ORIGINAL ARTICLE

CONTENTS

The role of lipid metabolism disorders and dysbiotic changes in metabolically dysfunction-associated steatotic liver disease combined with acne vulgaris

Iryna Smolanka , Yelyzaveta S. Sirchak
UZHHOROD NATIONAL UNIVERSITY, UZHHOROD, UKRAINE

ABSTRACT

Aim: To determine the features of hepatic lipid metabolism disorders and intestinal dysbiotic changes in patients with acne vulgaris and metabolic dysfunction-associated steatotic liver disease (MASLD)

Materials and Methods: The study included 109 patients: 59 with a combination of acne vulgaris and MASLD and 50 with acne only.

Results: It was found that patients with acne and MASLD are more likely to be overweight and have more pronounced changes in hepatic lipid metabolism. Also, patients with combined pathology showed a decrease in the number of the colonic normoflora , as well as an increase in the number of hemolytic forms of E. coli, Enterobacter, Citrobacter, Staphylococcus, Klebsiella, Clostridium, Proteus, Candida spp.

Conclusions: Our study highlighted that lipid profile determination and anthropometric examination are important factors in the diagnosis of patients with common acne and MASLD. Our data on changes in the composition of the colonic microflora in patients with comorbidities confirmed the existence of a relationship between the presence of intestinal dysbacteriosis and impaired skin homeostasis.

KEY WORDS: acne vulgaris; hepatic lipid metabolism disorders; intestinal dysbacteriosis; metabolic dysfunction-associated steatotic liver disease; microbiota; obesity

Wiad Lek. 2025;78(4):806-812. doi: 10.36740/WLek/203890 DOI

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease is now the most common cause of chronic liver disease in the Western world with a prevalence ranging from 5 to 33%[1]. This disease is characterized by the accumulation of fat in the liver. The histological changes are identical to alcoholic liver disease, but patients do not drink alcohol or drink it minimally [2]. Epidemiological studies [3] support a trend toward an increased incidence of MASLD in patients with insulin resistance (IR), which occurs in the context of obesity, type 2 diabetes mellitus (DM), and the presence of metabolic syndrome (MS). Some studies have shown that MASLD precedes the development of MS [4]. The literature has shown that MS is associated with inflammation, as well as increased levels of pro-inflammatory cytokines such as IL-6, TNF-α, and C-reactive protein[5,7]. The chronic inflammatory process of acne vulgaris is known to be associated with the development of MS [7,8]. In addition, other skin diseases with a similar pathogenesis as acne, such as psoriasis, rosacea and hidradenitis suppurativa, already have a well-established association with MS [7,8].

According to the literature, the pathogenesis of acne is explained by four key factors: excess sebum production, hyperproliferation of the bacteria *Cutibacterium acnes* (C. acnes, formerly called *Propionibacterium acnes*), hyperkeratinization of the sebaceous follicle, and inflammatory mechanisms [9]. Excess sebum production is mediated by several hormones, such as androgens, insulin, and insulin-like growth factor 1 (IGF-1) [10]. Moreover, there is increasing information about the relationship between acne and insulin resistance [11,12]. Interestingly, MASLD is found in patients with insulin resistance without obesity and without diabetes, which means that when developing in individuals with normal weight, it can be a predictor of early metabolic disorders and diseases [13,14].

The diseases mentioned above share common pathogenetic factors in their development and progression, therefore, they can combine and potentiate the development of each other.

AIM

Our study aimed to determine the features of hepatic lipid metabolism disorders and intestinal dysbiotic changes in patients with acne vulgaris and MASLD.

MATERIALS AND METHODS

Patients with acne vulgaris and MASLD were enrolled in the study. It was conducted from January 2019 to September 2024 at the clinical base of the Department of Propedeutics of Internal Diseases of the State Educational Institution "Uzhhorod National University".

The inclusion criteria were:

- age of at least 18 years;
- a diagnosis of mild or moderate acne vulgaris (2-3 points on the IGA scale) with the presence of symptoms for more than 6 months;
- a diagnosis of MASLD with or without liver fibrosis.

Exclusion criteria included: pregnancy; current use of any acne medications; use of oral or topical antibiotics or retinoids within last 2 months; alcoholic, viral (hepatitis B, C, D viruses), autoimmune liver fibrosis; Wilson-Konovalov's disease; hemochromatosis; chronic inflammatory bowel diseases (Crohn's disease, nonspecific ulcerative colitis); lactose intolerance; celiac disease; intestinal surgeries (including appendectomy with a duration of up to 6 months); colon cancer; doligosigma; colon diverticulosis; positive test for toxins A and B of Clostridium difficile bacteria in feces; type 1 diabetes mellitus; type 2 diabetes mellitus (decompensation stage); pulmonary tuberculosis (active form); psychiatric diseases; pregnancy and lactation; systemic autoimmune diseases; HIV; oncology.

STUDY DESIGN

109 participants were enrolled in the study with informed consent. 34 men (31.2 %), with an average age of 30.8±6.5 years and 75 women (68.8 %), with an average age - of 29.9±6.7 years.

Patients were divided into two groups:

- group 1 (n= 59) — patients with mild/moderate acne vulgaris and MASLD;
- group 2 (n= 50) — patients with mild/moderate acne vulgaris.

All participants were scheduled for the following examinations:

- assessment of the severity of acne using the Investigator's Global Assessment (IGA) scale;
- standardized photo-fixation of the face in three projections;
- blood lipidogram (plasma total cholesterol (TC), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), very low-density lipoproteins (VLDL), triglyceride (TG), atherogenic index) and insulin-like growth factor-1 (IGF-1) ;
- the liver function tests — alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), serum bilirubin;
- assessment on scales for the degree of liver fibrosis — NFS (NAFLD fibrosis score), FIB-4 (Fibrosis-4 index), Fibrotest, FibroIndex, Forns, APRI (AST to platelet ratio index);
- liver transient elastography;
- microbiological examination of feces;
- abdominal and pelvic ultrasonography.

The severity of acne was assessed by certified dermatologists who had previous experience with the scales that were used in the study. Diagnosis of MASLD and assessment of fibrosis severity was performed by a certified gastroenterologist according to scales and sonography in adherence to EASL–EASD–EASO clinical recommendations. Whole venous blood for lipid

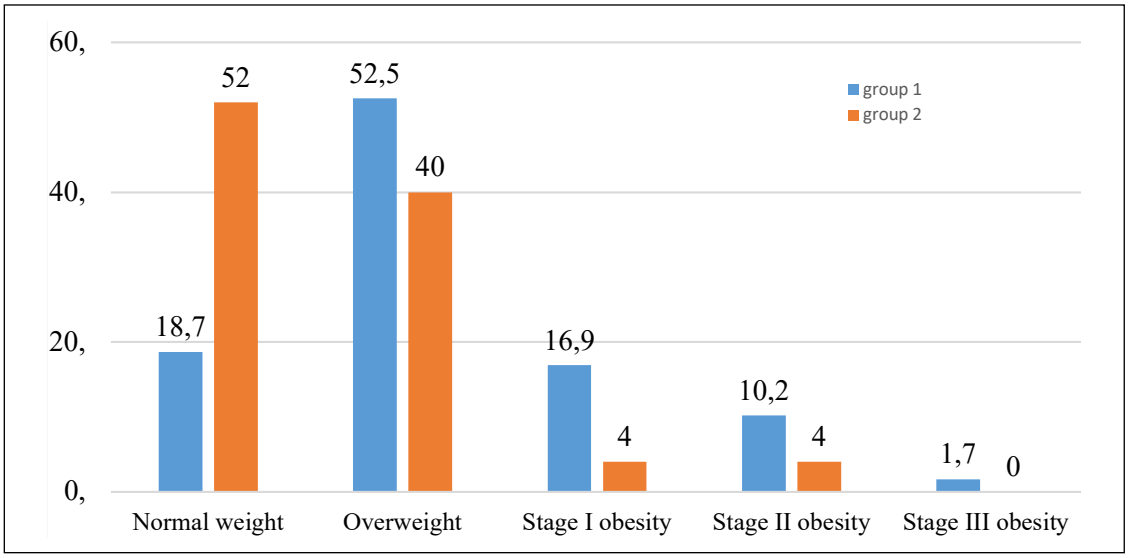

Fig. 1. Changes in BMI in patients with acne vulgaris and MASLD.

Table 1. Changes of indicators of quantitative and qualitative composition of colon microbiota

Indicator	Group 1 (n=59)	Group 2 (n=50)
***Bifidobacterium*: Control group 100.0 % (8.65±0.26)**		
frequency, %	67.8 %**	80.0 %*+
lg CFU/gr	5.88±0.09**	7.76±0.10*++
***Lactobacillus*: Control group 100.0 % (6.78±0.22)**		
frequency, %	66.8 %**	85.0 %*+
lg CFU/gr	5.01±0.10**	6.06±0.11*+
***E.coli* (with normal enzymatic properties): Control group 93.3 % (7.87±0.15)**		
frequency, %	71.2 %*	80.0 %*+
lg CFU/gr	5.28±0.07**	6.87±0.07*++
***E.coli* (haemolytic form): Control group 3.3 % (1.12±0.07)**		
frequency, %	20.3 %**	10.0 %*+
lg CFU/gr	4.77±0.15***	2.51±0.23**++
***Enterococcus*: Control group 90.0 % (7.56±0.11)**		
frequency, %	44.1 %***	70.0 %**++
lg CFU/gr	6.25±0.16*	7.08±0.08*+
***Enterobacter*: Control group 23.3 % (1.15±0.12)**		
frequency, %	42.4 %*	34.0 %*+
lg CFU/gr	4.66±0.09***	1.98±0.06**++
***Citrobacter*: Control group 26.7 % (1.47±0.09)**		
frequency, %	52.5 %**	30.0 %++
lg CFU/gr	3.56±0.12**	2.98±0.09*+
***Staphylococcus*: Control group 26.7 % (3.48±0.22)**		
frequency, %	64.4 %**	34.0 %++
lg CFU/gr	5.54±0.16**	4.12±0.06*++
***Klebsiella*: Control group 16.7 % (1.29±0.09)**		
frequency, %	45.8 %**	20.0 %+++
lg CFU/gr	3.29±0.18**	2.38±0.10**++
***Clostridium*: Control group 13.3 % (4.22±0.18)**		
frequency, %	32.2 %**	18.0 %++
lg CFU/gr	5.80±0.15**	4.98±0.09*+
***Proteus*: Control group 6.7 % (0.55±0.09)**		
frequency, %	28.8 %**	12.0 %*+
lg CFU/gr	2.50±0.08***	1.56±0.10**+
***Candida*: Control group 3.3 % (2.89±0.20)**		
frequency, %	15.3 %*	8.0 %
lg CFU/gr	4.98±0.09**	3.77±0.11*+

Note: the difference between the indicators in patients of groups 1 and 2 and the data of the control group is significant: * - $p<0.05$; ** - $p<0.01$; *** - $p<0.001$; the difference between the indicators in patients by groups 1 and 2 is significant: + - $p<0.05$; ++ - $p<0.01$; +++ - $p<0.001$.

profile determination was collected using appropriate vacutainers and the analysis was performed by the certified laboratory no later than one day after blood collection on Dimension EXL200. Faeces were collected in dry sterile dishes and delivered to the bacteriological laboratory no later than 2 hours after collection without the use of preservatives. The material was inoculated on a standard set of selective and differential diagnostic nutrient media for the isolation of aerobic and anaerobic microorganisms by the method of tenfold dilution (10^{-1}-10^{-9}). Changes in the quantitative and qualitative composition of the colon

Table 2. Severity of colon dysbiosis

The degree of CD	group 1 (n=59)	group 2 (n=50)
I degree of CD	20.3 %	64.0 %+
II degree of CD	64.4 %+	32.0 %
III degree of CD	15.3 %	4.0 %

Note: the difference between the indicators in patients by groups 1 and 2 is significant: + - p<0.01.

Table 3. The indicators of liver function in blood serum

Indicator	Group 1 (n=59)	Group 2 (n=50)
ALT, U/l	93.41±0.44**	32.14±0.32**
AST, U/l	76.14±0.56***	27.36±0.44***
TB, mmol/l	34.12±0.11*	28.29±0.27*
GGT, U/l	82.03±0.48**	30.67±0.38**
ALP, mmol/l	134.15±0.32**	124.16±0.42*

Note: the difference between the indicators in patients of groups 1 and 2 and the data of the control group is significant: * - p<0.05; ** - p<0.01; *** - p<0.001; the difference between the indicators in patients by groups 1 and 2 is significant: + - p<0.05; ++ - p<0.01; +++ - p<0.001.

Table 4. The indicators of blood lipid metabolism

Indicator	Group 1 (n=59)	Group 2 (n=50)
TC, mmol/l	3.56±0.08**++	2.09±0.07*
TG, mmol/l	7.84±0.09**++	5.98±0.08*
LDL, mmol/l	3.80±0.10**+	2.23±0.07*
VLDL, mmol/l	1.98±0.07**++	0.97±0.11*
HDL, mmol/l	0.99±0.07**+	1.44±0.09*

Note: the difference between the indicators in patients of groups 1 and 2 and the data of the control group is significant: * - p<0.05; ** - p<0.01; the difference between the indicators in patients by groups 1 and 2 is significant: + - p<0.05; ++ - p<0.01.

microflora were determined using the classification of intestinal dysbiosis by Kuvaeva-Ladodo (1991).

The analysis and processing of the results was carried out using the computer program Statistics for Windows v.10.0 (StatSoft Inc, USA) using parametric and nonparametric methods of evaluating the results.

RESULTS

In both groups, there were more women than men but the difference between them was not statistically significant (P=0.5). In Group 1 39 (66,1%) were female and 20 (33.8%) were male, Group 2 were 36 (68.9%) and 14 (31.1%), respectively. The mean ages of patients were 29.7±6.3 and 29.5±5.1, respectively, and there was no statistically significant difference (P=0.61). The age of participants ranged from 18 to 45 years.

BODY MASS INDEX

An anthropometric measurement found an increase in BMI in the vast majority of patients with acne vulgaris and MASLD (Fig. 1). Normal body weight was more often observed in patients from Group II (52% of patients). According to the results obtained, it was found that patients with acne vulgaris and MASLD are more likely to be overweight — obesity of all stages prevailed in Group 1.

CHANGES IN COLON MICROBIOTA

Changes in the quantitative and qualitative composition of the colonic microbiota were diagnosed during microbiological examination of feces. The study revealed a decrease in the number of normoflora (*Bifidobacterium and Lactobacterium, Escherichia coli* with normal enzymatic activity), as well as an increase in the number of hemolytic forms of *E. coli, Enterobacter, Citrobacter, Staphylococcus, Klebsiella, Clostridium, Proteus. and Candida spp* in patients from Group 1.

The changes in the quantitative and qualitative composition of the colon microbiota are presented in Table 1.

THE DEGREE OF COLON DYSBIOSIS

Our study showed that the majority of patients in Group 1 had an II degree of colon dysbacteriosis (CD), while the majority of patients in Group 2 had an I degree of

CD. The degree of colon dysbacteriosis in both groups is presented in Table 2.

THE LIVER FUNCTION TESTS

The results of biochemical blood tests indicate impaired liver function in patients with combined pathology (Group 1). An increase in cholestatic syndrome indicators (levels of ALP, GGT, TB) was observed in Group 1. Interestingly, in Group 2, an increase in serum bilirubin levels was detected while normal levels of AST and ALT (Table 3).

BLOOD LIPIDOGRAM

According to the results of the study, dyslipidemia was detected in both groups. However, these changes were more pronounced in patients with combined disease (Group 1). Table 4 shows the blood lipid metabolism indicators in patients of both study groups.

DISCUSSION

The immunomodulatory potential of the gut microbiota and its influence on distant organs has been increasingly investigated in recent years. Of particular interest are the gut-brain [16], gut-lung [17], gut-liver [18] and gut-skin axes [19,20]. Gut microbes can influence systemic inflammation, oxidative stress, glycemic control, and tissue lipid content [15] Epidemiological studies have assessed the distribution of the gut microbiota between healthy individuals and patients with MASLD. Some studies have observed a decrease in bacterial α- or β-diversity [21-23] microbial taxa, a meta-analysis of 54 studies (8894 participants) found a depletion of anti-inflammatory microbes (i.e. Ruminococcaceae and Coprococcus) and an enrichment of pro-inflammatory microbes (i.e. Fusobacterium and Escherichia) in patients with MASLD [14, 15]. Acne vulgaris has been postulated to have a gastrointestinal mechanism; however, little is known about gut microbiota dysfunction in this condition [16]. The results of our study also confirmed the information about changes in the quantitative and qualitative composition of microbiota in patients with combined pathology of acne vulgaris and MASLD. Statistically reliable data were obtained on a decrease in the number of normoflora (Bifidobacteria and Lactobacteria, Escherichia coli with normal enzymatic activity), as well as an increase in the number of hemolytic forms of E. coli, Enterobacter, Citrobacter, Staphylococcus, Klebsiella, Clostridium, Proteus and Candida spp.

According to the literature, it is known that liver dysfunction plays a significant role in the pathophysiology of chronic inflammatory skin disease. [24,25] Scientists actively discuss the relationship between MASLD and chronic inflammatory dermatological diseases. [24] One of the important indicators of MASLD is the progression of dyslipidemia. This disease leads to the development of the "lipid quartet" — a variant of highly atherogenic dyslipidemia with high titers of triglycerides, very low-density lipoproteins (VLDL), low-density lipoproteins (LDL) and low high-density lipoproteins (HDL) and high Plasma concentration of particles dangerous for the endothelium-intermediate density lipoproteins, the molecular weight of which is between VLDL and LDL [26, 27].

Dyslipidemia was observed in both study groups, which confirms the need for assessing serum lipid levels during acne treatments. According to our results, patients with combined pathology (acne and MASLD) have a worse serum lipid profile compared to patients who have only acne vulgaris.

According to clinical guidelines, the presence, duration and severity of obesity are associated with an increased risk of disease progression in MASLD [28]. Almost all patients are found to be obese (BMI>30 kg/m2) or overweight, which correlates with the degree of hepatic steatosis [29]. According to the literature, acne vulgaris is also known to be significantly associated with changes in lipid profile and body mass index (BMI) [30].

The results of our study also confirmed the relationship between increased BMI and impaired hepatic lipid metabolism. Anthropometric measurements revealed obesity in the vast majority of patients with combined pathology of acne vulgaris and MASLD. This suggests the need for additional examinations in patients with overweight and acne.

The main limitations of the research were the small sample size and cross-sectional study design. Future prospective multi-center studies with larger sample sizes are necessary to validate the results of our study.

CONCLUSIONS

Our study highlighted that altered serum lipid levels are common in patients with acne vulgaris and MASLD, indicating that lipid profile testing is an important factor in the diagnosis of these pathologies. As well as the importance of anthropometric examination, since most patients in the combined pathology group had an increased BMI. Noteworthy, our data obtained on changes in the quantitative and qualitative composition of the colonic microflora in patients with acne vulgaris and MASLD confirmed the relationship between the presence of intestinal dysbacteriosis and impaired skin homeostasis.

REFERENCES

1. Farrell GC, Larter CZ. Nonalcoholic fatty liver disease: from steatosis to cirrhosis. Hepatology. 2006;43(2):S99-S112. doi: 10.1002/hep.20973. DOI
2. Harsha Varma S, Tirupati S, Pradeep TVS et al. Insulin resistance and hyperandrogenemia independently predict nonalcoholic fatty liver disease in women with polycystic ovary syndrome. Diabetes Metab Syndr. 2019;13(2):1065-1069. doi: 10.1016/j.dsx.2018.12.020. DOI
3. Lazo M, Solga SF, Horska A et al. Fatty Liver Subgroup of the Look AHEAD Research Group. Effect of a 12-month intensive lifestyle intervention on hepatic steatosis in adults with type 2 diabetes. Diabetes Care. 2010;33(10):2156-63. doi: 10.2337/dc10-0856. DOI
4. Lonardo A, Ballestri S, Marchesini G et al. Nonalcoholic fatty liver disease: a precursor of the metabolic syndrome. Dig Liver Dis. 2015;47(3):181-90. doi: 10.1016/j.dld.2014.09.020. DOI
5. Adibi N, Robati RM. Skin and metabolic syndrome: A review of the possible associations. J Res Med Sci. 2021;26:16. doi: 10.4103/jrms.JRMS_585_20. DOI
6. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2005;365(9468):1415-28. doi: 10.1016/S0140-6736(05)66378-7. DOI
7. Misitzis A, Cunha PR, Kroumpouzos G. Skin disease related to metabolic syndrome in women. Int J Womens Dermatol. 2019;5(4):205-212. doi: 10.1016/j.ijwd.2019.06.030. DOI
8. Stefanadi EC, Dimitrakakis G, Antoniou CK et al. Metabolic syndrome and the skin: a more than superficial association. Reviewing the association between skin diseases and metabolic syndrome and a clinical decision algorithm for high risk patients. Diabetol Metab Syndr. 2018;10:9. doi: 10.1186/s13098-018-0311-z. DOI
9. Kircik LH. Advances in the Understanding of the Pathogenesis of Inflammatory Acne. J Drugs Dermatol. 2016;15(1):s7-10.
10. Arora MK, Yadav A, Saini V. Role of hormones in acne vulgaris. Clin Biochem. 2011;44(13):1035-1040. doi: 10.1016/j.clinbiochem.2011.06.984. DOI
11. Zacchè MM, Caputo L, Filippis S et al. Efficacy of myo-inositol in the treatment of cutaneous disorders in young women with polycystic ovary syndrome. Gynecol Endocrinol. 2009;25(8):508-13. doi: 10.1080/09513590903015544. DOI
12. Advani K, Batra M, Tajpuriya S et al. Efficacy of combination therapy of inositols, antioxidants and vitamins in obese and non-obese women with polycystic ovary syndrome: an observational study. J Obstet Gynaecol. 2020;40(1):96-101. doi: 10.1080/01443615.2019.1604644. DOI
13. Ivachevska VV. The effect of comprehensive treatment of patients with non-alcoholic fatty liver disease in combination with prediabetes on the lipid profile. Wiad Lek. 2021;74(4):957-760.
14. Ivachevska VV, Ivachevskyi MM, Hechko MM et al. Efficacy of comprehensive treatment of nonalcoholic fatty liver disease in patients with prediabetes. Wiad Lek. 2023;76(3):581-585. doi: 10.36740/WLek202303119. DOI
15. Bowe W, Patel NB, Logan AC. Acne vulgaris, probiotics and the gut-brain-skin axis: from anecdote to translational medicine. Benef Microbes. 2014;5(2):185-99. doi: 10.3920/BM2012.0060. DOI
16. Mayer EA, Nance K, Chen S. The Gut-Brain Axis. Annu Rev Med. 2022;73:439-453. doi: 10.1146/annurev-med-042320-014032. DOI
17. Budden KF, Gellatly SL, Wood DL et al. Emerging pathogenic links between microbiota and the gut-lung axis. Nat Rev Microbiol. 2017;15(1):55-63. doi: 10.1038/nrmicro.2016.142. DOI
18. Albillos A, de Gottardi A, Rescigno M. The gut-liver axis in liver disease: Pathophysiological basis for therapy. J Hepatol. 2020;72(3):558-577. doi: 10.1016/j.jhep.2019.10.003. DOI
19. De Pessemier B, Grine L, Debaere M et al. Gut-Skin Axis: Current Knowledge of the Interrelationship between Microbial Dysbiosis and Skin Conditions. Microorganisms. 2021;9(2):353. doi: 10.3390/microorganisms9020353. DOI
20. Navarro-López V, Núñez-Delegido E, Ruzafa-Costas B et al. Probiotics in the Therapeutic Arsenal of Dermatologists. Microorganisms. 2021;9(7):1513. doi: 10.3390/microorganisms9071513. DOI
21. Si J, Lee G, You HJ et al. Gut microbiome signatures distinguish type 2 diabetes mellitus from non-alcoholic fatty liver disease. Comput Struct Biotechnol J. 2021;19:5920-5930.doi: 10.1016/j.csbj.2021.10.032.
22. Iino C, Endo T, Mikami K et al.Significant decrease in Faecalibacterium among gut microbiota in nonalcoholic fatty liver disease: a large BMI- and sex-matched population study. Hepatol Int. 2019;13(6):748-756. doi: 10.1007/s12072-019-09987-8. DOI
23. Lee G, You HJ, Bajaj JS et al. Distinct signatures of gut microbiome and metabolites associated with significant fibrosis in non-obese NAFLD. Nat Commun. 2020;11(1):4982. doi: 10.1038/s41467-020-18754-5. DOI
24. Gau SY, Huang KH, Lee CH et al. Bidirectional Association Between Psoriasis and Nonalcoholic Fatty Liver Disease: Real-World Evidence From Two Longitudinal Cohort Studies. Front Immunol. 2022;13:840106. doi: 10.3389/fimmu.2022.840106. DOI
25. Bellinato F, Gisondi P, Mantovani A et al. Risk of non-alcoholic fatty liver disease in patients with chronic plaque psoriasis: an updated systematic review and meta-analysis of observational studies. J Endocrinol Invest. 2022;45(7):1277-1288. doi: 10.1007/s40618-022-01755-0. DOI

26. Yamaguchi K, Yang L, McCall S et al. Inhibiting triglyceride synthesis improves hepatic steatosis but exacerbates liver damage and fibrosis in obese mice with nonalcoholic steatohepatitis. Hepatology. 2007;45(6):1366-74. doi: 10.1002/hep.21655. DOI
27. Kleiner DE, Brunt EM. Nonalcoholic fatty liver disease: pathologic patterns and biopsy evaluation in clinical research. Semin Liver Dis. 2012;32(1):3-13. doi: 10.1055/s-0032-1306421. DOI
28. European Association for the Study of the Liver (EASL); European Association for the Study of Diabetes (EASD); European Association for the Study of Obesity (EASO). EASL-EASD-EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). J Hepatol. 2024;81(3):492-542. doi: 10.1016/j.jhep.2024.04.031. DOI
29. Carmiel-Haggai M, Cederbaum AI, Nieto N. A high-fat diet leads to the progression of non-alcoholic fatty liver disease in obese rats. FASEB J. 2005;19(1):136-8. doi: 10.1096/fj.04-2291fje. DOI
30. Sobhan M, Seif Rabiei MA, Amerifar M. Correlation Between Lipid Profile and Acne Vulgaris. Clin Cosmet Investig Dermatol. 2020;13:67-71. doi: 10.2147/CCID.S230617. DOI

CONFLICT OF INTEREST

The Authors declare no conflict of interest

CORRESPONDING AUTHOR

Iryna Smolanka
Uzhhorod National University
14 Universytetska St., 88000 Uzhhorod, Ukraine
e-mail: iryna.smolanka@gmail.com

ORCID AND CONTRIBUTIONSHIP

Iryna Smolanka: 0009-0005-6063-2936 A B D F
Yelyzaveta S. Sirchak: 0000-0001-6738-0843 A C F

A – Work concept and design, B – Data collection and analysis, C – Responsibility for statistical analysis, D – Writing the article, E – Critical review, F – Final approval of the article

RECEIVED: 17.10.2024
ACCEPTED: 22.03.2025