

The study included 88 (20 men, 68 women) patients with proximal femoral fracture aged 29 to 98 years (average age 72.97±13.17 years), among whom 27 cases of DVT were diagnosed by duplex ultrasound of the lower extremities before surgery, which was 30.7%. These 27 patients with preoperative DVT constituted group I; group II included 61 individuals without thromboembolic complications before surgery. The analysis of the relationship between preoperative DVT and clinical characteristics of patients was performed using univariate and multivariate regression analysis.

The most common risk factors for preoperative DVT in the setting of proximal femoral fractures are: female patients, individual anamnesis of VTE, severe pulmonary disease, VTE family history, lower extremity venous disease, osteoporosis, lower extremity inflammation. Significant risk factors for preoperative VTE in patients with proximal femoral fractures were found to be a Caprini thrombosis risk score of >10 and an increase in the time from injury to surgery of >6.5 days. The obtained results may be used to improve preoperative assessment algorithms for patients with proximal femoral fractures. The application of the identified criteria may facilitate timely diagnosis of venous thrombosis and optimize the strategy of preoperative preparation for surgical treatment. The Caprini VTE risk score and the preoperative waiting time are independent predictors of the preoperative DVT development, with an increase of one unit of measurement increasing the risk of thrombosis by 1.485 times (95% CI [1.075–2.051], $p=0.016$) and 1.166 times (95% CI [1.001–1.357], $p=0.048$), respectively.

Key words: proximal femoral fracture, preoperative venous thrombosis, risk factors.

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Автори заявляють про відсутність конфлікту інтересів.

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A – концепція роботи та дизайн, **B** – збір та аналіз даних, **C** – відповідальність за статичний аналіз, **D** – написання статті, **E** – критичний огляд, **F** – остаточне затвердження статті.

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MORPHOLOGICAL FEATURES OF LIVER INJURY IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOHEPATITIS WITH COMORBID CHRONIC KIDNEY DISEASE

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Whether chronic kidney disease (CKD) is accompanied by a distinct biopsy phenotype of metabolic dysfunction-associated steatohepatitis (MASH) remains insufficiently defined. The aim is to determine which liver histological features differ between MASH patients with stage I–III CKD and those without CKD. The study included 60 adults with MASH and class I obesity, 30 in each renal-status group. Three samples from segments V and VII were examined per patient. Steatosis, its acinar distribution, inflammation, hepatocellular degeneration, and fibrosis were recorded. Frequency differences were tested with Pearson's chi-square test; odds ratios (ORs) with 95% confidence intervals (CIs) were also calculated. CKD was accompanied by a higher frequency of S2-S3 steatosis (97% vs 53%; $p=0.0001$), microvesicular steatosis (83% vs 40%; $p=0.0006$), lobular inflammation >4 foci per field (43% vs 10%; $p=0.0035$), and multiple hepatocellular degenerative changes (77% vs 40%; $p=0.0040$). Panacinar steatosis and moderate/marked portal inflammation were likewise more common in the CKD group. F2-F3 fibrosis occurred in 47% versus 23%, but the comparison did not reach the prespecified significance threshold ($p=0.0581$). F4 fibrosis was not observed in either group. In this sample, CKD accompanied MASH with a broader steatotic distribution, stronger inflammatory activity, and more extensive cellular degeneration. The fibrosis comparison remained inconclusive.

Key words: metabolic dysfunction-associated steatohepatitis, chronic kidney disease, liver biopsy, steatosis, fibrosis.

Connection of the publication with planned research work.

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Introduction.

Metabolic dysfunction-associated steatotic liver disease (MASLD) comprises a histological continuum in which simple fat accumulation may coexist with inflammatory and hepatocellular changes characteristic of metabolic dysfunction-associated steatohepatitis (MASH) [1]. Fibrosis may develop as this tissue injury persists, and its stage carries more prognostic information than the amount of steatosis alone [2–4].

Kidney disease enters this clinical picture frequently. Population-based and meta-analytic data link steatotic liver disease with impaired renal function and subsequent chronic kidney disease (CKD) [5]. The overlap is biologically plausible because hepatic and renal injury share metabolic and systemic disturbances, including insulin resistance, inflammatory activation, oxidative stress, vascular dysfunction, and neurohumoral imbalance [6, 7].

Yet the available liver–kidney literature is dominated by serum markers, calculated scores, and elastography. These tools are appropriate for risk stratification, but they do not show where fat is distributed within the acinus, whether a microvesicular component is present, or how lobular, portal, and cellular lesions are arranged in the tissue [7, 8].

Biopsy can describe these features in the same specimen and therefore remains useful when a detailed morphological phenotype is required [8–10]. Direct biopsy comparisons of MASH according to renal status, however, are scarce.

We therefore examined whether stage I–III CKD was associated with a different pattern of steatosis, inflammation, hepatocellular change, and fibrosis in patients with MASH.

The aim of the study.

To compare liver histology in MASH according to the presence or absence of stage I–III CKD. Particular attention was directed to steatosis, inflammatory activity, hepatocellular alterations, and fibrosis.

Object and research methods.

Participants were 60 patients aged 35–57 years with MASH and class I obesity.

Renal status defined two groups of equal size. Group 1 comprised 30 patients without CKD (17 women, 57%; 13 men, 43%). Group 2 comprised 30 patients with stage I–III CKD (18 women, 60%; 12 men, 40%). In the latter group, CKD occurred in the setting of chronic complicated pyelonephritis during resolution of an exacerbation or with a latent course. Patients with nephrotic syndrome were not included.

CKD required structural and/or functional evidence of kidney damage persisting for at least three months. Clinical assessment considered albuminuria and/or proteinuria, urinary sediment changes, instrumentally documented structural abnormalities, and renal functional measures.

Serum creatinine was measured in all patients. Creatinine clearance was derived with the Cockcroft–Gault

equation, whereas eGFR was calculated by MDRD and CKD-EPI using an electronic calculator. CKD stage was assigned from eGFR together with persistent structural and/or functional evidence of kidney injury.

For histology, three cores were obtained from separate sites in liver segments V and VII. Percutaneous biopsy was performed under continuous ultrasound guidance and local anesthesia with a 16-G needle, using a right-sided approach through the eighth to tenth intercostal spaces between the anterior axillary and midclavicular lines.

All three cores were considered when assigning the patient's morphological characteristics. The statistical unit was the patient, not the individual biopsy fragment.

Specimens were fixed for 22 hours in 10% neutral buffered formalin, dehydrated through ascending ethanol concentrations, embedded in paraffin, and cut at 5 μ m. Routine sections were stained with hematoxylin and eosin. Connective tissue was assessed with hematoxylin-picrofuchsin by Van Gieson and with the Gomori method; argyrophilic fibers were demonstrated by Bodian silver proteinate impregnation. Neutral lipids were evaluated in frozen sections stained with Sudan scarlet according to A.S. Gordeladze.

Morphology was classified according to E.M. Brunt [9]. Steatosis grades were S1 ($\leq 33\%$ of hepatocytes), S2 ($>33\text{--}66\%$), and S3 ($>66\%$). Zone 3 predominance, zone 1 predominance, panacinar distribution, and a microvesicular component were recorded separately.

Lobular inflammation was categorized as <2 , 2–4, or >4 foci per field of view. Portal inflammation, microgranulomas, and large lipogranulomas were documented independently.

Hyaline-droplet, hydropic, and ballooning degeneration were assessed semiquantitatively. Acidophilic bodies, pigmented macrophages, megamitochondria, Mallory bodies, and glycogenated nuclei were also recorded and described as isolated or multiple according to their distribution in the material examined.

Fibrosis staging followed the Brunt system [9]: F0, no fibrosis; F1, perivenular, perisinusoidal and/or pericellular fibrosis in zone 3; F2, F1 changes plus portal fibrosis; F3, septal/bridging fibrosis; and F4, cirrhotic remodeling.

Slides were examined with a Delta Optical Evolution 300 Trino Plan LED microscope; an Olympus C740UZ camera was used for digital imaging.

All procedures complied with ICH Good Clinical Practice, applicable national ethical requirements, and the principles of the World Medical Association Declaration of Helsinki. The study protocol was reviewed and approved by the Bioethics Committee of Bukovinian State Medical University. Written informed consent was obtained from all participants before study participation.

Categorical variables are reported as n/N (%), with whole percentages because each group contained 30 patients. Pearson's chi-square test without continuity correction was used for frequency comparisons. Statistical significance was set at $p < 0.05$. ORs and 95% CIs were calculated to describe association strength. No formal multiplicity correction was applied; borderline p values were considered together with effect estimates and CIs. Calculations were performed in Primer of Biostatistics, Version 4.03.

Research results and their discussion.

The biopsy comparison showed a broader steatotic and inflammatory pattern in the CKD group. The principal between-group differences in steatosis and inflammatory activity are summarized in **table 1**.

S2–S3 steatosis and the microvesicular component produced the largest group contrasts. Panacinar spread was also more frequent with CKD, indicating that the difference concerned both the amount and the distribution of hepatic fat (**table 1**).

Inflammatory activity followed the same direction: high-grade lobular inflammation and moderate/marked portal inflammation were more common in the CKD group. The microgranuloma comparison ($p=0.0384$) warrants cautious interpretation because several morphological endpoints were tested (**table 1**).

Representative histological patterns are illustrated in **figures 1–3**. **Figure 1** shows S1 macrovesicular steatosis in MASH, whereas **figures 2 and 3** demonstrate S2 and S3 steatosis, respectively, in MASH with stage II CKD, accompanied by portal/periportal inflammation or centri-

lobular necrotic changes.

Cellular alterations and fibrosis were evaluated next, with the between-group comparisons presented in **table 2**.

Table 1 – Steatosis and inflammatory features by CKD status

Morphological feature	MASH, n=30	MASH + CKD, n=30	OR (95% CI)	p
S2–S3 steatosis	16/30 (53%)	29/30 (97%)	25.38 (3.05–211.11)	0.0001
Panacinar steatosis	6/30 (20%)	15/30 (50%)	4.00 (1.27–12.58)	0.0149
Microvesicular steatosis	12/30 (40%)	25/30 (83%)	7.50 (2.24–25.06)	0.0006
Lobular inflammation >4 foci/field of view	3/30 (10%)	13/30 (43%)	6.88 (1.71–27.75)	0.0035
Microgranulomas	12/30 (40%)	20/30 (67%)	3.00 (1.05–8.60)	0.0384
Moderate/marked portal inflammation	9/30 (30%)	20/30 (67%)	4.67 (1.57–13.87)	0.0045

Notes: values are n/N (%); OR compares MASH+CKD with MASH without CKD; CI, confidence interval; p, Pearson chi-square test.

Multiple hepatocellular degenerative changes and multiple glycogenated nuclei were significantly more frequent in patients with CKD (**table 2**).

Pigmented macrophages and megamitochondria were numerically more frequent with CKD, but neither comparison reached $p<0.05$.

F2–F3 fibrosis was found more often in the CKD group (47% vs 23%); however, $p=0.0581$ and the 95% CI for the OR included 1. F4 was not identified in either group (**table 2**).

A representative histological example of F3 fibrosis in MASH with stage III CKD is shown in **figure 4**.

Taken together, the quantitative comparisons in **tables 1 and 2** and the representative micrographs in **figures 1–4** highlight three observations. CKD was associated with a larger steatotic burden and wider acinar spread, with stronger lobular and portal inflammation, and with more extensive degenerative change in hepatocytes. The distinction between groups therefore emerged from the combination of lesions rather than from a single histological sign.

The steatosis findings are notable for both quantity and topology. S2–S3 was far more common with CKD, and panacinar involvement replaced the more limited distribution seen more often in patients without renal disease. Because the confidence interval around the OR

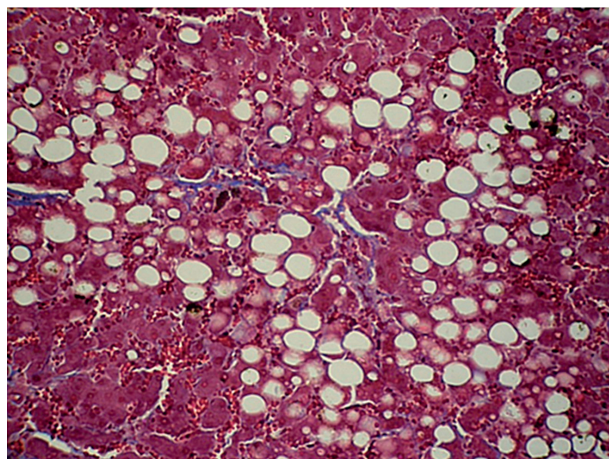


Figure 1 – MASH: macrovesicular steatosis in approximately 25% of hepatic parenchyma (S1). Stain: Slinchenko. Magnification: Obj. $\times 20$; Oc. $\times 10$.

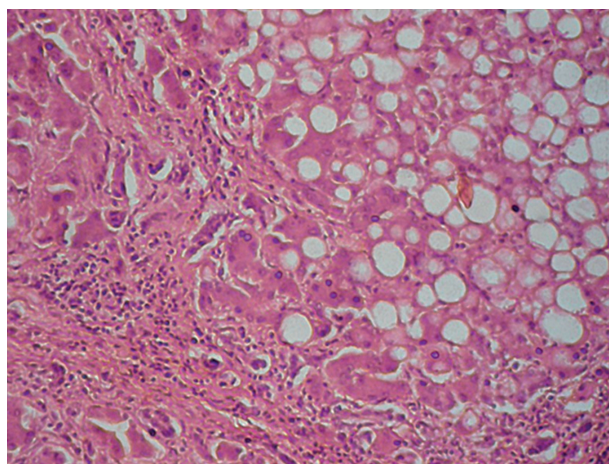


Figure 2 – MASH with stage II CKD: macrovesicular steatosis in approximately 45% of hepatic parenchyma (S2) with portal and periportal inflammation. Stain: hematoxylin and eosin. Magnification: Obj. $\times 20$; Oc. $\times 10$.

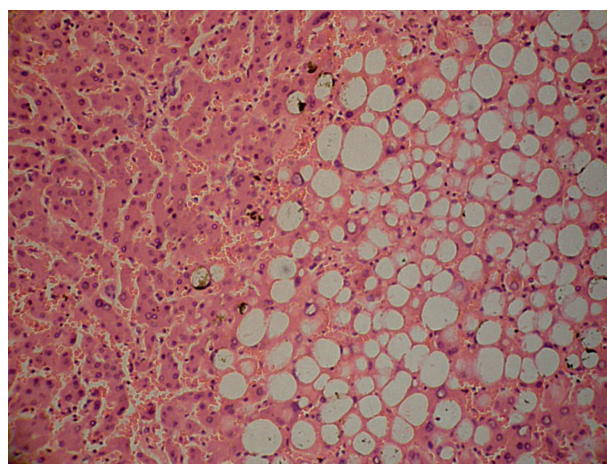


Figure 3 – MASH with stage II CKD: macrovesicular steatosis in approximately 75% of hepatic parenchyma (S3) with centrilobular hepatocellular necrosis extending into the lobule. Stain: hematoxylin and eosin. Magnification: Obj. $\times 20$; Oc. $\times 10$.

Table 2 – Hepatocellular alterations and fibrosis by CKD status

Morphological feature	MASH, n=30	MASH + CKD, n=30	OR (95% CI)	p
Multiple hepatocellular degenerative changes	12/30 (40%)	23/30 (77%)	4.93 (1.61–15.07)	0.0040
Multiple pigmented macrophages	6/30 (20%)	12/30 (40%)	2.67 (0.84–8.46)	0.0910
Multiple megamitochondria	3/30 (10%)	8/30 (27%)	3.27 (0.77–13.83)	0.0953
Multiple glycogenated nuclei	6/30 (20%)	15/30 (50%)	4.00 (1.27–12.58)	0.0149
F2–F3 fibrosis	7/30 (23%)	14/30 (47%)	2.88 (0.95–8.72)	0.0581

Notes: values are n/N (%). OR compares MASH+CKD with MASH without CKD; CI, confidence interval; p, Pearson chi-square test.

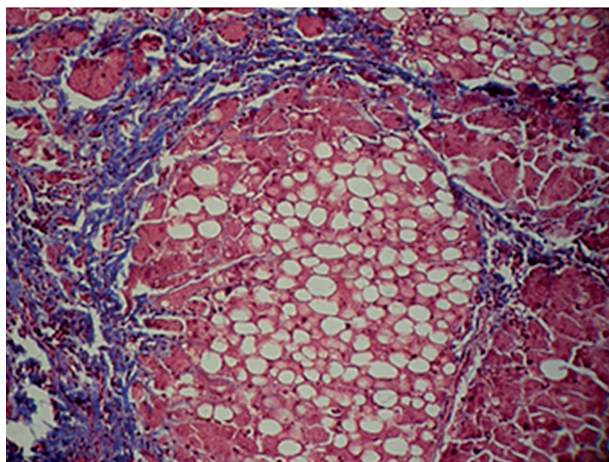


Figure 4 – MASH with stage III CKD: macrovesicular steatosis in approximately 54% of hepatic parenchyma (S2), marked perisinusoidal and centrilobular fibrosis in zone 3, and portal/periportal fibrosis with septa (F3). Stain: Slinchenko. Magnification: Obj. ×20; Oc. ×10

for S2–S3 was wide, the direction of this difference is more reliable than the exact size of the effect estimate.

The frequent microvesicular component adds a cellular dimension to this pattern. Disturbances in fatty-acid handling, β -oxidation, and mitochondrial metabolism are recognized in MASH [11, 12], but our study did not measure these pathways. The numerical excess of megamitochondria in the CKD group therefore cannot be taken as proof of mitochondrial dysfunction.

Inflammation also separated the groups. Both lobular and portal activity were greater in patients with CKD. Chronic inflammatory activation, oxidative stress, vascular abnormalities, and neurohumoral changes described in CKD offer a plausible biological background [6, 7], yet they remain explanatory hypotheses here because no circulating or tissue markers of those pathways were measured. Microgranulomas should not be overemphasized. Their p value was 0.0384, the lower limit of the OR confidence interval lay close to 1, and the analysis covered several histological endpoints without a formal multiplicity adjustment. This finding is best viewed as supportive rather than decisive.

The excess of multiple degenerative hepatocellular changes indicates that the CKD-associated phenotype extended beyond fat accumulation. The study category “multiple degenerative changes” was broader than the standardized concept of ballooning and should not be interpreted as a substitute for a ballooning score [8, 10].

These tissue data add a morphological layer to the epidemiological association between steatotic liver disease and CKD reported by Mantovani et al. and subsequent meta-analytic work [5, 13]. Most of that evidence is derived from clinical or non-invasive definitions; biopsy permits direct assessment of acinar fat distribu-

tion, microvesicular steatosis, compartment-specific inflammation, and cellular degeneration.

Fibrosis requires the most conservative reading. As shown in **table 2**, F2–F3 was almost

twice as frequent numerically in the CKD group, but the comparison remained above the $p < 0.05$ threshold and its OR confidence interval crossed 1. The data therefore support a numerical difference only, not a confirmed association with advanced fibrosis. **Figure 4** illustrates the morphology of F3 fibrosis in an individual case and should not be interpreted as group-level evidence of faster fibrogenesis.

They also say nothing about the rate of fibrogenesis. A single biopsy cannot establish progression. This distinction matters because fibrosis stage, rather than steatohepatitis activity alone, is closely related to long-term liver outcomes [4,14,15].

Clinically, the findings favor careful fibrosis risk stratification rather than wider use of biopsy. Current guidance prioritizes serum-based algorithms and liver-stiffness assessment for this purpose [2, 3]. A useful next step would be to relate the histological phenotype to eGFR, albuminuria, non-invasive fibrosis indices, and elastography, as suggested by recent liver–kidney evidence [16].

Interpretation is constrained by the study setting. Each group contained 30 patients, recruitment was from a single center, and morphology was sampled at one time point. Small or moderate effects may therefore have remained unresolved.

Generalizability is also limited: all participants had class I obesity, and CKD in the comorbid group occurred with chronic complicated pyelonephritis. Other CKD etiologies and metabolic phenotypes may show a different histological profile.

Finally, three cores from segments V and VII improved tissue coverage but could not remove the inherent sampling variability of liver biopsy.

Within these limits, the most reproducible signal was the joint presence of broader steatosis, more frequent microvesicular fat, stronger lobular and portal inflammation, and more extensive hepatocellular degeneration in patients with CKD. The fibrosis difference remained descriptive rather than statistically established.

Conclusions.

Stage I–III CKD accompanied MASH with a broader steatotic pattern, more active lobular and portal inflammation, and more widespread hepatocellular degeneration. F2–F3 fibrosis was more frequent numerically, but the difference was not statistically confirmed.

Prospects for further research.

Larger prospective studies should test these biopsy patterns across CKD stages and etiologies and relate them to eGFR, albuminuria, non-invasive fibrosis scores, and elastographic measurements.

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МОРФОЛОГІЧНІ ОСОБЛИВОСТІ УРАЖЕННЯ ПЕЧІНКИ ПРИ МЕТАБОЛІЧНО-АСОЦІЙОВАНОМУ СТЕАТОГЕПАТИТІ ЗА КОМОРБІДНОСТІ З ХРОНІЧНОЮ ХВОРОБОЮ НИРОК

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Резюме. Поєднання метаболічно-асоційованого стеатогепатиту (MASH) із хронічною хворобою нирок (ХХН) може супроводжуватися відмінностями структурного ураження печінки, однак безпосередніх біопсійних порівнянь залежно від ниркового статусу залишається недостатньо. Мета дослідження – порівняти морфологічні характеристики печінки у хворих на MASH з ХХН I–III стадій та без ниркової коморбідності. До дослідження включено 60 пацієнтів віком 35–57 років із MASH та ожирінням I ступеня. Першу групу становили 30 хворих без ХХН, другу – 30 пацієнтів із ХХН I–III стадій на тлі хронічного ускладненого пієлонефриту. У кожного пацієнта досліджували три біоптати печінки, отримані з різних ділянок V і VII сегментів. Оцінювали ступінь і ацинарний розподіл стеатозу, мікроезикалярний компонент, лобулярне та портальне запалення, дистрофічні зміни гепатоцитів і фіброз за критеріями Е.М. Brunt. Статистичною одиницею був пацієнт. Частоти порівнювали за критерієм хі-квадрат Пірсона; визначали відношення шансів та 95% довірчі інтервали. У групі з ХХН частіше реєстрували стеатоз S2–S3 – 97% проти 53% у пацієнтів без ХХН ($p=0,0001$), панацінарний розподіл – 50% проти 20% ($p=0,0149$) та мікроезикалярний компонент – 83% проти 40% ($p=0,0006$). Лобулярне запалення понад 4 вогнища у полі зору виявляли у 43% проти 10% ($p=0,0035$), а помірне або значне портальне запалення – у 67% проти 30% ($p=0,0045$). Множинні дистрофічні зміни гепатоцитів визначено у 77% пацієнтів із ХХН та у 40% без ХХН ($p=0,0040$); множинні глікогенізовані ядра – відповідно у 50% і 20% ($p=0,0149$). Частота F2–F3 фіброзу становила 47% у групі з ХХН і 23% у групі без ХХН, однак ця відмінність не досягла статистичної значущості ($p=0,0581$); F4 не виявлено. Таким чином, у дослідженій вибірці наявність ХХН I–III стадій при MASH асоціювалася з більш поширеним стеатозом, активнішими лобулярними та портальними запальними змінами і більшою поширеністю дистрофічних змін гепатоцитів. Щодо фіброзу встановлено лише чисельне переважання F2–F3 у пацієнтів із ХХН без статистичного підтвердження міжгрупової різниці.

Ключові слова: метаболічно-асоційований стеатогепатит, хронічна хвороба нирок, біопсія печінки, стеатоз, фіброз.

MORPHOLOGICAL FEATURES OF LIVER INJURY IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOHEPATITIS WITH COMORBID CHRONIC KIDNEY DISEASE

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Abstract. Direct biopsy comparisons of metabolic dysfunction-associated steatohepatitis (MASH) according to chronic kidney disease (CKD) status remain limited. The aim of this study was to compare liver morphology in pa-

tients with MASH with and without stage I–III CKD. Sixty patients aged 35–57 years with MASH and class I obesity were included. Thirty patients had no CKD, whereas 30 had stage I–III CKD associated with chronic complicated pyelonephritis. Three liver biopsy cores from separate areas of segments V and VII were examined for each participant. Steatosis grade and acinar distribution, microvesicular steatosis, lobular and portal inflammation, hepatocellular degenerative changes, and fibrosis were assessed according to the E.M. Brunt criteria. The patient, rather than each biopsy fragment, was the statistical unit. Group frequencies were compared using Pearson's chi-square test; odds ratios and 95% confidence intervals were calculated. S2–S3 steatosis was identified in 97% of patients with CKD and 53% of those without CKD ($p=0.0001$). Panacinar steatosis occurred in 50% versus 20% ($p=0.0149$), and a microvesicular component in 83% versus 40% ($p=0.0006$). Lobular inflammation exceeding four foci per field was recorded in 43% versus 10% ($p=0.0035$), while moderate or marked portal inflammation occurred in 67% versus 30% ($p=0.0045$). Multiple hepatocellular degenerative changes were present in 77% of patients with CKD and 40% without CKD ($p=0.0040$); multiple glycogenated nuclei were observed in 50% and 20%, respectively ($p=0.0149$). F2–F3 fibrosis was found in 47% of the CKD group and 23% of the non-CKD group, but the difference was not statistically significant ($p=0.0581$); F4 fibrosis was absent. Thus, in this cohort, stage I–III CKD accompanying MASH was associated with a broader steatotic pattern, greater lobular and portal inflammatory activity, and more extensive hepatocellular degeneration. The fibrosis finding remained numerical rather than statistically confirmed and should not be interpreted as evidence of faster fibrogenesis.

Key words: metabolic dysfunction-associated steatohepatitis, chronic kidney disease, liver biopsy, steatosis, fibrosis.

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Conflict of interest:

The authors declare no conflict of interest.

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ДИНАМІКА ЯКОСТІ ЖИТТЯ ТА ФУНКЦІОНАЛЬНОГО СТАНУ ПАЦІЄНТІВ ПІСЛЯ РАДИКАЛЬНОЇ ПРОСТАТЕКТОМІЇ ЗА ОПИТУВАЛЬНИКОМ EORTC QLQ-C30: ПОВІКОВИЙ АНАЛІЗ

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Радикальна простатектомія є одним із основних методів радикального лікування раку передміхурової залози, проте її результати визначаються не лише онкологічною ефективністю, а й характером післяопераційного відновлення, рівнем повсякденної активності та вираженістю симптомів. Метою дослідження було встановити особливості динаміки якості життя та функціонального стану пацієнтів різних вікових груп після радикальної простатектомії. Обстежено 214 хворих на рак передміхурової залози віком 52–80 років, яким виконано радикальну простатектомію з використанням лапароскопічної техніки. Оцінювання проводили за опитувальником EORTC QLQ-C30 до оперативного втручання, через 6 та 12 місяців після нього. Аналіз функціональних показників засвідчив їх істотне погіршення через 6 місяців після операції. Найбільш виражені зміни стосувалися фізичної витривалості, здатності виконувати повсякденні справи, професійної діяльності та брати участь у звичних видах дозвілля. Одночасно спостерігалось зростання симптомного навантаження, зокрема інтенсивності болю, слабкості, потреби у відпочинку, порушень