УДК 616.36-004-089
DOI http://doi.org/10.30978/MG-2024-1-43

ISSN 1727-5725 (Print)
ISSN 2521-649X (Online)

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Quantitative morphometry of compensated liver cirrhosis can aid to predict the outcomes of surgical treatment

Objective — to define quantitative morphometric characteristics of hepatic parenchyma lesions at compensated liver cirrhosis (CLC), which will allow more accurately to assess its functional reserves and predict the outcomes of surgical treatment.

Materials and methods. Intraoperative marginal liver biopsy was performed for 74 patients with CLC, who underwent surgical treatment. Morphological examinations with quantitative morphometry of intraoperative liver biopsies were done with the use of cytological analyzer with software «Integral-2MT» produced by the association «Kvant» (Kyiv). The connective tissue area (CTA), unchanged hepatocytes area (UHA), stromal-parenchymatous ratio (SPR), the volume of hepatocytes in the state of necrosis and/or necrobiosis (VHSNN), the volume of hepatocytes in the state of division (VHSD) were determined. The statistical analysis was performed by use of Statistica 12 software package.

Results. Three types of morphological pattern of CLC were isolated. Type I (type A) portal cirrhosis with weakly pronounced signs of parenchymatous and stromal reaction (mono-multilobular type, portal cirrhosis). The CTA was 66.73 ± 1.71 μm^2, SPR was 0.285 ± 0.019, UHA was 234.13 ± 11.5 μm^2, VHSNN was 11.21 ± 0.74 %, VHSD was 10.23 ± 0.57 %. Type II cirrhosis (type B) — cirrhosis of mono-multilobular type with moderately expressed signs of parenchymatous and stromal reaction. The CTA was 126.69 ± 12.5 μm^2, SPR was 0.617 ± 0.031, UHA was 205.34 ± 13.8 μm^2, VHSNN was 17.32 ± 0.63 %, VHSD was 15.43 ± 0.48 %. Type III cirrhosis (type C) — cirrhosis with pronounced signs of parenchymatous and stromal reaction, more often of multilobular type. The CTA was 240.16 ± 13.4 μm^2, SPR was 1.344 ± 0.089, UHA was 178.69 ± 18.7 μm^2, VHSNN was 23.97 ± 0.75 %, VHSD was 11.07 ± 0.58 %. Analysis of immediate results of surgical treatment demonstrated, that no one patient with A-type CLC died, while 9.5 % of patients with B-type and 25.0 % of patients with C-type CLC died in the early postoperative period.

Conclusions. Quantitative morphometry demonstrated that pathological changes in the liver at CLC are heterogeneous and can be categorized in to 3 types (A, B, C). Three types of morphological pattern at CLC, which were isolated, are characterized by significant differences in CTA, UHA, SPR, VHSNN, VHSD parameters. At transition of A-type cirrhosis into C-type, volume of hepatic parenchyma becomes to be decreased, while volume of connective tissue becomes to be increased. This is accompanied by decrease in UHA, increase in CTA, SPR and VHSNN. Surgical intervention in patients with type C morphological pattern of cirrhosis is associated with a high risk (25 %) of poor outcome, which indicates the need to limitations for indications to elective surgical operations in this category of patients. Thus, quantitative morphometry of liver biopsies in CLC patients can aid to predict the outcomes of surgical treatment.

Keywords: compensated liver cirrhosis, quantitative morphometry, morphological pattern, intraoperative liver biopsy, outcomes of surgical treatment.

Отримано • *Received* 28/12/2023. Прийнято до друку • *Accepted* 01/02/2024

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Liver cirrhosis is defined by its histological hallmark findings on liver biopsy (regenerative nodules surrounded by fibrotic tissue) and is considered as the final evolution stage of any progressive liver disease, irrespective of its etiology [10]. Cirrhosis is an important cause of morbidity and mortality in people with chronic liver disease (CLD) worldwide. In 2019, cirrhosis was associated with 2.4 % of global deaths [3]. Cirrhosis is associated with an increased surgical morbidity and mortality — studies in patients with underlying cirrhosis undergoing colorectal, gastric, or esophageal surgical procedures demonstrated nearly a three-fold increase in post-operative mortality rates with similarly increased rates of post-operative morbidity compared to patients without cirrhosis [4, 7, 8]. Clinical assessment supplemented by blood tests, imaging, liver stiffness measurement, endoscopy and assessment of portal pressure (derived from the hepatic venous pressure gradient) can facilitate risk stratification. Traditional prognostic scoring systems including the Child-Turcotte-Pugh and Model for End-stage Liver Disease are helpful but may overestimate surgical risk. Specific prognostic scores like Mayo Risk Score, VOCAL-Penn and ADOPT-LC can add precision to risk assessment [1]. Liver biopsy is still considered the gold-standard diagnostic method to identify the typical features of cirrhosis. Historically, liver biopsy has three major roles: for diagnosis, for assessment of prognosis (disease staging), and/or to facilitate therapeutic management decisions. It is probably the only method currently available that definitively distinguishes between the different pathological stages of CLD and helps identify factors associated with disease progression [2, 6, 9]. Over the past decade, imaging modalities and serological tests have emerged as important tools in the evaluation of liver diseases, in many cases supplanting the use of liver biopsy and histological examination. Nonetheless, the accuracy and diagnostic value of these methods may not always be conclusive, and the assessment of liver histology often remains the gold standard for diagnostic evaluation [5, 11].

Objective — to define quantitative morphometric characteristics of hepatic parenchyma lesions at compensated liver cirrhosis, which will allow more accurately to assess its functional reserves and predict the outcomes of surgical treatment.

Materials and methods

The study involved 74 patients with CLC, who underwent surgical treatment. In 33 (44.6 %) patients extraperitonization of right hepatic lobe with intraoperative laser irradiation was done, in 41 (65.4 %) patients — ligation of the left gastric artery and vein, splenic artery. In all cases intraoperative marginal liver biopsy was done — specimens not less than 1.5 × 1.5 × 1.5 cm in size were taken. By gender, the patients were distributed as follows: 49 (66.2 %) mails and 25 (33.8 %) femails. The age of the patients ranged from 17 to 66 years. The average age of the patients was 44.5 years. Thus, CLC commonly affects patients of working age from 31 to 60 years — 66 (89.1 %) patients.

In 30 (40.5 %) patients, a viral etiology of CLC was established, in 27 (44.7 %) patients — heavy alcohol consumption for 2 to 15 years, in 3 (7.9 %) patient the cause of the disease wasn't identified — there were no indications in the anamnesis about acute viral hepatitis, chronic alcoholism, malaria, contact with hepatotropic poisons in the past.

Mophological examinations with quantitative morphometry of intraoperative liver biopsies were done by use of cytological analyzer with software «Integral-2MT» produced by the association «Kvant» (Kyiv). The connective tissue area (CTA), unchanged hepatocytes area (UHA), stromal-parenchymatous ratio (SPR), the volume of hepatocytes in the state of necrosis and/or necrobiosis (VHSNN), the volume of hepatocytes in the state of division (VHSD) were determined. The statistical analysis was performed by use of Statistica 12 software package. The normality of data distribution was assessed with the Shapiro-Wilk test. Continuous data were presented as mean and standard deviation (M ± SD).

Results and discussion

When analyzing quantitative morphometric characteristics of intraoperative biopsies, we identified 3 types of morphological pattern of cirrhosis.

I type (type A). Portal cirrhosis with weakly pronounced signs of parenchymatous and stromal reaction (mono-multilobular type, portal cirrhosis).

Glisson's capsule is thickened, hyalinized. There are incomplete connective tissue septa with newly formed subcapsular bile ducts (Table 1).

Portal tracts are dilated, sclerosed, with focal hyalinosis of connective tissue. In single portal tracts — focal lymphoid-histiocytic infiltrates, not spreading to the liver lobules (Fig. 1).

Periductal and perivascular sclerosis is pronounced. Only in some portal tracts there is a diffuse lymphoid-histiocytic infiltration spreading through the border plate into the lobule throughout 2—3 layers of hepatocytes The hepatic trabeculae are not radially arranged in the forming false lobules, more often they are stretched approximately

Table 1. **Morphometrical characteristics of intraoperative liver biopsies in patients depending on CLC type**

Parameter	A-type	B-type	C-type
CTA, μm^2	66.73 ± 1.71	126.69 ± 12.5*	240.16 ± 13.4*#
UHA, μm^2	234.13 ± 11.5	205.34 ± 13.8	178.69 ± 18.7*
SPR, units	0.285 ± 0.019	0.617 ± 0.031*	1.344 ± 0.089*#
VHSNN, %	11.21 ± 0.74	17.32 ± 0.63*	23.97 ± 0.75*#
VHSD, %	10.23 ± 0.57	15.43 ± 0.48*	11.07 ± 0.58*#

Note. Differences are statistically significant ($p < 0.05$): * in comparison with A-type; # in comparison with B-type.

parallel to each other. Hepatocytes with weakly expressed parenchymal protein degeneration.

Sinusoids were predominantly slit-shaped, in some places their capillarization was expressed, lumen of sinusoids was poorly bloody. Closer to the sclerosed portal tracts there are bi- or trinuclear hepatocytes, or with one large hyperchromic *nucleus*, which indicates the preserved regenerative activity of the liver.

Type II (type B) — cirrhosis of mono-multilobular type with moderately pronounced signs of parenchymatous and stromal reaction.

The liver capsule is focally or diffusely thickened, hyalinized, in some places focally infiltrated with lymphoid-histiocytic elements, sometimes with admixture of leukocytes. In contrast to A-type of cirrhosis, in B-type cirrhosis infiltrates spread inside the liver lobules, penetrating through the outer border plate of hepatocytes. In this group, more extensive infiltration of portal tracts is observed, which makes up almost 1/2 of their volume. At the same time, lymphoid-histiocytic infiltrates spread along narrow and wide connective tissue septa from dilated portocaval tracts through the inner border plate between hepatic trabeculae (Fig. 2).

Lymphocytes in some places surround groups of periportal hepatocytes, causing their immune cytolysis or the formation of so-called «hepatocellular pseudoductules», from which later the formation of cholangioles is noted. In false lobules (monolobular or multilobular) there are phenomena of capillarization of sinusoids, in some places lymphoid or leukocytic focal infiltration is observed between hepatocytes in lobules (for example, in alcoholic cirrhosis focal infiltration of leucocytes around hepatocytes with fibrous or granular accumulations of alcoholic hyaline, so-called leukocyte chemotaxis around alcoholic hyaline).

Parenchymatous protein, including hydropic, degeneration with development of focal colliquative necrosis, small- or large-drop focal adipose degeneration are observed in hepatocytes in some places.

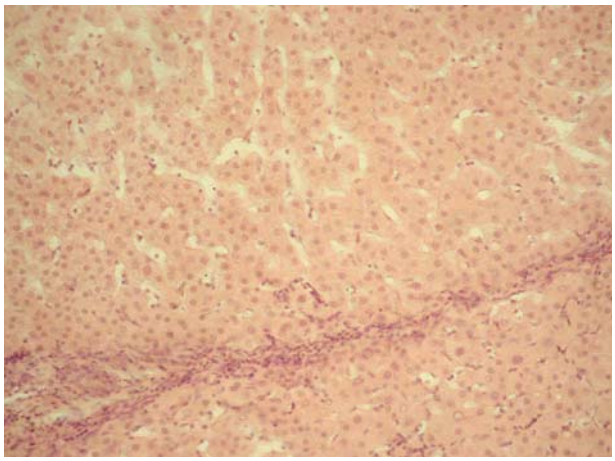

Fig. 1. **Type A cirrhosis. Narrow connective tissue layers with moderately pronounced lymphohistiocytic infiltration.** Hematoxylin and eosin staining. × 200

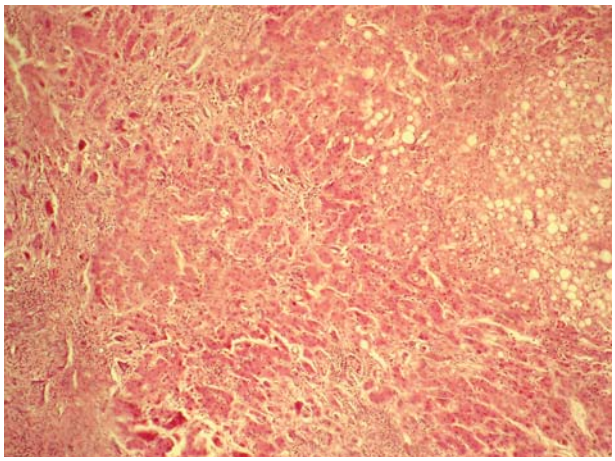

Fig. 2. **Type B cirrhosis. Wide connective tissue layers with diffuse lymphohistiocytic infiltrates penetrating into the lobules. Available areas of regenerating hepatocytes.** Hematoxylin and eosin staining. × 200

Periportal staircase-shaped or bridge-shaped portocentral single necrosis of hepatocytes with focal or diffuse lymphoid-cell infiltration is detected in some places. The VHSNN was 17.32 ± 0.63 %. In some portal tracts or wide septa, lymphoid-cell clusters resembled typical lymphatic follicles of lymph nodes.

Such structures testify most of all in favor of viral etiology of cirrhosis, because they are morphological expression of immunopathological processes, the greater degree of expression of which is caused by the action of antigenic viral determinants.

There are hepatocytes with manifestation of regenerative activity in the form of moderate number of dinuclear forms or hyperchromic nuclei increased in size, occasionally — 3-nuclear hepatocytes and hepatocytes with mitotic activity. The VHSD was 15.43 ± 0.48 %. If lymphoid-histiocytic type infiltrates appear in dilated and sclerosed portal tracts, there is an admixture of plasmatic cells in them.

III type (type C) — cirrhosis with pronounced signs of parenchymatous and stromal reaction, more often of multilobular type.

The liver capsule is thickened, partly hyalinized, in some places with focal or diffuse infiltration spreading on many narrow and wide connective tissue septa formed on the place of porto-portal, porto-central or centro-central bridge necrosis. Most of the dilated portal tracts with diffuse lymphoid-histiocytic infiltration and with admixture of plasmatic cells. In some portal tracts such infiltrates are found on the background of fibrosis of their connective tissue. In some places, typical lymphatic follicles are formed among the infiltrates. In one portal tract there may be up to 2—3 of them (Fig. 3).

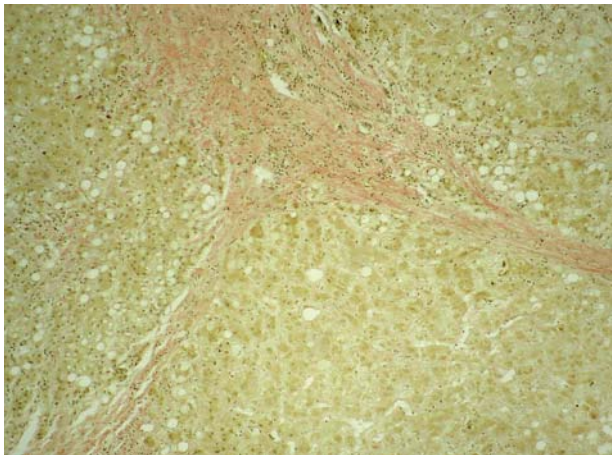

Fig. 3. **Type C cirrhosis. Wide connective tissue layers with focal lymphohistiocytic infiltrates. Large-droplet adipose degeneration of hepatocytes. Extensive necrosis, involving one to several lobules. Lack of hepatocyte regeneration.** Staining according to Van Gieson. × 200

From many portal tracts, lymphoid cell infiltrates spread not only between periportal hepatocytes through the hepatic border plate, but also penetrate to the central parts of lobules, causing immune cytolysis of hepatocytes with subsequent development of connective tissue septa in the places of hepatocyte death, which divide multilobular lobules into monolobular lobules and even into smaller pseudolobules.

In hepatocytes, pronounced parenchymal protein degeneration, up to baloney, focal or diffuse small-drop or large-drop adipose degeneration were observed. In separate observations, fibrous or granular alcoholic hyaline with pronounced leukocyte chemotaxis is found in hepatocytes.

In one observation, there were detected Councilman bodies — groups of hepatocytes with coagulation necrosis of cytoplasm, pushing out into the lumen of sinusoids. As a rule, they are found in viral hepatitis and cirrhosis after the reverse development of such hepatitis into cirrhosis.

The regenerative activity of hepatocytes was weakly expressed. In part of observations, many formed cholangioles from hepatocellular pseudoductules were found in portal and periportal fields of newly formed connective tissue among infiltrates.

Thus, at transition from type A to type C histological variant of cirrhosis the volume of liver parenchyma decreases, and the share of connective tissue increases, which is manifested by decrease of the UHA, increase of the CTA and, accordingly, SPR. As a result of these changes the functional reserve of the liver decreases, which is manifested by a decrease in the VHSD, an increase in the VHSNN.

However, it should be noted that the VHSD in the transition from A-type to B-type cirrhosis significantly increased, and in the transition from B-type to C-type significantly decreased, which we explain as follows.

In the morphological pattern of A-type cirrhosis, the development of connective tissue in the liver parenchyma is insignificant, so the existing mass of functioning hepatocytes is able to provide the function of the organ without significant disturbances. The VHSD in such morphological pattern is minimal, as there is no need for intensive renewal of the volume of functioning hepatocytes. The processes of necrobiosis and division are in balance. In B-type cirrhosis there is an increase in functional activity of cells and organ as a whole, as the processes of necrobiosis begin to prevail over the processes of division and the existing volume of functioning hepatocytes is not able to provide compensated function of the organ. In this connection, as a compensatory reaction of the organ to the increase of connective tissue,

Table 2. **Immediate outcomes of surgical treatment of CLC patients depending on the morphological type of cirrhosis**

Type of CLC	Mortality
A (n = 37)	0
B (n = 21)	2 (9.5 %)
C (n = 16)	4 (25.0 %)
Total (n = 74)	6 (8.1 %)

there is an increase in the VHSD. In C-type cirrhosis, there is further increase in the development of connective tissue in the liver, but the compensatory mechanisms that maintain the functional state of the organ are depleted. In addition, the replacement of a significant volume of hepatic parenchyma by connective tissue, dramatically reduces the number of hepatocytes that retain the ability to divide. These two factors lead to a reduction in the VHSD.

According to the results of quantitative morphometry, 37 (50 %) patients belong to type A, 21 (28.3 %) — to type B, and 16 (21.7 %) — to type C cirrhosis.

It seemed reasonable to us to study the relationship between the outcome of surgery and the type of morphological pattern of changes in the liver in this category of patients (Table 2). Poor outcomes of surgical treatment in all of cases were caused by postoperative acute decompensation of liver cirrhosis.

Conclusions

Quantitative morphometry demonstrated that pathological changes in the liver at CLC are heterogeneous and can be categorized in to 3 types (A, B, C). Selected 3 types of morphological pattern at CLC are characterized by statistically significant differences in CTA, UHA, SPR, VHSNN, VHSD. At transition of A-type cirrhosis into C-type, volume of hepatic parenchyma becomes to be decreased, while volume of connective tissue becomes to be increased. This is accompanied by decrease in UHA, increase in CTA, SPR and VHSNN. Surgical intervention in patients with type C morphological pattern of cirrhosis is associated with a high risk (25 %) of poor outcome, which indicates the need to limitations for indications to elective surgical operations in this category of patients. Thus, quantitative morphometry of liver biopsies in CLC patients can aid to predict the outcomes of surgical treatment.

Conflicts of interest: none.
Authorship contributions: conception and design, critical revision of the article — O. H. P.; acquisition of data — K. M. S., Y. O. S.; analysis, and interpretation of data, drafting the article — V. V. M., V. M. F.

References

1. Abbas N, Fallowfield J, Patch D, Stanley AJ, Mookerjee R, Tsochatzis E, Leithead JA, Hayes P, Chauhan A, Sharma V, Rajoriya N, Bach S, Faulkner T, Tripathi D. Guidance document: risk assessment of patients with cirrhosis prior to elective non-hepatic surgery. Frontline Gastroenterol. 2023 Mar 8;14(5):359-70. doi: 10.1136/flgastro-2023-102381. PMID: 37581186; PMCID: PMC10423609.
2. Chowdhury AB, Mehta KJ. Liver biopsy for assessment of chronic liver diseases: a synopsis. Clin Exp Med. 2023 Jun;23(2):273-85. doi: 10.1007/s10238-022-00799-z. Epub 2022 Feb 22. PMID: 35192111; PMCID: PMC10224864.
3. Huang DQ, Terrault NA, Tacke F, et al. Global epidemiology of cirrhosis — aetiology, trends and predictions. Nat Rev Gastroenterol Hepatol. 2023 Jun;20(6):388-98. doi: 10.1038/s41575-023-00759-2. Epub 2023 Mar 28. PMID: 36977794; PMCID: PMC10043867.
4. Johnson KM, Newman KL, Green PK, et al. Incidence and risk factors of postoperative mortality and morbidity after elective versus emergent abdominal surgery in a National Sample of 8193 patients with cirrhosis. Ann Surg. 2021 Oct 1;274(4):e345-e354. doi: 10.1097/SLA.0000000000003674. PMID: 31714310.
5. Khalifa A, Rockey DC. The utility of liver biopsy in 2020. Curr Opin Gastroenterol. 2020 May;36(3):184-91. doi: 10.1097/MOG.0000000000000621. PMID: 32097176.
6. Larrey D, Meunier L, Ursic-Bedoya J. Liver biopsy in chronic liver diseases: Is there a favorable benefit: risk balance? Ann Hepatol. 2017 Jul-Aug;16(4):487-9. doi: 10.5604/01.3001.0010.0272. PMID: 28612749.
7. Nevarez NM, Chang G, Porembka MR, et al. Presence of underlying cirrhosis is associated with increased in-hospital mortality and length of stay following pancreatoduodenectomy. HPB (Oxford). 2023 Oct 12:S1365-182X(23)01951-2. doi: 10.1016/j.hpb.2023.10.010. Epub ahead of print. PMID: 37867083.
8. Piecha F, Vonderlin J, Frühhaber F, et al. Preoperative TIPS and in-hospital mortality in patients with cirrhosis undergoing surgery. JHEP Rep. 2023 Sep 23;6(1):100914. doi: 10.1016/j.jhepr.2023.100914. PMID: 38074512; PMCID: PMC10698536.
9. Procopet B, Berzigotti A. Diagnosis of cirrhosis and portal hypertension: imaging, non-invasive markers of fibrosis and liver biopsy. Gastroenterol Rep (Oxf). 2017 May;5(2):79-89. doi: 10.1093/gastro/gox012. Epub 2017 Apr 17. PMID: 28533906; PMCID: PMC5421457.
10. Ripoll C, Groszmann R, Garcia-Tsao G, Grace N, Burroughs A, Planas R, Escorsell A, Garcia-Pagan JC, Makuch R, Patch D, Matloff DS, Bosch J; Portal Hypertension Collaborative Group. Hepatic venous pressure gradient predicts clinical decompensation in patients with compensated cirrhosis. Gastroenterology. 2007 Aug;133(2):481-8. doi: 10.1053/j.gastro.2007.05.024. Epub 2007 May 21. PMID: 17681169.
11. Sripongpun P, Pongpaibul A, Charatcharoenwitthaya P. Value and risk of percutaneous liver biopsy in patients with cirrhosis and clinical suspicion of autoimmune hepatitis. BMJ Open Gastroenterol. 2021 Aug;8(1):e000701. doi: 10.1136/bmjgast-2021-000701. PMID: 34362759; PMCID: PMC8351491.

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Кількісна морфометрія компенсованого цирозу печінки може допомогти спрогнозувати результати хірургічного лікування

Мета — визначити кількісні морфометричні характеристики ураження паренхіми печінки при компенсованому цирозі печінки (КЦП), що дасть змогу точніше оцінити її функціональні резерви та спрогнозувати наслідки хірургічного лікування.

Матеріали та методи. У 74 хворих на КЦП, які перенесли хірургічне лікування, виконано інтраопераційну крайову біопсію печінки. Мофологічні дослідження з кількісною морфометрією інтраопераційних біоптатів печінки проводили за допомогою цитологічного аналізатора з програмним забезпеченням «Інтеграл-2МТ» («Квант», Київ). Визначали площу сполучної тканини (ПСТ) і незмінених гепатоцитів (ПНГ), стромально-паренхіматозне співвідношення (СПС), об'єм гепатоцитів у стані некрозу та/або некробіозу (ОГСНН) і гепатоцитів у стані поділу (ОГСП). Статистичну обробку проводили за допомогою програмного комплексу Statistica 12.

Результати. Виділено три типи морфологічної картини КЦП. Тип I (тип А) — портальний цироз зі слабко виразними ознаками паренхіматозної та стромальної реакції (мономультилобулярний тип, портальний цироз), ПСТ — (66,73 ± 1,71) мкм^2, СПС — 0,285 ± 0,019, ПНГ — (234,13 ± 11,5) мкм^2, ОГСНН — (11,21 ± 0,74) %, ОГСП — (10,23 ± 0,57) %. Тип II (тип В) — цироз мономультилобулярного типу з помірно виразними ознаками паренхіматозної та стромальної реакції, ПСТ — (126,69 ± 12,5 мкм^2), СПС — 0,617 ± 0,031, ПНГ — (205,34 ± 13,8) мкм^2, ОГСНН — (17,32 ± 0,63) %, ОГСП — (15,43 ± 0,48) %. Тип III (тип С) — цироз з виразними ознаками паренхіматозної та стромальної реакції, частіше мультилобулярного типу, ПСТ — (240,16 ± 13,4) мкм^2, СПС — 1,344 ± 0,089, ПНГ — (178,69 ± 18,7) мкм^2, ОГСНН — (23,97 ± 0,75) %, ОГСП — (11,07 ± 0,58) %. Аналіз безпосередніх результатів хірургічного лікування показав, що жоден хворий із КЦП типу А не помер, тоді як 9,5 % хворих із типом В та 25,0 % хворих із типом С померли в ранній післяопераційний період.

Висновки. Кількісна морфометрія показала, що патологічні зміни в печінці при КЦП неоднорідні та можуть бути класифіковані на 3 типи (А, В, С). Виділені типи морфологічної картини характеризуються статистично значущими відмінностями за ПСТ, ПНГ, СПС, ОГСНН, ОГСП. При переході цирозу типу А в тип С об'єм печінкової паренхіми зменшується, а об'єм сполучної тканини збільшується. Це супроводжується зменшенням ПНГ, збільшенням ПСТ, СПС та ОГСНН. Хірургічне втручання у хворих із морфологічною картиною КЦП типу С супроводжується високим (25 %) ризиком несприятливого результату, що свідчить про необхідність обмеження показань до планових хірургічних операцій у цієї категорії пацієнтів. Кількісна морфометрія біоптатів печінки у хворих на КЦП може допомогти спрогнозувати наслідки хірургічного лікування.

Ключові слова: компенсований цироз печінки, кількісна морфометрія, морфологічна картина, інтраопераційна біопсія печінки, наслідки хірургічного лікування.

ДЛЯ ЦИТУВАННЯ

Petiunin OH, Makarov VV, Feskov VM, Smolianyk KM, Shpytalna YO. Quantitative morphometry of compensated liver cirrhosis can aid to predict the outcomes of surgical treatment. Modern Gastroenterology (Ukraine). 2024;1:43-48. http://doi.org/10.30978/MG-2024-1-43.