

Association between serum transaminase elevations and clinical course in patients with familial Mediterranean fever

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Abstract

Objective

To evaluate the clinical features, disease course, genetics, and treatment responses of familial Mediterranean fever (FMF) patients with elevated serum transaminase levels.

Methods

The charts of the FMF patients with at least one year of follow-up were retrospectively investigated for the presence of elevated serum transaminase levels. The genetic and clinical findings and disease course of identified patients were compared with a group of FMF patients with no transaminase elevations.

Results

A retrospective chart review included 780 registered patients diagnosed with FMF. Forty-two patients (5.3%) were excluded because of the insufficient follow-up duration, associated viral hepatitis, alcoholic hepatitis, and biopsy-proven liver amyloidosis. A total of 83 patients (11.2%) with elevated serum transaminase levels were identified, and their findings were compared with a control group of 655 patients with no transaminase elevation. FMF attacks began at a significantly older age (17.0 ± 12.9 vs. 12.4 ± 9.0 years, $p=0.01$), and the percentage of patients experiencing more frequent (≥ 3 attacks/month) attacks was higher (35.4% vs. 24.1%, $p=0.01$) in the elevated transaminase group.

No significant differences were observed between the two groups regarding age at diagnosis, delay in diagnosis, presenting features of FMF, CRP levels at baseline and during attacks, frequency of amyloidosis, and the colchicine dose, as well as the frequency of patients requiring anti-IL-1 treatments. The frequency of MEFV gene exon 10 mutations and p.M694V homozygosity did not differ significantly between the groups. Hepatosteatosis was observed by abdominal ultrasonography in 46.7% of the patients with elevated liver enzymes. A follow-up study after 6 years revealed that transaminase levels remained elevated in 21 out of 49 patients (42.9%), and one patient developed liver cirrhosis.

Conclusion

Elevated serum transaminase levels can be detected in approximately 11.2% of FMF patients, and this finding is not associated with colchicine use or manifestations of FMF. Ultrasonographic evidence of hepatosteatosis was observed in nearly half of the patients. Follow-up analysis showed that serum transaminase elevations may persist in 43% of the patients, which suggests further studies to determine whether this abnormality is a feature of FMF or not.

Key words

familial Mediterranean fever, MEFV, colchicine, liver toxicity, transaminase, amyloidosis

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Introduction

Familial Mediterranean fever (FMF) is the most common form of hereditary autoinflammatory disorders, which is caused by autosomal recessively inherited mutations of the *MEFV* gene. The disease is characterised by recurrent episodes of fever, sterile inflammation of serosal and synovial membranes, and erysipelas-like erythema (1). FMF predominantly affects Mediterranean populations, especially those of Middle Eastern origin (2). Colchicine is the mainstay of treatment, preventing the recurrence of inflammatory attacks and the development of AA amyloidosis (3). Mild elevations in liver enzymes have been described in FMF patients receiving colchicine, and these changes are generally considered to be drug-related and transient, improving after dose reduction or discontinuation of the drug (4).

Hepatomegaly is seen in approximately 5% of FMF patients. However, apart from the hepatic amyloidosis, liver involvement has not been a well-defined feature of FMF (5, 6). Various forms of liver involvement have been reported in the literature in association with FMF, including elevated liver enzymes, non-alcoholic fatty liver disease (NAFLD), hyperbilirubinaemia, Budd-Chiari syndrome, and cryptogenic cirrhosis (7). A recent study from the French National Reference Centre for FMF, involving 533 patients, reported confirmed chronic liver function test abnormalities in 12.4% of the cases (8). The same group also demonstrated steatosis, fibrosis, and non-alcoholic steatohepatitis in 10 (50%), 9 (45%), and 7 (35%) of the 20 FMF patients with elevated liver enzymes, as assessed by FibroMax (a non-invasive tool for assessing hepatic steatosis and fibrosis) and ultrasound (9). However, there is still limited data on the demographic features and presentation characteristics of FMF patients with elevated transaminase levels.

In the present study, we aimed to evaluate the clinical features, genetic findings, and treatment responses of FMF patients with elevated transaminase levels in comparison with those with no liver enzyme abnormalities.

Patients and methods

Patients

We retrospectively analysed medical records of 780 patients diagnosed with FMF and followed at a tertiary referral centre. All patients fulfilled Livneh *et al.*'s modified Tel-Hashomer criteria for the diagnosis (10). FMF patients over 18 years of age and with a follow-up duration of more than one year were included in the study. The characteristics of the patients, including demographics, clinical and laboratory findings at presentation and during the course of the disease, and the *MEFV* gene variations, were recorded using a standardised data collection form. All patients were on colchicine treatment. The study group was categorised into two arms: i) Patients with elevated serum liver enzymes and ii) FMF patients with normal serum liver enzyme levels. The elevated serum transaminase levels were defined as ALT ≥ 45 U/L and/or AST ≥ 42 U/L at least twice within 6 months. These cut-off values corresponded to the upper limits of normal used by our institutional laboratory during the study period.

These two groups were compared in terms of demographic characteristics, clinical features, genetic findings, and disease course. The patients with serum transaminase elevations were screened for other possible causes, and cases with viral hepatitis, alcoholic hepatitis, and biopsy-proven liver amyloidosis were excluded from the present study. The diagnosis of AA amyloidosis was confirmed using Congo red staining and immunohistochemistry. Among patients in the elevated transaminase group, who remained under regular follow-up visits during the past three years, the most recent transaminase levels were re-evaluated. The Ethics Committee of Istanbul University Faculty of Medicine approved the study protocol (2017-514).

Statistics

The results are presented as mean (with standard deviation) for continuous variables and as percentages for discrete variables. For the comparison of differences between groups, an independent t-test was applied for normally

Competing interests: none declared.

distributed continuous variables, and a chi-square test was applied for discrete variables. Multiple group comparisons were performed with the Kruskal-Wallis test with Bonferroni's correction. To address potential confounding factors associated with transaminase elevation, a multivariable logistic regression analysis was performed. Variables selected based on clinical relevance and univariate comparisons included sex, age at disease onset, follow-up duration, attack frequency, and family history of FMF. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. For all analyses, a *p*-value of <0.05 was considered significant. Statistical analyses were performed using SPSS 17.0 software (IBM Corp., NY, USA).

Results

Records of 780 patients with FMF were prepared, and 42 of them (5.3%) were excluded because of the insufficient follow-up duration, associated viral hepatitis, alcoholic hepatitis, or biopsy-proven liver amyloidosis. In the remaining patients, 83 patients (11.2%) with elevated serum transaminase levels were identified. Their findings were compared with those of 655 patients (83.5%) with normal serum transaminase levels as a control group.

Demographic features and presentation characteristics

Demographic features and presentation characteristics of the patients are given in Table I. There were 38 (45.8%) males and 45 (54.2%) females in the group with elevated transaminase levels, whereas 228 (34.8%) males and 427 (65.2%) females were in the group with normal transaminase levels (*p*=0.049). FMF attacks began at a significantly older age in the elevated transaminase group (17.0±12.9 years) compared to the normal transaminase levels group (12.4±9.0 years), (*p*=0.01). The mean diagnostic delay was 15.7±14.1 years in the elevated transaminase levels group, and 12.5±10.8 years in the control group (*p*=0.051).

There was no significant difference in the presenting manifestations of FMF, including fever, peritonitis, pleuritis, arthritis, and erysipelas-like erythema,

Table I. The comparison of presentation characteristics and laboratory findings of FMF patients with and without transaminase elevation.

Characteristic	Elevated transaminase (Study) group (n=83)	Normal transaminase (Control) group (n=655)	<i>p</i> -value
Males/Females, n	38/45	228/427	0.049
Age at the onset of FMF, mean ± SD years	17.2 ± 12.9	12.4 ± 9.0	0.01
Age at diagnosis, mean ± SD years	28.5 ± 13.7	25.5 ± 11.1	0.059
Diagnostic delay, mean ± SD years	15.7 ± 14.1	12.5 ± 10.8	0.051
Family history of FMF, n (%)	53 (63.8)	347 (53)	0.07
Family history of amyloidosis, n (%)	12 (14)	75 (11)	0.44
Baseline CRP, mean ± SD mg/L	4.6 ± 8.3	6.1 ± 8.9	0.13
CRP (during attack), mean ± SD mg/L	80.0 ± 70.1*	91.3 ± 70.1**	0.06
Attack duration, mean ± SD years	3.1 ± 1.7	3.02 ± 1.1	0.81
Fever, n (%)	78 (94)	578 (88.2)	0.16
Peritonitis, n (%)	74 (89.1)	608 (92.8)	0.12
Pleuritis, n (%)	38 (45.7)	320 (48.9)	0.25
Arthritis, n (%)	49 (59)	368 (56.2)	0.70
Erysipelas-like erythema, n (%)	11 (13.2)	68 (10.3)	0.88
Attack frequency (n, %)			
<1 attacks/month	19 (23.1)	206 (26.3)	0.11
≤ 2 attacks/month	34 (41.5)	302 (49.6)	0.21
≥ 3 attacks/month	29 (35.4)	147 (24.1)	0.01

CRP: C-reactive protein; SD: standard deviation.

*CRP levels during attacks were available for 70 (84.3%) patients elevated transaminase group.

**CRP levels during attacks were available for 614 (93.7%) patients in normal transaminase group.

between the two groups (Table I). However, patients with highly frequently recurring inflammatory attacks (≥3 attacks/month) were more common in the elevated liver enzyme group (35.4% vs. 24.1%, *p*=0.01).

The mean age of the FMF diagnosis (28.5±13.7 vs. 25.1±11.1 years), family history for FMF (63.8% vs. 53%), and family history for amyloidosis (14.6% vs. 11.7%) were not significantly different between the study and control groups. Mean attack duration was similar in both groups (3.1±1.7 days in the study, 3.02±1.1 in the control group). CRP levels during attacks were available for 70 patients (84.3%) in the elevated transaminase group and for 614 patients (93.7%) in the normal transaminase group, respectively. Mean values of the CRP noted during FMF attack and between attacks were not significantly different in the study (80±70.1 mg/dl and 4.6±8.3 mg/dl, respectively) and control groups (91.3±70 mg/dl and 6.1±8.9 mg/dl, respectively).

Genetic evaluation

The *MEFV* gene variant analysis was available in 73.8% of the elevated and 63.8% of the normal transaminase group. The frequencies of exon 10

variants were similar between the two groups (Table II). Homozygous M694V was found in 24.2% of patients in the elevated transaminase group and 30.8% in the control group.

Assessment of risk factors

associated with elevated liver enzymes

Body mass index (BMI) data were available for 57 patients (68.7%) in the elevated liver enzyme group. Among them, 16 (28.1%) had a normal BMI, 31 (54.4%) were overweight (BMI = 25–30), and 10 (17.5%) were classified as obese (BMI >30). Serum total cholesterol levels were measured in 56 patients in this group, and 13 (23.2%) had hypercholesterolaemia (total cholesterol >240 mg/dL). Hypertriglyceridaemia (serum triglycerides >200 mg/dL) was observed in 13 (21.7%) of the 60 patients with available data. Data for BMI and serum lipids were not available in the charts of the control group. To account for the significantly longer follow-up duration observed in patients with elevated transaminase levels, a multivariable logistic regression analysis was performed (Table IV). Male sex (OR 2.39, 95% CI 1.20–4.78, *p*=0.014), older age at disease onset (OR 1.05 per year, 95% CI 1.02–1.09, *p*=0.002),

Table II. Genetic testing results of FMF patients with and without transaminase elevation.

Genetic testing	Elevated transaminase group*	Normal transaminase group**	<i>p</i> -value
Exon 10 variants, n (%)			
One pathogenic variant	20 (32.3)	108 (25.8)	0.28
Two pathogenic variants	33 (52.2)	241 (57.7)	0.51
M694V homozygous	15 (24.2)	129 (30.8)	0.29

*MEFV analysis was available for 62 (73.8) patients in the elevated transaminase group.

**MEFV analysis were available for 418 (63.8) patients in the normal transaminase group.

Table III. Multivariable logistic regression analysis for elevated transaminases.

Variable	OR	95% CI	<i>p</i> -value
Male sex	2.53	1.14–5.60	0.023
Age at disease onset, per year	1.07	1.03–1.11	0.001
Follow-up duration, per year	1.16	1.10–1.22	<0.001
Frequent attacks*	0.56	0.23–1.41	0.223
Family history of FMF	1.74	0.74–4.07	0.202
AA amyloidosis	2.55	0.48–13.66	0.275
Exon 10 mutation	3.34	0.86–12.91	0.081

*Frequent attacks were defined as ≥ 3 attacks within 6 months.

Table IV. Disease course and treatment characteristics of FMF patients according to liver enzyme status.

Characteristics	Elevated transaminase group (n=83)	Normal transaminase group (n=655)	<i>p</i> -value
Follow-up duration, mean $\pm$ SD, years	14.9 $\pm$ 10.1	8.1 $\pm$ 6.7	<0.01
Colchicine dose, mean $\pm$ SD, mg/day	1.2 $\pm$ 0.49	1.15 $\pm$ 0.36	0.36
Colchicine resistance, n(%)	8 (61.5)	115 (92)	<0.01
Biologic treatments, n (%)			
Anakinra	13 (15.7)	125 (19.2)	0.43
Canakinumab	2 (15.4)	17 (13.6)	0.91
Presence of hepatosteatosis*, n (%)	37 (48.3%)	N/A	N/A
Presence of amyloidosis, n (%)	10 (12)	118 (18)	0.17
Persistence of transaminase elevation**, n(%)	21 (42.9%)	N/A	N/A

*Abdominal ultrasound was available for 34 (44.1%) patients in the elevated transaminase group.

**Recent transaminase levels were available for 49 (59.0%) of the 83 patients in the elevated transaminase group.

longer follow-up duration (OR 1.11 per year, 95% CI 1.07–1.16, $p < 0.001$), and family history of FMF (OR 2.22, 95% CI 1.07–4.60, $p = 0.032$) were independently associated with transaminase elevation. Attack frequency was not independently associated with transaminase elevation ($p = 0.926$). Carrying at least one exon 10 mutation did not reach conventional statistical significance (OR 3.34, 95% CI 0.86–12.91, $p = 0.081$), the magnitude of the observed effect suggests a possible association.

Disease course

The mean $\pm$ SD follow-up duration was 14.9 $\pm$ 10.1 years in the elevated

serum transaminase group and 8.1 $\pm$ 6.7 years in the control group, respectively ($p < 0.01$) (Table IV). The mean initial daily colchicine dose was 1.2 $\pm$ 0.49 mg/day in the elevated liver enzyme group and 1.15 $\pm$ 0.36 mg/day in the normal liver enzyme group ($p = 0.35$).

During the follow-up, 13 patients in the study group (15.7%) and 125 patients in the control group (19.2%) required an additional anti-IL-1 treatment (anakinra) on top of colchicine ($p = 0.43$). The main reason for starting anakinra was colchicine resistance in both groups (8 out of 13 patients (61.5%) in the study and 115 out of 125 patients (92.0%) in the control groups, $p < 0.01$). Among the

patients receiving anakinra, a switch to canakinumab was required in 2 patients (15.4%) of the study, due to anakinra-related skin reactions in both. In the control group, the treatment switched from anakinra to canakinumab in 17 patients (13.6%), most commonly (56.2%) due to inadequate response to anakinra.

Except for 7 of the 83 patients, all the patients with elevated serum transaminase levels had abdominal ultrasonography, and 37 (48.3%) of them had findings compatible with hepatosteatosis. No evidence of hepatic cirrhosis was reported in these patients.

The prevalence of renal AA amyloidosis was 14.6% in the study and 18.2% in the control groups ($p = 0.166$).

We re-evaluated the elevated liver enzyme group after approximately 6 years. We accessed the recent serum transaminase levels of 49 of the 83 patients (59.0%), who continued their regular follow-up during the past three years. Among these patients, 21 (42.9%) still had elevated transaminase levels, and 28 had levels within normal ranges. Two of the 83 patients underwent liver biopsy during this period, and both showed macrovesicular steatosis. Liver cirrhosis was identified in one patient.

Discussion

This cross-sectional retrospective study revealed that 11.2% of FMF patients followed at a dedicated FMF outpatient clinic of a tertiary referral centre had elevated serum transaminase levels, regardless of colchicine dose, viral and alcoholic hepatitis, or hepatic amyloidosis. Male patients were more frequent, and the mean age at onset of FMF attacks was significantly later in the elevated liver enzyme group. Despite the later onset, attacks were reported to be significantly more frequent among patients with elevated liver enzymes. The diagnostic delay was longer in the elevated liver enzyme group, but the difference was not statistically significant. There was no significant difference between the groups in terms of age at diagnosis, CRP values at baseline and during attack, and the distribution of the pathogenic MEFV variants.

Since patients followed for longer peri-

ods undergo more frequent laboratory assessments, a detection bias cannot be excluded. To address this issue, we performed a multivariable logistic regression analysis adjusting for clinically relevant variables. Longer follow-up duration remained independently associated with transaminase elevation, supporting the possibility that prolonged observation increases the likelihood of identifying liver enzyme abnormalities. However, male sex and older age at disease onset also remained independently associated with transaminase elevation after adjustment, suggesting that the observed findings cannot be explained solely by differences in follow-up duration. Exon 10 mutation carriage demonstrated a trend toward association with transaminase elevation, whereas M694V homozygosity was not independently associated. Although these findings do not establish a direct genetic determinant of liver involvement, they raise the possibility that patients carrying more severe MEFV genotypes may be predisposed to subclinical hepatic abnormalities. Given the limited number of patients with available genetic data and the retrospective design of the study, larger prospective cohorts are needed to clarify whether chronic inflammasome activation associated with severe MEFV variants contributes to persistent liver abnormalities in FMF.

The initial colchicine doses were comparable between the groups. Colchicine unresponsiveness was significantly more frequent in the normal liver enzyme group, and while the rate of amyloidosis was also higher in this group, this difference did not reach statistical significance.

The role of colchicine in the development of liver enzyme abnormalities could not be fully assessed in the present study. Although the initial colchicine doses were comparable between patients with and without transaminase elevation, detailed information regarding cumulative exposure, treatment duration, and maximum tolerated dose was not consistently available because of the retrospective design. Therefore, while our findings do not support an association between initial colchicine dose and transaminase elevation, a po-

tential contribution of long-term colchicine exposure cannot be completely excluded.

In the present study, nearly half of these patients (46.7%) had documented hepatosteatosis, which is in line with the previous study (9). Non-alcoholic fatty liver disease (NAFLD) is a common entity in the healthy population and is the most common cause of serum transaminase elevation in Western countries (11). The prevalence of NAFLD was estimated as 13-31% worldwide (12), but some studies reported higher prevalence rates of NAFLD (48-60%) from Turkey (13). The major risk factors associated with NAFLD include dyslipidaemia, obesity, and metabolic syndrome (11,13). A meta-analysis investigating epidemiological data about NASH reported that 51% and 69% of the patients had obesity and hyperlipidaemia, respectively (15). In our study group, the percentages of FMF patients with obesity and hyperlipidaemia were lower (17.5% for obesity and 23.2% for hyperlipidaemia), but these data were retrospective, and it may have overlooked some findings.

The interpretation of hepatosteatosis in our cohort should be made with caution. Although nearly half of the patients with elevated transaminase levels had ultrasonographic evidence of hepatosteatosis, BMI and metabolic data were not systematically available in the control group. Therefore, we were unable to determine whether the observed frequency of hepatosteatosis differed from that of FMF patients without transaminase elevation or whether it reflected the prevalence expected in the general population. Consequently, no firm conclusions can be drawn regarding a specific association between FMF and metabolic dysfunction-associated liver disease based on the present data. This impression was compatible with the previous study by Rimar *et al.* (15), investigating 27 FMF patients and 150 patients without FMF, who had transaminase elevations for more than 6 months and were diagnosed with NAFLD. Comparison of the two groups showed that FMF patients with NAFLD did not have findings of metabolic syndrome as much as matched controls

with NAFLD. Due to a higher proportion of NAFLD in FMF patients without overt metabolic syndrome, the authors also suggested that these findings might propose a pathogenic association between FMF itself and NAFLD (12).

Our recent study evaluating the potential utility of transient elastography (FibroScan) for assessing hepatic amyloidosis showed that FMF patients without amyloidosis had liver stiffness scores comparable to those with amyloidosis (6.8 vs. 7.15, respectively) (16). These findings highlight the need for further research to be able to characterise liver abnormalities in patients with FMF.

This study has several limitations, primarily related to its retrospective design. Incomplete or missing data in medical records limited the interpretation of findings, although re-evaluation of a subset of the patients provided an advantage over other retrospective analyses. An important limitation of our study was the significantly longer follow-up duration in the elevated transaminase group. Patients followed for a longer period are more likely to undergo repeated laboratory assessments, increasing the probability of detecting liver enzyme abnormalities. To address this potential source of bias, we performed a multivariable logistic regression analysis including follow-up duration and other clinically relevant variables. Although longer follow-up remained independently associated with transaminase elevation, male sex and older age at disease onset also retained independent associations. These findings suggest that the observed differences cannot be explained solely by longer observation time, although residual bias inherent to the retrospective design cannot be completely excluded. Another important limitation was the lack of systematically collected BMI and metabolic data in the control group. This prevented direct comparison of metabolic risk factors between patients with and without transaminase elevation and limited our ability to evaluate the contribution of metabolic syndrome-related mechanisms to hepatosteatosis.

To strengthen the current dataset, future studies should prospectively collect

comprehensive data, particularly on hepatotoxicity, hepatosteatosis, treatment regimens, established risk factors, and the long-term prognosis of patients with elevated serum transaminase levels. Molecular analyses of hepatic function in relation to pathogenic *MEFV* gene variants may also help elucidate the underlying mechanisms of this association.

In conclusion, this retrospective study found that approximately 11.2% of FMF patients had elevated transaminase levels but no association was observed between initial colchicine dose and the presence of transaminase elevation was observed, and ultrasonographic evidence of hepatosteatosis was present in nearly half of these patients. Although limited follow-up data suggest that this condition is generally non-progressive, these observations require validation in larger, prospective cohorts to clarify the potential pathogenic role of FMF in hepatosteatosis.

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