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Original Article

Etiology and clinical features of hypertransaminasemia in children: A retrospective study from a tertiary care center in Türkiye

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ABSTRACT

Objective: This study aimed to investigate the etiology of hypertransaminasemia in children and to evaluate the clinical characteristics of patients based on age and underlying causes.

Material and Methods: A total of 210 pediatric patients aged between 1 month and 18 years who presented to the Pediatric Gastroenterology Outpatient Clinic with hypertransaminasemia were retrospectively analyzed. Hypertransaminasemia was defined as AST >50 IU/L and ALT >45 IU/L on at least two separate occasions within two months.

Results: The mean age of the patients was 5.36±5.30 years and 50.5% were female. At presentation, 30.5% were asymptomatic, with fever being the most common symptom (23.4%). Physical examination findings were normal in 53.8% of cases. Mean AST and ALT values were significantly higher in symptomatic patients compared to asymptomatic ones ($p<0.001$). No significant difference were observed between age groups in terms of mean ALT and AST values ($p=0.290$, $p=0.190$). Hypertransaminasemia was mild in 55.7% of patients, moderate in 20.5%, and severe in 23.8%. An underlying etiology was identified in 61% of cases. The most common cause was infectious diseases, followed by metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic/genetic disorders. Severe hypertransaminasemia was significantly more frequent in infectious diseases than in idiopathic cases or MASLD, whereas mild hypertransaminasemia was more common in MASLD and idiopathic cases compared to infections ($p<0.001$).

Conclusion: Infectious diseases and MASLD remain the leading causes of hypertransaminasemia in children. Infectious etiologies contribute across all age groups, MASLD is strongly linked to obesity and adolescence. As elevated transaminase levels may be the only clinical clue, especially in asymptomatic patients with normal physical examinations, careful follow-up and comprehensive evaluation are essential to avoid potential complications.

Keywords: ALT, AST, infectious diseases, NAFLD, pediatric, transaminases

INTRODUCTION

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are intracellular enzymes found in hepatocytes and other cells and are called transaminases. AST is found not only in the liver but also in various extrahepatic tissues such as heart muscle, skeletal muscle, kidneys, pancreas, lungs, brain, erythrocytes, and leukocytes. ALT, on the other hand, is present in low concentrations in extrahepatic tissues and is considered more specific to hepatocellular damage (1, 2). Hypertransaminasemia occurs when these enzymes leak into the serum due to cellular injury or increased cell membrane permeability in affected tissues (1).

The degree of transaminase elevation does not always correlate with the severity of liver damage or prognosis (1). However, markedly elevated levels are typically associated with significant hepatocellular injury (1). For clinical interpretation, hypertransaminasemia is commonly classified into three categories: mild (<5× the upper limit of normal [ULN]), moderate (5-10×ULN), and severe (>10×ULN) (1). Mild to moderate elevations are often linked to chronic liver diseases such as chronic viral hepatitis, metabolic dysfunction-associated steatotic liver disease (MASLD), haemochromatosis, autoimmune hepatitis, alpha1-antitrypsin deficiency, Wilson's disease and celiac disease. Additionally drugs or non-hepatic causes including haemolysis, myopathy, thyroid disease, may contribute to hypertransaminasemia. Severe elevations ara

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often associated with acute viral hepatitis (e.g., A-E, herpes, EBV, CMV), drug or toxin induced liver injury, ischaemic hepatitis, autoimmune hepatitis, Wilson's disease, acute biliary obstruction, or acute Budd-Chiari syndrome (3,4).

In adults, the prevalence of elevated transaminase levels has been reported to be approximately 7.9%, with no identifiable etiology in many cases (5). Among asymptomatic adolescents, the prevalence ranges from 3.5% to 12.4% (3). However, the prevalence and etiology of hypertransaminasemia in children of all ages are not well defined, probably due to underestimation of the condition (3,5).

This study aimed to investigate the etiological spectrum and clinical characteristics of children who presented with hypertransaminasemia at a pediatric gastroenterology outpatient clinic.

MATERIALS and METHODS

This retrospective study included 210 pediatric patients aged between 1 month and 18 years who were admitted to the Pediatric Gastroenterology Outpatient Clinic of Kahramanmaraş City Hospital between June 2023 and December 2024 with hypertransaminasemia and who had elevated transaminases in at least two different measurements in the last two months. Patients with known chronic comorbidities such as chronic liver disease, chronic renal failure, congenital heart disease, type 1 diabetes mellitus, known genetic syndromes (e.g., Down syndrome), and myopathies were excluded from the study. For the purpose of statistical analysis, the highest recorded ALT and AST values were used.

Demographic data, presenting symptoms, clinical findings, laboratory results, imaging results and histopathological evaluations were collected from hospital electronic medical records and patient files. Hypertransaminasemia was defined as AST >50 IU/L and/or ALT >45 IU/L, in accordance with previously published national pediatric data (6,7). These fixed cut-off values were applied uniformly across all four age groups studied (1 month–2 years, 2–6 years, 6–12 years, and >12 years). Hypertransaminasemia was classified according to severity as mild (<5× upper limit of normal [ULN]), moderate (5–10× ULN), or severe (>10× ULN) (1).

Asymptomatic patients were identified incidentally, most often during routine laboratory evaluations such as preoperative evaluation, or general health checks. In some patients with non-specific complaints (e.g., sore throat, diarrhea), liver function tests were ordered as part of a broader evaluation for febrile or systemic illness. In cases with constipation or epistaxis, elevated transaminases were discovered incidentally, as these tests were performed as part of extended laboratory panels rather than targeted investigations.

Metabolic dysfunction associated steatotic liver disease (MASLD), the updated term for nonalcoholic fatty liver disease (NAFLD). The updated definition requires evidence of hepatic steatosis via imaging or biopsy along with at least one cardiometabolic risk factor. In this study, MASLD was diagnosed in patients with a body mass index (BMI) above the 85th percentile for age and sex (BMI z-score > +1), radiologic evidence of steatosis, and absence of other chronic liver diseases known to cause steatosis (8).

Drug-induced liver injury (DILI) was diagnosed according to the 2019 European Association for the Study of the Liver (EASL) Clinical Practice Guidelines, based on exclusion of other etiologies and at least one of the following criteria in patients with a relevant drug exposure history: (1) ALT ≥5×ULN; (2) ALP ≥2×ULN with elevated GGT and no bone disease; (3) ALT ≥3×ULN and total bilirubin >2×ULN (9).

The diagnosis of autoimmune hepatitis was based on the presence of interphase hepatitis, ALT, AST and serum IgG elevations and specific autoantibodies (10).

BMI Z-scores were calculated based on WHO growth standards. For children aged 0–5 years, malnutrition was defined as BMI Z-score < –2 SDS, overweight as > +2 SDS, and obesity as > +3 SDS. For those aged 5–19 years, malnutrition was defined as BMI Z-score < –2 SDS, overweight as > +1 SDS, and obesity as > +2 SDS. Values between these ranges were considered normal (11).

Statistical analysis

Statistical analyses were performed using IBM Statistical Package for the Social Sciences, version 21.0 (SPSS Inc., Armonk, NY, IBM Corp., USA). Categorical variables were expressed as numbers and percentages, while continuous variables were presented as mean±standard deviation. Normality of numerical data was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Categorical variables were compared using the Chi-square test when applicable, and the Mann-Whitney U test was used for non-normally distributed data. When significant differences were observed in Chi-square analyses involving more than two groups, post-hoc pairwise comparisons with Bonferroni correction were applied. Statistical significance was defined as a p-value of <0.050.

RESULTS

The mean age of the patients was 5.36±5.30 years (range:1 month-18 years), and 106 (50.5%) were female. At presentation, 30.5% of the patients were asymptomatic. The most common presenting complaint was fever (23.4%). Physical examination was normal in 113 patients (53.8%), while some had findings such as hepatomegaly, jaundice, or splenomegaly. Nutritional status varied, with both overweight/obesity and malnutrition observed (details in Table I).

Table I: Demographic and clinical characteristics of children with hypertransaminasemia n(%)

Age	
1 months-2 years	91 (43.3)
2-6 years	37(17.6)
6-12 years	51 (24.3)
>12 years	31 (14.8)
Gender	
Female	106 (50.5)
Male	104 (49.59
BMI z-score	
Normal	138 (65.7)
Malnourished	40 (19)
Overweight	19 (99
Obese	13 (6.2)
Complaints	
Fever	49 (23.4)
Sore throat	19 (9)
Jaundice	16 (7.6)
Diarrhea	13 (6.3)
Presentation due to overweight/obesity	12 (5.7)
Fatigue	11 (5.2)
Vomiting	11 (5.2)
Others (swelling in the neck, abdominal pain, constipation, failure to thrive, epistaxis, trauma, toxic ingestion, joint pain)	15 (7.1)
Asymptomatic	64 (30.5)
Physical examination findings	
Normal physical examinations	113 (53.8)
Upper respiratory and/or lower respiratory tract infection findings	43 (20.5)
Hepatomegaly	25 811.9)
Jaundice	16 (7.6)
Splenomegaly	13 (6.2)
Severity of hypertransaminasemia	
Mild	117 (55.7)
Moderate	43 (20.5)
Severe	50 (23.8)

Table II: Laboratory characteristics of children with hypertransaminasemia

	Asymptomatic	Symptomatic	p†
AST (IU/L)*	154.9±107.0	443.7±575.3	<0.001
ALT (IU/L)*	158.1±104.6	418.3±483.9	<0.001

**: (Mean±SD), †: Non parametric test*

Table III: Etiologies of Hypertransaminasemia in Children

Etiology	n (%)
Idiopathic	82 (39)
Infectious diseases	62 (29.5)
EBV	23 (29.5)
CMV	19 (29.5)
Sepsis	7 (29.5)
HAV	5 (29.5)
Rubella	3 (29.5)
Toxoplasma	2 (29.5)
Brucella	2 (29.5)
Influenza	1 (29.5)
MASLD	19 (9)
Metabolic/Genetic diseases	13 (6.2)
Wilson's disease	2 (6.2)
PFIC 8	1 (6.2)
SCYL 1 mutation	1 (6.2)
Johanson Blizzard syndrome	1 (6.2)
Glycogen storage type 3 disease	2 (6.2)
X-linked liver glycogenosis	1 (6.2)
Tyrosinemia type 1	1 (6.2)
Ornithine transcarbamylase deficiency	1 (6.2)
Hemachromatosis	1 (6.2)
Biotinidase deficiency	1 (6.2)
Alpha 1 antitrypsin deficiency	1 (6.2)
Autoimmune hepatitis	9 (4.3)
Muscular dystrophy	9 (4.3)
DILI (Pyrantel pamoate, sulfadiazine, isotretinoin, valproic acid, levetiracetam)	6 (2.9)
Celiac disease	4 (1.9)
Other (Trauma, mauriac syndrome, Maraş powder intake, mushroom poisoning)	4 (1.9)
Cholelithiasis and cholecystitis	2 (1)

Table IV: Severity of hypertransaminasemia classified by etiology

Etiology	Mild*	Moderate*	Severe*	Total (n)
Infectious diseases	20 (32.2)	15 (24.2)	27 (43.6)	62
MASLD	17 (89.5)	2 (10.5)	0 (0)	19
Metabolic/genetic disorders	8 (61.5)	0 (0)	5(38.6)	13
Idiopathic	43 (52.4)	16 (19.5)	23 (28.1)	82

: n(%)

The severity of hypertransaminasemia was mild in 117 (55.7%), moderate in 43 (20.5%) and severe in 50 (23.8%) patients (Table I). The mean ALT level was 342±427 IU/L (range 36-2.386 IU/L), and the mean AST level was 359±504 IU/L (range 27-3.042 IU/L). Mean AST and ALT levels were significantly higher in symptomatic patients compared to asymptomatic ones (p<0.001) (Table II). No significant differences in transaminase levels were observed among different age groups (ALT: p=0.290, AST: p=0.190).

An etiologic cause was identified in 128 patients (61%), with infectious diseases (e.g., EBV, CMV, sepsis, HAV, rubella, toxoplasmosis, brucellosis, influenza) being the most frequent cause, followed by MASLD and metabolic/genetic disorders (Table III). Among the metabolic/genetic group, rare conditions such as biotinidase deficiency were also observed. Although not typically considered a hepatic disorder, biotinidase deficiency has been reported to cause elevated liver enzymes in some patients (12).

Among patients diagnosed with autoimmune hepatitis, 66.7% had mild elevations. Similarly, 55.6% of muscular dystrophy patients, all patients with celiac disease, and 89.5% of those with MASLD exhibited mild hypertransaminasemia. In contrast, 43.6% of patients with infectious etiologies showed severe enzyme elevations (Table IV). Severe hypertransaminasemia was

Table V: Comparison of transaminase serum levels among the most common etiologies of hypertransaminasemia in children

	Etiology				p
	Idiopathic	Infectious diseases	MASLD	Metabolic/ Genetic diseases	
AST(IU/L)*	260.7±390.9	573.0±679.5	84.8±49.5	334.9±369.7	<0.001[1,2,3]
ALT(IU/L)*	293.5±414.5	499.9±546.7	134.4±58.6	243.0±226.6	<0.001[1,3]

***:** *(Mean±SD),* ***MASLD:** *Metabolic Dysfunction-associated Steatotic Liver Disease,* [1]*idiopathic vs. infectious diseases,* [2]*idiopathic vs. MASLD,* [3]*infectious diseases vs. MASLD*

Table VI: Distribution of hypertransaminasemia etiologies in children according to age groups.

	Age				p
	1 months-2 years*	2-6 years*	6-12 years*	>12 years*	
Etiology					
Idiopathic	46 (56.1)[a]	16 (51.6)[a,b]	15 (38.5)[a,b]	5 (20.8)[b]	
Infectious diseases	30 (36.6)[a]	10 (32.3)[a]	15 (38.5)[a]	7 (29.2)[a]	<0.001
MASLD	0 (0.0)[a]	1 (3.2)[a,b]	8 (20.5)[b,c]	10 (41.7)[c]	
Metabolic/Genetic diseases	6 (7.3)[a]	4 (12.9)[a]	1 (2.6)[a]	2 (8.3)[a]	

***:** *n (%), Different superscript letters (a, b, c) indicate significant pairwise differences between age groups (p<0.050, post-hoc Bonferroni corrected Chi-square test).*

significantly higher in infectious diseases than in the idiopathic group and MASLD, and in metabolic/genetic diseases than in MASLD. Mild hypertransaminasemia was significantly higher in MASLD and idiopathic group compared to infectious diseases (p<0.001). In infectious diseases, both AST and ALT levels were found to be elevated while the lowest values were observed in the MASLD group (Table V).

Infectious diseases contributed to hypertransaminasemia across all age groups at similar rates (p=0.718). MASLD was rarely observed in younger children but showed a marked increase with age: 52.6% of all MASLD cases occurred in patients older than 12 years, and within the >12 year age group, 41.7% were diagnosed with MASLD. Metabolic/genetic disorders, while overall rare, were most frequently encountered in the 1 month–2 years age group (46.1%). The proportion of patients with idiopathic hypertransaminasemia was highest in the 1 month–2 years (56.1%) and 2–6 years (51.6%) groups and decreased with age (20.8% in >12 years). The most common etiologies were idiopathic in the youngest two age groups, idiopathic and infectious diseases in the 6–12 years group, and MASLD in adolescents over 12 years (Table VI). Additionally, MASLD was identified in 59.4% of patients classified as overweight or obese.

Abdominal ultrasonography was performed in all patients, and imaging was normal in 67.7% of children for liver parenchyma and bile ducts. Hepatomegaly was detected in 25 (11.9%) patients, hepatosteatosis in 21 (9.3%), splenomegaly in 13 (6.2%), heterogeneity in liver parenchyma in 8 (3.5%), cholelithiasis and cholecystitis in 2 (1%), and hepatic haemangioma in 1 (0.4%) patient. Liver biopsy was performed in 13 (6.2%) patients and histopathological results contributed to the diagnosis in 11 (84.6%) patients. Liver biopsy confirmed autoimmune hepatitis in nine patients and Wilson's disease in two patients. In two other patients—one with SCYL1 mutation and one with ornithine transcarbamylase (OTC) deficiency—the biopsy revealed nonspecific and nondiagnostic histological changes, including macrovesicular steatosis, pericellular fibrosis, hydropic degeneration and atrophic changes in hepatocytes, moderate lymphocytic infiltration in portal areas, and mild bile duct injury.

DISCUSSION

The increased use of routine biochemical testing has led to a rise in incidental findings of hypertransaminasemia. The symptoms accompanying hypertransaminasemia are highly variable (13). While Sanrı et al. (7) reported obesity as the most common presenting complaint (29.1%), and Serdaroğlu et al. (8) found fatigue in 53.4% of cases, this study showed that 30.5% of patients were asymptomatic. Fever was the most frequent presenting symptom (23.4%), and physical examination was unremarkable in 53.8% of patients. This highlights that hypertransaminasemia may remain clinically silent and the importance of routine monitoring for laboratory abnormalities even in the absence of symptoms or abnormal physical findings.

The prevalence of idiopathic hypertransaminasemia varies among studies. Serdaroğlu et al. (7) reported idiopathic hypertransaminasemia in 6.4% of cases. In the cohort by Iorio et al. (13), idiopathic hypertransaminasemia was observed in 135 out of 425 patients (31.7%), including both those with transient (<6 months) and persistent (>6 months) enzyme elevation. Çeltik et al. (15) reported a rate of 27.1% in the neonatal period, with 12% in their overall cohort. In our study, no etiological cause was identified in 39% of patients.

In underdeveloped countries, hepatitis A virus (HAV) infection remains the leading cause of hypertransaminasemia in children.

Conversely, in developed countries, MASLD is the primary cause, followed by hepatobiliary, genetic and autoimmune diseases (3). The etiological spectrum of hypertransaminasemia in our cohort largely reflects both local epidemiology and global trends. Infectious diseases emerged as the most common cause (29.5%), consistent with previous studies from Türkiye reporting similar rates 34% in Serdaroğlu et al. (7) and 34.4% in Çeltik et al. (15). MASLD was the second leading cause in our study (9%), in line with global data showing its rising prevalence in children and adolescents.

Hepatotropic viruses (hepatitis A, B, C, E, and non-A-E viruses) and systemic febrile infections such as EBV and CMV frequently cause moderate to severe elevations in aminotransferase levels (3,4,16). In addition to viral infection, bacterial sepsis or parasite infections may also lead to hepatitis (17). In our study, the leading etiological infectious cause was EBV infection (11%), followed by CMV infection (9%) and bacterial sepsis (3.4%). HAV infection was observed less frequently (2.4%) because hepatitis A vaccination in the national immunization program since 2012. Among patients with infectious etiologies, 43.6% exhibited severe and 24.2% moderate hypertransaminasemia. Interestingly, infectious etiologies were observed at similar frequencies across all age groups in our study. This suggests that infectious diseases remain a substantial and age-independent contributor to pediatric hypertransaminasemia in our population, despite improvements in vaccination coverage and public health measures.

MASLD has become an increasingly prevalent cause of hypertransaminasemia in children and adolescents (8). This trend has been underscored by recent consensus statements and pediatric guidelines, which highlight MASLD as one of the most significant chronic liver diseases of childhood (8,18,19). A global meta-analysis estimated MASLD prevalence at 7.6% in the general pediatric population and approximately 36% among children with obesity (20). An autopsy study revealed that histological MASLD prevalence ranges from 0.7% in 2-4 year olds to 17.3% in 15-19 year olds (21). Mild to moderate hypertransaminasemia is commonly observed in MASLD (22). In our study, similar to the literature, 89.5% of the MASLD group had mild hypertransaminasemia and 52.6% were >12 years of age. MASLD was present in 59.4% of our overweight/obese patients. Recent meta-analyses indicate that the prevalence of MASLD among children with obesity is approximately one-third (around 34.2%), though this varies across regions. This condition is now recognized as a significant public health concern. Particularly worrisome is the pediatric-onset form, since early manifestation leads to a longer duration of exposure to metabolic risk factors and thereby increases the likelihood of lifelong complications (19).

Congenital metabolic and genetic diseases are more prevalent in pediatric patients compared to adults, accounting for 20–30% of liver diseases in infancy and childhood (3). Iorio et al. (13) reported a 12% prevalence of genetic diseases among children with isolated hypertransaminasemia. In our study, metabolic/genetic diseases were responsible for 6.2% of children with hypertransaminasemia and were mostly observed in the 1 month-2 years age group (46.1%), similar to the literature.

Autoimmune hepatitis has a lower prevalence than viral hepatitis and MASLD (4, 23). The most common non-hepatic cause of transaminase elevation is muscle diseases. In case of muscle disease, ALT and AST elevation is accompanied by creatine kinase (CK) elevation, which indicates muscle destruction (3). The prevalence of autoimmune hepatitis was 4.3%, and the prevalence of muscular dystrophy in our study was also 4.3%.

Drug-induced liver injury (DILI) is an important but often unrecognised cause of hypertransaminasemia in children. The true incidence of pediatric DILI is unknown, but children are estimated to account for approximately 10% of all reported cases. Most cases are subclinical, presenting only with laboratory abnormalities. The most frequently implicated hepatotoxic drugs in children are antibiotics and antiepileptics (24,25). In our study, DILI was rare (2.9%) and most often related to antiepileptic drugs.

The limitations of our study are that it is retrospective in design, macro AST was not studied, and transaminase cut-off values reflecting our population are not known. In addition, we used fixed cut-off values (AST >50 IU/L and ALT >45 IU/L) rather than age- and sex-specific thresholds.

CONCLUSION

This study highlights that infectious diseases and MASLD remain the leading causes of pediatric hypertransaminasemia in Türkiye. The age-independent role of infections, the strong association of MASLD with adolescent obesity, and the high proportion of asymptomatic cases represent the distinctive contributions of this study. These results emphasize the need for clinicians to remain vigilant even in asymptomatic children and support public health strategies that combine infection control with urgent efforts to prevent and manage childhood obesity.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Kahramanmaras Sutcu Imam University Medical Research (16.12.2024, reference number: 34/05).

Contribution of the authors

Study conception and design: Dr. Melike Arslan; data collection: Dr. Melike Arslan; analysis and interpretation of results: Dr. Melike Arslan; draft manuscript preparation: Dr. Melike Arslan

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Conflict of interest

The authors declare that there is no conflict of interest.

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