high prevalence of hepatic steatosis on abdominal ultrasound, observed in over 60% of pediatric and 56% of adult patients, suggests that fatty liver disease may represent an additional mechanism of liver injury, likely related to disrupted lipid metabolism during chronic energy deprivation.

Interestingly, the significantly higher aspartate aminotransferase (AST) levels observed in the adult cohort may reflect a longer duration of illness, increased catabolic activity, or greater muscle breakdown. This finding suggests that liver involvement in anorexia nervosa is not only a function of age or chronicity but rather a dynamic physiological response to nutritional status. The lack of consistent clinical or demographic predictors reinforces the importance of routine liver function testing in all patients with restrictive eating disorders, as hepatic abnormalities may be present even in those who appear clinically stable. The systematic exclusion of alternative etiologies, including infectious, autoimmune, and metabolic liver diseases, further reinforces the association between severe malnutrition and hepatic dysfunction observed in our sample.

One of the most clinically relevant findings of this study is the strong correlation between nutritional status and liver function, particularly in the pediatric population. Children and adolescents with severe malnutrition (BMI z-score ≤ −2.5) had a significantly higher prevalence of hypertransaminasemia. Conversely, early initiation of structured nutritional support was associated with rapid and sustained improvements in liver enzyme, emphasizing the critical role of early diagnosis and intervention. Given the greater hepatic plasticity observed in younger patients, timely nutritional rehabilitation may prevent long-term organ damage.

Beyond its somatic consequences, liver dysfunction in anorexia nervosa may also carry important neuropsychiatric implications that warrant careful consideration. In severely malnourished patients, particularly adults with BMI <13 kg/m² or children with BMI z-score <−3, subtle signs of minimal hepatic encephalopathy may emerge, including cognitive slowing, affective instability, and reduced treatment engagement. Although rarely observed, these manifestations may reflect mild hyperammonemia or neurotransmitter disturbances, as previously described [23]. These findings suggest that hepatic dysfunction may serve not only as an indicator of systemic deterioration, but also as a contributing factor to cognitive and behavioral resistance during the refeeding process [23]. In our pediatric cohort, the full normalization of transaminase levels after nutritional rehabilitation reinforces the potential role of hepatic recovery in promoting both physical and psychological treatment efficacy.

Our data support the implementation of systematic liver function monitoring as a standard component in the management of eating disorders and emphasize the value of comprehensive, multidisciplinary approaches that integrate somatic and psychiatric care.

Transient elevations in liver enzymes are common during the refeeding phase and should not be misinterpreted as signs of treatment failure. Instead, these changes likely represent physiological metabolic adaptations to nutritional rehabilitation and should be managed within a structured, closely monitored clinical framework. Clinicians should also recognize that cognitive recovery may lag behind biochemical normalization by one to two weeks [24, 25], highlighting the need for ongoing psychological support even after hepatic parameters have returned to normal.

The consistent association between BMI recovery and liver enzyme normalization further supports the integration of hepatic function parameters into clinical risk stratification and treatment planning, especially in severely malnourished patients or those with limited early weight gain. In pediatric populations, where organ resilience is typically greater, timely and coordinated intervention may help to prevent long-term complications. Informing patients and families about the potential delay between liver recovery and psychological improvement is essential for aligning expectations and supporting long-term treatment adherence.

Elevated AST levels in adults may reflect more prolonged hepatic stress or cumulative metabolic burden, reinforcing the need for early detection and aggressive nutritional intervention in younger patients to mitigate long-term organ damage.In addition to macronutrient repletion, emerging evidence underscores the importance of correcting micronutrient deficiencies in malnourished populations. While our study did not assess micronutrient status, existing literature highlights the role of vitamin and trace element supplementation in supporting hepatic and systemic recovery. Particular attention has been paid to nutraceuticals, including myo-inositol and multinutrient strategies, which have shown potential benefits in women's health and the prevention of obstetric complications such as gestational diabetes and preterm birth [27-29]. On the other hand, particular attention must be reserved to the quality of the studies carried out because it is inevitable that there may be a conditioning of companies in response to the strong needs of patients [30]. Although data on the use of supplements and vitamins in anorexia nervosa are limited, their appropriate integration may improve the effectiveness of nutritional rehabilitation and support liver recovery in this vulnerable population [31-34]. This study presents some limitations, including its retrospective, single-center design and the failure to assess micronutrients or vitamins, which could play a role in liver function and overall metabolic status. Despite these limitations, the study provides potentially useful information for clinicians on the prevalence, clinical significance, and reversibility of liver dysfunction in malnourished patients with anorexia nervosa.

CONFLICT OF INTEREST STATEMENT

All the authors report no relevant conflicts of interest for this article.

AUTHORS' CONTRIBUTION

Conceptualization: R.I., V.D.C., F.D.D., M.I.S., M.P.R., L.V.; methodology R.I., V.D.C., F.D.D., M.P.R., L.V. software: V.D.C., F.F., M.L.; writing—original draft preparation: V.D.C., F.D.D., R.I., F.F., M.I.S. All authors have read and agreed to the published version of the manuscript.

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