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Association Between E-FABP Levels, Disease Severity, and Metabolic Parameters in Hidradenitis Suppurativa

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ABSTRACT

Introduction: Studies have shown that hidradenitis suppurativa (HS) is associated with obesity, dyslipidemia, hypertension, metabolic syndrome, and some autoinflammatory diseases. Studies on E-FABP, a member of the fatty acid binding protein family, suggest that this protein may be associated with psoriasis, obesity, metabolic syndrome, atherosclerosis, and autoimmune diseases.

Objectives: The aim of this study was to compare serum E-FABP levels in patients with HS and in healthy control subjects and to investigate the relationship between disease severity and metabolic parameters and serum E-FABP levels.

Methods: Forty-four HS patients and 45 healthy subjects were included in the study. In the patient and control groups, hs-CRP, routine biochemistry parameters, and serum lipid profiles were measured. Serum E-FABP levels were also determined by ELISA method. Blood pressure, pulse rate, and body measurements were also recorded in the patient and control groups. Obtained data were analyzed with SPSS 22.0.

Results: Serum E-FABP levels were found to be statistically significantly higher in the HS group than in healthy controls. However, no significant correlation was found between serum E-FABP levels and disease severity scores. The effect of E-FABP on the development of HS was determined by multivariate logistic regression analysis independent of age, sex, BMI, and hs-CRP. It was determined that the increase in E-FABP level increased the risk of HS development independently of other risk factors.

Conclusions: According to this study, E-FABP may be associated with the pathogenesis of HS rather than with disease severity or metabolic conditions.

Introduction

Hidradenitis suppurativa (HS) is a chronic inflammatory disease associated with multiple comorbidities, characterized by the formation of recurrent painful nodules, abscesses, and sinus tracts with foul-smelling discharge, associated with hair follicles in intertriginous areas [1]. Although the pathogenesis of the disease has not been fully elucidated, factors such as genetic predisposition, hormonal and immune disorders, microbiome disorders, follicular occlusion, smoking, and obesity are thought to play a role in the pathogenesis of the disease [2,3]. Inflammation, resulting in follicular hyperkeratosis and dilatation, follicle rupture, and changes in tissue architecture, plays a key role in the pathogenesis of HS [4]. Studies suggest that HS is associated with obesity, dyslipidemia, hypertension, atherosclerosis, metabolic syndrome, diabetes, Crohn's disease, ankylosing spondylitis, and some autoinflammatory diseases [5-7].

Epidermal fatty acid binding protein (E-FABP), a member of the fatty acid binding protein family, is mostly found in the epidermis, but it has also been shown to be widely expressed in immune cells such as T lymphocytes and macrophages, and E-FABP regulates their immunological functions [8]. In a study on psoriasis, E-FABP was found to be associated with inflammation and disease severity [9]. Zhang et al.'s study on mice showed that the increase in E-FABP expression in macrophages triggered skin inflammation through cytokines such as IFN-γ and IL-17 [10]. In addition, studies suggest that E-FABP may be associated with obesity, metabolic syndrome, atherosclerosis, and autoimmune diseases [11-13].

Objectives

To our knowledge, there are no data in the literature on the relationship between E-FABP and HS. The role of E-FABP in the pathogenesis of HS is unknown. The aim of this study was to compare serum E-FABP levels in patients with hidradenitis suppurativa, an inflammatory skin disease, and healthy controls and to investigate the relationship between serum E-FABP levels and disease severity and metabolic parameters.

Methods

This study was approved by the local ethics committee with the decision dated 28.09.2022 and numbered 265. The signed informed consent form was obtained from all participants before inclusion in the study.

Our study included 44 patients (15 females, 29 males) who were diagnosed with HS according to clinical diagnostic criteria, aged between 18-65 years, and who attended the Department of Dermatology and Venereal Diseases. The control group consisted of 45 healthy volunteers (16 females, 29 males) aged 18 years and older who attended our clinic for any skin problem, who did not have any inflammatory skin or systemic disease, and who did not use any systemic medication according to the anamnesis and examination results.

The patient group included individuals who had no inflammatory disease other than HS, who had not received systemic treatment for hidradenitis suppurativa in the preceding two months, had not received topical treatment for HS in the preceding one month, and had not used any medication in the previous week. Additionally, pregnant and breastfeeding women, those with diabetes mellitus, active or past malignancy history, chronic kidney, liver, heart failure, additional inflammatory and autoimmune diseases, those with active infection, and those who did not approve the voluntary informed consent form were not included in the study.

Included in the patient and control groups were individuals with similar characteristics in terms of number, sex, age, and body mass index (BMI). Patients were questioned about their height, weight, disease duration, age at onset, family history, presence of additional systemic disease, and active medication use. Blood pressure, pulse, waist circumference, and hip circumference measurements were taken from all participants.

Patients were grouped as mild, moderate, or severe HS according to both Hurley staging and International Hidradenitis Suppurativa Severity Score System (IHS4) scoring. Hurley stage I was evaluated as mild disease, stage II as moderate, and stage III as severe disease (7, 26, and 11 patients, respectively). According to the IHS4 score, ≤3 points were evaluated as mild, 4–10 points as moderate, and ≥11 points as severe disease (eight, 28, and eight patients, respectively). All patients were divided into two groups as obese and non-obese according to the BMI recommended by the World Health Organization (WHO) (13 and 31 patients, respectively).

Patients were divided into two groups as having and not having metabolic syndrome, those who met or did not meet at least three criteria for the diagnosis of metabolic syndrome according to waist circumference, blood pressure measurement results, laboratory results, and NCEP-ATPIII diagnostic criteria (12 and 32 individuals, respectively).

Biochemical Analyses

Venous blood samples taken after at least 12 hours of fasting were centrifuged at 3000 rpm (revolutions per minute) for 10 minutes. Fasting blood glucose (FBG), blood urea nitrogen (BUN), alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatinine, serum lipid profile, and hs-CRP levels were analyzed. Quantitative measurement of serum E-FABP level was performed by the sandwich enzyme-linked immunosorbent assay (ELISA) method according to the working procedure of the commercial kit (Elabscience Human Fatty Acid Binding Protein 5, Epidermal, ELISA Kit, Catalog no: E-EL-H1086, Wuhan, China).

Statistical Analysis

SPSS Statistics 22.0 program was used to analyze the data. The values of the descriptors are presented as mean ± standard deviation or median (interquartile range) according to the distribution feature. Categorical variables are given as frequency and percentage. The Shapiro-Wilk test was used to evaluate the conformity of continuous variables to normal distribution. In the comparison of two independent groups, the independent samples T-test was used for variables showing normal distribution, and the Mann-Whitney U test was used for variables not showing normal distribution. Kruskal-Wallis tests were used for comparisons of more than two groups. The significance of the linear relationship between E-FABP and other variables was determined using the Spearman correlation test. P-value was 0.05, and small differences were considered statistically significant.

Results

There was no significant difference between the two groups in terms of sex distribution, age, height, weight, BMI, waist circumference, or hip circumference ($P>0.05$). In the patient group, which had a median disease duration of 5 years, SBP, pulse, hs-CRP, and triglyceride levels were found to be statistically significantly higher compared to healthy controls, while HDL-C levels were found to be statistically significantly lower. There was no significant difference between the two groups in terms of FBG, AST, ALT, BUN, Cre, LDL-C, TC, or DBP levels (Table 1).

The median values of serum E-FABP levels in the patient and control groups were found to be 11.47 (10.56) and 7.61 (9.17), respectively, and a statistically significant difference was found between the two groups in terms of serum E-FABP levels ($P=0.006$). (Figure 1). According to the severity of the disease, no significant difference was found between the mild, moderate, and severe groups in terms of serum E-FABP levels according to both Hurley and IHS4 staging modalities ($P=0.578$ and $P=0.684$, respectively).

In the patient group, E-FABP levels of both the obese and non-obese patients were found to be significantly higher than the healthy control group ($P=0.041$, $P=0.016$). However, no statistically significant difference was found between the serum E-FABP levels of the obese and non-obese participants in the patient group and between the serum E-FABP levels of those with and without metabolic syndrome in the patient group ($P=0.949$ and $P=0.649$, respectively).

In the patient group, correlations between E-FABP and height, weight, BMI, waist circumference, hip circumference, blood pressure, pulse rate, disease severity modalities, hs-CRP, BUN, Cre, AST, ALT, FBG, serum lipid profile, and pack/year in smokers were examined; no correlation was found with E-FABP in terms of these parameters (Table 2).

In the correlations of Hurley and IHS4 scores with hs-CRP, cigarettes (pack/year), weight, waist circumference, BMI, pulse, systolic blood pressure and diastolic blood pressure parameters; a positive correlation was found between Hurley stage and hs-CRP, weight, waist circumference. A positive correlation was found only between IHS4 score and pulse rate (Table 2).

The effects of serum E-FABP and hs-CRP levels on the development of HS were evaluated using univariate logistic regression analysis. It was determined that the increase in E-FABP and hs-CRP levels was effective in the development of HS ($P=0.011$ and $P=0.017$, respectively). The independent effect of this relationship with E-FABP on other risk factors known to play a role in the development of HS (age, sex, BMI) and hs-CRP was evaluated using multivariate logistic regression analysis (model 1 and model 2). It was determined that the increase in E-FABP levels increased the risk of HS development independently of other risk factors (Table 3).

Discussion

This study is the first to examine the relationship between serum E-FABP levels and HS; serum E-FABP levels were found to be significantly higher in the patient group compared to healthy controls. In addition, the patient group, which was further divided into as obese and non-obese, found that E-FABP levels of both obese and non-obese patients were

Table 1. Descriptive, clinical, and laboratory findings in HS patients and healthy controls (HC).

	HS	HC	p
Sex Female (n, %) Male (n, %)	15 (34.1) 29 (65.9)	16 (35.6) 29 (64.4)	0.885
Age (year)*	31.48 ± 9.55	31.56 ± 7.69	0.966
Disease duration	5 (7)	-	-
BMI	27.72 (8.40)	27.14 (4.86)	0.485
IHS4	6 (6)	-	-
Hurley	2 (1)	-	-
FBG	93.50 (15)	93 (13)	0.579
Cre*	0.78 ± 0.16	0.80 ± 0.16	0.527
ALT	19 (15)	18 (18)	0.850
AST	17 (7)	17 (7)	0.931
TC*	195.30 ± 48.61	190.28 ± 30.60	0.563
TG	126 (91.14)	84 (55.50)	**0.001**
LDL-C*	120.20 ± 42.09	118.02 ± 26.08	0.770
HDL-C	46.39 (15.50)	50 (13.87)	**0.034**
hs-CRP	3.37 (5.33)	1.32 (2.25)	**0.001**
WC*	98.52 ± 17.16	97.40 ± 12.54	0.726
HC*	109.95 ± 10.45	107.76 ± 8.74	0.284
SBP	118.77 (20)	110 (10)	**0.031**
DBP	80 (10)	80 (10)	0.641
Pulse*(beats/mn)	81.66 ± 12.61	76.58 ± 10.75	**0.044**
E-FABP	11.47 (10.56)	7.61 (9.17)	**0.006**

Data expressed as median (±IQR) and *mean (±SD); IHS4: International Hidradenitis Suppurativa Severity Score System; BMI: body mass index (body weight (kg)/height (cm)^2; FBG: fasting blood glucose; Cre: creatinine; ALT: alanine aminotransferase; AST: aspartate aminotransferase; hs-CRP: high sensitive c-reactive protein; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TC: total cholesterol; TG: triglyceride; WC: waist circumference; HC: hip circumference; SBP: systolic blood pressure; DBP: diastolic blood pressure; mn: minute

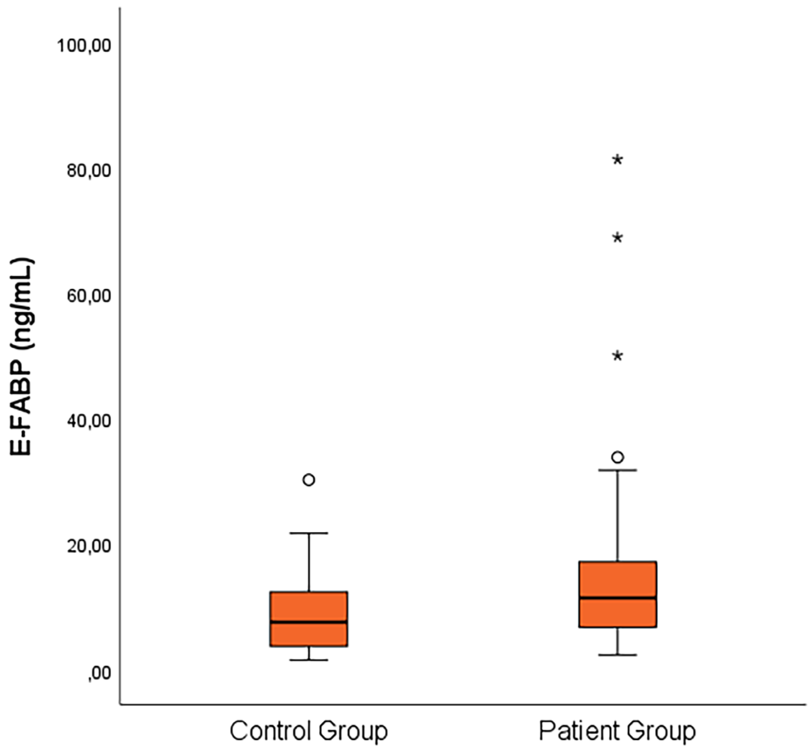

Figure 1. Serum E-FABP levels in the patient and control groups.

Table 2. Correlations of serum E-FABP levels with disease severity modalities and metabolic parameters.

	hs-Crp	IHS4	Hurley	HDL-C	TG	WC	BMI	Pulse
E-FABP								
r	-0.101	0.035	-0.159	0.252	0.091	-0.070	-0.017	-0.061
p	0.514	0.822	0.301	0.099	0.557	0.652	0.911	0.694
hs-Crp								
r		0.265	0.427	**-0.378**	**0.427**	0.284	0.265	0.091
p		0.082	**0.004**	**0.011**	**0.004**	0.062	0.082	0.557
IHS4								
r			0.743	-0.077	0.195	0.151	0.170	**0.331**
p			<0.0001	0.620	0.203	0.328	0.269	**0.028**
Hurley								
r				-0.143	0.148	**0.338**	0.295	0.209
p				0.355	0.338	**0.025**	0.052	0.172
HDL-C								
r					-0.273	-0.184	-0.205	0.107
p					0.073	0.231	0.182	0.489
TG								
r						**0.340**	0.260	0.063
p						**0.024**	0.088	0.682
WC								
r							**0.901**	0.143
p							**<0.0001**	0.354
BMI								
r								0.145
p								0.309

Abbreviations: hs-CRP: high sensitive c-reactive protein; IHS4: International Hidradenitis Suppurativa Severity Score System; HDL-C: high-density lipoprotein cholesterol; TG: triglyceride; WC: waist circumference; BMI: body mass index; r: Spearman correlation coefficient

Table 3. Effect of E-FABP and its levels on HS disease.

Models	OR (95% CI)	*P*
Univariate model	1.076 (1.017-1.140)	*P*=0.011
Multivariate model 1*	1.075 (1.016-1.139)	*P*=0.013
Multivariate model 2**	2.268 (1.414-3.639)	*P*=0.001

Abbreviations: OR; odds ratio, CI; confidence interval, *Model 1: Adjusted for age, sex, BMI. *Model 2: Adjusted for age, sex, BMI, and hs-CRP.

significantly higher than the healthy control group, but no significant difference was found between the median E-FABP levels of patients with metabolic syndrome and the median E-FABP levels of the healthy group in the patient group, which was further divided into those with and those without metabolic syndrome.

Inhibition of E-FABP, which is highly expressed in psoriatic epidermis, significantly reduced keratinocyte differentiation and caused significant changes in psoriatic markers. These data support the role of E-FABP in keratinocyte differentiation [14]. In the initial stage of HS pathogenesis, increased ductal keratinocyte production, overstimulated by various factors in genetically predisposed individuals, is thought to contribute to follicular obstruction and early inflammation [15]. Because E-FABP plays a strong role in keratinocyte differentiation, we think that E-FABP may play a role in the pathogenesis of HS by increasing follicular hyperkeratinization, which causes follicular obstruction in the early stages of HS disease.

Different studies have found that E-FABP has a significant relationship with skin inflammation [10,16,17]. In a study conducted on mice by Zhang et al., the authors reported that high levels of E-FABP expression in macrophages in obese mice fed a high-fat diet may have promoted inflammation by producing cytokines such as IL-1β, IL-18, IFN-γ, and IL-17. It has also been reported that E-FABP-deficient mice are completely resistant to high-fat diet-induced skin lesions. Based on these findings, the authors showed that the increase in E-FABP is a critical regulator of skin inflammation [10]. It is thought that high-level expression of E-FABP in immune cells may also pave the way for the development of autoimmune diseases by promoting inflammation mediated by IL-17 and IFN-γ [13]. Using experimental autoimmune encephalomyelitis modeling, mice specifically lacking epidermal FABP were shown to be protected against the development of encephalomyelitis, and dendritic cells lacking E-FABP were defective in supporting Th1 and Th17 responses [13].

Hidradenitis suppurativa is known to exhibit pathogenetic features of both neutrophilic dermatoses and autoinflammatory disorders (inflammasome-mediated IL-1β dominance). Th1 and Th17 immune cells also make a strong contribution to the pathogenesis of hidradenitis suppurativa [18]. We believe that the E-FABP molecule may also play a role after the initial stage of HS pathogenesis due to its connection with the IL-1β and IL-17 pathways in the studies mentioned above and that similar pathways play an important role in the pathogenesis of HS. In the experimental autoimmune encephalomyelitis model mentioned above, a novel E-FABP inhibitor was identified, and further analyses in MS animal models demonstrated therapeutic benefit in improving clinical symptoms. Therefore, these data suggest that this newly identified inhibitor may be a novel drug candidate for the treatment of MS and other autoimmune diseases [13]. Developing a similar inhibitor model for HS, which does not yet have an effective treatment, may offer new hope for treatment. In addition, monitoring serum E-FABP levels in HS disease with currently available IL-17 inhibitor treatments may provide a new guide in elucidating disease pathogenesis and monitoring treatment responses.

In different studies, it was observed that E-FABP levels were higher in the serum of psoriatic patients or in skin with psoriatic lesions compared to the healthy control group, and it was determined that E-FABP levels decreased significantly in the serum or in skin with lesions after clinical treatment of psoriatic disease [9,19–21]. In the study of Kozlowska et al., E-FABP levels were found to be correlated with the disease severity score and CRP levels [9]. In this study, similar to our study, the patient group was divided into obese and non-obese; in both groups, serum E-FABP levels were found to be statistically significantly higher than the serum E-FABP levels of the control group. Although the BMIs of the patient and control groups were similar in our study, the fact that serum E-FABP levels of both the obese and non-obese groups in the patient group were significantly higher than the E-FABP levels of the healthy control group suggests that E-FABP is related to the pathogenesis of HS disease rather than the obesity factor.

There are a limited number of studies conducted on different populations investigating serum E-FABP levels and metabolic diseases in humans; the correlations of serum E-FABP levels with BMI, waist circumference, FBG, TG, HDL-C, LDL-C, CRP, SBP, DBP, and other metabolic and inflammatory parameters in these studies are contradictory, and different results have been found [12,22–25]. However, in general, it is seen that E-FABP levels are positively correlated with waist circumference, TG levels, and CRP levels and negatively correlated with HDL-C. In our study, no correlation was found between E-FABP levels and these metabolic or inflammatory parameters. Therefore, studies with a larger number of unique populations are needed to further elucidate the relationship between E-FABP and metabolic parameters.

In our patient group, SBP, pulse, hs-CRP, and triglyceride levels were found to be statistically significantly higher, while HDL-C levels were found to be statistically significantly lower compared to healthy controls. A positive correlation was found between Hurley stage, one of the disease severity modalities, and hs-CRP, weight, and waist circumference. However, a positive correlation was found only between IHS4 score and pulse rate. According to all these results, it is seen that HS may be associated with low HDL-C and high TG levels, and heart rate is affected as the disease severity increases. This supports the view that HS patients may also be at risk in terms of cardiac disease. In addition, according to our study, being overweight and having a high waist circumference can also be considered as risk factors for severe disease. In an HS study by Miller et al., higher hs-CRP levels were found in the patient group compared to the control group, and the authors stated that this situation indicated the systemic inflammation burden in HS disease rather than infection [26]. In our study, we concluded that hs-CRP is an important risk factor for the development of HS disease in the univariate logistic regression analysis performed with hs-CRP levels (P=0.017). According to all these results, high hs-CRP levels suggest that there is inflammation in the background of the disease and that this inflammation may play an important role in both disease severity and disease pathogenesis.

The effect of E-FABP on the development of HS was determined by multivariate logistic regression analysis independent of age, sex, BMI, and hs-CRP. It was determined that the increase in E-FABP level increased the risk of HS development independently of other risk factors. (Table 3). Therefore, according to this study, E-FABP may be related to the pathogenesis rather than to the severity of hidradenitis suppurativa disease or metabolic conditions. However, studies on large case series at both serum and tissue levels are needed to elucidate this relationship.

The limitations of our study are that the serum was not examined simultaneously in tissue and that the number of cases was relatively small. In addition, the fact that inflammatory cytokines such as IL-17 and IL-1β, which may be associated with E-FABP, which plays a role in the etiopathogenesis of the disease, were not studied simultaneously in our study is an important limitation for the elucidation of the pathogenesis.

The strengths of our study include that it is an original study examining E-FABP in HS patients. HS patients were evaluated using two different disease staging systems (Hurley and IHS4).

Conclusions

In conclusion, E-FABP levels in hidradenitis suppurativa patients were examined in detail in our study, which aimed to reveal the relationship with disease severity and accompanying metabolic disorders. E-FABP levels were found to be statistically significantly higher in the patient group than in the control group, but no significant relationship was found with obesity, metabolic syndrome, or disease severity. E-FABP was found to be an independent risk factor in the development of HS.

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